Objectives  
After completing this article, readers should be able to:

1. Determine the extent and depth of a burn injury.
2. Delineate how to evaluate a child who has an acute burn injury.
3. Know when to suspect abuse in a burned child.
4. Describe the resuscitation of a burned child.
5. Recognize the metabolic and immune response to a burn injury.
6. Discuss how to evaluate and manage an inhalation injury.

Definitions
It is very important for the clinician caring for a burn victim to know the depth, or degree, of burn. A first-degree burn is superficial, dry, painful to touch, and heals in less than 1 week. A second-degree burn is partial thickness and pink or possibly mottled red. It exhibits bullae or frank weeping on the surface. It usually is painful unless classified as deep and heals in 1 to 3 weeks. Second-degree burns commonly are caused by scald injuries and result from brief exposure to the heat source. A third-degree burn is the most serious. It appears pearly white, charred, hard, or parchmentlike. The dead skin (eschar) is white, tan, brown, black, and occasionally red. Superficial vascular thrombosis can be observed; there also can be focal tissue loss with prolonged exposure and a soapy-looking lesion that is found in alkali burns.

A first-degree burn is exemplified by prolonged exposure to sunlight. Second- and third-degree burns can result from contact with hot fluids (scald) or hot objects, such as an iron (contact burn), flames (flame burn), high-voltage electricity (electrical burn); various chemical agents, including acid and alkali (chemical burn); or very cold objects or environments (frostbite).

The physician also must determine the extent of injury. The determination of total body surface area (TBSA) involved in a burn injury is shown in the Figure.

Epidemiology
Two thirds of burn injuries in children younger than 4 years of age are scald burns, one fifth are from contact with hot substances, and the remainder are flame burns. For children between the ages of 6 months and 2 years, spillage of hot liquids, such as coffee and other hot foods, is the most common scenario. Hot water burns occur primarily in the bathroom and generally are more extensive than hot food burns. Most microwave-associated burns in children are due to exposure to the hot foods within the appliance.

A subset of burns in young children is due to child abuse (10% to 30% overall) by parents, siblings, or caretakers. Affected children may exhibit other signs of abuse or neglect, such as poor body hygiene, fractures, bruises, or malnutrition. Cigarette burns constitute the greatest number of thermal injuries in the abused population, usually are limited in extent, and may not necessitate hospitalization for care, but clearly require confinement of the child if abuse is suspected. Scald burns that exhibit a straight-line distribution suggest immersion; such appearance on the feet, posterior legs, buttocks, and hands suggest abuse. These burns often are associated with trauma, such as fractures. Cases of placing small children inside microwave ovens also have been reported. In these cases,
the injuries are full-thickness, and most are demarcated close to the source of the microwave. In cases of contact burn with a hot iron, burns on the dorsal surface of the hand often indicate abuse; those on the palm usually suggest neglect.

Contact with hot items in the home or fires are the chief causes of burn injury among children and young adolescents. Such burns are associated with contact with curlers and curling irons, room heaters, ovens, ranges, gasoline, and fireworks. Electrical burn injuries are likely to result from contact with household electrical cords or current in wall outlets. Males are at greater risk of burn injury and death than are females; children younger than 4 years of age, especially those who have disabilities, are at the greatest risk.

Pathogenesis
Once sustained, the burn injury, especially full-thickness burns that occupy 40% or more TBSA, give rise to many complications. The most immediate are burn shock and burn edema, as well as inhalation injury, if sustained. Within a few days of the burn injury, other responses are detected, including hypermetabolism, systemic inflammatory response syndrome (SIRS), and sepsis.

Acute Changes: Burn Shock and Burn Edema
Burn injury results in loss of fluid from the intravascular space and excessive fluid accumulation in the interstitial space, resulting in hypovolemia and swelling of the burned skin. When burns exceed 25% TBSA, noninjured tissues also swell. The cause of the fluid shift is believed to be the presence of various mediators stimulated by the burn injury (Table 1). These mediators also impair cardiac contractility and increase vascular resistance, creating a scenario for hypovolemia, hypoperfusion, tissue ischemia, renal failure, cardiovascular collapse, and death, if aggressive resuscitation therapy is not initiated early.

Electrical burns cause additional complications. Cardiac arrhythmia, including ventricular fibrillation, can occur at the time and site of the injury, as can myocardial damage. Tissues that are most resistant to electric current, such as bone, sustain the greatest heat injury, and soft tissue next to the bone frequently is damaged. Myoglobinuria, renal failure, and neurologic damage, including Guillain-Barré syndrome, transverse myelitis, amyotrophic lateral sclerosis, paresis, and paralysis, can develop up to 2 years following an electrical burn. Eye injuries complicate 5% to 20% of electrical burns; cataracts are the most common complications.

Hypermetabolism
Children who have large burns experience an increase in energy expenditure and protein metabolism of approximately 50% after the first few days following injury. This results in negative nitrogen balance and depletion of body protein stores, which can last for 9 months following the burn. This state results in weight loss of up to 20%, impaired growth velocity, muscle wasting, impaired immunity, delayed wound healing, and markedly reduced bone formation, with acute skeletal bone loss and increased risk of fracture. Epinephrine, cortisol, and glucagon are produced in excess and mediate the increased gluconeogenesis, glycogenolysis, muscle breakdown, and bone loss. They also antagonize the anabolic effects of insulin and growth hormone. The body is less able to use fat for fuel, and muscle becomes an energy source.

SIRS
SIRS occurs as a result of burn injury, and symptoms range from tachypnea and tachycardia, leukocytosis, and
fever to hypotension, shock, and multisystem organ failure. The hyperactive immune response causes a generalized inflammation that damages healthy tissue as well as infected burn wounds. Microvascular permeability leads to decreased tissue oxygenation, and blood flow is reduced due to microthrombi. During this reaction, the intestinal and, possibly, the respiratory barriers to infection are damaged, allowing the entry of additional bacteria into the circulation, known as bacterial translocation.

Coexisting with SIRS is another reaction termed the counter anti-inflammatory response syndrome (CARS), which attempts to limit the damage created by inflammatory cells. However, the CARS reaction also promotes immunosuppression. These reactions are generated by chemokines, which are located in the macrophages and endothelial cells, and attract inflammatory cells to the site of damage. The inflammatory cells produce cytokines, which also stimulate the inflammatory response and tissue destruction. Differentiating SIRS and CARS from sepsis, which is the leading cause of death in burns, is extremely challenging.

Inhalation Injury
Inhalation of the toxins associated with flame smoke accounts for 80% of burn-related deaths. These include carbon monoxide and hydrogen cyanide, which can potentiate tissue hypoxia and acidosis. It is important to know both the source of the fire and the nature of the gases it produces to treat the injury adequately. In addition, the inhaled hot gases cause oropharyngeal edema in the same way that they affect other tissues, as discussed previously. Also, inhalation of various chemicals, such as aldehydes, can cause contact damage to the trachea and bronchi, resulting in inflammation and formation of fibrin casts that can obstruct the lower airway completely and lead to decreased oxygenation due to pulmonary vascular vasoconstriction.

### Diagnosis and Management of the Burn Wound
The appearance of the skin should be sufficient to classify the depth and extent of the burn. It is important to obtain a complete history of the burn injury because certain sources, such as alkali, may require additional treatment to stop the penetration of skin layers.

The superficial burn wound that extends to less than 10% TBSA usually can be treated on an outpatient basis unless abuse is suspected. Therapy consists of applying a cotton gauze occlusive dressing to protect the damaged skin from bacterial contamination, eliminate air movement over the wound (thus reducing pain), and decrease water loss. Dressings are changed daily. A topical antimicrobial agent should be applied to the wound prior to the dressing for prophylaxis. The agent used most commonly is silver sulfadiazine, which has activity against *Staphylococcus aureus*, *Escherichia coli*, *Klebsiella* sp, *Pseudomonas aeruginosa*, *Proteus* sp, and *Candida albicans*. The primary adverse effect of silver sulfadiazine is leukopenia, which occurs in 5% to 15% of treated patients. However, there is no increase in the incidence of infection. Daily clinical inspection and wound culture, if necessary,

### Table 1. Mediators of Burn Shock and Edema

<table>
<thead>
<tr>
<th>Mediator</th>
<th>Source</th>
<th>Effect</th>
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<tbody>
<tr>
<td>Histamine</td>
<td>Mast cells from burned skin</td>
<td>Increases capillary permeability, arteriolar dilatation, and venular contraction</td>
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<tr>
<td>Prostaglandins</td>
<td>Arachidonic acid released from burned tissue and inflammatory cells</td>
<td>PGE2, PGI2: potent vasodilators; increase microvascular permeability</td>
</tr>
<tr>
<td>Thromboxanes</td>
<td>Platelets in the burn wound</td>
<td>Thromboxanes A2 and B2: vasoconstrictors; contribute to tissue ischemia</td>
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<tr>
<td>Kinins</td>
<td>Inflammatory cells</td>
<td>Increase venular permeability</td>
</tr>
<tr>
<td>Serotonin</td>
<td>Inflammatory cells</td>
<td>Vasoconstrictor; reduces blood flow to burn wounds</td>
</tr>
<tr>
<td>Catecholamines</td>
<td>Adrenal medulla</td>
<td>Vasoconstrictor; contributes to wound ischemia, increased systemic vascular resistance</td>
</tr>
<tr>
<td>Oxygen radicals</td>
<td>Burned tissue</td>
<td>Increase vascular permeability and burn edema</td>
</tr>
<tr>
<td>Platelet aggregation factor</td>
<td>Burn wound platelets</td>
<td>Increases capillary permeability</td>
</tr>
<tr>
<td>Angiotensin II and Vasopressin</td>
<td>Renal juxtaglomerular cells</td>
<td>Vasoconstrictors; may be responsible for intestinal ischemia (angiotensin) and increased systemic vascular resistance (vasopressin)</td>
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</table>
should determine when the wound is healed, but such wounds generally heal in fewer than 2 weeks. Also, the application of Biobrane® by experienced practitioners can be of value in partial-thickness burns. The product consists of a coat of cellophane topped by a protein that adheres to the wound and protects it while healing.

**Initial Treatment of a Child Who Has Extensive Burns**

Treatment of a child who has major burn injury consists of fluid resuscitation to prevent shock, early excision and grafting of the burn wound coupled with early nutrition support, identification of airway involvement due to inhalation injury, and measures to treat sepsis. Additionally, with knowledge of the nature of the burn and any potential chemical exposure or electrical injury, more specific measures can be taken to diagnose acute complications, such as cardiac arrhythmias, or to use antidotes for toxic inhalants.1

**Initial Management**

The victim must be removed from the scene, the burning extinguished, any jewelry removed (sources of heat), and in the case of a chemical burn, any saturated clothing removed and skin thoroughly irrigated with water, taking care not to spread any chemical to unburned skin. In addition, the patient’s chest should be exposed to assess respiratory effort, and pulse should be obtained. Any accompanying injuries, such as fractures, also should be assessed. Intravenous access should be obtained as quickly as possible. Determination of past medical history, allergies, and current medications at the scene is advisable if time permits.

Once the nature and extent of injury are assessed, fluid resuscitation is begun. Fluid administration, usually through at least two large-bore intravenous catheters, begins with Ringer’s lactate 5,000 mL/m² burned surface area per day, with an additional 2,000 mL/m² TBSA per day of Ringer’s lactate and 5% dextrose for maintenance. The first half of the fluid load is infused over the first 8 hours postburn; the remainder is infused over the ensuing 16 hours. The infusion rates should be adjusted to maintain a urine flow of 1 mL/kg per hour. During the second 24 hours, fluid administration is reduced to 3,750 mL/m² surface area burn, with 1,500 mL/m² TBSA per day as maintenance, which almost always can be administered in the form of enteral feeding through a feeding tube. This is known as the Galveston formula.

**Early Excision and Grafting**

Early excision and grafting of the burn wound should be performed at a burn center. The rationale for rapid closure of all unequivocal deep burns is to reduce wound infection, fluid loss, and concomitant complications. Most burn surgeons close deep wounds within 1 to 2 weeks of the injury.

**Early and Aggressive Nutrition Support**

With early and aggressive nutrition support, burn centers have been able to reduce the resting energy expenditure from 1.6 to 2 times normal to less than 1.5 times normal. Because other processes beyond hypermetabolism, such as fluid loss, sepsis, and inflammation, contribute to energy expenditure, intensive nutrition support can help minimize protein catabolism and weight loss. A high-carbohydrate diet is needed because of the relative inabil-

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**The superficial burn wound that extends to less than 10% TBSA usually can be treated on an outpatient basis unless abuse is suspected.**
Recognition of Airway Involvement

A history of closed-space exposure to smoke should raise suspicion of inhalation injury. Physical signs that are most suggestive of airway injury are hoarseness and stridor, which indicate partial obstruction of the airway and a risk for total obstruction. Patients who exhibit these signs require immediate endotracheal intubation. Patients who are obtunded or comatose may have been exposed to carbon monoxide, which is diagnosed by measurement of blood carboxyhemoglobin concentrations. However, the correlation between inhalation injury and symptoms and signs often is poor, which necessitates bronchoscopic examination during evaluation at the burn center. Intravenous radionuclide scans using xenon $^{133}$ can identify small airways that are affected by inhalation injury. Areas retaining xenon for more than 90 seconds are assumed to be obstructed. Treatment of inhalation injury is supportive. With combined burn and inhalation injury, fluid requirements are increased by 40% to 75% in the first 24 hours postinjury. For carbon monoxide exposure, patients should receive 100% oxygen until the blood carboxyhemoglobin levels are less than 10%.

Patients who have hypoxia require supplemental oxygen. Chest physiotherapy and spirometry generally are used to treat airway secretions. Wheezing is an indication for diagnostic bronchoscopy to determine whether there is plugging of the airway or bronchospasm and edema. If there is plugging, therapeutic bronchoscopy and aerosolized heparin and humidification are administered. For bronchospasm, treatment initially is with nebulized beta$_2$ agonists and, if needed, intravenous aminophylline. If respiratory failure ensues, the treatments of choice are intubation, mechanical ventilation, and tracheostomy. If ventilation remains inadequate, permissive hypercapnia may stimulate respiratory effort.

Sepsis

Burn wound sepsis is defined as the site showing proliferating microorganisms that exceed $10^5$/g tissue and invasion of underlying unburned tissue. Bacteremia is indicated by the transient presence of microorganisms in the blood; sepsis is defined as invasion of the blood by pathogenic bacteria from local foci of infection, such as the burn wound. Sepsis frequently is accompanied by hyperthermia, hypothermia, and prostration.

Table 2 lists the symptoms and signs associated with sepsis with gram-positive and gram-negative organisms. Some of the more common infections seen with severe burn injury include pneumonia, subacute bacterial endocarditis, catheter infections, thrombophlebitis, suppura-

<table>
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<th>Features of Bacterial Sepsis Following Large Burns</th>
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<tbody>
<tr>
<td><strong>Gram-negative</strong></td>
</tr>
<tr>
<td>Temperature usually $10^4^\circ F (40^\circ C)$ or higher</td>
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<tr>
<td>Gradual onset</td>
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<tr>
<td>White blood cell count usually $&gt;20 \times 10^7$/ml; blood cultures may be negative</td>
</tr>
<tr>
<td>Wounds usually $&gt;20 \times 10^9$/L; blood cultures may be negative</td>
</tr>
<tr>
<td>Burn wound biopsy with $&gt;10^5$ organisms per gram of tissue</td>
</tr>
<tr>
<td>Ileus, decreased blood pressure and urine output, anorexia, irritability</td>
</tr>
<tr>
<td><strong>Gram-positive</strong></td>
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<tr>
<td>Temperature increased or normal, followed by hypothermia</td>
</tr>
<tr>
<td>White blood cell count either low or high; blood cultures may be negative</td>
</tr>
<tr>
<td>Wounds develop focal gangrene</td>
</tr>
<tr>
<td>Burn wound biopsy with $&gt;10^5$ organisms per gram of tissue</td>
</tr>
<tr>
<td>Ileus, decreased blood pressure and urine output, obtundation</td>
</tr>
<tr>
<td><strong>Features of Bacterial Sepsis Following Small Burns</strong></td>
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<tr>
<td><strong>Gram-positive</strong></td>
</tr>
<tr>
<td>Temperature increased or normal, followed by hypothermia</td>
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[Table 2: Features of Bacterial Sepsis Following Large Burns]

*Pediatrics in Review Vol.25 No.12 December 2004 415*
tive chondritis and sinusitis, urinary tract infections, cholecystitis, and intestinal infections. Antibiotic therapy should be based on sensitivity of the organism(s) involved.

Prognosis
Despite the seriousness of large burn injury in children, the prognosis for survival is good and improving steadily. Recent data analyzed at the United States Army Institute for Surgical Research showed that adults older than 50 years of age who had burns of less than 50% TBSA accounted for 19% of the admissions to burn units, but greater than 50% of the deaths. In contrast, children younger than 4 years of age who were similarly burned accounted for 19% of admissions but only 12.5% of the deaths. Other challenges remain, including physical and emotional rehabilitation, reconstructive surgery, treatment of the hypermetabolic state with anabolic agents such as growth hormone, and reintegration of children into their communities. Work is ongoing in these and other areas.

Suggested Reading

PIR Quiz
Quiz also available online at www.pedsinreview.org.

1. A 9-month-old boy is brought to the emergency department for burns involving his feet and lower legs. According to the father, the boy sustained his injuries when he was being bathed in the bathtub and the father accidentally turned on the hot water. Which of the following is most suggestive of nonaccidental (child abuse) injury?
   A. Blister formation.
   B. Myoglobinuria.
   C. Pearly white surface.
   D. Splash burns.
   E. Straight-line circumferential burns.

2. You are examining a 1-year-old girl during a health supervision visit. As a part of anticipatory guidance, you discuss burn injuries in children. Which of the following is the most common source of accidental burn injuries at this age?
   A. Chemical burns
   B. Contact with hot surfaces.
   C. Electrical burns.
   D. Exposure to microwave.
   E. Spillage of hot liquids.

3. A 4-year-old girl is brought to the emergency department after being rescued from a house fire. Physical examination shows: rectal temperature 96.8°F (36°C), heart rate of 100 beats/min, respiratory rate of 24 breaths/min, and blood pressure of 110/70 mm Hg. She has medium-pitched inspiratory stridor and suprasternal retractions. Air entry is equal on both sides. She is obtunded and responds to painful stimuli by appropriate withdrawal. Her face is covered with soot. No skin burns are noted. Supplemental oxygen is provided by a face mask. Pulse oximetry shows an oxygen saturation of 100%. Intravenous access is obtained. Which of the following is the most appropriate next step?
   A. Administration of 20 mL/kg Ringer’s lactate.
   B. Computed tomography of head, neck, and chest.
   C. Endotracheal intubation.
   D. Fiberoptic bronchoscopy.
   E. Radionuclide xenon scan.
4. An 8-year-old boy is brought to the emergency department after suffering burn injuries while lighting a firecracker at a backyard barbecue party. Physical examination shows third-degree burns on 60% of his body surface involving the trunk and lower extremities. His rectal temperature is 96.8°F (36°C), heart rate is 180 beats/min, respiratory rate is 48 breaths/min, and blood pressure is 90/60 mm Hg. Which of the following is the most accurate statement regarding his metabolic state over the next several weeks?

A. Decreased cortisol level.
B. Decreased energy requirements.
C. Increased epinephrine and glucagon levels.
D. Increased nitrogen retention.
E. Increased utilization of fat as the energy source.

Note: In the October 2004 issue of *Pediatrics in Review*, on page 357, question 12 repeated the value PCO₂, 50 mm Hg; one of the values should have been PO₂, 50 mm Hg.