Optimal care of the burn patient requires not only specialized equipment but also, more important, 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 [see 7:15 Management of the Burn Wound]. 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 debride 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 [see 7:15 Management of the Burn Wound], an acute increase in stinging 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 epithelialized 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

<table>
<thead>
<tr>
<th>Table 1 American Burn Association Criteria for Burn Injuries That Warrant Referral to a Burn Unit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Partial-thickness burns of greater than 10% of total body surface area</td>
</tr>
<tr>
<td>Third-degree burns</td>
</tr>
<tr>
<td>Electrical burns, including lightning injury</td>
</tr>
<tr>
<td>Chemical burns</td>
</tr>
<tr>
<td>Inhalation injury</td>
</tr>
<tr>
<td>Burn injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or increase mortality</td>
</tr>
<tr>
<td>Burns with concomitant trauma</td>
</tr>
</tbody>
</table>
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—scarving, contractures, and rehabilitation difficulties [see 7:17 Rehabilitation of the Burn Patient]—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.

Fluid Management

In the late 1960s, Charles Baxter developed objective criteria for resuscitation of the thermally injured patient. 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. 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 [see 7:15 Management of the Burn Wound] 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 [see 7:16 Miscellaneous Burns and Cold Injuries].

Table 2  Criteria for Outpatient Management of Burn Patients

<table>
<thead>
<tr>
<th>Outpatient Management Appropriate</th>
<th>Outpatient Management Inappropriate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with small burns* who have demonstrated understanding of wound care, pain control, and therapy</td>
<td>Abused patients</td>
</tr>
<tr>
<td>Demented patients</td>
<td>Intoxicated patients</td>
</tr>
<tr>
<td>Homeless patients</td>
<td>Patients with comorbid conditions</td>
</tr>
<tr>
<td>Patients with a language barrier</td>
<td></td>
</tr>
</tbody>
</table>

*1%–5% of total body surface area.
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.

Colloid administration (albumin or fresh frozen plasma) after the capillary leak has closed (12 to 72 hours) may facilitate resuscitation in the patient with persistent low urine output 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 intravascular leak and resuscitation volume in patients who are not responding to resuscitation. Indications for plasmapheresis include a sustained MAP of less than 60 mm Hg and a UOP of 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), insensitive 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 detection and measurement of insensitive 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 Tables 3 and 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. 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. Whereas Baxter suggested that 12% of patients would require more than 4.3 ml/kg/% TBSA resuscitation fluid, subsequent reports suggest that more than 55% of patients receive this amount of fluid. 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. Sustained intra-abdominal hypertension is often fatal. Bedside decompressive laparotomy can alleviate ACS and can be performed safely through burn wounds, and its use should be considered in patients with hemodynamic instability, hyperventilation, and elevated abdominal pressures. Whether the patient survives, however, depends on the comorbid conditions that led to the requirement for large resuscitative volumes.

### 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 Tables 5 and 6].

### INHALATION INJURY

Inhalation injuries occur in approximately one third of all major burns, and mortality is more than double that of cutaneous burns. Curiously, isolated inhalation injuries do not result in high mortality. 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

### Table 3  Acute Physiologic Changes during Burn Resuscitation

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Comment</th>
<th>Goal</th>
<th>Signs of Underresuscitation</th>
<th>Signs of Overresuscitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluid volume</td>
<td>Fluid input generally exceeds output during the early post-burn period as edema develops</td>
<td>Urine output: adults, 30 ml/hr; children &lt; 20 kg, 1.0–1.5 ml/kg/hr</td>
<td>Low urine output</td>
<td>Urine output &gt; 30 ml/hr; hyperglycemia must be excluded</td>
</tr>
<tr>
<td>Body weight</td>
<td>An accurate dry weight is necessary for estimation of resuscitation fluid requirements</td>
<td>Weight will increase because of intravascular leak and resuscitation volume</td>
<td>Weight approaches dry weight</td>
<td>Massive weight gain from anasarca</td>
</tr>
<tr>
<td>Body temperature</td>
<td>Hyperthermia may indicate a hyperdynamic state</td>
<td>Normothermia</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Electrocardiographic status</td>
<td>Dysrhythmias are uncommon in young patients but may complicate management of older patients</td>
<td>Normal sinus rhythm</td>
<td>Tachycardia may reflect intravascular contraction</td>
<td>Dysrhythmias may reflect poor oxygenation, electrolyte imbalance, or pH abnormality</td>
</tr>
</tbody>
</table>
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]. Hyperbaric oxygen therapy has been touted as a superior treatment for quickly reducing carboxyhemoglobin levels, but data are controversial and the studies are generally poorly controlled. Hyperbaric oxygen therapy may be appropriate in a controlled setting. Hyperbaric oxygen therapy 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), hypoxemia with metabolic acidosis (three cases), respiratory acidosis (four cases), hyperglycemia, which may misleadingly increase urine output

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Comment</th>
<th>Goal</th>
<th>Signs of Underresuscitation</th>
<th>Signs of Overresuscitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum creatinine and blood urea nitrogen</td>
<td>Normal baseline values rule out preexisting renal disease, which reduces urine output reliability as an index of tissue perfusion</td>
<td>Normal values</td>
<td>Rising values may reflect underresuscitation or acute tubular necrosis</td>
<td>May be normal</td>
</tr>
<tr>
<td>Hematocrit and hemoglobin</td>
<td>Significant blood loss from incorrectly performed surgical interventions such as escharotomies or central venous line placement may lower values</td>
<td>Should approach normal</td>
<td>May be elevated with severe intravascular depletion; this is typical with delayed resuscitation</td>
<td>May be low in patients with excessive intravascular volumes</td>
</tr>
<tr>
<td>White blood cell count (WBC)</td>
<td>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 leukopenia increases, neutropenia resolves without treatment with stimulatory factors</td>
<td>Normal values</td>
<td>May increase</td>
<td></td>
</tr>
<tr>
<td>Blood glucose</td>
<td>Increased release of catecholamines in burn patients may lead to hyperglycemia; diabetic patients may require insulin</td>
<td>Levels maintained at ≤ 120 mg/dl</td>
<td>Hyperglycemia, which may misleadingly increase urine output</td>
<td>Hypoglycemia, especially in infants (&lt; 20 kg), who have decreased glycogen stores</td>
</tr>
<tr>
<td>Electrolytes</td>
<td>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</td>
<td>Normal electrolyte levels</td>
<td>―</td>
<td>―</td>
</tr>
<tr>
<td>Plasma protein and myoglobin levels</td>
<td>Patients with very deep burns or electrical burns may have elevated plasma myoglobin levels</td>
<td>Decreased albumin level within the first 8 hr after burn injury may be normal</td>
<td>Myoglobinemia may result from prolonged underresuscitation and tissue ischemia</td>
<td>Myoglobinemia may result if excessive resuscitation leads to compartment syndrome; escharotomy should be performed to minimize rhabdomyolysis</td>
</tr>
<tr>
<td>Prothrombin time, partial thromboplastin time, and platelet count</td>
<td>Initial values are useful to determine whether the patient has preexisting hepatic or hematologic disease</td>
<td>Normal</td>
<td>Prolonged shock and underresuscitation may lead to disseminated intravascular coagulation; coagulation factors and platelets may be needed in such cases</td>
<td>Unrecognized compartment syndrome and delayed escharotomy may cause tissue ischemia and disseminated intravascular coagulation; a dropping platelet count may indicate heparin-induced thrombocytopenia</td>
</tr>
</tbody>
</table>

**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 oropharyngeal cavity. 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 man-
ifest 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.

**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. Tracheo-bronchial 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. 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 resus-

**Table 6** Mechanisms of Pulmonary Dysfunction and Indications for Mechanical Ventilation

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Best Indicator</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inadequate alveolar ventilation</td>
<td>$P_{CO_2}$ and pH</td>
</tr>
<tr>
<td>Inadequate lung expansion</td>
<td>Tidal volume, respiratory rate, VC</td>
</tr>
<tr>
<td>Inadequate respiratory muscle strength</td>
<td>MIF; MVV; VC</td>
</tr>
<tr>
<td>Excessive work of breathing</td>
<td>$V_t$ required to keep $P_{CO_2}$ normal; $V_t/V_l$; respiratory rate</td>
</tr>
<tr>
<td>Unstable ventilatory drive</td>
<td>Breathing pattern, clinical setting</td>
</tr>
<tr>
<td>Severe hypoxemia</td>
<td>$P(A-a)O_2$; $P_{CO_2}/P_{O_2}$; $P_{CO_2}/F_{CO_2}$; $Qs/Qt$; minute ventilation</td>
</tr>
</tbody>
</table>

MIF—maximum inspiratory force  
MVV—maximum voluntary ventilation  
$P(A-a)O_2$—alveolar-to-arterial PO$_2$ gradient  
$P_{CO_2}/F_{CO_2}$—ratio of arterial $P_{CO_2}$ to inspired $O_2$  
$Qs/Qt$—intrapulmonary right-to-left shunt fraction  
VC—vital capacity  
$V_t/V_l$—dead space fraction  
$V_t$—minute ventilation

citation failures in burn victims with concomitant inhalation injury.

Inhalation injury can often be a clinical diagnosis. Lower airway injury can be confirmed by bronchoscopy or xenon-133 ventilation-perfusion scan, but these modalities do not change therapeutic choices or clinical outcome.

**ACUTE LUNG INJURY AND ACUTE RESPIRATORY DISTRESS SYNDROME**

Understanding of the pathophysiology of ARDS has improved since its initial description in the late 1960s, 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. ARDS is an independent risk factor for death in burn patients. Mortality in burn patients with ARDS is attributable to overwhelming sepsis and

**Table 7** Clinical Manifestations of Carbon Monoxide Poisoning

<table>
<thead>
<tr>
<th>Carboxyhemoglobin Level (%)</th>
<th>Clinical Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 10</td>
<td>None</td>
</tr>
<tr>
<td>15–25</td>
<td>Nausea, headache</td>
</tr>
<tr>
<td>30–40</td>
<td>Confusion, stupor, weakness</td>
</tr>
<tr>
<td>40–60</td>
<td>Coma</td>
</tr>
<tr>
<td>&gt; 60</td>
<td>Death</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Carboxyhemoglobin Half-life</th>
<th>Treatment Modality</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 hr</td>
<td>Room air</td>
</tr>
<tr>
<td>45–60 min</td>
<td>100% oxygen</td>
</tr>
<tr>
<td>20 min</td>
<td>100% oxygen at 2 atm (hyperbaric oxygen)</td>
</tr>
</tbody>
</table>
TABLE 9  Clinical Findings Associated with Specific Inhaled Products of Combustion

<table>
<thead>
<tr>
<th>Source</th>
<th>Product of Combustion</th>
<th>Clinical Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Organic matter</td>
<td>Carbon monoxide, carbon dioxide</td>
<td>Poor tissue oxygen delivery, narcosis</td>
</tr>
<tr>
<td>Wood, paper, anhydrous ammonia</td>
<td>Nitrogen oxides (NO, NO₂)</td>
<td>Airway mucosal irritation, pulmonary edema, dizziness</td>
</tr>
<tr>
<td>Polyvinyl chloride (plastics)</td>
<td>Hydrogen chloride</td>
<td>Airway mucosal irritation</td>
</tr>
<tr>
<td>Wool, silk, polyurethane (nylon)</td>
<td>Hydrogen cyanide</td>
<td>Respiratory failure, headache, coma</td>
</tr>
<tr>
<td>Petroleum products (gasoline, kerosene, propane, plastics)</td>
<td>Carbon monoxide, nitrogen oxide, benzene</td>
<td>Airway mucosal irritation, coma</td>
</tr>
<tr>
<td>Wood, cotton, paper</td>
<td>Aldehydes</td>
<td>Airway mucosal irritation, lung parenchyma damage</td>
</tr>
<tr>
<td>Polyurethane (nylon)</td>
<td>Ammonia</td>
<td>Airway mucosal irritation</td>
</tr>
</tbody>
</table>

multiple organ failure rather than to respiratory failure alone.²⁵

Clinically, ARDS is characterized by pulmonary edema, refractory hypoxemia, diffuse pulmonary infiltrates, and 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.²⁴ Advanced age is also an important risk factor for the development of ARDS—indeed, one small retrospective study has suggested that age is the only independent major predisposing factor for ARDS.²⁶ Curiously, acute lung injury rarely develops in patients with inhalation injury but without cutaneous burns.²⁷,²⁴

Inflammatory Mediators in ARDS with Burn Injuries

Local and systemic inflammatory mediators released in response to burn injury include platelet-activating factor, interleukins (IL-1, IL-2, IL-6, and IL-8), prostaglandin, thromboxane, leukotrienes, hematopoietic growth factors (granulocyte-macrophage colony-stimulating factor, macrophage colony-stimulating factor, and granulocyte colony-stimulating factor), cell adhesion molecules (intercellular cell adhesion molecule–1, endothelial-leukocyte adhesion molecule–1, and vascular cell adhesion molecule–1), and nitric oxide (NO).²⁹,³⁰ Systemic levels of circulating tumor necrosis factor–α (TNF–α) and IL-1 correlate with ARDS severity. IL-2 promotes multisystem organ edema, lung neutrophil sequestration, and platelet activation through alterations in microvascular permeability. 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.³¹ 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, a phagocytic cell that 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.

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. 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.³² Pharmacologic approaches to treating ARDS in burn patients parallel those used in other critically ill surgical patients and are addressed elsewhere [see 8:5 Pulmonary Insufficiency].

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 exacerbates underlying lung injury and perpetuates systemic inflammation. These effects can be minimized by maintaining low tidal volumes and peak pressures and by applying positive end-expiratory pressure. 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 utilizes low inspiratory volumes (4 to 6 ml/kg) to keep peak inspi-
ventilated with conventional means. The volume-preset, assisted with low tidal volumes had a 22% lower mortality than patients tolerated. Capnia may be reduced by using hyperventilation, although respiratory acidosis with a pH as low as 7.20 is permitted. The early resuscitative phase may be the optimal time to initiate ventilation. In patients with large burns and inhalation injury, it may be useful or the tidal volume can be titrated to 7 to 8 ml/kg, provided that peak inspiratory pressures are below 40 cm H2O. Pressure support levels between 5 and 20 cm H2O can be titrated to keep the respiratory rate below 35 breaths/min and may be useful for weaning.

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, 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.

**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 (P/F) early in the course of ARDS. Some data suggest that prone positioning in conjunction with NO administration may improve arterial oxygenation. 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. 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. Early implementation of permissive hypercapnia 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. Improved oxygenation and pulmonary toilet has been reported in patients treated early with HFPV, 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.

**Nitric oxide 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. Clinically, inhaled NO may be useful in burn patients with severe acute lung injury in whom conventional ventilatory support is failing. The safety of inhaled NO in these patients is indicated by low methemoglobin levels and absence of hypotension attributable to the NO. Strong, immediate, and sustained improvement in the P/F ratio and reduction in pulmonary arterial mean pressure in response to NO seem to correlate with survival. However, the use of inhaled NO has been reported in only small numbers of burn patients, and a prospective study is warranted.

**Corticosteroids** The use of corticosteroids in the treatment of burns is problematic because of the negative effect these agents have on wound healing. Nevertheless, there is some evidence that rescue treatment with corticosteroids in the late chronic fibroproliferative phase of ARDS may decrease mortality and lower the P/F ratio in patients with smoke inhalation injury. The relative risks and benefits of corticosteroids 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, 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.

**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. 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, 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, trachea–innominate artery fistulas, tracheoesophageal fistulas, and posttracheostomy dysphagia. 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. 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. Nevertheless, tracheostomy has been reported to provide no benefit for early extubation or overall outcome for burn patients. One 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. Percutaneous dilatational tracheostomy may provide a reasonable, less invasive approach for patients who are likely to need prolonged ventilatory support. 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 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 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. 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. At least one study has demonstrated that in pediatric burn patients, physical examination is the most reliable tool for evaluating the source of fever.

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 modern medical literature of tetanus in patients who had received immunization during childhood.

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 may well be contraindicated in patients with burns. 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. Two mainstays of both prevention and treatment is daily washing with soap and water 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.

Nutrition

In the early 1970s, Curreri and others 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. 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 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. 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.

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. 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 1]. Probably the most widely used formula is the Harris-Benedict equation, which estimates basal energy expenditure according to gender, age, height, and weight. 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, 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.

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. 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.

The so-called metabolic cart is a portable gas analyzer that quantifies volumes of inspired O2 and expired CO2. This result can also be indirectly measured in patients with pulmonary artery catheters in place by using the Fick equation:

\[
kcal/day = \frac{[(3.9 \times Vo_2) + (1.1 \times VCO_2)]}{6.96}
\]

This result can also be indirectly measured in patients with pulmonary artery catheters in place by using the Fick equation:

\[
kcal/day = \text{cardiac output} \times (\text{arterial Po}_2 - \text{venous Po}_2) \times 10 \times 6.96
\]

ENTERAL NUTRITION

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

Anemia

Because acute blood loss is uncommon in a patient with an isolated burn injury, a rapidly decreasing hematocrit during resusc-
tation 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 [see 7:15 Management of the Burn Wound], operative blood loss has decreased, as has the need for transfusion.86,87 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. For an otherwise healthy patient who does not need surgery, a hematocrit as low as 20% may be tolerated. However, patients with inhalation injury or ARDS may benefit from the greater oxygen-carrying capacity afforded by a higher hematocrit. 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.88 At least one prospective study suggests that administration of recombinant erythropoietin in acutely burned patients does not prevent anemia or decrease transfusion requirements.89

Pain Management

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

Background pain is the discomfort that burn patients experience throughout the 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 anti-inflammatory 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 are 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 citrate80 work well for wound care; I.V. 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.

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.91,92

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.93 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 intensive 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, or cetirizine may relieve itching. There are also promising data on the use of doxepin ointment as a topical treatment for itching of healed wounds.94 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,95 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.96

At the University of Washington 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. On the basis of these data, patients with burns larger than 20% TBSA receive prophylaxis with subcutaneous unfractionated heparin, 5,000 U twice a day.
84. Demling RH, DeSanti L: Oxandrolone induced lean mass gain during recovery from severe burns is maintained after discontinuation of the anabolic steroid. Burns 29:793, 2003