Trauma Reports

PRACTICAL, EVIDENCE-BASED REVIEWS IN TRAUMA CARE

Volume 14, Number 4 **July/Aug 2013**

Authors:

Creagh Boulger, MD, Assistant Professor of Emergency Medicine, The Wexner Medical Center at The Ohio State University, Columbus, OH.

Andrew Retzinger, MD, Clinical Instructor, Department of Emergency Medicine, The Wexner Medical Center at The Ohio State University, Columbus, OH.

Howard Werman, MD, Professor of Emergency Medicine, The Wexner Medical Center at The Ohio State University, Columbus, OH.

Peer Reviewer:

John Cheng, MD, Assistant Professor, Department of Pediatrics, Division of Emergency Medicine, Emory University School of Medicine, Children's Healthcare of Atlanta, Atlanta, GA.

Extra Hour of CME Credit available in this issue!

Take our 2013 Reader Survey! See details on page 15.

Statement of Financial Disclosure

To reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Dr. Dietrich (editor in chief), Dr. Boulger (author), Dr. Retzinger (author), Dr. Werman (author), Dr. Cheng (peer reviewer), and Ms. Behrens (nurse reviewer) report no relationships with companies related to this field of study. Ms. Mark (executive editor), and Ms. Hamlin (managing editor) report no relationships with companies related to the field of study covered by this CME activity.

AHC Media

Pediatric Burns: Current Standard for Assessment and Management

Pediatric burns, with all of their challenging aspects, are a common injury faced by emergency medicine physicians. Burn injuries are painful for the patient, distressing to the parent, and often raise some difficult questions for the physician in regard to recognition and assessment of non-accidental trauma and the clinical dilemma of disposition. The authors review the current standard for recognition, evaluation, and management of pediatric burn injuries.

— Ann M. Dietrich, MD, Editor

Introduction

The purpose of this article is to review the epidemiology, pathophysiology, evaluation, differential diagnosis, management, and complications associated with pediatric burns. The article identifies risk factors for pediatric patients affected by burns, the mechanism of burn injury, and the morbidity and mortality associated with pediatric burns. Additionally, this article discusses basic physiology and definitions of the various subclasses of burns, as well as the unique pathophysiology of the pediatric population. A systemized approach to the initial evaluation of the pediatric burn patient is presented, with a discussion of other less common but potentially life-threatening diseases that present in a manner similar to burn injuries. The authors address the key laboratory studies necessary when managing the pediatric burn patient. In addition, the article discusses the various components of pediatric burn treatment, from the initial stabilization and basic management to considerations of fluid administration, use of blood products, antibiotics, nutrition and, ultimately, of surgical care. The authors address potential complications of burn injuries and provide a brief discussion on their management. Finally, the article discusses disposition of the pediatric burn patient, including criteria for admission and transfer.

Epidemiology

Burns are a significant cause of morbidity and mortality throughout the world. Children comprise 29% of all burn victims.[1,2] Burns are a leading cause of accidental death in the pediatric population; they are the third leading cause of accidental death after motor vehicle deaths and drowning.[1] Burn injuries account for nearly 120,000 ED visits for patients younger than the age of 20.[1,3] Although potentially life-threatening, the majority of pediatric burns are minor and do not require admission.[4] One large study cited an overall mortality of less than 3% for burn injuries.[5] However, mortality rates are higher for children younger than 4 years of age.[6]

Burns are defined as caused by a variety of sources, including heat, electricity, chemicals, radiation, and friction. This article will focus primarily on assessment and management of thermal burns.

Children typically develop their motor skills before abstract thinking and reasoning. Thus, as they become more mobile and explore their environment, they often put themselves in danger from many sources that can lead to burns.

Executive Summary

- Young children require as little as 15% total body surface area (TBSA) to trigger a systemic response. As they age, the percentage needed to trigger an inflammatory response increases.
- Children involved in closed-space fires are more likely to sustain inhalation injury as the result of their relative immobility and impaired ability to escape.
- First-degree burns are typically moist, red, painful, and without blistering and are not included in BSA calculations.
- The practitioner must maintain a suspicion for nonaccidental trauma (NAT) when the history and physical exam do not correlate with findings, when there is a delay in seeking care, when there is concern for neglect or lack of supervision, or when specific burn patterns are present.

Common mechanisms for burn injuries in children often include biting electric cords, exposure to hot bath water, and spilling of hot liquids. Among pediatric burn injuries, scalds are the most common form of burns worldwide.[4,7,8] These frequently occur in the home, primarily in the kitchen and bathroom, and most commonly affect children younger than the age of 5 years. In addition to age, crowded homes, unsupervised play, low socioeconomic status, younger unmarried mothers, and lack of maternal education all are associated with increased risk for scald injuries.[9] Among children older than the age of 5 years, flame and fire-related injuries become more prominent. In the pediatric population, as with adults, males tend to be more commonly affected than females.[1]

Table 1. Temperature/Time Exposure Relationships

Temperature Moritz (°F/°C)	Time to achieve third-degree burn (seconds)
155/68	1
140/60	5
127/52	60
124/51	180
120/48	300
100/37	Safe for bathing

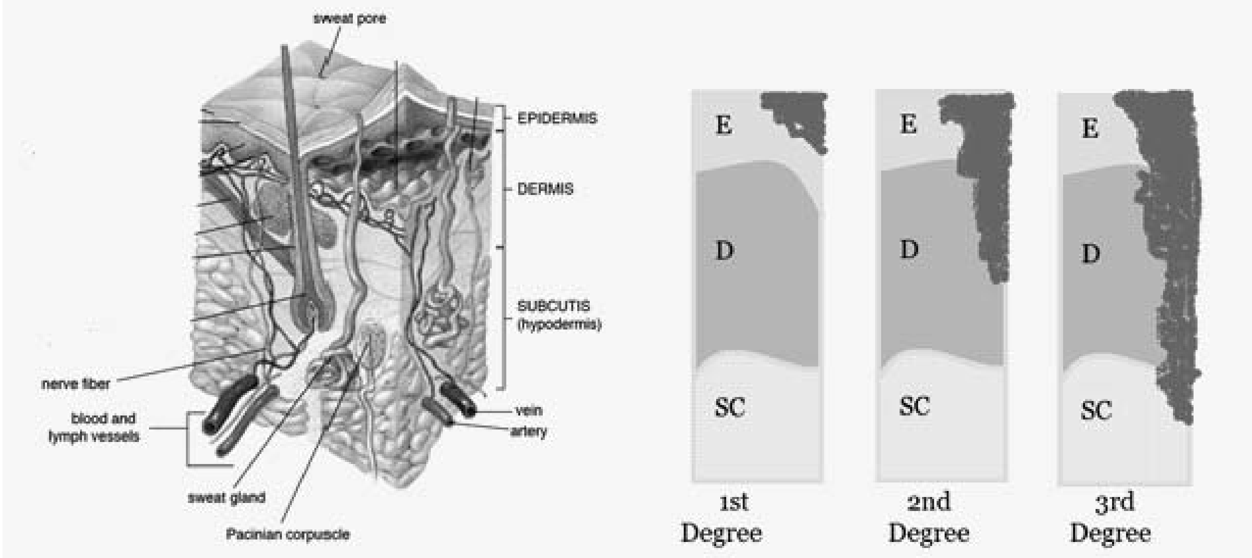
Pathophysiology

A common truth in medicine is that "children are not little adults." This principle applies to pediatric burn injuries as well. Children have thinner skin and less developed mechanisms of thermoregulation than adults; this is especially true for infants. As a result, children tend to get deeper burns, lose heat more rapidly, have more insensible fluid loss, and require less exposure time to produce significant damage. In addition, children have higher overall fluid requirements than their adult counterparts. They also have more volatile immune systems that allow them to mount a greater systemic inflammatory response to burn injuries. In children, even small burns can trigger this diffuse immune response.

Young children require as little as 15% total body surface area (TBSA) to trigger a systemic response. As they age, the percentage needed to trigger an inflammatory response increases.[10] This response usually occurs in the latter portion of the first 24 hours and can manifest as shock or systemic inflammatory response syndrome (SIRS). Components of the inflammatory response include tissue release of cytokines, free radicals, and prostaglandins. As a result of this release of chemical mediators and inflammatory markers, the capillary bed becomes "leaky," resulting in decreased intravascular volume and local edema. Secondly, this massive release of chemicals results in neutropenia, malfunctioning T-cells, and subsequent suppression of the immune system. The hematologic system is also affected, with red blood cells (RBCs) being destroyed by free radicals, direct thermal injury, and formation of microthrombi at the site of the burn. Several other organ systems are affected by the systemic response. The gastrointestinal system responds with increased permeability of the gut and decreased motility. The cardiovascular system is impacted by increasing catecholamine output, producing hypermetabolic state and subsequent myocardial depression.[11,12]

The systemic response to burns occurs in a relatively predictable manner. The "ebb" occurs in the first 48 hours and usually includes decreases in cardiac output (CO), decreased oxygen consumption, and hyperglycemia secondary to impaired glucose tolerance. Over the next five days or so is a state referred to as "flow." In the flow state, the previously depressed functions gradually rise to a catabolic state, then plateau throughout wound healing and recovery. This catabolic state can

Figure 1. Burn Depth



Key:
E = epidermis; D = dermis; SC = subcutis

persist long beyond the initial recovery, resulting in muscle loss, growth delay, and poor wound healing.[13,14]

The skin is the body's first line of defense. Burns occur when the skin comes into contact with hot or caustic material. Temperatures as low at 40° Celsius can cause significant injury to pediatric patients secondary to the thin nature of their skin. In addition, damage to the tissue can continue even after the source of the burn is removed. As a result of the burn injury, the tissues coagulate and have decreased blood flow, leading to hypoxia, accumulation of toxins, and further tissue damage.

The depth of the burn is determined by several factors, including the source of the burn, time exposed to the source, pressure of the source, and the temperature. Exposure to higher temperature under significant pressure over a longer period of time will likely cause a deeper, more severe burn. In 1947, Moritz established temperature/time exposure relationships.[15] These experiments were conducted in normal adults and, to this day, remain the reference standard for all burn injuries. One must take into account that given the unique feature of the pediatric integument system, these exposure times may be markedly reduced in this population. Diller et al attempted to address this in 2006 and came to the conclusion that a correction factor should be applied to children. These authors suggested a proposed correction of 3-4°F reduction in temperature from Moritz's original work.[16] In addition, Moritz's exposure times are in regard to third-degree burns, and one must consider that deep second-degree burns have comparable severity.[15] (*See Table 1.*)

The nomenclature for describing burns has changed in the last several years and will be addressed and defined here. (*See Figure 1.*)

The least invasive class of burns is first degree. This type of burn does not go deeper than the epidermis. First-degree burns, also referred to as superficial burns, are typically moist, red, painful, and without blistering. These rarely require medical treatment. Calculations of body surface area affected should not include first-degree burns. Second-degree burns can be further subdivided into partial and full thickness burns. Partial-thickness second-degree burns, also known as superficial partial-thickness burns, extend beyond the epidermis but only involve the top layers of the dermis. These burns are moist, red, and painful with blistering. A classic example of this type of injury is a blistering sunburn. These burns typically do not involve the vasculature or nerves, so they have intact sensation and will blanch. Full-thickness second-degree burns, also known as deep partial thickness burns, involve the dermis but extend into much deeper levels. They are often moist, red, hemorrhagic, blistering, and painful. Deep partial-thickness burns typically have diminished sensation secondary to nerve damage and often have delayed capillary refill secondary to vascular damage. Depending on the extent of surface area involved, deep second-degree burns may be treated surgically like third-degree burns. Second-degree burns are included in BSA estimates for the purpose of triage and fluid resuscitation. Third-degree burns, also known as full-thickness burns, extend beyond the dermis into the subcutaneous tissue. In contrast to superficial and partial-thickness burns, these burns are insensate and dry. The skin will often have a

Table 2. Lund-Browder Method

	0-1 year	1-4 years	5-9 Years	10-14 years	15-Adult
Head	19	17	13	11	9
Thigh (each)	5.5	6.5	8	8.5	9
Leg (each)	5	5	5.5	6	6.5
Torso	26	26	26	26	26
Arm (each)	4	4	4	4	4
Forearm	3	3	3	3	3
Hand	3	3	3	3	3
Buttock	5	5	5	5	5
Genitalia	1	1	1	1	1
Foot (each)	3.5	3.5	3.5	3.5	3.5
Neck	1	1	1	1	1
Numbers presented as percentage TBSA					

Table 3. Rule of Nines

	Adult	Child
Head	9	18
Torso (front and back)	18	28
Arm (each)	9	9
Leg (each)	18	14
Genitalia	1	1
Numbers presented as percentage TBSA		

black or white, waxy appearance. Treatment of these burn injuries often requires surgical debridement and grafting. The final class of burns is fourth degree. These burns extend beyond the subcutaneous tissue into the deep bone and muscle. These burns appear very similar to third-degree burns on clinical examination.

Deeper burns have predictable patterns of injury. The center of the burn is referred to as the zone of coagulation. This area sustains irreversible damage and consists of non-viable tissue. The zone of ischemia is the layer adjacent to the zone of coagulation. Tissue in this region is subject to reversible microvascular injury. This zone is the most responsive to resuscitation. If adequately resuscitated, tissue in this area may be recoverable; if not, it can become necrotic and devitalized. The outermost zone is the zone of hyperemia. This area is subject to vasodilatation in response to the local inflammatory changes. The tissue in this area does not suffer irreversible injury and is usually viable. If inadequate fluid resuscitation is given, any of these zones are prone to expand.

The depth of the burn and structures affected allows the physician to classify the burn as mild, moderate, or severe. This, unfortunately, allows for a discrepancy between physicians and doesn't account for the dynamic nature of the burn.

In addition to the burn injury, the physician must consider whether an inhalation injury is present. Inhalation injuries should be suspected if the burn occurred in a closed space, especially when there are clinical findings of stridor, hoarseness, drooling, facial burns, soot in the sputum or airway, or singed nasal hair. As the heat from closed-space fires circulates through the airway, it causes edema to the tissue. The amount of edema can be potentiated by any concomitant toxins released from the burning material. Toxins may also produce direct damage to the tissue from their chemical properties. In response to this insult, the body mounts an inflammatory response, which includes neutrophils, cytokines, and free radicals. After the acute injury, the vasculature becomes leaky, producing an acute respiratory distress syndrome-like (ARDS) picture; the patient develops hypoxia and has bilateral infiltrates on chest radiograph. This reaction, in addition to the systemic effects of the burn, makes the patient prone to develop pneumonia. In the early stage, *Staphylococcus aureus* is the most common culprit, whereas later

Pseudomonas predominates. Since children more commonly suffer from scald injuries, they have a lower incidence of inhalation injury than their adult counterparts. Despite this fact, the incidence of inhalation injury is still about 30% in pediatric burn patients.[5,17,18] However, children involved in closed-space fires are more likely to sustain an inhalation injury as the result of their relative immobility and impaired ability to escape. When they sustain inhalation injuries, pediatric patients suffer more extensive damage because of their higher minute ventilation and smaller airways. Almost 50% of deaths from burn injuries can be attributed to inhalation injury. These deaths are usually the result of carbon monoxide toxicity and hypoxia.[19]

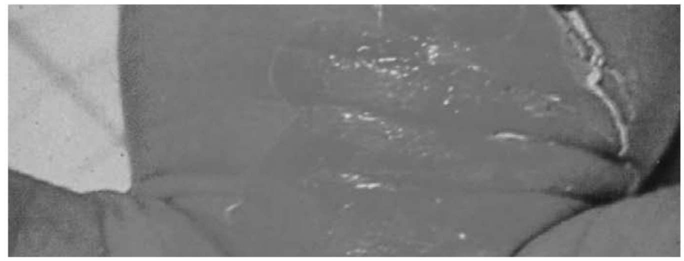
In addition to the increase in morbidity and mortality, inhalation injuries also can significantly alter management. When inhalation injury is present, the managing physician must increase the volume of fluids administered.[20] The details of evaluation and management of these injuries will be discussed in subsequent sections.

Differential Diagnosis

Typically, a history of fire or exposure to a source (chemical, electrical, thermal, or radiation) is available upon patient presentation. However, in the rare occurrence it is not available, other conditions should be considered that may appear similar to a burn. Some of these conditions are staphylococcal scalded skin syndrome (*see Figure 2*), toxic shock syndrome, Kawasaki's disease, Stevens-Johnson syndrome, and toxic epidermal necrolysis. While an extensive discussion of these diseases is beyond the scope of this paper, the authors mention them as a consideration in evaluation of the patient with a burn-like picture if history is unavailable.

Additionally, the practitioner must maintain a suspicion for nonaccidental trauma (NAT) when the history and physical exam do not correlate with findings, when there is a delay in seeking care, when there is concern for neglect or lack of supervision, or when specific burn patterns are present (these will be discussed in the next section). The incidence of abuse and neglect as a cause of pediatric burns varies from almost 2-25%. The true incidence is likely higher since the reported numbers were generated using data collected with varying methods. Standard definitions of abuse often exclude neglect.[21,22,23] If the practitioner suspects abuse, social workers and the local department of family and children's services need to be contacted, as suspicion of child abuse falls under the mandatory reporting laws in the United States. The specifics of these laws vary slightly by state. It is vital that the practitioner be aware of his or her state's policies. Specific state policies can be found online at www.childwelfare.gov.

Figure 2. Staphylococcal Scalded Skin Syndrome



Initial Evaluation

When evaluating the pediatric burn patient, a thorough history is critical. Components of the history should include time of occurrence, source of burn, duration of exposure, situation leading up to exposure, as well as methods of treatment prior to arrival. In addition, a thorough past medical history is essential, including tetanus status, co-morbidities such as respiratory disease, immune compromising diseases, diabetes, and cardiovascular problems. These historical components are essential because they portend a higher morbidity and mortality and may help guide treatment and the decision to transfer.

As with any critically ill or injured patient, it is imperative to proceed in a methodical and organized manner when assessing a pediatric burn patient. The American College of Surgeons (ACS) and American Burn Association (ABA) suggest following the primary and secondary surveys as described in the Advanced Trauma Life Support (ATLS) course.[24]

Begin with an assessment of the airway. In doing so, the practitioner must not only consider current airway compromise, but also should take note of concerning findings and anticipate a possible decline in airway patency. Emergent and immediate control of the airway with endotracheal intubation should be performed if any of the following are present: altered mental status, hypoxia, stridor, drooling, oropharyngeal edema, marked tachypnea, or apnea. Additional concerning findings include hoarse voice, oropharyngeal erythema and edema, singed facial hairs and soot in oropharynx, or carbonaceous sputum. These should prompt frequent repeated assessments of the child's airway. Methods of airway control will be discussed further in the management section.

Once the airway is assessed, the practitioner should assess the quality and quantity of respirations. One should note the presence of wheezing, rales, or stridor, as well as tachypnea and increased work of breathing, including the use of accessory muscles. Assess for burns on the chest and neck. Large burns to the chest and neck, especially those that are circumferential, may be

Figure 3. Grill Burn

Table 4. Grades of Ocular Burns

Grade	Appearance	Prognosis
I	Corneal ulceration, no limbal ischemia	Good
II	Steamy cornea with some limbal ischemia and injection of conjunctiva	Good
III	Corneal opacification, loss of iris detail, moderate amount of limbal ischemia	Guarded
IV	Cornea opaque, iris and pupil obscured, large amount of limbal ischemia	Poor*
*Improving prognosis with current day therapies		

constrictive and significantly compromise ventilation. If significant burns involving the chest are present, one may need to progress to escharotomy, which will be discussed under management.

The next area to be addressed is the circulatory system. Assessment of the circulation includes checking pulses and capillary refill, especially in extremities affected by the burn. If pulses are absent or there is a marked delay in capillary refill, one should again consider escharotomy to restore perfusion to the affected limb. Another means of assessing circulation is by measuring blood pressure. Most burn patients do not initially present with hypotension. It should be noted, however, that early in the course of a burn, hypotension is rare and should prompt good fluid resuscitation as well as an assessment for internal bleeding. SIRS and sepsis are later findings. The appropriate manner of resuscitation and fluid administration will be discussed later, but is similar to other trauma assessments. Until the airway, breathing, circulation, disability, and exposure, including removal of all clothing and a thorough inspection of the skin (i.e., primary survey), are assessed, managed, and stabilized, one should not proceed. After full exposure, one should do a thorough assessment of the burn. This will be described in more detail below with the secondary survey.

Evaluation of the Burn

The next step of assessment is an estimation of the extent of burn injury. This is important in guiding therapeutic interventions and triage decisions and often correlates with the potential morbidity and mortality. There are several validated methods for estimating the extent of burns in the pediatric population. Two commonly used methods are the Lund-Browder method and rule of nines. (*See Tables 2 and 3.*) As a result of their disproportionately large heads, children cannot accurately be assessed using the same means as adults. As a general rule, the pediatric patient's palm, including fingers, provides an estimate equivalent of about 1% TBSA. While all burns need be documented, only superficial partial thickness burns and those that are deeper second-, third-, and fourth-degree burns are included in calculations for fluid administration. In general, the initial estimation of TBSA involvement is often incorrect secondary to inexperience with burn estimation and failure to fully clean and expose the patient. Also burns can often evolve in the first 24 hours.[25]

When assessing burn injuries, the practitioner should pay special attention to the shape and extent of the burn. Specific patterns should raise concern for NAT and neglect. Some of these patterns include burns in the shape of a hot object, such as the imprint of a utensil or curling iron, as well as cigarette burns and lighter burns. (*See Figure 3.*) A stocking glove distribution of burns arises from the child being forcefully placed into hot water. This pattern should raise suspicion for submersion injury and abuse. Finally, genital burns should cause the practitioner to consider NAT, as previously mentioned.

Following full burn assessment, one should proceed with a complete secondary survey of the patient. This should include palpation of all bony surfaces as well as an assessment of pelvic stability. In addition, one should assess the abdomen for distention and tenderness and carefully examine the genitourinary region. Concomitant trauma is not uncommon in burn patients and can be missed if a thorough survey is not performed.

Special consideration should be given to the eye examination, especially in chemical and flash burns. Ocular burns can be broken down into four categories. Grades 1 and 2 are minor with small ulceration, chemosis, and conjunctival erythema. Grades 3 and 4 affect the deeper structures and appear often with corneal opacification, dark thrombosed vasculature, and iridocyclitis. (*See Table 4.*) To fully assess for ocular involvement, one should stain the eyes with fluorescein and look with a slit lamp or Wood's lamp. Increased uptake suggests corneal involvement. When ocular injury is suspected, one should begin with copious irrigation of the eye using 500-1000 mL of normal saline, clean water, or other irrigation fluid for at least 15 minutes to a goal of neutral pH of 7. If ocular involvement is discovered, one should treat with a topical ocular antibiotic and arrange close follow-up within 24 hours with an ophthalmologist. The use of ocular steroids should be discussed. More severe eye injuries (grades 3 and 4) require emergent ophthalmology consultation and often result in surgical exploration and debridement.[26]

Diagnostic Testing

After a full assessment of the burn victim is complete, the practitioner should consider further diagnostic testing. This may include chest radiographs, imaging of any injured areas, and CT scan of the head in patients with an altered mental status. Laboratory studies to consider include carboxyhemoglobin, cyanide levels, type and cross, complete blood count, chemistries, urinalysis, urine myoglobin, creatinine kinase, coagulation studies, and blood gases.

Early Management

Airway Management. Management of the burned pediatric patient proceeds in a stepwise progression, beginning with management of the airway. The physician must be aware of the dynamic nature of the airway in a burned child. Failure to consider inhalation injury and improper or delayed management of the critical airway is one of the most frequent causes of adverse events in the pediatric burn patient.[27] The presence of inhalational injury dramatically increases mortality in burn victims,[28,29] and in the acute setting, burn-specific insults can result in abrupt airway collapse. External injuries can also complicate the airway with eschar formation of the chest or circumferential burns of the neck constricting the airway and impeding ventilation mechanics.[30,31]

If there is concern for impending airway collapse, a definitive airway should be established. Conventional induction and paralyzing agents are acceptable, with the caveat that succinylcholine should be avoided in the patient who presents 48 hours after injury, as severe burns can result in delayed onset hyperkalemia.[32] (*See Table 5.*) In the pediatric patient who does not require intubation, there is still a role for sedation and analgesia, particularly in the acute phase of decontamination, cleaning, and debridement of burns. The effectiveness of ketamine or various opioids in concert with benzodiazepines has been well demonstrated.[33,34] (*See Table 5.*) There is also increasing evidence that dexmedetomidine can serve as an effective replacement for benzodiazepines.[35,36]

Approximately 80% of fire-related deaths are due not to the injury itself, but to the inhalation of toxic products[28], most notably carbon monoxide (CO) and hydrogen cyanide gases (CN). Various toxins have a profound effect on lung parenchyma, both via direct alveolar

Table 5. Analgesics, Sedatives, and Paralytics for Pediatric Burn Patients

Drug	Intravenous Dosages
Analgesics	
Morphine	0.1-0.2 mg/kg
Fentanyl	1.0-3.0 ug/kg
Ketorolac	0.5 mg/kg (max dose 30 mg)
Hydromorphone	0.015 mg/kg
Sedatives	
Midazolam	0.05-0.10 mg/kg
Diazepam	0.2 mg/kg (max dose 10 mg)
Lorazepam	0.05 mg/kg (max dose 2 mg)
Diprivan	1.0-2.0 mg/kg (loading), 50-100 ug/kg/min (infusion)
Etomidate	0.15 mg/kg (sedation); 0.3 mg/kg (induction)
Dexmetomidine[37]	0.5-2 ug/kg (sedation); 0.1-2 ug/kg/hr (infusion)
Other	
Ketamine	0.5-2.0 mg/kg
Paralytics	
Succinylcholine	1.0-2.0 mg/kg
Vecuronium	0.1-0.2 mg/kg
Rocuronium	0.5-1.0 mg/kg

Table 6. Common Antidotes for Cyanide Toxicity

Antidote	Pediatric Dosing
Hydroxocobalamin	70 mg/kg up to adult dose of 5 g, administered IV over 15 minutes, then a second dose of the same given over 15-120 minutes, as clinically indicated
Sodium thiosulfate	500 mg/kg up to the adult dose of 12.5 g, administered IV over 10-30 minutes, then a second administration of half the initial dose given at 2 hours if symptoms persist
Sodium nitrite	Administer 0.2 mL/kg of 3% sodium nitrite solution intravenously, not to exceed 300 mg; repeated at half the initial dose if symptoms of cyanide toxicity persist/reappear

destruction and impedance of ciliary function. Exposure to smoke triggers an inflammatory cascade that increases bronchial vessel permeability and leads to pulmonary edema. This localized destruction, in combination with the subsequent sloughing of necrotized respiratory epithelium and interstitial congestion, results in hypoxia.

In addition to direct alveolar and bronchial effects, compounds such as CO and CN are absorbed and produce systemic toxicity. The increased affinity of CO for hemoglobin shifts the oxygen dissociation curve to the left, impairing diffusion of oxygen into the tissues. Management of CO toxicity involves initial supportive care, primarily through the administration of 100% humidified, normobaric oxygen, which reduces the elimination half-life of CO from 5 to 6 hours to approximately 30 to 90 minutes. Further reduction in half-life to 30 minutes is possible with the utilization of hyperbaric oxygen therapy (HBOT).[38] The goal of HBOT is to prevent delayed neuropsychological sequelae in the form of cognitive impairments and affective disorders that manifest days to months after exposure.

There are limited data to indicate exactly when HBOT is indicated in pediatric patients.[39,40] Studies in adult populations suggest benefits in patients with acute CO exposure in patients with loss of consciousness, and those with higher COHb levels at presentation.

CN uncouples oxidative phosphorylation within mitochondria, derailing aerobic metabolism and resulting in formation of lactic acid. CN is managed with high-flow oxygen as well as concomitant administration of one of the four classes of antidote. These include hydroxocobalamin, sodium thiosulfate, dicobalt edetate, and methemoglobin-forming compounds.[41] (*See Table 6.*)

The mainstay for refractory hypoxia from inhalational injury-related pulmonary edema is ventilator support. The goal is to improve oxygenation while minimizing ventilator-induced lung injury. Unfortunately, limited data exist to suggest which settings lead to the best outcomes in the pediatric population. One 2004 survey produced no consensus among pediatric burn centers regarding the application of various ventilator modes in the setting of acute respiratory failure, as well as the use of cuffed endotracheal tubes and the timing of tracheostomy.[42]

Small studies have demonstrated the effectiveness of less conventional ventilator therapies, including high-frequency percussive ventilation, high-frequency oscillatory ventilation,[43] extracorporeal membrane oxygenation (ECMO) in refractory patients,[44] and even the use of surfactant as an adjunct,[45] but further study is needed to elucidate the effectiveness of these alternative therapies. A full discussion of these therapies is beyond the scope of this article.

Fluid Resuscitation

As with adults, adequate fluid resuscitation and maintenance of euvolemia is a critical component in the management of pediatric burns. The causes of hypotension in burn patients are multifactorial and include evaporative fluid losses, extravascular fluid shifts, and inflammatory responses related to the burn itself, as well as from subsequent infection. Inadequate resuscitation leads to renal and hepatic dysfunction.[46,47] Restoring and maintaining adequate perfusion can prevent late complications, including multisystem organ failure. The efficacy of fluid resuscitation in burned children is dependent on time to intervention. Prompt initiation is associated with a lower incidence of sepsis and renal failure, fewer deaths with cardiac arrest, and lower overall mortality.[48]

The Parkland formula provides an early estimate for 24-hour fluid volume requirements for resuscitation. Fluid requirements are calculated by multiplying 4 milliliters per kilogram body mass (ml/kg) by the estimated percentage total body surface area (TBSA) of second- and third-degree burns for the first 24 hours (4 mL/kg/% BSA). Half of the calculated volume is administered in the first 8 hours following injury and the remainder during the subsequent 16 hours.

Although the Parkland formula is well established as a guideline for fluid resuscitation, it has been recently challenged.[49] Multiple studies have demonstrated a subset of patients in which the Parkland Formula inaccurately estimates necessary replacement.[50,51] Identification of these specific populations in which high fluid volumes have produced poor outcomes has changed the basis of concern from inadequate resuscitation of burn patients to over-resuscitation.[52]

The clinical consequences of over resuscitation, first described by Pruitt[53] as "fluid creep" are manifest in the form of ARDS, pneumonia, bloodstream infections, multiple organ failure, compartment syndromes of both the abdomen and non-traumatic limbs, and death.[54,55] Recognition of this complication has led to the utilization of alternative products for initial resuscitation, including hypertonic fluids and colloids,[56,57] the clinical effectiveness of which has not yet been conclusively demonstrated. Despite this, a number of burn centers use colloid replacement therapy in pediatric patients in whom isotonic crystalloid replacement does not prove adequate. Colloids have not demonstrated a survival benefit in these patients.[58,59,60] Limited data suggest that using invasive hemodynamic monitoring to guide fluid resuscitation results in a lower incidence of sepsis and mortality at a similar infection rate, and that using such monitoring achieves similar rates of urine output in critically burned patient with less volume.[61]

As most severe burns are complicated by anemia, blood products are viewed as an often necessary supplement to intravenous crystalloids for volume replacement. However, blood transfusions are not a benign therapy, and associated complications may occur. Some studies have demonstrated a correlation between increased transfusion products in trauma and increased mortality[62] and, as such, the current trend is to minimize transfused blood products.[63] The details of transfusion ratios and techniques are beyond the scope of this article.

Figure 4. Large Second- and Third-Degree Burns to Torso and Extremities with Escharotomy

Photo courtesy of Dr. Colin Kaide

Figure 5. Close-up of Escharotomy Incision

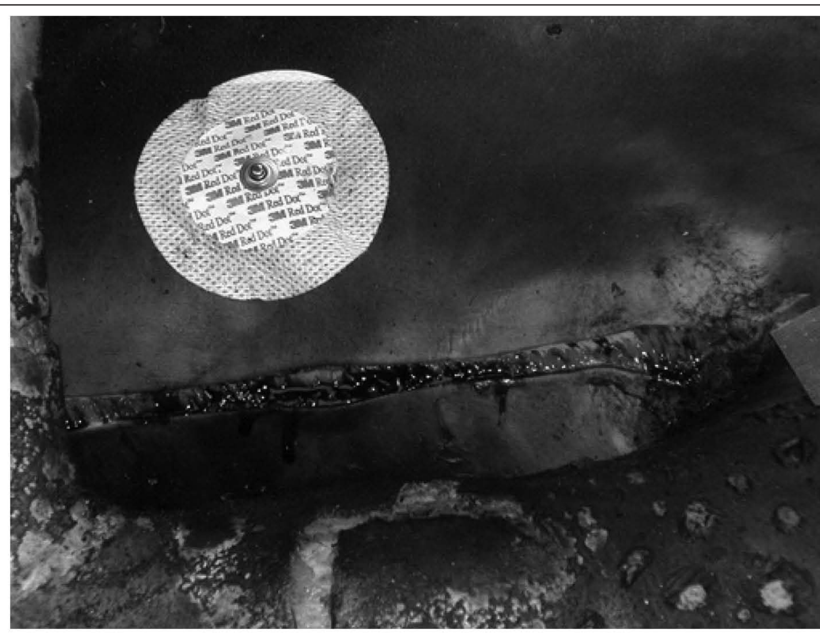


Photo courtesy of Dr. Colin Kaide

Escharotomy

Escharotomy is the surgical release of restrictive, inelastic tissue when such tissue begins to compromise physiological function (e.g., neurovascular function in an extremity, tracheal obstruction, or severely limited chest wall motion). Prior to commencing, the patient should receive proper analgesia, either local

Table 7. ABA Indications for Transfer to a Burn Center

- Second-degree burns (partial thickness) of greater than 10% of the body surface area (BSA)
- Third-degree burns (full thickness) in any age group
- Burns involving:
  - Signs or symptoms of inhalation injury
  - Respiratory distress
  - The face
  - The ears (serious full-thickness burns or burns involving the ear canal or drums)
  - The mouth and throat
  - Deep or excessive burns of the hands, feet, genitalia, major joints, or perineum
- Electrical injury or burn (including lightning)
- Burns associated with trauma or complicating medical conditions
- Chemical burns
- Burn injury in patients who will require special social, emotional, or rehabilitative intervention
- Burned children in hospitals without qualified personnel or equipment for the care of children

or systemic, as time allows. For the physician unfamiliar with the technique, the incision should be made to the depth of the subcutaneous fat.

For limbs, the lateral and medial aspects of the involved extremity are incised with a scalpel 1 cm proximal to the burned area, extending 1 cm distal to the burn. Neck escharotomy should be performed longitudinally along the lateral posterior aspect of the neck to avoid the critical vascular structures. In the case of full-thickness circumferential chest burns, the incision should extend from the clavicle to the costal margin bilaterally and link transverse incisions as dictated by the area of the burn.[68] (*See Figures 4 and 5.*)

Wound

The primary early complications of burn wounds are volume losses and infection secondary to interruption of the dermal barrier. Later, there is disruption of mechanical function related to contracture formation. Once the injured area has been cleared of obstructive debris and/or any chemical agents that might augment injury, efforts should be directed at cleansing the wound and debriding necrotic tissue. These steps allow for better visualization of the wound, identification of wound margins, and the removal of devitalized tissue that has the potential to serve as a nidus for infection. Cleaning typically consists of gentle mechanical scrubbing with soap and water. When blisters are encountered, the practitioner should attempt to leave them intact, since unroofing them may lead to slightly higher infection rates and more patient discomfort.

Tetanus vaccination status should be addressed and updated as necessary. Before the advent of effective tetanus prophylaxis, 20% of the patients sustaining major thermal injury died of tetanus infection.[69]

Burns with an intact epidermis do not require antimicrobial prophylaxis or complex dressings, but for patients with deeper burns, consideration should be given to dressing type and antimicrobial agents. Maintaining wounds at low colonization levels reduces the frequency and duration of septic episodes caused by wound contamination from skin flora.[70] Systemic antimicrobial agents do not reach burn wounds in sufficient concentrations due to thrombosis of capillaries and wound edema, which compresses local vasculature. Therefore, their use is not routinely recommended. However, topical agents do successfully delay colonization of organisms and keep wound flora to a minimum. When preparing a patient for transfer to a burn center, a clean or sterile dry towel/sheet should be placed over the burned area without any additional medications or dressings.

Historically, application of antimicrobial agents such as mafenide acetate, 1% silver sulfadiazine, or bacitracin in combination with synthetic bandages has been the standard of care,[71] but these agents are slowly being replaced by more advanced artificial skin and biosynthetic dressings.

Biosynthetic dressings such as Mepilex, Biobrane, beta-glucagon collagen, and hydrocolloid improve wound healing by promoting early epithelialization. Occasionally, the application of these will be requested by the burn specialist. Some, such as Mepilex Ag, have the added benefit of intrinsic antimicrobial properties and can be used independently of topical antibiotics. Early studies suggest they provide a wide range of benefits in the form of less-frequent dressing changes, facilitation of outpatient versus inpatient therapy, increased patient comfort, and decreased incidence of operative intervention.[72,73,74] Studies have not consistently demonstrated a benefit of one dressing type over another, and practice varies from institution to institution.

Ultimately, those burns that are too extensive or complicated to be managed with dressings require definitive surgical management in the form of excision and reconstruction using skin grafts. Early excision has consistently been shown to decrease hospital length of stay and mortality.[75,76,77]

Nutrition

Nutrition is of critical importance

following a severe burn. The goal is to provide nutritional supplementation within 48 hours of injury. Extensive burn trauma leads to both a hypermetabolic and catabolic state, in which the patient is at risk for malnutrition and progressive weight loss for an interval extending for at least 9–12 months after injury.[78] This discrepancy between protein/energy supply and demand manifests clinically with delayed wound healing, muscle wasting, and immunocompromise during recovery. A number of regulatory molecules contribute to this consumptive state, and future therapies will likely attempt to combat muscle breakdown via a combination of anabolic proteins, anabolic steroids, and anti-catabolic agents.[79] The details of the complex caloric needs of the pediatric burn patient are beyond the scope of this article.

Psychological Support

In the pediatric patient, the physical damage of a severe burn injury is often accompanied by psychological stress. Acutely, this presents as internalizing behaviors such as anxiety and withdrawal, and externalizing behaviors, such as those characterized by oppositional defiant disorder. These represent healthy coping mechanisms, which may help children deal with their situation.[87]

More concerning is the potential to develop behaviors or attitudes that are dysfunctional. Children must deal with a host of complications. In addition to anxiety and post-traumatic stress disorder (PTSD) symptoms, they may have difficulty with social interaction, self-esteem issues, or problems with body image (related to scarring).[88] This is compounded by whatever functional deficits they have acquired as a result of the burn injury itself. The authors of one study concluded that 15–20% of children developed a negative psychosocial outcome as a result of burn trauma.[89]

An increasing body of literature reveals extensive psychosocial and familial challenges and suggests that there is utility in recruiting professional psychological help for pediatric trauma survivors and their families in the aftermath of a burn injury.[90]

Complications

Infection is the most common complication of pediatric burns.[91,92] Infectious complications include urinary tract infection, pneumonia, wound infection, and line infection. The practitioner should have a high suspicion for infection in the pediatric burn patient with fever, leukocytosis, malaise, rash, or altered mental status. Urinary tract infections (UTIs) due to prolonged Foley catheter use are more common in pediatric burn patients than adults and are the leading complication of burns in children younger than age 16 years.[2,92] Cellulitis closely follows UTIs in rates of complication.[2] In addition to the common pathogens *Staphylococcus* and *Streptococcus*, one should be aware of pathogens unique to burn patients, such as *Pseudomonas*, yeast, and fungus. As with other hospitalized and immune-compromised patients, providers should be cognizant of the potential for resistant organisms when choosing antimicrobials.[93,94] The third most common infection is pneumonia. As with non-burned patients, the risk of pneumonia is increased with mechanical ventilation.[93] In addition, inhalation injury alone also increases the risk of pneumonia.[95]

Pediatric burn victims are also prone to septicemia and fungemia, with sepsis being the leading cause of mortality in pediatric burn patients.[94,95]

As expected, larger, deeper burns and indwelling lines increase the risk for nosocomial infection.[96] Treatment for these conditions includes antibiotics and supportive care. Prophylactic antibiotics are not indicated.[93] Prevention consists of early wound closure, infection control programs, minimizing ventilator days, and meticulous catheter care. In addition, the use of central lines should be reserved to the ICU setting or in cases in which peripheral access cannot be obtained.

Renal failure is a less common and often less severe complication of pediatric burns. The etiology is often secondary to inadequate resuscitation of the burn patient. Given the fact that most children do not have underlying kidney disease, renal function tends to recover well with fluid resuscitation.[97]

In contrast to renal failure, abdominal compartment syndrome (ACS) is a phenomenon that develops in burn patients as a result of adequate or over-resuscitation. ACS is present when intra-abdominal pressure consistently measures greater than 20 mm Hg and there are signs of end organ damage. Burns induce ABS through several mechanisms, including over-resuscitation, direct compression due to circumferential burns, and increased mesenteric vascular resistance and subsequent inflammatory cascades. Early recognition and prompt treatment of ACS is essential to minimizing end organ damage, restoring perfusion, and improving outcomes.[98,99]

Disposition

The decision to discharge, admit, or transfer the pediatric burn patient is one that is not always clear. To assist in this, the ABA has established guidelines for transfer to a tertiary burn center. (*See Table 7.*)

The majority of pediatric burns are minor, as previously discussed. Admission rates for pediatric burns are relatively low, ranging from 4-10%. Most first-degree burns and burns less than 10% often can be sent home with close outpatient follow-up. Social work should be involved in burns suspicious for NAT or the patient should be admitted for further investigation to prevent exposing the child to further abuse and injury.[7,100-104]

Prevention

The prevention of pediatric burns by making parents and children aware of environmental contributors is as important as the recognition and evidence-based management of burn injuries. The ABA has made a concerted effort in the area of burn prevention, as have many pediatric advocacy groups and local burn

Table 8. Pearls of Pediatric Burns

- Consider NAT as a cause of burn.
- Early, aggressive airway control is essential.
- Use the rule of 9s to assess TBSA of second-, third-, and fourth-degree burns. If unable to remember rule of 9s, the patient's palm equals 1% TBSA.
- Resuscitate using Parkland formula
  - 4 mL/kg/% TBSA
  - half of this volume over first 8 hours, and second half over the next 16 hours
- Be knowledgeable about transfer criteria and transfer early and appropriately.

centers. Efforts include suggesting a lower temperature setting of 120°F on residential water heaters to reduce the incidence of scald injuries. Simple measures such as teaching parents to turn pot handles to the back, being cognizant of electrical cord location, using non-slip mats, not heating infant bottles in a microwave, applying outlet covers, and never leaving children unsupervised in the kitchen are being employed to reduce burn injuries. As practitioners, it is important to review and reinforce these concepts when one encounters parents with a child, especially one with a burn, to prevent future injury.

Conclusion

Burns are a significant problem affecting the pediatric population. In addition to the physical disfigurement of burn injuries, they carry a psychological burden that can leave a lifelong impact on the child and family. Pediatric burn patients can be very complex. An organized approach to the assessment and management of the pediatric burn patient assures the best outcome. Early control of the airway and appropriate fluid management are critical determinants of good outcomes for this population. Proper assessment and evaluation of the burn injury ensures patients are dispositioned to the adequate level of care. It is crucial that the physician understand the unique medical and psychological complications of pediatric burns, enabling early recognition and intervention to minimize long-term disability. (*See Table 8.*)

References

1. Centers For Disease Control. National Center for Injury Preventions and Control [Online]. Available: www.cdc.gov/ncipc.wisquars. Accessed 25 March 2013.
2. American Burn Association. National Burn Repository Annual Report. American Burn Association, Chicago, IL, 2012.
3. D'Souza A, Nelson N, McKenzie L. Pediatric burn injuries treated in US emergency departments between 1990 and 2006. *Pediatrics* 2009;1424;124.
4. Carlsson A, Udén G, Håkansson A, et al. Burn injuries in small children: A population-based study in Sweden. *J Clin Nurs* 2006;129:15.
5. Barrow R, Spies M, Barrow L, et al. Influence of demographics and inhalation injury on burn mortality in children. *Burns* 2004;72:30.
6. Cuccurullo S. Physical Medicine and Rehabilitation Board Review, New York, NY: Demos Medical Publishing, 2004.
7. Drago D. Kitchen scalds and thermal burns in children five years and younger. *Pediatrics* 2005.
8. Mille RS, Bessey P, Schurr M, et al. National Burn Repository 2005: A ten-year review. *J Burn Care Res* 2006;27:411.
9. Delgado J, Ramirez-Cardich M, Gilman R, et al. Risk factors for burns in children: Crowding, poverty and poor maternal education. *Injury Prevention* 2002;8:38-41.
10. Haberal M, Abali AES, Karakayali H. Fluid management in major burn injuries. Indian *J Plast Surg* 2010;43:29-36.
11. Reiss E, Pearson E, Artz C. The metabolic response to burns. *J Clin Invest* 1956;35:62-77.
12. Cuthbertson D, Angeles Valero Zanuy M, León Sanz M. Post-shock metabolic response. 1942. *Nutr Hosp* 2001;16:176-182.
13. Herndon D, Tompkins R. Support of the metabolic response to burn injury. *Lancet* 2004;363:1895-1902.
14. Jeschke M, Gauglitz G, Kulp G, et al. Long-term persistence of the pathophysiologic response to severe burn injury. 2011 *PONE*, online.
15. Moritz A, Herriques FC. Studies of thermal injuries: II. The relative importance of time and surface temperature in the causation of cutaneous burns. *Am J Pathology* 1947;23:695-720.
16. Diller K. Adapting adult scald safety standards to children. *J Burn Care & Research* 2006;27:314-322.
17. Fidkowski CW, Fuzaylov C, Sheridan RL, et al. Inhalation burn injury in children. *Pediatric Anesthesia* 2009;19:147-154.
18. Clark WR. Smoke inhalation: Diagnosis and treatment. *World J Surgery* 1992;16:24-29.
19. Serebrisky D, Nazarian EB, Connolly H. Inhalation Injury. Medscape. Available: http://emedicine.medscape.com/article/1002413-overview#a0199. Accessed 15 April 2013.
20. Endorf F, Gamelli R. Inhalation injury, pulmonary perturbations, and fluid resuscitation. *J Burn Care Res* 2007;28:80-83.
21. Chester D, Jose R, Aldlyam E, et al. Non-accidental burns in children — are we neglecting neglect? *Burns* 2006;32:222-228.
22. Deitch E, Staats M. Child abuse through burning. *J Burn Care Rehabil* 1982;89-94.
23. Keen J, Lendrum J, Wolman B. Inflicted burns and scalds in children. *BMJ* 1975;4:268-269.
24. American Burn Association. Advanced Burn Life Support, Chicago, IL: American Burn Association, 2007.
25. Chan Q, Barzi F, Cheney L, et al. Burn size estimation in children: Still a problem. *Emerg Med Australas* 2012;24:181-186.
26. Kuckelkorn R, Schrage N, Keller G, et al. Emergency treatment of chemical and thermal eye burns. *Acta Ophthalmol Scand* 2002;80:4-10.
27. Gore DC. Assessment of adverse events in the demise of pediatric burn patients. *J Trauma* 2007;63:814.
28. El-Helbawy RH. Inhalation injury as a prognostic factor for mortality in burn patients. *Ann Burns Fire Disasters* 2011;24:82-8.
29. Turegun M. The last 10 years in a burn centre in Ankara, Turkey: An analysis of 5264 cases. *Burns* 1997;23:584-590.

30. Grevious MA. Burn scar contractures of the pediatric neck. *J Craniofac Surg* 2008;19:1010-1015.

31. Rose AS. Contracture related airway obstruction (CRAO) treated successfully with incisional release. *Int J Pediatr Otorhinolaryngol* 2011;75:286-288.

32. Martyn JA. Succinylcholine-induced hyperkalemia in acquired pathologic states: etiologic factors and molecular mechanisms. *Anesthesiology* 2006;104:158-169.

33. Norambuena C. Oral ketamine and midazolam for pediatric burn patients: A prospective, randomized, double-blind study. *J Pediatr Surg* 2013;48:629-634.

34. Bayat A. Analgesia and sedation for children undergoing burn wound care. *Expert Rev Neurother* 2010;10: 1747-1759.

35. Fagin A. A comparison of dexmedetomidine and midazolam for sedation in severe pediatric burn injury. *J Burn Care Res* 2012;33:759-763.

36. Lin H. Use of dexmedetomidine for sedation in critically ill mechanically ventilated pediatric burn patients. *J Burn Care* Res2011;32:98-103.

37. Phan H. Clinical uses of dexmedetomidine in pediatric patients. *Paediatric Drugs* 2008;10:49-69.

38. Leikin FPJB In: *Poisoning and Toxicology Handbook*, 4th ed. 2008: 768-770.

39. Martin JD. Recognition and management of carbon monoxide poisoning in children. *Clin Ped Emerg Med* 2000;1: 244-250.

40. Baum CR. What's new in pediatric carbon monoxide poisoning? *Clinical Pediatric Emergency Medicine* 2008;9:43-46.

41. Lawson-Smith P. Cyanide intoxication as part of smoke inhalation — a review on diagnosis and treatment from the emergency perspective. *Scand J Trauma Resusc Emerg Med* 2011;3:14.

42. Silver GM. A survey of airway and ventilator management strategies in North American pediatric burn units. *J Burn Care Rehabil* 2004;25:435-440.

43. Greathouse ST. High-frequency oscillatory ventilators in burn patients: Experience of Riley Hospital for Children. *J Burn Care Res* 2012;33:425-435.

44. Askegard-Giesmann JR. Extracorporeal membrane oxygenation as a lifesaving modality in the treatment of pediatric patients with burns and respiratory failure. *J Pediatr Surg* 2010;45:1330-1335.

45. Sen S, Tung K, Palmieri T, et al. Surfactant therapy for acute respiratory distress in severe pediatric burn injury: A case series. *J Burn Care Res* 2012;33: e88-91.

46. Jeschke MG. Mortality in burned children with acute renal failure. *Arch Surg* 1998;134:752-756.

47. Wolf SE. Mortality determinants in massive pediatric burns. *Ann Surg* 1997;5:554-69.

48. Barrow RE. Early fluid resuscitation improves outcomes in severely burned children. *Resuscitation* 2000;45:91.

49. Cartotto RC. How well does the Parkland formula estimate actual fluid resuscitation volumes? *J Burn Care Rehabil* 2002;23:258-265.

50. Kaups KL. Base deficit as an indicator of resuscitation needs in patients with burn injuries. *J Burn Care Rehabil* 1998;19:346-348.

51. Friedrich JB Is supra-Baxter resuscitation in burn patients a new phenomenon? *Burns* 2004;30:464-466.

52. Rogers AD, Karpelowsky J, Millar AW, et al. Fluid creep in major pediatric burns. *Eur J Pediatr Surg* 2010;20:133-138.

53. Pruitt BAJ. Protection from excessive resuscitation: Pushing the pendulum back. *J Trauma* 2000;49:567–568.

54. Klein MB, Hayden D, Elson C, et al. The association between fluid administration and outcome following major burn: a multicenter study. *Ann Surg* 2007;245:622-628.

55. Tuggle D, Skinner S, Garza J, et al. The abdominal compartment syndrome in patients with burn injury. *Acta Clin Belg* Suppl. 2007;1:136-140.

56. Saffle JI. The phenomenon of "fluid creep" in acute burn resuscitation. *J Burn Care Res* 2007;28:382-395.

57. Atiyeh BS, Dibo SA, Ibrahim AE, et al. Acute burn resuscitation and fluid creep: It is time for colloid rehabilitation. *Ann Burns Fire Disasters* 2012;25:59-65.

58. Bunn F, Roberts IG, Tasker R, et al. Hypertonic versus near isotonic crystalloid for fluid resuscitation in critically ill patients. *Cochrane Database Syst Rev* 2004;CD002045.

59. Alderson P, Bunn F, Li Wan Po A, et al. Human albumin solution for resuscitation and volume expansion in critically ill patients. *Cochrane Database Syst Rev* 2004; CD001208.

60. Faraklas I, Lam U, Cochran A, et al. Colloid normalizes resuscitation ratio in pediatric burns. *J Burn Care Res* 2011;32:91-97.

61. Kraft R, Herndon DN, Branski LK, et al. Optimized fluid management improves outcomes of pediatric burn patients. *J Surg Res* 2013;181:121-8.

62. Malone DL, Dunne J, Tracy JK. Blood transfusion, independent of shock severity, is associated with worse outcome in trauma. *J Trauma* 2003;54:898-907.

63. Palmieri TL, Lee T, O'Mara MS, et al. Effects of a restrictive blood transfusion policy on outcomes in children with burn injury. *J Burn Care Res* 2007;28:65-70.

64. Palmieri TL, Greenhalgh DG, Sen S. Prospective comparison of packed red blood cell-to-fresh frozen plasma transfusion ratio of 4: 1 versus 1: 1 during acute massive burn excision. *J Trauma Acute Care Surg* 2013;74:76-83.

65. Mitra B, Mori A, Cameron PA. Fresh frozen plasma (FFP) use during massive blood transfusion in trauma resuscitation. *Injury* 2010;41:35-39.

66. Zehtabchi S, Nishijima DK. Impact of transfusion of fresh-frozen plasma and packed red blood cells in a 1:1 ratio on survival of emergency department patients with severe trauma. *Acad Emerg Med* 2009;16:371-378.

67. Curinga G, Jain A, Feldman M, et al. Red blood cell transfusion following burn. *Burns* 2011;37:742-752.

68. Bethel CA. Burn care Procedures. In: Roberts and Hedges, *Clinical Procedures in Emergency Medicine*, 5th ed. 2010: 713-714.

69. Moncrief JA. Burns. II. Initial treatment. *JAMA* 1979;242:179-182.

70. Barret JP, Dardano AN, Heggers JP, et al. Infestations and chronic infections in foreign pediatric patients with burns: Is there a role for specific protocols? *J Burn Care Rehabil* 1999;20:482-486.

71. Demling RH, Lalonde C. *Burn Trauma*, New York (NY): Thieme Medical Publishers Inc.; 1989.

72. Lesher AP, Curry RH, Evans J. Effectiveness of Biobrane for treatment of partial-thickness burns in children. *J Pediatr Surg* 2011;46:1759-1763.

73. Delatte J, Evans J, Hebra AV. Effectiveness of beta-glucan collagen for treatment of partial-thickness burns in children. *J Pediatr Surg* 2001;36: 113-118.

74. Martin FT, O'Sullivan JB, Regan PJ. Hydrocolloid dressing in pediatric burns may decrease operative intervention rates. *J Pediatr Surg* 2010;45:600-605.

75. Munster AM, Smith-Meek M, Sharkey P. The effect of early surgical intervention on mortality and cost-effectiveness in burn care, 1978-91. *Burns* 1994;20: 61-64.

76. Ong YS, Samuel M, Song C. Meta-analysis of early excision of burns. *Burns* 2006;32:145-150.

77. Tompkins RG, Burke JF, Schoenfeld DA. Prompt eschar excision: A treatment system contributing to reduced burn mortality. A statistical evaluation of burn care at the Massachusetts General Hospital (1974-1984). *Ann Surg* 1986;204: 272-281.

78. Hart DW, Wolf SE, Mlcak R. Persistence of muscle catabolism after severe burn. *Surgery* 2000;128:312-319.

79. Pereira C, Murphy K, Jeschke M. Post burn muscle wasting and the effects of treatments. *Int J Biochem Cell Biol* 2005;37:1948-1961.

80. Chan MM, Chan GM. Nutritional therapy for burns in children and adults. *Nutrition* 2009;25:261-269.

81. Prelack K, Dwyer J, Dallal GE. Growth deceleration and restoration after serious burn injury. *J Burn Care Res* 2007;28:262-268.

82. Dylewski ML, Baker M, Prelack K. The safety and efficacy of parenteral nutrition among pediatric patients with burn inju-

ries. *Pediatr Crit Care Med* 2013;14: 120-125.

83. Khorasani EN, Mansouri F. Effect of early enteral nutrition on morbidity and mortality in children with burns. *Burns* 2010;36:1067-1071.

84. Murphy KD, Lee JO, Herndon DN. Current pharmacotherapy for the treatment of severe burns. *Expert Opin Pharmacother* 2003;4:369-384.

85. Hurt RT, Frazier TH, McClave SA. Stress prophylaxis in intensive care unit patients and the role of enteral nutrition. *JPEN J Parenter Enteral Nutr* 2012;36:721-731.

86. Pilkington KB, Wagstaff MJ, Greenwood JE. Prevention of gastrointestinal bleeding due to stress ulceration: A review of current literature. *Anaesth Intensive Care* 2012;40:253-259.

87. Kazak AE, Kassam-Adams N, Schneider S. An integrative model of pediatric medical traumatic stress. *J Pediatr Psychol* 2006;31:343-355.

88. Bakker A, Maertens KJ, Van Son MJ. Psychological consequences of pediatric burns from a child and family perspective: A review of the empirical literature. *Clin Psychol Rev* 2013;33:361-371.

89. Tarnowski KJ, Rasnake LK, Gavaghan-Jones MP. Psychosocial sequelae of pediatric burn injuries: A review. *Clinical Psychology Review* 1991;11:399-418.

90. Martin-Herz SP, Zatzick DF, McMahon RJ. Health-related quality of life in children and adolescents following traumatic injury: A review. *Clin Child Fam Psychol Rev* 2012;15:192-214.

91. Fenlon S, Nene S. Burns in Children. Continuing Education in Anaesthesia. *Critical Care & Pain* 2007;7:76-80.

92. Schlager T, Sadler J, Weber D, et al. Hospital-acquired infections in pediatric burn patients. *South Med J* 1994;87: 481-484.

93. Weber J, McManus A. Infection control in burn patients. *Burns* 2004;30:A16-24.

94. Geyik M, Aldemir M, Hosoglu S, et al. Epidemiology of burn unit infections in children. *Am J Infect Control* 2003;31:342-346.

95. Sheridan R. Sepsis in pediatric burn patients. *Pediatr Crit Care Med* 2005;6: .

96. Fekih Hassen A, Ben Khalifa S, Raddaoui K, et al. Risk factors for nosocomial infection in pediatric burn patients. *Ann Fr Anesth Reanim* 2012;31:591-595.

97. Palmieri T, Lavrentieva A, Greenhalgh D. An assessment of acute kidney injury with modified RIFLE criteria in pediatric patients with severe burns. *Intensive Care Medicine* 2009;35:2125-2129.

98. Carlotti APCP, Carvalho WB. Abdominal compartment syndrome: A review. *Pediatric Crit Care Med* 2009;10: 115-120.

99. Malbrain M, Cheatham M, Sugrue M, et al. The abdominal compartment syndrome. In: *Surgical Intensive Care Medicine*. Springer; 2010:507-527.

100. Chipp E, Walton J, Gorman D, et al. A 1 year study of burn injuries in a British emergency department. *Burns* 2008;34:516-520.

101. DeKoning E, Hakenewerth A, Platts-Mills T, et al. Epidemiology of burn injuries presenting to North Carolina emergency departments in 2006–2007. *Burns* 2009;35:776-782.

102. Fagenholz P, Sheridan R, Harris N, et al. National study of emergency department visits for burns injuries, 1993 to 2004. *J Burn Care Res* 2007;28:681-690.

103. Khan A, Rawlins J, Shenton A, et al. The Bradford Burn Study: The epidemiology of burns presenting to an inner city emergency department. *Emerg Med J* 2007;24:564-566.

104. Rawlins J, Khan A, Shenton A, et al. Epidemiology and outcome analysis of 208 children with burns attending an emergency department. *Pediatr Emerg Care* 2007;23:289-293.

105. Norbury WB. In: *Total Burn Care*, 3rd ed. New York, NY: Saunders Elsevier: 420-433.

106. Drago D. Kitchen scalds and thermal burns in children five years and younger. *Pediatrics* 2005;10:115.

107. Barrow R, Spies M, Barrow LN, et al. Influence of demographics and inhalation injury on burn mortality in children. *Burns* 2004;30:72-77.

CNE/CME Objectives

Upon completing this program, the participants will be able to:

- discuss conditions that should increase suspicion for traumatic injuries;
- describe the various modalities used to identify different traumatic conditions;
- cite methods of quickly stabilizing and managing patients; and
- identify possible complications that may occur with traumatic injuries.

CNE/CME Instructions

HERE ARE THE STEPS YOU NEED TO TAKE TO EARN CREDIT FOR THIS ACTIVITY:

1. Read and study the activity, using the provided references for further research.

2. Log on to **www.cmecity.com** to take a post-test; tests can be taken after each issue or collectively at the end of the semester. *First-time users will have to register on the site using the 8-digit subscriber number printed on their mailing label, invoice, or renewal notice.*

3. Pass the online tests with a score of 100%; you will be allowed to answer the questions as many times as needed to achieve a score of 100%.

4. After successfully completing the last test of the semester, your browser will be automatically directed to the activity evaluation form, which you will submit online.

5. **Once the completed evaluation is received, a credit letter will be e-mailed to you instantly.** You will no longer have to wait to receive your credit letter.

CME/CNE Questions

1. A burn that involves the dermis and epidermis is classified as:
   A. first degree
   B. second degree
   C. third degree
   D. fourth degree

2. A burn that is insensate is classified as:
   A. first degree
   B. second degree
   C. third degree
   D. fourth degree

3. Which of the following *does not* require transfer to a burn center?
   A. a child with genital burns
   B. a child with second-degree burns over the entire anterior chest and right anterior thigh
   C. a child with circumferential second-degree burns of a finger
   D. All of the above should be sent to a pediatric burn center.

4. A 5-year-old child has a second-degree splash burn to the anterior chest, anterior right thigh, and anterior right upper extremity. What is the TBSA?
   A. 10%
   B. 15%
   C. 20%
   D. 25%

5. If the previously mentioned 5-year-old weighs 20 kg, what is the total amount of fluids the child should receive in the first 24 hours?
   A. 800 mL
   B. 1000 mL
   C. 1600 mL
   D. 2000 mL

6. Which of the following is an indication for early intubation?
   A. hoarse voice
   B. carbonaceous sputum
   C. drooling
   D. all of the above
7. Which of the following is an indication for escharotomy?
   A. circumferential extremity burn
   B. poor ventilatory compliance with significant chest wall burn
   C. abdominal wall burn
   D. all of the above
8. Children involved in closed-space fires are more likely to sustain inhalation injury as the result of their relative immobility and impaired ability to escape.
   A. true
   B. false
9. Young children require as little as what percentage total body surface area (TBSA) to trigger a systemic response?
   A. 1%
   B. 5%
   C. 25%
   D. 15%
10. Which of the following statements is true regarding nutritional needs of burn patients?
   A. Patients are only at risk for malnutrition and progressive weight loss for the first three months after injury.
   B. Patients may experience symptoms such as delayed wound healing, muscle wasting, and immunocompromise during recovery.
   C. Extensive burn trauma leads to a hypermetabolic state, but not a catabolic state.
   D. Extensive burn trauma leads to a catabolic state, but not a hypermetabolic state.
11. Which of the following factors in the history and physical exam of burn injuries should cause the practitioner to maintain suspicion of non-accidental trauma?
   A. when the history and physical exam do not correlate with findings
   B. when there is a delay in seeking care
   C. when there is concern for neglect or lack of supervision
   D. when specific burn patterns are present
   E. all of the above
12. Which of the following is the leading complication of burns in children younger than 16 years of age?
   A. urinary tract infection from prolonged Foley catheter use
   B. wound infection
   C. pneumonia
   D. line infection

Extra Hour of CME Credit Available in this Issue

This issue contains material for an additional 1 hour of *AMA PRA Category 1 Credit*™.

Trauma Reports Reader Survey Now Online

This year, we're going digital with our annual *Trauma Reports* reader survey – and giving away a free publication to subscribers who take it. To participate, go to the Web address at the bottom of this message and enter your responses. When you're done, you'll receive a PDF of our new 29-page publication, *The Physician's Guide: How Not to Get Sued*.

Thanks in advance for sharing your thoughts about *Trauma Reports* and how we might better meet your needs as a subscriber.

Here's the Web address for the survey: https://www.surveymonkey.com/s/TraumaReports

Trauma Reports™ (ISSN 1531-1082) is published bimonthly by AHC Media, a division of Thompson Media Group, LLC, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Senior Vice President / Group Publisher: Donald R. Johnston

Executive Editor: Shelly Morrow Mark

Managing Editor: Leslie Hamlin

POSTMASTER: Send address changes to Trauma Reports,
P.O. Box 105109, Atlanta, GA 30348.

Subscriber Information

Customer Service: 1-800-688-2421

Customer Service E-Mail:
customerservice@ahcmedia.com

Editorial E-Mail:
shelly.mark@ahcmedia.com

World Wide Web page:
http://www.ahcmedia.com

FREE to subscribers of *Emergency Medicine Reports* and *Pediatric Emergency Medicine Reports*

Subscription Prices

United States

$249 per year. Add $17.95 for shipping & handling

Multiple Copies

Discounts are available for group subscriptions, multiple copies, site-licenses or electronic distribution. For pricing information, call Tria Kreutzer at 404-262-5482.

All prices U.S. only. U.S. possessions and Canada, add $30 postage plus applicable GST.

Other international orders, add $30.

Accreditation

AHC Media is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

AHC Media designates this enduring material for a maximum of 2.5 *AMA PRA Category 1 Credits*™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Approved by the American College of Emergency Physicians for a maximum of 2.5 hour(s) of ACEP Category I credit.

AHC Media is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation.

This activity has been approved for 1.5 nursing contact hours using a 60-minute contact hour.

Provider approved by the California Board of Registered Nursing, Provider # 14749, for 1.5 Contact Hours.

This is an educational publication designed to present scientific information and opinion to health professionals, to stimulate thought, and further investigation. It does not provide advice regarding medical diagnosis or treatment for any individual case. It is not intended for use by the layman. Opinions expressed are not necessarily those of this publication. Mention of products or services does not constitute endorsement. Clinical, legal, tax, and other comments are offered for general guidance only; professional counsel should be sought for specific situations.

This CME/CNE activity is intended for emergency, family, osteopathic, trauma, surgical, and general practice physicians and nurses who have contact with trauma patients.

It is in effect for 24 months from the date of publication.

In Future Issues | **Blast Injuries**

AHC Media

Dear *Trauma Reports* Subscriber:

This issue of your newsletter marks the start of a new continuing medical education (CME) semester and provides us with an opportunity to remind you about **the procedures for earning CME and delivery of your credit lette**r.

Trauma Reports, sponsored by AHC Media, provides you with evidence-based information and best practices that help you make informed decisions concerning treatment options and physician office practices. Our intent is the same as yours — the best possible patient care.

Upon completion of this educational activity, participants should be able to:

- discuss conditions that should increase suspicion for traumatic injuries;
- describe the various modalities used to identify different traumatic conditions;
- cite methods of quickly stabilizing and managing patients; and
- identify possible complications that may occur with traumatic injuries.

The American Medical Association, which oversees the Physician's Recognition Award and credit system and allows AHC Media to award *AMA PRA Category 1 Credit ™*, has changed its requirements for awarding *AMA PRA Category 1 Credit ™*. Enduring materials, like this newsletter, are now required to include an assessment of the learner's performance; the activity provider can award credit only if a minimum performance level is met. AHC Media considered several ways of meeting these new AMA requirements and chose the most expedient method for our learners.

HERE ARE THE STEPS YOU NEED TO TAKE TO EARN CREDIT FOR THIS ACTIVITY:
1. Read and study the activity, using the provided references for further research.
2. Log on to **www.cmecity.com** to take a post-test; tests can be taken after each issue or collectively at the end of the semester. *First-time users will have to register on the site using the 8-digit subscriber number printed on their mailing label, invoice, or renewal notice.*
3. Pass the online tests with a score of 100%; you will be allowed to answer the questions as many times as needed to achieve a score of 100%.
4. After successfully completing the last test of the semester, your browser will be automatically directed to the activity evaluation form, which you will submit online.
5. **Once the completed evaluation is received, a credit letter will be e-mailed to you instantly.** You will not have to wait to receive your credit letter!

This activity is valid 24 months from the date of publication. The target audience for this activity includes emergency medicine and family physicians.

If you have any questions about the process, please call us at (800) 688-2421, or outside the U.S. at (404) 262-5476. You can also fax us at (800) 284-3291, or outside the U.S. at (404) 262-5560. You can also email us at: customerservice@ahcmedia.com.

On behalf of AHC Media, we thank you for your trust and look forward to a continuing education partnership.

Sincerely,

Lee Landenberger
Continuing Education Director
AHC Media