CHAPTER 63
Burn Injury: Thermal and Electrical

WINSTON T. RICHARDS, DAVID J. HALL, MARTIN D. ROSENTHAL, and DAVID W. MOZINGO

INTRODUCTION

Burn injury accounts for 40,000 hospital admissions/year, including 30,000 admissions to hospitals with specialized burn centers. More than 60% of the 40,000 hospitalizations for burn injuries in the United States each year are now admitted to 127 hospitals with specialized burn centers. Thirty-eight percent of the admissions exceeded 10% total body surface area (TBSA), and 10% exceeded 30% TBSA involvement. Most included severe burns of such vital body areas as the face, hands, and feet; interestingly, 69% of burn patients were male. Data from the National Hospital Discharge survey of 2010, as well as the National Inpatient Sample 2012, and the American Burn Association National Burn Repository 2013 report suggest that the causes of the burns include fire (43%), scald (34%), contact with a hot object (9%), electrical (4%), and chemical (3%) (1).

Burn injury has a systemic effect on the patient, and each organ system responds to this injury in a predictable manner proportional to the extent of burn. The current literature supports the following physiologic responses in each organ system.

BURN PHYSIOLOGY AND THE EFFECTS ON ORGAN SYSTEMS

Cardiovascular System

Immediately after a burn injury cardiac output (CO) becomes depressed, systemic vascular resistance (SVR) increases, and the patient's capillary permeability increases. Each of these occurs in a burn size- and time-dependent fashion.

During the late 1960s and early 1970s, animal studies by Moncrief, Asch, and Wilmore clearly defined the physiologic response of the heart and systemic and pulmonary vasculature to burn injury (3–4). These experiments showed a burn size- and severity-dependent depression in CO, which resolved over time, and could be altered with fluid resuscitation and the addition of vasoactive medications. They also showed a time-dependent increase in SVR and pulmonary vascular resistance (PVR), which resolved with fluid resuscitation as well as with the administration of vasoactive medication.

Burn injury causes loss of the skin's fluid barrier function. These resultant fluid losses, as well as increased capillary permeability, lead to hypovolemia and burn shock. The loss of fluid leads to decreased cardiac preload and ultimately, decreased CO. Integumentary fluid losses and tissue edema drive the cardiovascular response to burn injury, but improvement in cardiac function parameters achieved by fluid resuscitation do not fully resolve this deficit; there is evidence linking a myocardial depressant factor to multiple inflammatory mediators (5–8).

Cardiac depression after a thermal injury is mediated by the interaction of multiple inflammatory and anti-inflammatory molecular signals. Early and late inflammatory mediators influence myocardial contraction and relaxation in the first 24 to 48 hours after burn injury (9,10). Factors including tumor necrosis factor-α (TNF-α), Fas ligand, interleukin (IL)-1, IL-18, IL-6, macrophage migration inhibitory factor (MIF), high mobility group box 1 (HMGB-1) chemokines, and caspase-1 have been implicated in the loss of cardiac contractility as well as myocardial relaxation, leading to decreased CO after injury.

By contrast, the anti-inflammatory pathways involving IL-10, transforming growth factor (TGF)-β, and soluble TNF receptor lead to the resolution of the initial myocardial depression. Resuscitation of the burn patient addresses the cardiac depression, with administered fluid bolstering the intravascular volume by replacing losses and anticipating future deficits. Multiple resuscitation strategies exist for fluid management in the burn patient, which are discussed separately below. Myocardial depression can be marginally supported pharmacologically with the use of inotropic agents. Further research toward more direct management of the decreased contractility could target the inflammatory mediators released after burn injury (8).

Pulmonary System

Burn injury has multiple effects on the pulmonary system. Depending on the mode of injury, including inhalation and direct burns to the chest wall, these effects may be manifest as alterations in respiratory rate, tidal volume, gas exchange, and even long-term effects on pulmonary function. Burn injury not only affects the lungs in a direct manner, but complications of these injuries may add to further dysfunction. Pneumonia and tracheal irritation from prolonged intubation are some of the complicating factors that affect the burn patient (2).

The pulmonary response to burn injury is characterized by transient pulmonary hypertension, decreased lung compliance, and hypoxia. These functional changes are mediated by multiple factors including inflammation, acid–base imbalance, airway injury, and chest wall restriction. The body's pH balance highlights the close interaction between the pulmonary and renal systems in addressing disturbances caused by burn injury. Studies of anti-inflammatory treatment prior to burn injury altered the pattern of decreased compliance, pulmonary hypertension, and hypoxia, suggesting a cause-and-effect relationship (11).

With relatively small TBSA burns, respiratory rates increased, peaking by postburn day 8 and returning to control levels by about 3 weeks postburn. Along with these respiratory rate changes, an increase in tidal volume and minute ventilation were also recorded; these returned to control levels in approximately 3 weeks (11).

Chest burns with their concurrent edema formation showed notable restrictive effects on pulmonary function during the
first 3 days postinjury; these effects resolved over time. During the patient's initial resuscitation, decreased chest wall compliance may lead to restricted ventilation and increased airway pressures; burn wound escharotomy of the chest wall is indicated in these situations to improve chest wall compliance.

Further effects of burn injury on pulmonary function have been attributed to burn wound infection, which may lead to lung dysfunction secondary to inflammatory mediator release from around the burned tissue, possibly mediated by thromboxane A2; management of the inflammatory process initiated by burn injury may prove to be helpful in modulating this effect (12).

Complications associated with intubation after burn injuries include ventilator-associated pneumonia (VAP) and tracheal stenosis. Protocols for the reduction/prevention of VAP have been extensively reviewed and are currently being used in many hospitals. The judicious management of ventilator support and endotracheal intubation addresses postinjury tracheal stenosis. Tracheal decannulation at the earliest opportunity afforded by the patient's physiology is important in reducing this complication. Presently, a generalized benefit from the VAP bundle has not been clearly defined in the burn patient population, and requires further study (13–15).

Smoke inhalation injury affects between 10% and 30% of burn patients. This injury is caused by the inhalation of hot gases and products of incomplete combustion; when present, an inhalation injury is associated with increased mortality up to 20% over that predicted by age and extent of burn alone. Although in the past, increased fluid resuscitation was advocated for patients with inhalation injury, this did not correlate well with fluid requirements during resuscitation; interestingly, a low initial PaO2/FIO2 ratio does so correlate. An increased TBSA injury is associated with increased pneumonia rates, and the presence of pneumonia in patients with an inhalation injury results in a higher mortality rate. Pneumonia and inhalation injury increase length of stay over inhalation injury alone; this is discussed further below (16).

In the late 1980s, high-frequency percussive ventilation (HFPV) was used to treat patients with severe inhalation injury; the results were promising, and further studies followed. By 2007, it had been noted that HFPV for inhalation injury did not change mean ventilator days, intensive care unit (ICU) length of stay, hospital length of stay, or incidence of pneumonia. Despite the similarities in the HFPV and conventional ventilator groups, a decrease in both overall morbidity and mortality in a subset of patients with less than or equal to 40% TBSA burned was noted. This finding led to the recommendation for further study in a randomized and controlled fashion (17–28).

Renal System

Renal failure has been reported in up to 20% of burn injury patients, with a clinical picture related to the size and severity of burn injury. Multifactorial in nature, the time course of renal failure falls into early or late categories. The mortality of patients developing severe renal failure with burn injury approaches 80%. Early acute renal failure appears to be directly associated with clinical events such as delayed resuscitation, under-resuscitation, hypotension, and rhabdomyolysis.

Increased burn severity is associated with a higher incidence of acute renal failure. Rhabdomyolysis, a rare consequence of flame burns, but often associated with electrical injury, has a direct association with renal failure. Late episodes of acute renal failure are more commonly associated with sepsis, toxic drugs, and pre-existing medical conditions.

Treatment of acute renal failure in the burn patient and other patient populations involves multiple steps. Adequate fluid resuscitation initiated as early as possible during the time course of the burn injury will reduce the presentation of early acute renal failure. Monitoring urine output as a measure of renal perfusion allows the clinician to maintain adequate fluid input. Late episodes of acute renal failure can be addressed by treating the underlying cause. Sepsis from invasive vascular access, wounds, pneumonia, or urinary tract should be controlled by replacing the lines, excising the burn wound, and providing adequate antibiotic therapy for pneumonia and urinary tract infection. When needed, renal replacement therapy should be instituted and has shown benefit. The application of CVVH in adult patients with severe burns and acute kidney injury was associated with a decrease in 28-day and hospital mortality (29).

Gastrointestinal Tract and Liver

Ileus presents in patients with burns exceeding 20% TBSA. Despite this problem, using the gut with some—even minimal—level of feeding is advocated early after injury, and is usually well tolerated. Tube feeding should be instituted for patients with large burns where the patient is unable to tolerate sufficient intake by mouth to sustain the markedly increased nutritional requirements (30).

Gastroduodenal stress ulceration has been a hallmark of early burn care, with significant bleeding or perforation complicating the care of extensively burned individuals. This complication markedly decreased with the use of antacids, H2-antagonists, and proton pump inhibiting medications; their use has become routine in the management of the thermally injured patient (31).

Perforation

Poor perfusion of the gut during resuscitation may lead to segmental ischemia in the watershed areas of the intestine. With this condition, there is a risk for developing necrosis and subsequent perforation; vasoactive medications used during burn resuscitation or during prolonged septic events may increase this risk (32). Clinicians may use physical examinations, laboratory studies, and x-rays to monitor this complication.

Normal gut flora has been identified as an infection source in the burn patient as well as in other critically injured patients (33–35). Maintaining gut integrity with nutritional support in the form of glutamine supplementation has been proposed and studied in the burn patient. A second approach to this problem, selective gut decontamination, has been proposed and tested in animal models (36–38). Selective gut decontamination has been shown to reduce bacterial translocation from the gut in burned rats (39). With this reduction in translocation, immunosuppression was reduced and the cardiac response to subsequent septic challenge was improved. Clinical success with this approach is not well documented (40–44).

Intra-abdominal hypertension, as defined by a bladder pressure of 30 mmHg or greater is the precursor to the abdominal compartment syndrome. Respiratory compromise with increasing peak airway pressures, renal compromise with decreased
renal perfusion and urine output, and increased mortality among burn patients are the features of this syndrome (45). Burn patients with 30% or greater TBSA injury, requiring fluid resuscitation over and above the standard calculated rates are at risk for this complication (46). We recommend monitoring bladder pressures in each patient with burns greater than 30% and initiating therapeutic maneuvers for those patients with pressures of greater than 30 mmHg (47).

Therapy for intra-abdominal hypertension follows a graded response. Reduced fluid administration, sedation, and neuromuscular blocking agent (NMBA) use for the patient are the initial treatments. Escharotomies and peritoneal drainage make up the next most invasive line of management and, ultimately, abdominal decompression through a laparotomy incision may be needed to relieve the symptoms. In the severely burned population, abdominal compartment syndrome has a high mortality rate and should be addressed urgently when recognized (48–50).

Central Nervous System

Burn injury and resuscitation in an ovine model showed that cerebral autoregulation adjusted to the hemodynamic changes caused by burn injury. Autoregulation of cerebral blood flow was effective to a point and then began to fail as resuscitation proceeded, suggesting that the cerebrovascular system has a limited reserve to tolerate the effects of burn injury (51–53).

Endocrine System

There is a graded response of the endocrine system after burn injury. Hormone levels are directly related to the TBSA involved; these levels rise and fall in a time-dependent fashion from the onset of injury. Burn injury is characterized by a painful incident followed by a significant inflammatory response, with fluid losses and shifts occurring both near the burn wound itself and systemically. Each part of the endocrine system reacts to regain or maintain its preburn state.

The hypothalamus responds by secreting antidiuretic hormone (ADH) which acts on the collecting ducts of the kidney to facilitate the reabsorption of water into the blood. This reduces the volume of urine formed while retaining water in response to losses of fluid from the intravascular space. The anterior pituitary releases adrenal corticotrophic hormone (ACTH), which stimulates the release of the mineralocorticoid aldosterone and glucocorticoid cortisol. Aldosterone acts on the kidney to promote retention of sodium ions in the blood. Water follows the salt and helps maintain normal blood volume and pressure. Glucocorticoids increase blood sugar levels through the stimulation of gluconeogenesis. This elevation in blood glucose levels is thought necessary to supply the increased metabolic demand of the injured body. Cortisol and other glucocorticoids also have a potent anti-inflammatory effect on the body (54–62). They depress the immune response, especially cell-mediated immune responses.

The adrenal medulla releases the tyrosine-derived neurotransmitters adrenaline and noradrenaline into the blood. This response is associated with the autonomic nervous system—sometimes called the fight or flight response—and leads to multiple effects, some of which are an increase in the rate and strength of the heartbeat, resulting in increasing blood pressure, and the shunting of blood from the skin to the skeletal muscles, coronary arteries, liver, and brain. With the release of adrenaline and noradrenaline, blood sugar rises and the metabolic rate is increased; bronchial dilation occurs; pupils dilate; and blood clotting time is decreased. This autonomic response also leads to increased ACTH secretion from the anterior lobe of the pituitary.

The elevations in stress-related hormones are noted in a time- and burn size–dependent manner after injury. This hormone response follows a pattern associated with the ebb and flow of the burn injury process. An initial increase in the hormone levels in response to fluid shifts and inflammation resolves over time as the patient’s fluid balance returns to normal, and the burn wounds close.

Several medications are available that can modify the endocrine response to burn injury. A recent prospective, double-blind, randomized single-center study on the effect of oxandrolone on the endocrine, inflammatory, and hypermetabolic responses during the acute phase of burn injury suggested that this treatment shortened the length of acute hospital stay, maintained lean body mass, and improved body composition and hepatic protein synthesis while having no adverse effects on the endocrine axis postburn. This study, and another using beta-blockade to modify the metabolic response after burn injury, are at the heart of attempts to improve the outcome of patients after a severe burn injury (63,64).

Hematopoietic System

Typically, the red blood cell (RBC) mass in the burn patient declines in a burn size- and severity-dependent fashion. This is initially related to the burn injury itself and thereafter decreased RBC production, increased RBC damage, and RBC losses from therapeutic interventions. Although surgery and phlebotomy account for the iatrogenic loss of RBCs, several possible causes have been explored for the injury-related loss of blood in the burn patient. Initial heat injuries to the RBCs, as well as sequestration of blood in the burn eschar, are early factors in the loss of RBCs and the decline in hemoglobin and hematocrit. Damage to RBCs secondary to the inflammatory response to the burn wound is a later developing cause for blood loss in these patients (65–70).

In 1973, Loebl (69) described studies of RBC half-life in burn victims and healthy volunteers. RBCs from the burn patient had a normal half-life when transfused into healthy volunteers. The same RBCs had a decreased half-life in the burn victim and, when normal RBCs were transfused into a burn victim, they acquired a similar decrease in their half-life; this study suggested a humoral or inflammatory process driving the loss of red cells.

Later studies have linked this loss in RBCs to a process mediated by inflammation and the release of toxic oxygen–free radicals, representing a nonspecific mechanism for the destruction of red cells. Immune system–mediated processes for the destruction of RBCs in burn patients have also been postulated, but Coombs testing in these patients has failed to reveal a definite link to immune-mediated blood loss. Studies by Poslusny et al. in a mouse model show a decrease in bone marrow hematopoietic commitment to erythroid cells and an increase in myeloid cells. This accounted for a decrease in RBC production after burn injury. Further study is needed to identify the same patterns in burn patients (71,72).
Immune System

Infection remains a major complicating factor of burn injury (73–76). Burned skin loses its barrier function against the environment, and normal skin flora and environmental pathogens are able to gain access to the system. Risk of infection and its complications are directly related to the size of the burn injury and additional factors, such as inhalation injury and pre-existing medical conditions. Burn injury leads to compromised immune function, resulting in increased susceptibility to sepsis and multiple organ system failure.

Immune system dysfunction occurs on the cellular and humoral levels. Multiple avenues for this effect have been investigated. Currently, macrophages, T cells, other lymphocyte subpopulations, and humoral factors such as opsonins, immunoglobulins, protease inhibitors, toll-receptors, and chemoattractant factors have been implicated in the process. Some combination of all of the above factors, related to their natural interaction, produces a weakened immune system susceptible to infection entering through multiple avenues (77–79).

In general, burn patients are assailed by bacteria from multiple directions. Damaged skin, its barrier function destroyed, is the most obvious portal. Burn-related hypotension and peripheral vasoconstriction may permit intestinal hypoperfusion, with normal gastrointestinal flora becoming a source of proinflammatory mediators, pathogens, and toxins that contribute to multi-organ failure. Other clinically relevant pathways include cannulation of the respiratory, vascular, and genitourinary systems, providing ready access for bacteria into the compromised host; these are considered nosocomial infections (80).

Musculoskeletal System

Loss of muscle mass and bone are notable in severely burned patients. Bone loss is, in part, due to an increase in glucocorticoids that inhibit bone formation and osteoblast differentiation, hypercalcemia secondary to hypoparathyroidism, and vitamin D deficiency. Muscle loss is secondary to an intense catabolic state initiated by the inflammatory response to burn injury and is fueled by the need to repair the surface injury suffered. Propranolol administration in pediatric patients reduces thermogenesis, cardiac work, resting energy expenditure, and peripheral lipolysis; it also increases skeletal muscle protein anabolism. These effects are being studied in adult burn patients (81–86). A randomized, double-blinded, placebo-controlled study by Klein and Herndon (82) suggested that intravenous pamidronate administration may help preserve bone mass in children with over 40% TBSA burns. Follow-up to that study 2 years later showed a sustained improvement in bone mineral content as measured by dual-energy radiograph absorptiometry (83). This effect was attributed to a decrease in the glucocorticoid-mediated effect on bone mass.

FLUID RESUSCITATION

Fluid resuscitation addresses the clinical picture of burn shock. Multiple resuscitation regimens have been developed to overcome the cardiac depression, vasoconstriction, and hypovolemia associated with acute burn injury. Most, if not all, of the current formulae have been developed through retrospective review of the fluid requirements of burn patients. Aided by the use of the rule of nines and Lund-Browder charts, a patient’s TBSA burn and weight measurements are used to determine their initial fluid requirement. One-half of the fluid requirements calculated are given in the first 8 hours after the burn injury, with the remaining amount administered over the subsequent 16 hours. A slight variation in the composition of the resuscitation fluid is present in the different formulae; there are also differences in the addition of colloid to the initial resuscitation scheme (87–98); the most commonly used formulae are noted in Table 63.1.

Colloid infusion during the resuscitation of acutely injured patients has been debated for some time. Acute burn injury leads to capillary permeability, which allows loss of intravascular albumin into the interstitial spaces of the acutely injured patient. Currently, the application of albumin or fresh-frozen plasma is considered after the initial 8-hour period postburn in an attempt to avoid loss of the colloid secondary to capillary permeability (99–102).

Adequate resuscitation is measured by end-organ perfusion. Currently, exact measures of end-organ perfusion are being developed and tested. A surrogate measure of the success of fluid administration is the measurement of urine output. Renal function is highly dependent on renal blood flow, which can be adequately assessed by the rate of urine output. Most practitioners view a urine production rate of 0.5 to 1.0 mL/kg ideal (or adjusted) body weight per hour as adequate. Secondary measures of perfusion are also important in the resuscitation plan. The combination of blood pressure, heart rate, oxygenation, and central venous pressure are used in tandem to determine the adequacy of treatment.

Measures of adequate resuscitation, such as blood pressure, heart rate, and central venous pressure used alone, must be monitored cautiously. Postburn tissue edema will decrease the accuracy of cuff blood pressure measurements, and the vasoconstriction caused by catecholamine release will adversely affect the accuracy of indwelling arterial lines. Central venous pressure measurements require the interaction of multiple physiologic and environmental parameters to provide the practitioner with meaningful measurements.

Patients who begin to lag in their urine output during resuscitation should have their fluid rates adjusted. Urinary rates of less than one third the predicted value based on the patient body weight over two consecutive hours should prompt an increase in intravenous fluid administration. On the other hand, patients running one third or more over their expected urine output may benefit from decreased fluid administration. A graded increase or decrease of the intravenous fluid rate of 20% per hour is a measured and conservative response to these situations. Those patients who do not respond as expected to calculated fluid administration or who require more than 6 mL/kg per percent TBSA fluid administration
should be considered for more invasive monitoring, such as pulse-waveform analysis (FlowTrack, PICCO, LiDCO, and bedside Echo) or pulmonary artery catheter monitoring. With measurements obtained through this more advanced monitoring, a decision can be made to either support the CO or reduce the SVR, or both. Small doses of hydralazine may be used to reduce peripheral resistance in situations where the CO remains low. Hydralazine doses on the order of 0.5 mg/kg have been shown to be effective when used in this situation. In animal models of burn injury, sodium nitroprusside and verapamil decreased SVR and supported CO with good effect. This approach should be used with caution in the severely burn-injured patient to avoid further tissue hypoperfusion, which may exacerbate the condition of the partially burned tissues, increase fluid creep, worsen tissue edema, and may precipitate abdominal compartment syndrome (103).

At-risk patients for volume over-resuscitation include pediatric patients with an increased TBSA to weight, patients with inhalation injuries, electrical injuries, intoxication, and delayed fluid resuscitation. Fluid creep during resuscitation is worsened by exceeding the Parkland formula early or pushing high urine output during goal-directed therapy. Other factors that contribute to increased fluid administration include sedation and pain management with opioids, propofol, and benzodiazepines, or patient factors like morbid obesity (104–106).

Insensible water losses become more significant with larger TBSA burns. Evaporative water losses from the open wounds/burns usually peak on the third day postburn and then trail off until the wounds are completely closed. An estimation of insensible water losses may be calculated as follows:

\[
\text{Insensible water loss (in mL/h)} = (125 + \% \text{TBSA burned}) \times \text{BSA (in m²)}
\]

The body surface area may be estimated using the formula of DuBois and DuBois as follows:

\[
\text{BSA} = \left(\frac{W^{0.425} \times H^{0.725}}{100} \right) \times 0.007184
\]

where \( W \) is the weight in kilograms and \( H \) is the height in centimeters. Initially, the replacement fluid is free water initially and then it is altered based on electrolyte measurements (107,108).

**ELECTRICAL INJURY**

Electrical injuries can be divided into those due to low voltage (<1,000 volts) and those due to high voltage (≥1,000 volts) (109). These injuries have varying patterns: Low-voltage injuries range from the circumoral injuries noted in children who have bitten home electrical cables to deaths caused by dropping electrical appliances in a bathtub full of water; high-voltage injuries have a range that includes the more severe episodes of instant death, massive tissue loss, and secondary clothing ignition. Some of the less dramatic injuries include thermal injury, central nervous system–related trauma, and fractures. With high-voltage injuries, there is a high ratio of limb amputations, highlighting the danger of this modern-day source of power. Herein, we will concentrate on high-voltage injuries.

Electrical energy interacts with human anatomy following the basic principles of physics. Current flowing through tissue is related to the voltage drop across the resistance of that tissue. Heat produced by this current can be represented mathematically as follows:

\[
J \ (\text{heat in Joules}) = I^2 \times R \times T
\]

where \( I \) is the current, \( R \) is the resistance, and \( T \) is the time in seconds. Body tissues have differing electrical resistances. Given the above equation, it appears that differing tissues would create varying degrees of heat and subsequent damage; interestingly, clinical findings do not wholly support this concept. The highest resistance is found in the bone, fat, and tendons, whereas the lowest resistance has been identified in the muscles, blood, and nerves; skin has an intermediate resistance. Clinical findings in electrically injured patients support the idea that the body represents a volume conductor with a resistance on the order of 500 to 1,000 ohms. In this model, the relative differences in tissue resistance are small enough that the body is considered a single resistor. Heat generated by the current flowing through the resistor is related to the cross-sectional area of the entry or exit wound and the local anatomy.

Contact wounds on the hands and feet are common. Each of the contact areas might have a low cross section, releasing more heat in that area; as the current crosses the “bottleneck” areas of the ankles and wrists, there may be more tissue damage generated at those sites. At its most extreme, heat released by high-voltage injuries produces coagulation necrosis of the tissues and varying other effects on the organs as the electricity passes through.

Arc injuries are less common, but just as destructive. Electricity can travel 2 to 3 cm/10,000 volts, and may travel 10 ft or more to its target. Temperatures at the contact points range from 2,000 to 4,000°C, with spikes of up to 20,000°C; this intense temperature leads to severe and deep tissue damage.

Electrical injuries have specific organ effects in addition to the thermal injury described above. With high-voltage injuries, cardiac standstill and ventricular fibrillation are the most lethal cardiac injuries. Other electrocardiographic (ECG) findings and rhythm changes that have been reported include atrial fibrillation, focal ectopic arrhythmias, supraventricular tachycardia, right bundle branch block, and nonspecific ST-T segment changes. These clinical findings are thought to be associated with direct myocardial muscle damage, coronary vasospasm, and coronary endarteritis.

Renal injury may be direct, although this is rare, or may take on the more familiar form of acute renal failure secondary to rhabdomyolysis. Large quantities of muscle protein, hemoglobin, and other tissue proteins released from the tissues coagulated by high voltage and current are filtered into the renal tubules, causing acute renal failure with oliguria or polyuria. Up to 15% of patients injured in high-voltage accidents will suffer from this type of renal injury.

Central nervous system injury ranges from the devastating effect of high voltage and current on the brain and brainstem, leading to instant death or to more subtle findings. Altered levels of consciousness with varying degrees of recovery have been reported, while progressive neurologic deterioration has been noted in both the central and the peripheral nervous systems. With high-voltage injuries, progressive deterioration of the microvascular nutrient vessels to the nerves has been identified, and is thought to lead to ischemia, necrosis, and fibrosis of the injured nerve; progressive loss of function can be seen as a late developing problem (110).
INHALATION INJURY

The pathophysiology associated with smoke inhalation injury falls into three broad categories: (i) upper airway injury; (ii) asphyxiant gases and hypoxic environments; and (iii) carbonaceous particle deposition (113–116).

The diagnosis of inhalation injury is based on several site-specific and clinical findings surrounding each burn patient. For example, closed space injuries or explosions are some of the circumstances surrounding inhalation injury. Noxious fumes noted at the scene by the scene responders, as well as facial burns noted on the patient, are other findings consistent with inhalation injury. Clinical findings on examination, such as large burns, carbonaceous sputum, hoarseness, or an abnormal lung examination, are associated with this injury, and elderly patients are more susceptible to inhalation injury.

Once inhalation injury is suspected, upper airway pathology should be expected. Heated smoke or ambient air may injure the supraglottic airway from the lips to the vocal cords. This injury may occur abruptly, leading to significant edema and swelling of the face and oropharynx, as well as affecting the region around the vocal cords.

True heat injury below the cords is rare, with the exception of steam injuries or ignition of flammable gases in the airway. Signs and symptoms of a true airway burn injury include hoarseness; stridor and/or wheezing; carbonaceous sputum; singed nasal hair, eyebrows, or facial hair; and edema or inflammatory changes in the upper airway. The resultant upper airway edema formation can threaten the airway and the patient’s breathing.

Asphyxiant gases and hypoxic environments lead to the second area of pathology associated with inhalation injury. In fires involving structures, the ambient oxygen level markedly decreases; this lack of oxygen may lead to carbon monoxide generation, as this molecule is a byproduct of incomplete combustion in the burning structure. Carbon monoxide binds tightly to hemoglobin, reducing the amount of oxygen delivered to end organs, which may cause a hypoxic injury. Aside from carbon monoxide, other toxic gases can be released by the flames. Cyanide generation is associated with burning plastics; this molecule is highly lethal. Treatment of carbon monoxide intoxication includes a high concentration of oxygen and, sometimes, hyperbaric oxygen. Cyanide poisoning treatment includes delivery of oxygen, as well as a three-part regimen to bind the cyanide compound in the blood.

The third broad area of pathology associated with smoke inhalation injury is related to the deposition of carbonaceous particles in the airway. The flame-generated toxins that are bound to the carbon particles will slowly be released after the latter are deposited in the lungs, inducing a chemical tracheal bronchitis. This effect is manifested by impaired ciliary function and edema, significant inflammation, and ulceration or necrosis of the respiratory epithelium. The clinical sequelae of tracheal bronchial injury include bronchorrhea, broncho-spasm, distal airway obstruction, and atelectasis, as well as pneumonia. Epithelial injury leads to sloughing of the mucosa and blockage of the airways with this cellular debris. Air trapping in this situation leads to atelectasis and the development of barotrauma and pneumonia.

Treatment for each area of pathology is based on standard clinical practice. When presented with a patient who is suspected of having an inhalation injury and upper airway edema, endotracheal intubation to protect the airway should be performed early to avoid the consequences of airway compromise. Patients suffering from carbon monoxide exposure, and showing clinical signs of intoxication, should be treated with 100% oxygen to displace the molecule from hemoglobin. If immediately available, and if the patient is stable, high levels of carboxyhemoglobin and severe neurologic symptoms should be treated with hyperbaric oxygen therapy. The tracheal-bronchial injury associated with inhalation injury and carbonaceous particle deposition should be treated with humidified oxygen by face mask, and frequent examination of the airway should be performed to evaluate for signs of compromise that may require endotracheal intubation.

Cyanide toxicity presents clinically with lethargy, nausea, headache, weakness, and coma. Cyanide combines with cytochrome oxidase, thereby blocking oxygen use and inhibiting high-energy phosphate compound production. Cyanide toxicity begins at 0.1 μg/mL of serum and quickly leads to death at concentrations of 1 μg/mL. Laboratory studies show a decreased arteriovenous oxygen difference with severe metabolic acidosis; this acidosis is unresponsive to fluids and oxygen administration.

S-T segment elevations may be seen on the patient’s electrocardiogram, mimicking a myocardial infarction. Treatment of this condition includes the administration of 100% oxygen and a three-part medication regimen. Initially, the administration of amyl nitrate pearls, by inhalation for 15 to 30 seconds every minute, is followed by 10 mL 3% sodium nitrate solution (300 mg) intravenously over 3 minutes, repeated at one-half the dosage in 2 hours if symptoms persist or recurrent signs of toxicity are present. Sodium thiosulfate is the final medication, given in a dose of 50 mL of a 25% solution (12.5 g) intravenously over 10 minutes, repeated at half the dosage in 2-hour intervals if persistent or recurrent signs of toxicity are present.

INFECTION CONTROL

Historically, mortality from burn injury was associated with burn shock (117–119); the loss of fluid through the burned and damaged skin, as well as fluid shifts related to the release of inflammatory mediators, led to hypovolemia and unresponsive end-organ failure. As our understanding of the injury suffered during burns improved, so did the survivability of these injuries. Currently, burn shock is well controlled by our fluid resuscitation regimens. The new challenge in burn injury is the concurrent development of infection in a compromised host. Burn injury is associated with a burn size–dependent depression in the immune system in which bacterial, fungal, and viral elements are better able to breach the defense mechanisms of the body, thereby worsening the injury.
As fluid resuscitation techniques advanced, wound colonization and sepsis became a leading factor in morbidity and mortality. The advent of topical antibiotic/anti-infective agents addressed this new area of pathology. Subsequent movement toward early wound excision and skin grafting led to improvements in the rates of wound sepsis and its complications. Although the burn wound has become less of a risk for infection, other portals of entry have persisted in plaguing the burn patient. With our current methods of intubating the respiratory, vascular, and genitourinary systems, we expose the burn patient to other portals of entry for pathogens.

**NUTRITION**

Burn patients present to the ICU in a severe inflammatory state. This state, along with a wide variety of prehospital factors and premorbid conditions, provides the practitioner with a challenging nutritional problem. Nutritional support for the burn patient can be addressed in several steps: first, an assessment of the patient’s initial nutritional status, and second, monitoring of his or her nutritional status throughout the hospital course, and adjustments based on the monitoring measures. By using a combination of variables, including burn size and severity, time from injury, physical parameters such as age, weight, and the presence of other medical factors, nutrition support can be tailored to each burn patient.

Nutritional assessment begins with measurement of the patient’s weight, estimation of the patient’s calorie and protein needs, measurement of the patient’s serum albumin, prealbumin, and C-reactive protein levels (120–133). Pre-existing illnesses should prompt the practitioner to make adjustments in the rate of feeding, use of additional medications, and the need for additional nutrient support.

Glutamine supplementation has been shown to improve morbidity and mortality when administered to critically ill patients. This response appears to improve with increasing doses of the amino acid, and parenteral administration appears to have an improved response over enteral administration. Not all of the trials reported to date have shown a definitive benefit, and the general consensus is that a large randomized controlled trial would be needed to confirm or refute the benefits of glutamine administration (134,135).

Physiologically, glutamine affects the immune system, the anti-oxidant status, glucose metabolism, and heat shock protein response. These physiologic effects appear to provide benefit with regard to gastrointestinal mucosal integrity, wound healing, gram-negative bacteremia and infection—including with *Pseudomonas*—as well as a reduction in mortality, and possibly cost savings to burn patients. At the time of this writing, a consensus has not been reached on the length of time for glutamine therapy, the optimal dose, and definite safety aspects of the supplementation of glutamine in critically ill and burn patients. Most studies suggest that clinically important differences appear to commence at doses over 0.2 g/kg body weight per day, and most trials have used 15 to 30 g/day glutamine supplementation.

**WOUND MANAGEMENT**

With the exception of chemical burns, for which prompt water irrigation to remove the offending agent is required, no specific treatment of the thermal burn wound is needed in the prehospital setting (113,136,137). The patient should be covered with a clean sheet and blanket to conserve body heat and minimize burn wound contamination during transport to the hospital. The application of ice or cold-water soaks, when initiated within 10 minutes after burning, may reduce tissue heat content and lessen the depth of thermal injury. If cold therapy is used, care must be taken to avoid causing hypothermia; this is accomplished by limiting this form of therapy to 10% or less of the body surface and only for the time required to produce analgesia.

Following admission to the burn center, definitive care of the burn wound can begin. Daily wound care involves cleansing, debridement, and dressing of the burn wound. On the day of injury, the burn wound is best cleansed by means of hydrotherapy, a practice that has been used in the treatment of burn patients for many years and remains an integral part of current treatment plans. Hydrotherapy is accomplished by means of showering or use of a spray table. Showering is often used for ambulatory patients who remain capable of independent, or near-independent, wound care. Patients who are near to discharge are encouraged to use the shower, especially if showering is to be used at home.

Use of a spray table is generally reserved for newly admitted patients, those with limited mobility, or those with large open wounds. The patient is placed on the table, and the wounds are washed and rinsed with running water. As an alternative, a stretcher or plinth can be placed over a Hubbard tank. The patient is placed on the stretcher, and the wounds are washed and rinsed as described previously.

Wound debridement involves the removal of all loose tissue, wound debris, and eschar (nonviable tissue); debridement of a burn wound is accomplished through mechanical, chemical (enzymatic), or surgical means.

Surgical, or sharp, debridement requires the use of scalpels and scissors to debride wounds of loose, necrotic tissue. Care must be taken to avoid excessive debridement that results in bleeding and pain. Bleeding may indicate injury to the healthy underlying tissue. In most instances, sharp debridement should be carried out in the operating room to ensure adequate debridement and hemostasis. Definitive management of third-degree burn wounds and deep second-degree injuries may require tangential excision of the eschar or necrotic tissue followed by wound closure with skin grafts or other wound closure adjuncts. This process will not be reviewed here.

Multiple topical antimicrobial agents are used in burn wound care. Mafenide acetate (SulfaMylon), silver sulfadiazine (Silvadene), and silver nitrate are the three most commonly used topical antimicrobial agents for burn wound care. Each agent has specific limitations and advantages with which the physician must be familiar to ensure patient safety and optimal benefit. Mafenide acetate and silver sulfadiazine are available as topical creams to be applied directly to the burn wound, whereas silver nitrate is applied as a 0.5% solution in occlusive dressings. Either cream is applied in a half-inch (about one-third of a centimeter) layer to the entire burn wound in an aseptic manner after initial debridement, and reapplied at 12-hour intervals or as required to maintain continuous topical coverage. Once daily, all of the topical agents should be cleansed from the patient using a surgical detergent disinfectant solution and the burn wounds examined by the attending physician. Silver nitrate is applied as a 0.5% solution in multilayered occlusive dressings that are changed twice daily.
Mafenide acetate burn cream is an 11.1% suspension in a water-soluble base. This compound diffuses freely into the eschar, owing to its high degree of water solubility. Mafenide is the preferred agent if the patient has heavily contaminated burn wounds or has had burn wound care delayed by several days. This agent has the added advantage of being highly effective against gram-negative organisms, including most *Pseudomonas* species. Physicians using this agent must be aware of several potential clinical limitations associated with its use. Hypersensitivity reactions occur in 7% of patients, and pain or discomfort of 20 to 30 minutes duration is common when it is applied to partial-thickness burn wounds. This agent is also an inhibitor of carbonic anhydrase, and a diuresis of bicarbonate is often observed after its use. The resultant metabolic acidosis may accentuate postburn hyperventilation, and significant acidemia may develop if compensatory hyperventilation is impaired. Inhibition of this enzyme rarely persists for more than 7 to 10 days, and the severity of the acidosis may be minimized by alternating applications of mafenide with silver sulfadiazine cream every 12 hours (138).

Silver sulfadiazine burn cream is a 1% suspension in a water-miscible base. Unlike mafenide, silver sulfadiazine has limited solubility in water and, therefore, limited ability to penetrate into the eschar. The agent is most effective when applied to burns soon after injury to minimize bacterial proliferation on the wound's surface. This agent is painless on application, and serum electrolytes and acid–base balance are not affected by its use. Hypersensitivity reactions are uncommon; an erythematous maculopapular rash sometimes seen subsides on discontinuation of the agent. Silver sulfadiazine occasionally induces neutropenia by a mechanism thought to involve direct bone marrow suppression; white blood cell counts usually return to normal following discontinuation. With continual use, resistance to the sulfonamide component of silver sulfadiazine is common, particularly in certain strains of *Pseudomonas* and many *Enterobacter* species. However, the continued sensitivity of microorganisms to the silver ion of this compound has maintained its effectiveness as a topical antimicrobial agent.

A 0.5% silver nitrate solution has a broad spectrum of antibacterial activity imparted by the silver ion. This agent does not penetrate the eschar, because the silver ions rapidly precipitate on contact with any protein or cationic material. Use of this agent is not associated with more intense wound pain, except from the mechanical action required for dressing changes. The dressings are changed twice daily and moistened every 2 hours with the silver nitrate solution to prevent evaporation from increasing the silver nitrate concentration to cytotoxic levels within the dressings. Transeschar leaching of sodium, potassium, chloride, and calcium should be anticipated, and these chemical constituents should be appropriately replaced. Hypersensitivity to silver nitrate has not been described. Mafenide acetate, silver sulfadiazine, and 0.5% silver nitrate are effective in the prevention of invasive burn wound infection; however, because of their lack of eschar penetration, silver nitrate soaks and silver sulfadiazine burn cream are most effective when applied soon after burn injury.

**PAIN MANAGEMENT**

Because burn pain is variable in its degree and time course, reliance on a single analgesic regimen is unreliable at best and unsuccessful at worst. Conversely, the diverse spectrum of burn patients—adult versus children, large burns versus small, ICU nursing versus ward setting—makes the routine individualization of analgesic plans overwhelming and impractical. Our recommendation is to determine an analgesic regimen for each individual patient based on two broad categories: the assessed clinical need for analgesia and the limitations imposed by the patient.

The first step is to address background, procedural, postoperative, and breakthrough pain treatment separately, and then consider individual drug choices based on patient limitations. To reinforce this type of approach to analgesic management, detailed institutional guidelines to help physicians and nurses choose and administer specific analgesics are recommended.

**Background Pain**

In general, because it is a pain of continuous nature, background pain is best treated with mild-to-moderately potent, longer-acting analgesics administered so that plasma drug concentrations remain relatively constant throughout the day. Examples include the continuous IV infusion of fentanyl or morphine (with or without patient-controlled analgesia [PCA]), oral administration of long-acting opioids with prolonged elimination (methadone) or prolonged enteral absorption (sustained-release morphine, sustained-release oxycodone), or oral administration on a regular schedule of short-acting oral analgesics (oxycodone, hydromorphone, codeine, acetaminophen). Such analgesics should almost never be administered on an as-needed (PRN) basis during the early and middle phases of hospitalization.

**Procedural Pain**

In contrast, procedural pain is significantly more intense but shorter in duration; therefore, analgesic regimens for procedural pain are best composed of more potent opioids that have a short duration of action. Intravenous access is helpful in this setting, with short-acting opioids (fentanyl) offering a potential advantage over more longer-acting agents (morphine, hydromorphone). When intravenous access is not present, orally administered opioids (morphine, hydromorphone, oxycodone, codeine) are commonly used, although their relatively long duration of action (2 to 6 hours) may potentially limit postprocedure recovery for other rehabilitative or nutritional activities. Oral transmucosal fentanyl and nitrous oxide are useful agents when IV access is not present due to their rapid onset and short duration of action.

**Postoperative Pain**

Postoperative pain deserves special mention because increased analgesic needs should be anticipated following burn wound excision and grafting. This is particularly true when donor sites have been harvested, as these are often the source of increased postoperative pain complaints. In contrast, pain from excised/grafted burns may increase, decrease, or not change postoperatively compared to preoperatively. Typically, this increased analgesic need in the postoperative period is limited to 1 to 4 days following surgery before returning to, or falling below, preoperative levels.
Breakthrough Pain

Breakthrough pain occurs at rest when background analgesic therapy is inadequate. Breakthrough pain occurs commonly in burn patients, particularly in early stages of hospitalization until a stable, appropriate, and individualized pharmacologic regimen can be determined for each patient. Analgesia for breakthrough pain can be provided with IV or oral opioids. When breakthrough pain occurs repeatedly, it is an indication to re-evaluate and likely increase the patient’s background pain analgesic regimen, as it may be inadequate in terms of analgesic dose and/or frequency. Tolerance develops rapidly in these patients and may initially manifest as breakthrough pain.

Patient Limitations

As stated above, the presence of intravenous access directly influences analgesic drug choice, particularly in children. Similarly, patients who are endotracheally intubated and ventilated are somewhat protected from the risk of opioid-induced respiratory depression; thus, opioids may be more generously administered in these individuals, as is often required for painful burn debridements. Also, individual differences in opioid efficacy should be considered in all patients, including opioid tolerance in patients requiring prolonged opioid analgesic therapy or in those with pre-existing substance abuse histories.

An appropriate rationale is to titrate the drug dose to the desired effect, rather than to rely on a particular textbook dose for all patients. Because of the development of drug tolerance with prolonged medical use or recreational abuse of opioids (i.e., increasing drug doses are required to attain adequate levels of analgesia), opioid analgesic doses needed for burn analgesia may exceed those recommended in standard dosing guidelines. Furthermore, because of cross-tolerance, tolerance to one opioid analgesic usually implies tolerance to all opioid analgesics. One clinically relevant consequence of drug tolerance is the potential for opioid withdrawal to occur during inpatient burn treatment. Thus, the period of inpatient burn care is not an appropriate time to institute deliberate opioid withdrawal or detoxification measures in tolerant patients, as such treatment ignores the very real analgesic needs—background pain and procedural pain—of these patients. Similarly, when reductions in analgesic therapy are considered as burn wounds close, reductions should occur by careful tapering, rather than abrupt discontinuation of opioids, to prevent the acute opioid withdrawal syndrome.

Anxiolysis in the Treatment of Burn Pain

Current aggressive therapies for cutaneous burns, together with the qualities of background and wound care pain, make burn care an experience that normally induces anxiety in a large proportion of adult and pediatric patients. Anxiety, in itself, can exacerbate acute pain. This has led to the common practice in many burn centers of using anxiolytic drugs in combination with opioid analgesics. Intuitively, this practice seems particularly useful in premedicating patients for wound care, to diminish the anticipatory anxiety experienced by these patients prior to and during debridement. Low-dose benzodiazepine administration significantly reduces burn wound care pain scores and narcotic requirements. It appears that the patients most likely to benefit from this therapy are not those with high trait or premorbid anxiety, but rather those with high state or the time of the procedure anxiety, or those with high baseline pain scores. Other nonpharmacologic anxiolysis techniques, such as hypnosis and behavioral therapy, could also be considered.

TEAM APPROACH TO BURN PATIENTS

The management of the burned patient is a multidisciplinary effort of burn care professionals to provide optimal care to the burn patient. This multidisciplinary care spans the early resuscitative phases of care through the long-term rehabilitation and reconstructive phases. The Burn Center Director coordinates all activities of the multidisciplinary care of the critically ill burn patients. Team members include burn surgeons, plastic and reconstructive surgeons, critical care specialists, anesthesiologists, critical care burn nurses, physical therapists, occupational therapists, clinical nutritional specialists, psychologists, social workers, and pastoral care support personnel. This multidisciplinary approach affords the patients and their families state-of-the-art resources for optimal outcome, education, and rehabilitation. This concept of team care, originating in the 1950s when the first burn centers opened, has persisted to this day and is a model of coordinated, interdisciplinary, outcome-driven patient care.

Key Points

- Role of inflammation and inflammatory mediators in the depression of cardiac function and increase in capillary permeability.
- Smoke inhalation and its effect on mortality from burn injury.
- Renal replacement therapy and its potential role in modulating the inflammatory effects of burn injury.
- Modulating the hypermetabolic response to burn injury medically as well as reducing the loss of bone density in patients with severe burn injuries.
- Addressing the effects of burn injury on the immune system and reducing the incidence of infection through VAP bundles, catheter management, and improving the rate of wound closure. Further study in supporting the immune system despite the burn injury.

References


