14 MANAGEMENT OF THE PATIENT WITH THERMAL INJURIES

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Optimal care of the burn patient requires not only specialized equipment but also, more importantly, a team of dedicated surgeons, nurses, therapists, nutritionists, pharmacists, social workers, psychologists, and operating room staff. Burn care was one of the first specialties to adopt a multidisciplinary approach, and over the past 30 years, burn centers have decreased burn mortality by coordinating prehospital patient management, resuscitation methods, and surgical and critical care of patients with major burns. Detailed practice guidelines for burn patients, as well as lists of the resources needed in a burn center, have been developed.[1,2]

Where to Treat Burn Patients

Patients with critical burns, as defined by the American Burn Association [*see Table 1*], should be transferred to a specialized burn center as soon as possible after their initial assessment and resuscitation. A community general or plastic surgeon with an interest in burns could manage moderate burns that do not involve functionally significant body sites. However, even patients with small burns benefit from the expertise of a specialized burn care team. Furthermore, the burn center's focused approach facilitates patient and family education, reentry into society, long-term rehabilitation needs, and reconstructive surgical needs.

OUTPATIENT VERSUS INPATIENT MANAGEMENT

Outpatient management may be appropriate for small burns (1 to 5% of total body surface area [TBSA]) that do not involve joints or vital structures. However, successful outcomes in such cases require a well-organized plan and clear communication with the patient and family. Many outpatient management plans fail because insufficient teaching during a short visit to an emergency department leads to inadequate pain control, wound infection, and limited movement.

Three important reasons for hospitalizing a patient with a burn injury are wound care, physical therapy, and pain management. A short hospital stay immediately after the injury gives the burn team the opportunity to teach the patient how to properly clean and dress the burn; this is especially important for burns to the extremities. A therapist should assess patient movement and educate the patient about expected activity levels and exercise programs. Background pain (pain experienced with ordinary daily activities) and procedural pain (pain experienced during wound care) should be carefully assessed, and analgesic medications should be titrated to the individual patient's pain levels.

Complex burn wound management is discussed in detail elsewhere. For outpatient management, however, simplicity is the key to success. Patients and their families are unlikely to manage complicated dressing plans. For outpatient burn care, once-daily dressing changes are adequate. A common misconception is that these wounds must be cleaned with sterile saline. In fact, burns can be effectively washed during a daily shower or bath with regular tap water and nonperfumed soap. A second misconception is that the patient must scrub the wound to débride all the superficial exudates. Simply wiping the wound with a soapy washcloth to remove the topical ointment and wipe away the bacteria that have accumulated over the past day provides adequate care. Intact blisters can be left as a protective wound cover if they do not prevent movement of a joint. Dressings must allow full range of motion.

Physical therapy is an essential component of burn management. A common misconception is that burns over joints should be immobilized to promote healing. Actually, immobilization of extremities leads to swelling, which worsens burn wound pain and increases the risk of wound infection. Patients with hand burns must be taught exercises to maintain range of motion. Likewise, patients with foot burns must ambulate without assistive devices so that normal muscle contraction can facilitate lymphatic drainage of the lower extremity. Patients must be taught to elevate burned extremities when they are not actively exercising.

Inadequate pain management is a frequent reason for return visits to the emergency department or readmission to the hospital. Often inadequate pain control results from poor patient understanding of how to care for the burn (e.g., excessive scrubbing during wound care or inactivity and subsequent swelling). Although a healing partial-thickness burn may become more painful as the epithelial buds begin to emerge and healing progresses, an acute increase in stinging

***Table 1* American Burn Association Criteria for Burn Injuries that Warrant Referral to a Burn Unit**

Partial-thickness burns of greater than 10% of total body surface area
Third-degree burns
Electrical burns, including lightning injury
Chemical burns
Inhalation injury
Burn injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or increase mortality
Burns with concomitant trauma

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pain may be the first sign of a superficial wound infection. This is an indication that the burn should be evaluated for signs of infection, including erythema and breakdown of a previously epithelized wound; cellulitis may or may not surround an infected burn. Systemic antibiotics and a change in the topical antimicrobial agent are indicated in this situation.

Socioeconomic issues can be important contraindications to outpatient management of a burn wound [*see Table 2*]. Any suggestion of abuse—of a child or an adult—mandates admission for full evaluation of the home situation by the burn team; if the history and the burn distribution are consistent with a nonaccidental injury or potential neglect, the patient must be referred to protective services. Likewise, suggestion of a self-induced burn injury should trigger admission for psychological evaluation. For example, the presence of multiple small cigarette burns in various phases of healing is an absolute indication for admission to the hospital for psychological evaluation, even though the burns themselves may be easily cared for at home with small adhesive bandages. Although language barriers are not an absolute indication for hospital admission, there must be assurance that patients fully understand the treatment plan before they leave the emergency department. Underinsured and homeless patients may not have the resources to care for a wound outside the hospital and should be admitted for initial wound care and planning for transfer to a facility where they have access to a daily shower. Finally, the success of outpatient burn wound management depends on the ability to arrange a follow-up visit with an outpatient health care provider who can assess the outcome.

For patients with large burns, transition from inpatient to outpatient status is based on the same principles listed above. When burn pain can be controlled with oral medication and the patient and family can provide wound care, perform range-of-motion exercises, and manage splints and other assistive devices, outpatient management is appropriate. In some cases, daily or weekly outpatient therapy sessions to maintain range of motion may be included. If there are concerns about nonhealing wounds, weekly follow-up visits with the burn surgeon may be indicated initially. Because of possible long-term sequelae—scarring, contractures, and rehabilitation difficulties—the burn team should follow burn patients for 1 to 2 years after injury; longer follow-up may be necessary for patients with persistent contractures and scar formation. Prolonged follow-up is especially important with young children, who may encounter difficulties as they grow and may therefore require periodic monitoring until adulthood.

***Table 2* Criteria for Outpatient Management of Burn Patients**

Outpatient Management Appropriate	Outpatient Management Inappropriate
Patients with small burns* who have demonstrated understanding of wound care, pain control, and therapy	Abused patients Demented patients Intoxicated patients Homeless patients Patients with comorbid conditions Patients with a language barrier

*1–5% of total body surface area.

Fluid Management

In the late 1960s, Charles Baxter developed objective criteria for resuscitation of the thermally injured patient.[3,4] The Baxter formula (also known as the Parkland formula) calls for the infusion, over 24 hours, of 3 to 4 mL of crystalloid per percentage of TBSA burned. Half of this volume is delivered during the first 8 hours after injury, and the other half is delivered over the subsequent 16 hours. It is important to remember that this is an estimate of need; individual patients may have higher or lower fluid requirements depending on their overall condition and comorbidity. Continuous monitoring and reliance on objective clinical outcomes must dictate patient management.

The reliability of the Parkland formula directly depends on accurate assessment of burn depth and percentage of TBSA affected.[5] There are two formulas for quick estimation of burn size. One is the commonly used Rule of Nines: each arm is considered to be 9% of TBSA, each leg 18%, the anterior trunk 18%, the posterior trunk 18%, and the head 9% [*see Figure 1*]. Another easy method involves using the patient's full palm, including digits, to represent 1% of TBSA. First-degree burns should not be included in the calculation of burned areas.

Despite improvements in invasive monitoring techniques, the most reliable measures of adequate tissue perfusion for burn resuscitation continue to be mean arterial pressure (MAP) and adequate urine output (UOP). MAP should be maintained above 60 mm Hg to ensure adequate cerebral perfusion. For an otherwise healthy adult, a UOP of 30 mL/hr should be adequate; for a child, 1.0 to 1.5 mL/kg/hr should suffice. No evidence supports the use of pulmonary arterial (PA) catheter measurements for routine resuscitation; in fact, reliance on PA catheters may lead to overresuscitation and contribute to the development of fluid-related complications (see below). Use of diuretics and inotropes should be restricted to patients with underlying comorbidity, especially preexisting cardiac disease. Use of inotropes will not stop the leak of plasma into the extravascular space but may lead to ischemia in the wound, resulting in conversion of a partial-thickness wound into a full-thickness wound. Use of mannitol may be appropriate for patients with myoglobinuria who require an osmotic diuretic to maintain a UOP of 100 mL/hr.

Although the first 24 hours after a burn is usually considered the resuscitative phase of a burn injury, stabilization of the flux of mediators and closure of capillary leaks, in fact, take place on a continuum, occurring gradually from 12 to 48 hours after the burn injury. As capillary leakage resolves, the amount of fluids needed to maintain a MAP of 60 mm Hg and a UOP of 30 mL/hr should progressively decrease. A patient with both a large, deep burn and a profound inhalation injury or a patient in whom resuscitation has been delayed may require significantly more fluid than predicted by the Parkland formula to maintain blood pressure and UOP. It is common for a patient with combined thermal and inhalation injury to require 1.5 times Parkland or more.

Baxter's early work showed that capillary leak may persist for up to 24 hours postburn[4] and that plasma expansion during this period was independent of the type of fluid given.[3] More recently, however, it has been suggested that although endothelial dysfunction and capillary leak are present within

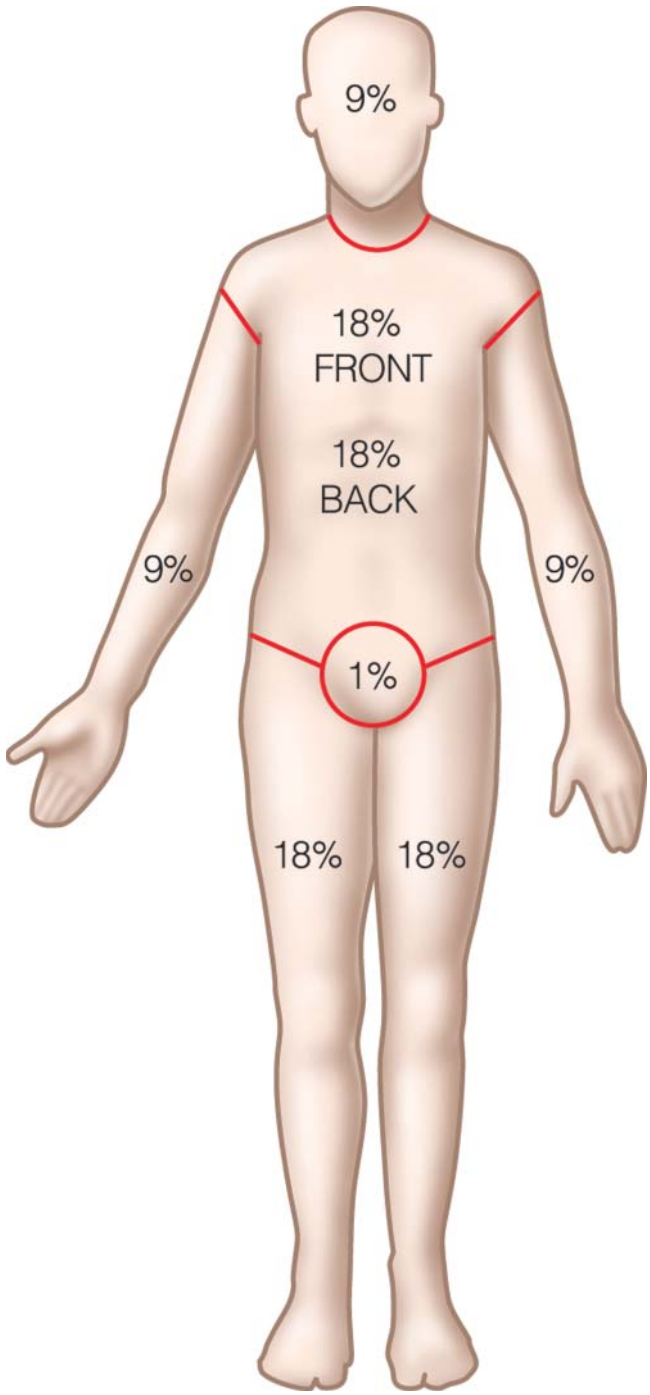

Figure 1 **The size of a burn can be estimated by means of the Rule of Nines, which assigns percentages of total body surface to the head, the extremities, and the front and back of the torso.**

2 hours of burn injury, they last for a median of 5 hours, a period that is much shorter than previously described.[6] Colloid administration (albumin or fresh frozen plasma) after the capillary leak has closed (12 to 48 hours) may facilitate resuscitation in the patient with persistent low UOP and hypotension despite adequate crystalloid delivery. In such cases, the formula used is 5% albumin, 0.3 to 0.5 mL/kg/% TBSA burned over 24 hours. Alternatively, plasmapheresis may reduce intravenous fluid requirements in patients who are not responding to resuscitation.[7] Indications for plasmapheresis include a sustained MAP of less than 60 mm Hg and a UOP less than 30 mL/hr in a patient with ongoing fluid needs that exceed twice the estimated volume requirements. Early plasmapheresis (12 to 24 hours after injury) may decrease the incidence of complications from administration of excessive fluid (see below). Why plasmapheresis works is unknown, but, theoretically, the process should remove inflammatory mediators that cause vasodilatation and capillary leak.

Once resuscitation is complete (24 to 48 hours after injury), insensible losses and hyperthermia associated with a hyperdynamic state may indicate the need for ongoing fluid administration. The route of administration can be intravenous or, preferably, enteral. Reliable daily weights can be extremely valuable for the detection and measurement of insensible fluid loss or fluid retention.

Along with MAP and UOP, several laboratory variables can be used to ensure that patients are receiving appropriate amounts of resuscitation fluid [*see Table 3 and Table 4*].

COMPLICATIONS OF FLUID ADMINISTRATION

Before the development of current resuscitation formulas, inadequate resuscitation was a common cause of death in burn patients as a result of decreased tissue perfusion and subsequent multiorgan failure.[8] In addition, this ischemia caused conversion of the burn to a deeper injury, thereby increasing surgical requirements. However, there are also complications associated with overresuscitation, or so-called fluid creep.[9,10] Whereas Baxter suggested that 12% of patients would require more than 4.3 mL/kg/%TBSA resuscitation fluid,[4] subsequent reports suggested that more than 55% of patients receive this amount of fluid.[8,9,11] Excessive fluid resuscitation increases the risk of complications, including poor tissue perfusion, compartment syndrome involving the abdomen or extremities, pulmonary edema, and pleural effusion.

Abdominal compartment syndrome (ACS) is an increasingly well-recognized posttraumatic complication that occurs in patients who require extensive fluid resuscitation. Increased abdominal pressure decreases lung compliance and impedes lung expansion, resulting in elevated airway pressures and hypoventilation. The classic presentation includes high peak airway pressures, decreased venous return, oliguria, and intra-abdominal pressures exceeding 25 mm Hg.[12] Sustained intra-abdominal hypertension is often fatal. Bedside decompressive laparotomy can alleviate ACS and can be performed safely through burn wounds,[13] and its use should be considered in patients with hemodynamic instability, hypoventilation, and elevated abdominal pressures. Whether the patient survives, however, depends on the comorbid conditions that led to the requirement for large resuscitative volumes.

To avoid potential complications of overaggressive fluid resuscitation, many institutions have developed protocols that guide the intensive care unit (ICU) nurse to adjust the amount of fluid given to a patient based on UOP and MAP, such that if the hourly UOP is low, the fluids are increased, and if the UOP is high, the fluids are decreased.

Table 3 Acute Physiologic Changes during Burn Resuscitation

Measurement	Comment	Goal	Signs of Underresuscitation	Signs of Overresuscitation
Fluid volume	Fluid input generally exceeds output during the early postburn period as edema develops	Urine output: adults, 30 mL/hr; children < 20 kg, 1.0–1.5 mL/kg/hr	Low urine output	Urine output > 30 mL/hr; hyperosmolar diuresis from hyperglycemia must be excluded
Body weight	An accurate dry weight is necessary for estimation of resuscitation fluid requirements	Weight will increase because of intravascular leak and resuscitation volume	Weight approaches dry weight	Massive weight gain from anasarca
Body temperature	Hyperthermia may indicate a hyperdynamic state	Normothermia	—	—
Electrocardiographic status	Dysrhythmias are uncommon in young patients but may complicate management of older patients	Normal sinus rhythm	Tachycardia may reflect intravascular contraction	Dysrhythmias may reflect poor oxygenation, electrolyte imbalance, or pH abnormality

Table 4 Acute Biochemical and Hematologic Changes during Burn Resuscitation

Measurement	Comment	Goal	Signs of Underresuscitation	Signs of Overresuscitation
Serum creatinine and blood urea nitrogen	Normal baseline values rule out preexisting renal disease, which reduces urine output reliability as an index of tissue perfusion	Normal values	Rising values may reflect underresuscitation or acute tubular necrosis	May be normal
Hematocrit and hemoglobin	Significant blood loss from incorrectly performed surgical interventions such as escharotomies or central venous line placement may lower values	Should approach normal	May be elevated with severe intravascular depletion; this is typical with delayed resuscitation	May be low in patients with excessive intravascular volumes
White blood cell count (WBC)	The initial WBC may vary, depending on the stress response and cell margination; the absolute value is not particularly useful during the early postburn period; once leukopoiesis increases, neutropenia resolves without treatment with stimulatory factors	Normal values	May increase	May decrease, but this generally represents margination of circulating neutrophils into the wound
Blood glucose	Increased release of catecholamines in burn patients may lead to hyperglycemia; diabetic patients may require insulin	Levels maintained at ≤ 120 mg/dL	Hyperglycemia, which may misleadingly increase urine output	Hypoglycemia, especially in infants (< 20 kg), who have decreased glycogen stores
Electrolytes	Electrolyte status depends on the type of crystalloid used for resuscitation; hypernatremia and hyponatremia can be avoided by resuscitation with lactated Ringer solution; use of normal saline should be avoided because it can lead to hyperchloremic acidosis	Normal electrolyte levels	—	—
Plasma protein and myoglobin levels	Patients with very deep burns or electrical burns may have elevated plasma myoglobin levels	Decreased albumin level within the first 8 hr after burn injury may be normal	Myoglobinemia may result from prolonged underresuscitation and tissue ischemia	Myoglobinemia may result if excessive resuscitation leads to compartment syndrome; escharotomy should be performed to minimize rhabdomyolysis
Prothrombin time, partial thromboplastin time, and platelet count	Initial values are useful to determine whether the patient has preexisting hepatic or hematologic disease	Normal	Prolonged shock and underresuscitation may lead to disseminated intravascular coagulation; coagulation factors and platelets may be needed in such cases	Unrecognized compartment syndrome and delayed escharotomy may cause tissue ischemia and disseminated intravascular coagulation; a dropping platelet count may indicate heparin-induced thrombocytopenia

Taken to the extreme, this includes creation of a computer program that titrates fluid rate according to UOP.[14,15] Early reports are promising and suggest a low incidence of complications.

Airway Management

Abnormal pulmonary function commonly complicates the management of thermally injured patients. It may result from inhalation injury or from the systemic response to the burn. Understanding the management of pulmonary dysfunction in the thermally injured patient requires a working knowledge of pulmonary function measurements and of pulmonary pathophysiology [*see Table 5 and Table 6*].

INHALATION INJURY

Inhalation injuries occur in approximately one third of all major burns, and mortality is more than double that of cutaneous burns.[16–18] Curiously, isolated inhalation injuries do not result in high mortality.[19] Presumably, the combination of inhalation injury and cutaneous thermal injury creates a double insult in which recurrent or persistent bacteremia aggravates the pulmonary injury.

Three distinct components of inhalation injury exist: carbon monoxide (CO) poisoning, upper airway thermal burns, and inhalation of products of combustion. Diagnosis of an inhalation injury requires a thorough history of the circumstances surrounding the injury and is often suggested by fire in a closed space, carbonaceous sputum, and an elevated carboxyhemoglobin level (> 15%).

Carbon Monoxide Poisoning

CO injury is the most commonly recognized form of inhalation injury and the most common cause of death in inhalation injury. Clinical signs and symptoms of CO toxicity correlate with arterial carboxyhemoglobin levels, which can be used to quickly and precisely determine the degree of CO intoxication [*see Table 7*]. CO intoxication can be easily treated with 100% inhaled oxygen, which rapidly accelerates the dissociation of CO from hemoglobin [*see Table 8*].

Table 5 Measures of Pulmonary Function

Measurement	Normal Values	Abnormal Values Indicating Need for Mechanical Ventilation
Tidal volume (V_T), mL/kg	5–8	< 5
Vital capacity (V_C), mL/kg	65–75	< 10; < 15*
Forced expiratory volume in 1 s (FEV_1), mL/kg	50–60	< 10
Functional residual capacity (FRC), % of predicted value	80–100	< 50
Respiratory rate (f), breaths/min	12–20	> 35
Maximun inspiratory force (MIF), cm H_2O	80–100	< 20; < 25; < 30*
Minute ventilation (V_E), L/min	5–6	> 10
Maximum voluntary ventilation (MVV), L/min	120–180	< 20; < ($2 \times V_E$)*
Dead space fraction (V_D/V_T), %	0.25–0.40	> 0.60
$Paco_2$, mm Hg	36–44	> 50; > 55*
Pao_2, mm Hg	75–100 (breathing room air)	< 50 (room air); < 70 (mask O_2)*
Alveolar-to-arterial Po_2 gradient [$P(A\text{-}a)o_2$], mm Hg	25–65 (breathing 100% O_2)	> 350; > 450*
Arterial-alveolar Po_2 ratio (Pao_2/P_Ao_2)	0.75	< 0.15
Arterial Po_2-inspired O_2 (Pao_2/F_Io_2) mm Hg	350–450	< 200
Intrapulmonary right-to-left shunt fraction (Qs/Qt), %	≤ 5%	> 20%; > 25% > 30%*

F_Io_2 = fraction of inspired oxygen; $Paco_2$ = arterial carbon dioxide tension; Pao_2 = arterial oxygen tension; P_Ao_2 = alveolar oxygen tension.
*More than one value indicates lack of uniform agreement in the literature.

Table 6 Mechanisms of Pulmonary Dysfunction and Indications for Mechanical Ventilation

Mechanism	Best Indicator
Inadequate alveolar ventilation	$Paco_2$ and pH
Inadequate lung expansion	Tidal volume, respiratory rate, VC
Inadequate respiratory muscle strength	MIF; MVV; VC
Excessive work of breathing	V_E required to keep Pco_2 normal; V_D/V_T; respiratory rate
Unstable ventilatory drive	Breathing pattern, clinical setting
Severe hypoxemia	$P(A\text{-}a)o_2$; Pao_2/P_Ao_2; P_Ao_2/F_Io_2; Qs/Qt

F_Io_2 = fraction of inspired oxygen; MIF = maximum inspiratory force; MVV = maximum voluntary ventilation; $P(A\text{-}a)O_2$ = alveolar-to-arterial Po_2 gradient; $Paco_2$ = arterial carbon dioxide tension; Pao_2/F_Io_2 = ratio of arterial Po_2 to inspired O_2; P_Ao_2/F_Io_2 = ratio of alveolar Po_2 to inspired O_2; Qs/Qt = intrapulmonary right-to-left shunt fraction; VC = vital capacity; V_D/V_T = dead space fraction; V_E = minute ventilation.

Table 7 Clinical Manifestations of Carbon Monoxide Poisoning

Carboxyhemoglobin Level (%)	Clinical Manifestations
< 10	None
15–25	Nausea, headache
30–40	Confusion, stupor, weakness
40–60	Coma
> 60	Death

Hyperbaric oxygen therapy has been touted as a superior treatment for quickly reducing carboxyhemoglobin levels,[20] but the data are controversial and the studies are generally poorly controlled.[21] Hyperbaric oxygen therapy may be appropriate in a patient with impaired neurologic status and a markedly elevated carboxyhemoglobin level (> 25%). However, the risks (including barotrauma) associated with isolation in a hyperbaric chamber may be too significant for a burn patient (> 10% TBSA) undergoing resuscitation. In one study of 10 patients with combined inhalation injury and burns treated acutely with hyperbaric oxygen, seven patients survived, but the complications included aspiration (two cases), cardiac arrest (two cases), hypovolemia with metabolic acidosis (three cases), respiratory acidosis (four cases), cardiac dysrhythmia (three cases), and eustachian tube occlusion (two cases).[22] Consensus is growing that when cardiac arrest complicates inhalation injury, the result is uniformly fatal regardless of aggressive therapy, including hyperbaric oxygen therapy.[23]

Upper Airway Thermal Injury

Direct thermal damage tends to occur in the upper airway rather than in the lower airway because the oropharyngeal cavity has a substantial capacity to absorb heat. Upper airway thermal injury constitutes an important indication for intubation because it is mandatory to control the airway before airway edema develops during resuscitation.

The diagnosis of upper airway thermal injury is achieved with direct laryngoscopic visualization of the oropharynx. The decision whether to intubate should be based on visual evidence of pharyngeal burns or swelling or carbonaceous sputum coming from below the level of the vocal cords. If a patient is phonating without stridor, intubation can often be delayed. Singed facial and nasal hair does not constitute an adequate independent indication for intubation.

Treatment of upper airway injuries includes hospital admission for observation and provision of humidified oxygen, pulmonary toilet, bronchodilators as needed, and prophylactic endotracheal intubation as indicated. Upper airway thermal burns usually manifest within 48 hours after injury, and airway swelling can be expected to peak at 12 to 24 hours after injury. A patient with a true upper airway burn will likely require airway protection for 72 hours. A short course of steroids may facilitate earlier resolution of airway edema in a patient with small cutaneous burns, but a patient with a burn larger than 20% TBSA should not be treated with steroids because of the risk of infection and failure to heal. The decision whether to extubate can be based on pulmonary weaning criteria but also on the presence of an air leak around the endotracheal tube.

Table 8 Half-Life of Carbon Monoxide–Hemoglobin Bonds with Inhalation Therapy

Carboxyhemoglobin Half-Life	Treatment Modality
4 hr	Room air
45–60 min	100% oxygen
20 min	100% oxygen at 2 atm (hyperbaric oxygen)

Lower Airway Burn Injury

Burn injury to the tracheobronchial tree and the lung parenchyma results from combustion products in smoke [*see Table 9*] and, under unique conditions, inhaled steam. Numerous irritants in smoke or the vaporized chemical reagents in steam can cause direct mucosal injury, leading to mucosal slough and bronchial edema, bronchoconstriction, and bronchial obstruction. Tracheobronchial mucosal damage also leads to neutrophil chemotaxis and release of inflammatory mediators into the lung parenchyma, accentuating the injury with exudate formation and microvascular permeability. Inflammatory occlusion of terminal bronchioles and necrosis of the mucosa render the airway and pulmonary parenchyma susceptible to infection with resulting pneumonitis, which further increases mortality. Together, these may progress to pulmonary edema, pneumonia, and acute respiratory distress syndrome (ARDS). Reduced myocardial contractility secondary to smoke-toxin inhalation may also contribute to resuscitation failures in burn victims with concomitant inhalation injury.

Inhalation injury can often be a clinical diagnosis.[18] Lower airway injury can be confirmed by bronchoscopy or xenon-133 ventilation-perfusion scan,[24] but these modalities have not traditionally changed therapeutic choices or clinical outcome.[25] There is no standard definition of inhalation injury, but a bronchoscopic grading scheme has been created, based on Abbreviated Injury Score criteria [*see Table 10*],[26] derived from findings on initial bronchoscopy. The scale grades the degree of airway injury based on progressively severe endoluminal damage. Interestingly, bronchoalveolar lavage findings on initial bronchoscopy have demonstrated a high incidence of bacterial contamination, typically gram-positive cocci that would commonly be associated with community-acquired pneumonia, and often in high quantitative colony counts.[27]

ACUTE LUNG INJURY AND ACUTE RESPIRATORY DISTRESS SYNDROME

Understanding of the pathophysiology of ARDS has improved since its initial description in the late 1960s,[28] and ARDS-related deaths were lower in the period 1995 through 1998 than in the period 1990 through 1994; however, 40 to 70% of patients with ARDS still die of the disease.[29] ARDS is an independent risk factor for death in burn patients.[30] Mortality in burn patients with ARDS is attributable to overwhelming sepsis and multiple organ failure rather than to respiratory failure alone.[31]

Clinically, ARDS is characterized by pulmonary edema, refractory hypoxemia, diffuse pulmonary infiltrates, and

Table 9 Clinical Findings Associated with Specific Inhaled Products of Combustion		
Source	**Product of Combustion**	**Clinical Effect**
Organic matter	Carbon monoxide Carbon dioxide	Poor tissue oxygen delivery Narcosis
Wood, paper, anhydrous ammonia	Nitrogen oxides (NO, NO_2)	Airway mucosal irritation, pulmonary edema, dizziness
Polyvinyl chloride (plastics)	Hydrogen chlorine	Airway mucosal irritation
Wool, silk, polyurethane (nylon)	Hydrogen cyanide	Respiratory failure, headache, coma
Petroleum products (gasoline, kerosene, propane, plastics)	Carbon monoxide, nitrogen oxide, benzene	Airway mucosal irritation, coma
Wood, cotton, paper	Aldehydes	Airway mucosal irritation, lung parenchyma damage
Polyurethane (nylon)	Ammonia	Airway mucosal irritation

altered lung compliance. Pathologically, it is distinguished by diffuse alveolar epithelial damage with microvascular permeability and subsequent inflammatory cell infiltration into the lung parenchyma, interstitial and alveolar edema, hyaline membrane formation, and, ultimately, fibrosis.

The development of ARDS is presaged by high fluid resuscitation requirements, reflecting increased microvascular permeability and leading to increased pulmonary edema. ARDS commonly develops within 7 days after injury. The likelihood of death is significantly increased in patients with a multiple organ dysfunction score of 8 or higher and a lung injury score of 2.76 or higher. In one review, burn patients with inhalation injury had a 73% incidence of respiratory failure (with hypoxemia, multiple pulmonary infections, or prolonged ventilator support) and a 20% incidence of ARDS, whereas patients without inhalation injury had a 5% incidence of respiratory failure and a 2% incidence of ARDS.[30] Advanced age may also constitute an important risk factor for the development of ARDS; one small retrospective study has suggested that age is the only independent major predisposing factor for ARDS.[32] Curiously, acute lung injury rarely develops in patients with inhalation injury without cutaneous burns.[33,34]

Inflammatory Mediators in ARDS with Burn Injuries

Local and systemic inflammatory mediators released in response to burn injury include platelet-activating factor, interleukins, prostaglandin, thromboxane, leukotrienes, hematopoietic growth factors, cell adhesion molecules, and nitric oxide (NO).[35,36] Systemic levels of circulating tumor necrosis factor–α (TNF-α) and interleukin-1 correlate with ARDS severity. Clinical studies have correlated infection—but not isolated inhalation injury—with increased IL-2 levels, which reemphasizes the potential significance of the double insult inflicted by the combination of a burn and an inhalation injury.[37] Some data suggest that relative imbalances in levels of inflammatory mediators may be more important than absolute values.

An important cell in the inflammatory cycle is the pulmonary alveolar macrophage, which produces reactive oxygen intermediates (ROIs) as a means of killing microorganisms. In animal models, addition of a burn injury to a smoke insult exaggerates lipid peroxidation and hypoproteinemia, implicating reactive oxygen species in the pathophysiology of ARDS. With systemic inflammation, unchecked ROI production may lead to local tissue injury. ROIs damage cells by direct oxidative injury to cellular proteins and nucleic acids, as well as by inducing lipid peroxidation, which leads to the destruction of the cell membrane. ROIs are generated under conditions of ischemia-reperfusion (as with failed resuscitations), which occurs when the flow of oxygenated blood is restored to ischemic tissue such as unexcised eschar. During ischemia, there is increased activity of xanthine oxidase and increased hypoxanthine production; when reperfusion reintroduces oxygen, the xanthine oxidase and hypoxanthine generate ROIs, which cause more tissue injury.

Table 10 Bronchoscopic Criteria Used to Grade Inhalation Injury	
Grade	**Bronchoscopic Findings**
0 (no injury)	Absence of carbonaceous deposits, erythema, edema, bronchorrhea, or obstruction
1 (mild injury)	Minor or patchy areas of erythema, carbonaceous deposits in proximal or distal bronchi (any combination)
2 (moderate injury)	Moderate degree of erythema, carbonaceous deposits, bronchorrhea, with or without compromise of the bronchi (any combination)
3 (severe injury)	Severe inflammation with friability, copious carbonaceous deposits, bronchorrhea, bronchial obstruction (any combination)
4 (massive injury)	Evidence of mucosal sloughing, necrosis, endoluminal obliteration (any combination)

Management of ARDS

In spite of 30 years of advances in ARDS treatment, patients with ARDS still must depend on mechanical respiratory support—not treatment—as the primary therapeutic intervention while the alveolar epithelium repairs itself, the capillary permeability resolves, and the lung heals. Restricting fluids to prevent further edema formation has increased survival.[38] The most encouraging strategy to prevent lung injury and increase survival has been low tidal volume mechanical ventilation, commonly called lung protective ventilation, with or without high levels of positive end-expiratory pressure (PEEP).[39,40] Pharmacologic approaches to treating ARDS in burn patients parallel those used in other critically ill surgical patients and are addressed elsewhere.

For most patients with pulmonary complications from thermal injury, conventional ventilatory approaches will be

adequate. However, the population at risk for development of ARDS may need more sophisticated management to reduce barotrauma and pulmonary infection in the minimally compliant lung with increased airway pressures. In the past, conventional ventilator management of inhalation injury and ARDS, which emphasizes normalization of blood gases, promoted high rates of barotrauma—that is, ventilator-induced lung injury that is physiologically and histopathologically indistinguishable from ARDS itself. Overdistention and cyclic inflation of injured lung exacerbate underlying lung injury and perpetuate systemic inflammation. These effects can be minimized by maintaining low tidal volumes and plateau pressures and by applying PEEP. Hence, the use of alternative modes of ventilation (e.g., volume-limited ventilation with or without inverse-ratio ventilation, prone positioning, and tracheal gas insufflation) has increased in patients at risk for ARDS. No single approach is likely to benefit all patients, and adjustment of ventilatory controls must be based on individual clinical responses.

Lung-protective ventilation Lung-protective ventilation uses low inspiratory volumes (4 to 6 mL/kg) to keep peak plateau pressures below 30 cm H_2O. This strategy is limited by the accumulation of CO_2 (so-called permissive hypercapnia), although respiratory acidosis with a pH as low as 7.20 is tolerated.

The Acute Respiratory Distress Syndrome Network Study (ARDSNet) found that ARDS patients ventilated with low tidal volumes had a 22% lower mortality than patients ventilated with conventional means.[40,41] The volume-preset, assist-control mode is recommended for tidal volume control, and the respiratory rate should be slowly increased as tidal volume is reduced to maintain minute ventilation and prevent acute hypercapnia. Tidal volume can be increased for severe acidosis ($pH < 7.15$). Alternatively, a sodium bicarbonate infusion may be initiated to correct pH and tracheal gas insufflation can be used to help clear CO_2 in cases of severe hypercarbia. Ventilator inspiratory flow should be optimized to minimize dyspnea. If dyspnea results in asynchronous breaths, sedation may be necessary or the tidal volume can be titrated to 7 to 8 mL/kg provided that plateau pressures are below 30 cm H_2O.

One study of children with burns found that low tidal volume ventilation was associated with low incidences of ventilator-induced lung injury and respiratory-related deaths,[42] which supports the use of this modality in thermally injured patients. In fact, in patients with large burns and inhalation injury, it may be warranted to use low tidal volume ventilation before ARDS develops. The early resuscitative phase may be the optimal time to initiate this approach.

Positive end-expiratory pressure The ARDSNet comparing ventilation with lower tidal volumes to traditional tidal volumes for acute lung injury employed a PEEP ladder with increasing amounts of PEEP for increasing fraction of inspired oxygen (F_IO_2), from a low of 5 mm Hg PEEP to a high of 24 mm Hg PEEP.[40,41] The higher respiratory rate associated with the smaller tidal volumes of the ARDSNet strategy has been found to generate a higher total PEEP as a result of the addition of intrinsic PEEP (from shortened expiration time), and this has been postulated as a possible mechanism for the 22% reduction in mortality.[43] Acute lung injury is a heterogeneous disease, and different etiologies of lung injury likely benefit from different approaches to ventilatory management. In patients with a focal distribution of loss of aeration, there is a low potential for alveolar recruitment and susceptibility to alveolar hyperventilation with high levels of PEEP. In such patients, examination of static lung compliance and plasma concentrations of IL-6, IL-8, and tumor necrosis factor receptor were decreased with a more conservative PEEP strategy than the ARDSNet protocol.[39] In contrast, a patient with inhalation injury and significant truncal thermal burns undergoing fluid resuscitation exhibits a more diffuse lung injury pattern with significant decrease in the chest wall compliance and more pleural effusions. Talmor and colleagues have suggested that such patients may benefit from placement of an esophageal balloon and titration of the tidal volume and PEEP according to the measured transpulmonary pressure.[44]

Prone positioning Changing a patient's position from supine to prone is emerging as a simple and inexpensive strategy to improve gas exchange in acutely injured lungs. Studies report that despite concerns about airway protection, this is a safe intervention that may improve the ratio of arterial oxygen pressure to fraction of inspired oxygen (Pa_{O_2}/F_IO_2) early in the course of ARDS.[45] Protti and colleagues used computed tomography to show that decreased arterial carbon dioxide tension (Pa_{CO_2}) following prone positioning is related to lung recruitability and, accordingly, severity of lung injury.[46] Some data suggest that prone positioning in conjunction with NO administration may improve arterial oxygenation.[47] However, no clinical trials have examined the use of prone positioning in burn patients. If prone positioning has a significant effect, this positive result presumably would be evident during operative procedures when a patient with an acute lung injury is placed in this position (e.g., for excision of a posterior torso burn). Furthermore, prone positioning may be relatively contraindicated in a patient with a burned head who is at extreme risk for loss of control of the airway because of facial swelling and difficulty securing an endotracheal tube.

Extracorporeal membrane oxygenation Few centers have experience with extracorporeal membrane oxygenation (ECMO), and published information on its use for the treatment of ARDS in patients with inhalation injury and burns is mostly confined to anecdotal case reports.[48] Given its experimental nature and its high cost, ECMO is reserved for patients in whom other ventilatory modalities fail. Although ECMO has been shown to increase survival in some children with large burns and severe acute lung injury, patients with higher ventilator requirements before undergoing ECMO generally do not survive, suggesting that if ECMO is to be successful, it must be instituted early to prevent barotrauma and irreversible lung injury.[49] Early implementation of permissive hypercapnia and lung protective ventilation may be equally effective.

High-frequency percussive ventilation High-frequency percussive ventilation (HFPV) is another strategy for maintaining low peak pulmonary pressure and preventing alveolar overdistention. HFPV has the added advantage of

facilitating mucosal clearance of tracheobronchial casts that occlude the airway and predispose to pulmonary infection. Although HFPV is usually described as rescue therapy for patients in whom conventional therapy has failed, there is some evidence that it can reduce mortality and the incidence of pneumonia in patients with inhalation injury.[50,51] Improved oxygenation and pulmonary toilet have been reported in patients treated early with HFPV,[52] which suggests that a larger-scale prospective trial is warranted to determine whether the benefits of HFPV justify the added cost and effort of maintaining multiple types of ventilators and credentialing for respiratory therapists.

A similar method, high-frequency oscillatory ventilation, may have no impact on burn mortality. However, it may have a role in the supportive management of burn patients with severe oxygenation failure that is unresponsive to conventional ventilation.[53]

NO inhalation Endogenously produced NO plays an important role in the changes in systemic and pulmonary microvascular permeability seen in an animal model of combined smoke inhalation and third-degree burn.[36] Clinically, inhaled NO may be useful in burn patients with severe acute lung injury causing refractory hypoxemia in whom conventional ventilatory support is failing.[54] Low methemoglobin levels and absence of hypotension attributable to the NO indicate the safety of inhaled NO in these patients. Strong, immediate, and sustained improvement in the $Pa_{O_2}/F_{I}O_2$ and reduction in PA mean pressure in response to NO seem to correlate with survival. However, many studies have failed to show a survival advantage with inhaled NO, and a prospective study is warranted.

Corticosteroids The use of corticosteroids in the treatment of burns is potentially problematic because of the negative effect these agents have on wound healing. Nevertheless, there is evidence that early initiation of low- to moderate-dose corticosteroids for severe ARDS continued for 14 to 21 days before tapering may decrease the mortality and morbidity outcomes without increased adverse reactions.[55] Encouragingly, comparison of the effects of dexamethasone, methylprednisolone, and hydrocortisone on wound healing in an animal model has shown that unlike dexamethasone and hydrocortiscone, methylprednisolone did not significantly impair wound healing.[56] Thus, in a patient with severe ARDS, methylprednisolone may be the corticosteroid of choice to lessen the morbidity and mortality of severe lung injury. Larger multicenter studies, particularly those including burn patients, could strengthen the findings from meta-analyses of the existing studies and more definitively answer questions about risks and benefits.

Nebulized heparin Numerous pathologic airway alterations occur with inhalation injury and acute lung injury/ARDS. Augmented bronchial circulation, bronchospasm, and airway obstruction from cast formation from mucus secretions, denuded epithelial cells, inflammatory cells, and fibrin act to impair pulmonary function. Therefore, prevention or dissolution of fibrin cast formation with its resultant ventilation/perfusion mismatch, increased atelectasis, and associated inflammation could significantly improve pulmonary function.[57] Desai and colleagues have reported that nebulized heparin combined with *N*-acetylcysteine significantly decreased reintubation rates, incidence of atelectasis, and mortality in children with massive burn and smoke inhalation injury.[58]

TRACHEOSTOMY VERSUS ENDOTRACHEAL INTUBATION

Transmural airway inflammation from inhaled gases and heat necessitates endotracheal airway protection, yet the use of endotracheal tubes in such cases may be complicated by tracheal pressure necrosis. Hence, survivors of inhalation injury may develop laryngotracheal strictures. One report suggests that there is a 5.5% incidence of tracheal stenosis in patients with burns and inhalation injury.[59] The relative risks and benefits of tracheostomies and endotracheal intubation have been debated since the early 1970s. Each modality has its own advantages and complications. Nasotracheal intubation is the least advantageous form of airway protection because of its association with paranasal sinusitis,[60] as well as pressure necrosis of the alar rim of the burned nose, which is nearly impossible to reconstruct. Therefore, nasotracheal intubation should be avoided unless absolutely necessary.

Tracheostomies are also associated with complications, including tracheal malacia, tracheal stenosis, tracheoinnominate artery fistulas, tracheoesophageal fistulas, and posttracheostomy dysphagia.[61] However, complications associated with tracheostomy may relate to previous long-term endotracheal intubation and to the underlying pathophysiology, suggesting that if tracheostomy is to be done, it should be done early on. Furthermore, the tracheostomy tube should be removed at the earliest possible time. In a 1985 study of airway management, tracheal stenosis and tracheal scar granuloma formation were reported to be more frequent and more severe after tracheostomy than after translaryngeal intubation.[62] As expected, the duration of tube placement significantly affected the development of permanent damage, leading to the conclusion that initial respiratory support with translaryngeal tubes is preferable for up to 3 weeks. Burn patients who undergo tracheostomy before postburn day 10 may have a lower incidence of subglottic stenosis with no difference in pneumonia incidence when compared with orally intubated patients.[63] Nevertheless, tracheostomy has been reported to provide no benefit for early extubation or overall outcome for burn patients.[49] A major consideration in deciding whether to perform a tracheostomy has been the presence of eschar at the insertion site, which complicates tracheostomy-site care and increases the risk of airway infection. Several investigators reported the use of percutaneous tracheostomy in burn patients to be safe and cost effective; in one study, percutaneous tracheostomy had a lower incidence of stoma-site infections and pulmonary sepsis.[64] Thus, percutaneous dilatational tracheostomy may provide a reasonable, less invasive approach for patients who are likely to need prolonged ventilatory support.[65] This procedure can be safely performed at the bedside, at one quarter the cost of a conventional tracheostomy. Given ongoing controversies over the relative risks and benefits of endotracheal intubation and tracheostomy in burn patients and the rarity of complications from intubation in our own practice, we perform tracheostomies only when multiple attempts at extubation have failed;

these failures usually occur because the patients cannot protect their airway.

Temperature Regulation

Because the burn patient has lost the barrier function of the skin, temperature regulation is an important goal of successful management. Keeping a patient warm and dry is a major goal during resuscitation, especially during the pre–burn center transport of patients. This includes maintaining a warm ambient temperature. Large evaporative losses[66] combined with administration of large volumes of intravenous fluids that are at room temperature or colder may accentuate the hypovolemia, which will complicate the patient's overall course and may lead to disseminated intravascular coagulopathy. Mild hyperthermia may occur in the first 24 hours as a result of pyrogen release or increased metabolic rate[67] and may cause tachycardia that misleadingly suggests hypovolemia. Because infection is unlikely early on, especially within the first 72 hours after injury, elevated temperatures should be treated with antipyrogens to control the energy expenditure associated with increased catabolism.[68] About 72 hours after injury, patients with thermal injuries commonly develop a hyperdynamic state, the systemic inflammatory response syndrome (SIRS), which is characterized by tachycardia, hypotension, and hyperthermia—classic signs of sepsis that in this case do not have an infectious source.

Although patients with burns are likely to have elevated temperatures and may even have elevated white blood cell counts, fevers in burn patients are not reliable indicators of infections.[69] At least one study has demonstrated that in pediatric burn patients, physical examination is the most reliable tool for evaluating the source of fever.[69]

Infection Control

Infection is a major potential problem for patients with large thermal injuries. In one review, up to 100% of such patients developed an infection from one or more sources during the hospital stay. It is important to apply sound epidemiologic practice to treating infections, both to limit development of opportunistic infections in individual patients and to achieve good infection control in the burn unit itself.

Tetanus prophylaxis has been standard for patients admitted for any type of trauma, primarily because the disease is so devastating and its prevention so simple. There are a few cases in the modern medical literature of tetanus in patients who received immunization during childhood.[70]

For many years, all patients admitted with burn injuries received antibiotic prophylaxis against gram-positive organisms. This practice often led to the development of gram-negative bacterial infections or, even worse, fungal infections. Studies have now verified that prophylactic antibiotics not only are unnecessary but also may well be contraindicated in patients with burns.[71] Therefore, treatment of infections in patients with burns should be based on clinical judgment and supportive laboratory and radiologic findings.

The wound is a primary source of infection for patients with burns. The mainstay of both prevention and treatment is daily washing with soap and water and application of a topical broad-spectrum antimicrobial agent. As soon as it becomes evident that a burn wound will not heal, excision and grafting should be performed. Preferably, the decision to proceed with surgery should be made before postburn day 21. For patients who undergo surgery, perioperative antibiotics may reduce postoperative wound infection.[71]

Nutrition

In the early 1970s, Curreri and Luterman recognized that patients with major thermal injury experience hypermetabolism, with an increased basal metabolic rate, increased oxygen consumption, negative nitrogen balance, and weight loss; hence, these patients have exaggerated caloric requirements.[72] Furthermore, inadequate caloric intake can be associated with delayed wound healing, decreased immune competence, and cellular dysfunction.

A patient with a large burn may lose as much as 30 g of nitrogen a day because of protein catabolism. Not only is urinary excretion of urea nitrogen increased, but also large amounts of nitrogen are lost from the wound itself. Therefore, total urea nitrogen levels do not accurately reflect all nitrogen losses in burn patients.[73] A patient with a small burn (< 10% TBSA) may lose nitrogen at a rate of 0.02 g/kg/day. A moderate burn (11 to 29% TBSA) may be associated with nitrogen losses equaling 0.05 g/kg/day. A large burn (≥ 30% TBSA) may result in the loss of as much as 0.12 g/kg/day, which may be equivalent to daily losses of 190 g of protein or about 300 g of muscle. Thus, a highly catabolic patient with a large burn will typically require 2 g/kg/day of protein to maintain a positive nitrogen balance.

Catabolism generally continues until wounds have healed. However, once a patient becomes anabolic, preburn muscle takes three times as long to regain as it took to lose.[74,75] Therefore, a patient in whom it takes 1 month for burn wounds and donor sites to heal may need 3 or more months to regain preburn weight and muscle mass. These statistics underscore the importance of accurately estimating each patient's caloric needs during hospitalization. Over the years, a number of equations have been developed to estimate caloric needs [*see Table 11*]. Probably the most widely used formula is the Harris-Benedict equation, which estimates basal energy expenditure according to gender, age, height, and weight:

Men BEE: 13.75 × Weight + 5.00 × Height
− 6.76 × Age + 66.5
Women BEE: 9.56 × Weight + 1.85 × Height
− 4.68 × Age + 65.5

Table 11 **Formulas for Estimating Caloric Needs in Burn Patients**

Harris-Benedict Formula
Basal energy expenditure (BEE)* × activity factor† = calories needed daily
Curreri Formula
25 kcal / kg + 40 kcal/% TBSA burned = calories needed daily

TBSA = total body surface area.
*Women: BEE = 65.5 + 9.6 (weight in kg) + 1.8 (height in cm) − 4.7 (age in years). Men: BEE = 66.5 + 13.8 (weight in kg) + 5.0 (height in cm) − 6.8 (age in years).
†For burns, the activity factor is 2, which may overestimate caloric needs for patients with smaller burns.

The basal energy expenditure is then multiplied by an activity factor that reflects the severity of injury or the degree of illness; for burns, this multiplier is 2, the maximal factor for this formula. The limitation of the Harris-Benedict equation is that it may overestimate caloric needs for patients with burns smaller than 40% TBSA. A formula specific for patients with burns is the Curreri formula,[76] which is based on patient weight and burn size; this formula may overestimate caloric needs for patients with large burns and therefore is best used for patients with burns less than 40% TBSA.[77,78]

Ongoing evaluation of metabolic status of the burn patient is necessary to take into account changes in wound size and clinical condition. Metabolic demands decrease with burn healing or grafting; on the other hand, donor sites create new wounds, which may increase catabolic rates. Development of infection or ARDS greatly increases catabolism and may alter caloric needs.[79] Simple assessment of nitrogen requirements can be determined by measuring 24-hour total urea nitrogen levels in the urine. However, this does not account for nitrogen lost from the wound itself. Serum albumin levels are notoriously unreliable markers of adequate nutrition because they lag behind clinical progress; they are especially known to be low in patients with burns larger than 20% TBSA.[80] Transthyretin (also known as prealbumin, although it is not related to albumin in structure or function) levels correlate more closely with catabolic status,[81] and a trend over several weeks may indicate whether the patient's caloric needs are being met.[82] C-reactive protein levels also provide an indication of the patient's general inflammatory state; high levels may correlate with increased catabolism.[83] For intubated patients, indirect calorimetry may be helpful in measuring caloric needs but may not be more exact than the Curreri formula.[83] The so-called metabolic cart is a portable gas analyzer that quantifies volumes of inspired O_2 and expired CO_2 and calculates nutritional requirements according to the following formula:

$$\text{kcal/day} = ([3.9 \times \dot{V}O_2] + [1.1 \times \dot{V}CO_2]) \times 1.44$$

where $\dot{V}O_2$ is the volume of oxygen consumed and $\dot{V}CO_2$ is the volume of carbon dioxide consumed. This result can also be indirectly measured in patients with PA catheters in place by using the Fick equation:

$$\text{kcal/day} = \text{cardiac output} \times (\text{arterial } PO_2^- \text{ venous } PO_2) \times 10 \times 6.96$$

ENTERAL NUTRITION

As early as 1976, the benefits of enteral nutrition over parenteral nutrition had already been identified for patients with functional gastrointestinal systems.[84] The problems of prolonged ileus and Curling stress ulcers in burn patients have been largely eliminated by early feeding.[85] Multiple studies have shown that patients with major thermal injury can receive adequate calories within 72 hours after injury.[86] At the University of Washington Regional Burn Center, tube feeding is started a median of 5 hours after admission.

There is ongoing debate about the benefits of gastric feeding versus duodenal feeding. Although feeding distal to the pylorus should pose less aspiration risk, one study found evidence of enteral formula in pulmonary secretions of 7% of patients receiving gastric feeds compared with 13% of patients receiving transpyloric feeding.[87] Hence, for burn patients with high caloric needs, the benefit of decreased aspiration with transpyloric feeds may only be theoretical and may be offset by the delay in feeding for confirmation of tube placement; such confirmation is necessary because these tubes can easily flip back into the stomach.

Continuation of tube feedings during surgery in intubated patients who require multiple operations is a safe way to maximize caloric intake and decrease wound infection. There is no need to stop feedings for anesthesia induction and endotracheal intubation in the patient with a secure airway[88]; however, intraoperative positioning, especially if the patient will be prone during surgery, may necessitate stopping feedings preoperatively. Mayes and colleagues have presented data that support continuation of tube feedings in critically ill burn patients undergoing decompressive laparotomy.[89]

GLUCOSE LEVELS

Both hyperglycemia and hypoglycemia are associated with increased mortality and morbidity in critically ill patients. Improved survival in surgical ICU patients maintained with tight blood glucose control of 80 to 110 mg/dL[90] led to the widely practiced policy of tight glycemic control. The association of poor glucose control with bacteremia, reduced graft take, and higher mortality in pediatric burn patients has further supported this practice.[91] Based on the current literature, it is our policy to maintain blood glucose levels as close to normal as possible, without evoking unacceptable glucose fluctuations, hypoglycemia, or hypokalemia, and therefore aim for a range of 100 to 180 mg/dL.

ALBUMIN LEVELS

Burn patients characteristically have hypoalbuminemia that persists until wounds are healed and the rehabilitation phase of recovery has begun. In fact, patients with large burns have serum albumin levels that average 1.7 g/dL and never exceed 2.5 g/dL.[80] Management of hypoalbuminemia is controversial, but there is general agreement that once burn resuscitation is complete, infusion of exogenous albumin to serum levels above 1.5 g/dL does not affect burn patient length of stay, complication rate, or mortality.[92]

NUTRITIONAL SUPPLEMENTS

Specialized nutritional formulas with purported effects on metabolic rate and immunologic status have garnered a great deal of interest as adjuncts in the management of critically ill and injured patients.[93] Much of the information on nutritional requirements for critically ill patients was derived from an animal burn model,[94] however, and studies on the efficacy of specialized nutritional supplements in humans have generated contradictory data. A randomized trial of nutritional formulas that were intended to enhance immune status and that included essential amino acids and omega-3 fatty acids showed no clinical advantage in burn patients.[95] However, the addition of glutamine supplementation to an enteral nutrition regimen has been shown to decrease hospital and ICU length of stay as well as mortality in adult burn patients, perhaps explained by its trophic influence on intestinal epithelium and on maintenance of gut integrity.[96] Other nutritional or metabolically active supplements that have demonstrated promise in promoting anabolism in burn

patients include insulin, recombinant human growth factor, the anabolic steroid oxandrolone, and propranolol.[97] Oxandrolone in particular has produced marked improvements in weight gain, return to function, and length of hospital stay[98]; however, its use should be cautioned in nonburn patients as surgical patients have not shown the same benefit.[99] Early administration of antioxidant supplementation with α-tocopherol and ascorbic acid has been shown to reduce the incidence of organ failure and shorten ICU length of stay in critically ill surgical patients.[100] Whether this is true for burn patients remains to be demonstrated, but the relatively low cost and the low risk of complications make this an attractive intervention for burn patients at risk for ARDS.

Anemia

Because acute blood loss is uncommon in a patient with an isolated burn injury, a rapidly decreasing hematocrit during resuscitation should prompt an evaluation for associated injuries. Procedures during resuscitation, such as central venous line placement or escharotomies, should not be associated with significant blood loss.

Anemia was a major problem in burn management before early excision and grafting became commonplace. As excision techniques have become more sophisticated, operative blood loss has decreased, as has the need for transfusion.[101,102] Nevertheless, excision and grafting may be associated with large blood loss, and the operating team must be prepared for intraoperative blood transfusion.

Decisions about transfusion must be based on the patient's age, overall condition, and comorbidity. The risks of viral transmission and transfusion reactions, as well as the cost, must also be carefully considered. Based on the findings of the Transfusion Requirements in Critical Care (TRICC) trial, for an otherwise healthy patient who does not need surgery, a hematocrit as low as 20% may be tolerated.[103] Patients with inhalation injury or ARDS may benefit from the greater oxygen-carrying capacity afforded by a higher hematocrit. Studies of the effects of prolonged storage of blood have shown that storage alters corpuscle and cytosol and thereby impairs oxygen-carrying capacity. Blood transfusion can lead to impaired oxygen uptake and can be proinflammatory. Additionally, each transfusion has been shown to have an associated increased infectious risk. Therefore, for patients with large burns who require multiple operations for excision and grafting, every effort should be made to limit blood loss and decrease phlebotomy to conserve blood and decrease the need for blood transfusions. Additionally, patients with large burns and anticipated blood loss during hospitalization should probably receive iron supplements.

Given that the literature contains some indication that erythropoietin levels may be elevated in patients with large burns, the benefit of exogenous erythropoietin is debatable.[104] At least one prospective study suggests that administration of recombinant erythropoietin in acutely burned patients does not prevent anemia or decrease transfusion requirements.[105]

Pain Management

Pain management for patients with burn injuries can be challenging. The simplest approaches work best; polypharmaceutical drug administration is likely to confuse both patients and health care providers and should therefore be avoided. Burn patients experience several different classes of pain: background, breakthrough, and procedural. Each patient responds to a different approach.

Background pain is the discomfort that burn patients experience day and night. It is best treated with long-acting pain relievers. For a hospitalized patient with large burns, methadone or controlled-release morphine sulfate may be the most appropriate choice for background pain. In an outpatient with a small burn, a nonsteroidal antiinflammatory drug (NSAID) may be optimal; if excision and grafting are planned, the NSAID should be stopped at least 7 days before surgery to permit recovery of platelet function.

Breakthrough pain results when activities of daily living exacerbate burn-wound discomfort. Short-acting narcotics or acetaminophen is used to alleviate breakthrough pain. Persistent breakthrough pain indicates that the dose of the long-acting medication should be increased.

Procedural pain is the discomfort that patients experience during wound care and dressing changes. This usually requires treatment with a short-acting narcotic. For inpatients with larger burns, oral narcotics or transmucosal fentanyl citrate[106] works well for wound care; intravenous morphine or fentanyl is used for uncontrolled pain. For outpatients, oxycodone (5 to 15 mg) works well for daily wound care.

Anxiety related to wound care is an underdiagnosed and undertreated source of discomfort that is often construed as pain, especially in children. Therefore, patients with large burns requiring wound care once or twice a day should be evaluated to determine whether they would benefit from a short-acting anxiolytic agent for procedures. Reports have suggested encouraging results in treating neuropathic hyperalgesia and allodynia in the burn wound or donor site with gabapentin, finding rapid reduction in neuropathic pain with no severe adverse reactions.[107]

Learning to accurately assess pain in burn patients can help prevent complications related to excessive narcotic use, such as prolonged sedation, delirium, and, more urgently, loss of airway control. This is especially true in young children and elderly patients, who may have decreased ability to tolerate narcotics.[108,109]

Nonpharmacologic approaches are also an important component of pain management in burn patients. Hypnosis—administered either by trained health care providers or, more efficiently, by patients themselves—has proved to be a useful tool for reducing narcotic use in patients with burns.[110] Another distraction modality that has shown promise and garnered significant publicity has been virtual reality. Although it is not a standard of care for all patients admitted with burn injuries, preliminary observations suggest that use of virtual reality can enhance patient comfort during wound care and intense therapy.

Discomfort in the healed wound may persist for months after injury. In general, narcotics do not control such symptoms; exercise and deep massage are more effective. Itching can be a pervasive long-term symptom for which there is no reliable topical or systemic therapy. Diphenhydramine, cyproheptadine, cetirizine, and gabapentin may relieve itching. There are also promising data on the use of doxepin ointment as a topical treatment for itching of healed wounds.[111] Keeping the wound moist with a topical salve may be as effective as other pharmacologic approaches.

Deep Vein Thrombosis Prophylaxis

The incidence of deep vein thrombosis (DVT) and, thus, the need for DVT prophylaxis in patients with thermal injury have never been clearly defined. Whereas some studies report DVT in as many as 25% of all hospitalized burn patients and advocate DVT prophylaxis,[112] others report that thromboembolism is responsible for only 0.14% of deaths in burn patients and does not warrant the potential complications of anticoagulation therapy.[113] At the University of Washington Regional Burn Center, a quality assurance review found that in patients with burns larger than 20% TBSA, clinically evident thromboembolic disease occurred in 9% of those who received prophylaxis with unfractionated heparin and in 18% of those who received low-molecular-weight heparin, perhaps as a result of a greater tendency for the low-molecular-weight heparin to be held. On the basis of these data, patients with burns larger than 20% TBSA receive prophylaxis with subcutaneous unfractionated heparin, 5,000 U three times a day.

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