

Burns Management

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A healthy six-month-old female presents to the OR for treatment of burns on the right side of her body. Her grandmother laid her down onto their apartment heater for approximately 30 minutes. The child's right side of the face, neck, shoulder, arm, hand, abdomen, leg, and foot are burned. Her lung fields are clear. Her first set of vital signs are: HR: 174, BP: 76/37; SpO₂: 100% on room air.

How Do Pediatric Burns Usually Occur?

Burns are a leading cause of in-home harm to children. Infants who are not yet walking are often burned when they are placed in contact with hot surfaces, or as a result of a hot liquid spill. Mobile toddlers are able to pull a cup containing hot liquid off a table, chew on an electrical cord, or accidentally step on a hot surface. Adolescent burns may involve gasoline and fire. Overall, 70% of pediatric burns are associated with hot liquids.

What Are the Different Classifications of Burns?

Initial classification is based on the type of burn:

- **Thermal burns:** the depth of the skin injury is related to contact temperature, duration of contact, and thickness of the skin involved. There may be airway damage from hot smoke inhalation;
- **Cold exposure burns** such as frostbite occur when bodily tissues are frozen;
- **Chemical burns** cause caustic reactions, alteration of the pH, disruption of cell membranes, and/or toxic effects on metabolic processes. Acid burns cause tissue coagulation whereas alkali burns cause liquefaction necrosis;
- **Electric current burns** are transformed into thermal injury as the current passes through

poorly conducting tissues. They may cause severe fractures, hematomas, visceral injury, and skeletal and cardiac muscle injury which can lead to pain, myoglobinuria, and dysrhythmias or other ECG abnormalities;

- **Inhalation burns** are typically caused by steam or fire;
- **Radiation burns** result from radiofrequency energy or ionizing radiation that causes damage to skin and tissues (e.g., sunburn).

Burns are further classified based on their penetrating depth:

- **Superficial (first-degree) burns** involve only the epidermal layer of the skin. These burns do not blister but are painful, dry, and blanch with pressure. These injuries are self-healing and require about one week to heal.
- **Partial thickness (second-degree) burns** can be classified as either superficial or deep. Superficial partial thickness burns are painful, red, weeping and also blanch with pressure. They blister within 24 hours, and do not typically scar, although skin pigmentation may change. Deep partial thickness burns extend deeper into the dermis and include damage to the hair follicles and glandular tissues. They are painful only to pressure and almost always blister. They are wet or waxy dry and have a mottled discoloration to them. They do not blanch with pressure. Without skin graft or infection, they heal in three to nine weeks.
- **Full thickness (third-degree) burns** destroy all the layers of the dermis and injure underlying subcutaneous tissue. Burn eschar typically remains intact. If the eschar is circumferential, it can compromise the area it is surrounding. These burns are usually painless. The skin appears along the spectrum of waxy white to charred black. The skin is dry, inelastic, and does not blanch with pressure. Blisters do not develop. Without

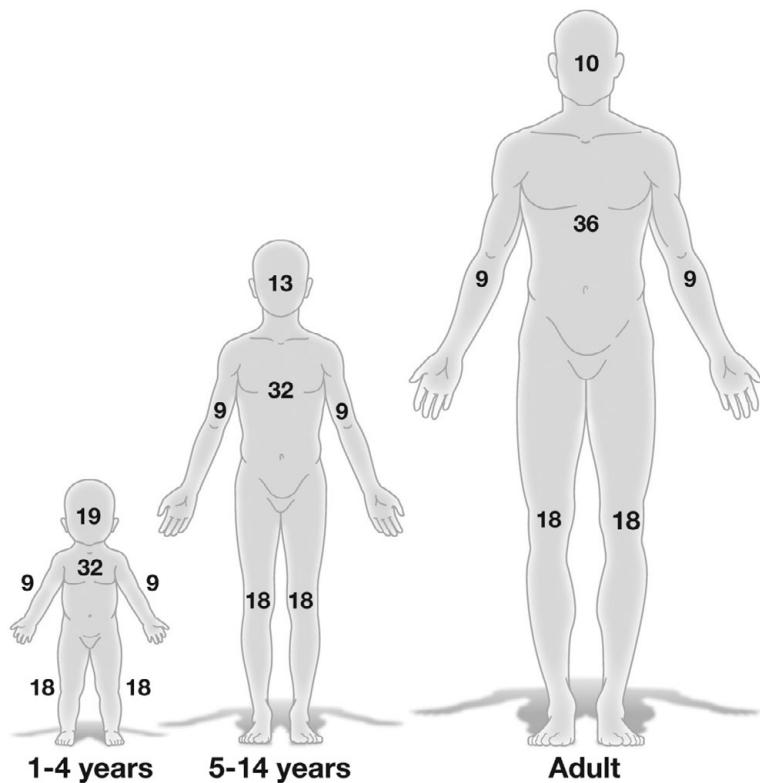


Figure 16.1 Schematic representation of body surface area (BSA) variability between children and adults. Reproduced with permission from: Litman RS, *Basics of Pediatric Anesthesia*, Philadelphia, 2014. Illustration by Rob Fedirko.

surgery, these burns heal with contractures. These burns scar severely with contractures. Surgery is required for this healing process.

- **Fourth-degree burns** are deep and potentially life-threatening burns that extend from the skin down to the fascia, muscle, and/or bone.

What Is the Estimated Percent Total Body Surface Area Burned on This Patient?

Burns can be classified according to the percentage body surface area (BSA) involved. The total percentage BSA derives from the “Rule of Nines,” which is different in children than in adults since the pediatric head accounts for a larger percentage of BSA (Figure 16.1).

A *major burn* is defined as: (1) a second-degree burn with greater than 10% BSA or second-degree burn for children over 10 years old with greater than 20% BSA; (2) a third-degree burn with greater than 5% BSA; or (3) a second- or third-degree burn of the hands, feet, perineum, or major joints, electrical or chemical burns, inhalational injury, or burns in

patients with preexisting medical conditions. The American Burn Association recommends that patients suffering from a major burn should be referred to a certified burn center.

Morbidity and mortality increase with increasing size and depth of the burn. Inhalational smoke injury and early shock are associated with greater mortality. The risk of mortality increases when the burn extends to greater than 60% BSA; yet there have been patients who have survived with a 90% burn. Severely burned patients may survive the initial insult, only to succumb to a secondary complication (e.g. infection). The age of the burned child is important for his or her survival, because of better prognosis with increasing age.

What Are Your Initial Anesthetic Concerns?

The initial anesthetic management of the pediatric burn patient should focus on airway evaluation and management, provision of adequate oxygenation and ventilation, as well as restoring circulatory volume. Burn patients will have difficulty regulating body temperature, so heat lamps and blankets should be used to

maintain euthermia. There are pharmacokinetic implications after third-degree burns because of the up-regulation of postsynaptic receptors, which can lead to acute hyperkalemia with succinylcholine administration. Wound care is usually deferred until after the acute resuscitation phase.

IV access can be difficult in cases of severe burns covering large surface areas necessitating central access. Intraosseous access can be an important tool in these patients and should be used when appropriate to avoid delay in resuscitation.

What Are the Implications for Airway Management?

Prior to tracheal intubation, the highest possible concentration of oxygen by face mask should be used. Tracheal intubation is indicated if there is evidence of inadequate ventilation, singed nasal hair, facial or upper airway edema, hoarseness, or stridor. Patients with stridor or other signs of upper airway obstruction should have tracheal intubation performed prior to the development of facial and upper airway edema, which can impede future tracheal intubation. In adults and older children, awake fiberoptic intubation is preferred if upper airway edema or injury is suspected; the extent of airway injury can be examined at the same time. Uncooperative young children will require tracheal intubation following induction of general anesthesia. Low levels of PEEP may help avoid pulmonary edema.

Respiratory failure and asphyxia may result from inhalation of toxic fumes, chemical injury from smoke, upper airway edema and, in the later stages, eschar formation on the chest wall. Inhalation of toxic chemicals can cause increased airway secretions, irritability, capillary leaking, and pulmonary edema. Clinical manifestations include hypoxia, hypercapnia, dyspnea, bronchospasm, cough, and stridor. Circumferential burns of the chest wall can cause restrictive respiratory failure for which an escharotomy may be required.

Postintubation complications may include increased secretions, bronchospasm, atelectasis, airway edema, and bronchopneumonia.

Why Are Severe Burns of the Neck and Chest Important to the Anesthesia Care Provider?

Patients with neck and chest burns are at risk for airway compromise as the eschars can become

severely restrictive. Escharotomies are performed to avoid airway compression in the case of neck burns or restriction of the thoracic cavity in the case of circumferential chest burns which may result in atelectasis, pneumonia, or respiratory insufficiency.

Escharotomies may be required in the limbs as tight eschars may cause vascular compromise and loss of distal pulses.

What Is Carbon Monoxide Toxicity?

Carbon monoxide (CO), which is a byproduct of combustion and present in smoke, is responsible for the majority of deaths from smoke inhalation. CO has a 200 times greater affinity for hemoglobin than oxygen, leading to tissue hypoxia. CO will shift the oxygen dissociation curve to the left, impairing oxygen offloading to tissues. CO also interferes with mitochondrial uncoupling of oxidative phosphorylation and reduces ATP production, causing a metabolic acidosis. Pulse oximetry will not reflect CO toxicity and it will show normal oxygen saturation. Thus, carboxyhemoglobin must be specifically measured as a component of a blood gas analysis. Clinical symptoms are directly proportional to CO levels.

Low CO levels cause mild CNS symptoms and high CO levels can cause coma. The four-hour half-life of CO can be decreased to 40 minutes with 100% oxygen therapy. Severe CO intoxication can be treated with hyperbaric oxygen therapy; however, there is little evidence that it prevents permanent neurological deficits.

What Is Cyanide Toxicity?

Cyanide toxicity is commonly seen in victims who inhale or absorb combustible synthetic materials, like burning of plastic materials with elevated nitrogen content. Cyanide binds to cytochrome oxidase and impairs tissue oxygenation by converting intracellular aerobic metabolism to anaerobic metabolism. An elevated venous oxygen level and a lactic acidosis that does not improve with oxygen administration are suggestive of cyanide toxicity. Often, the patient will present with high lactic acidosis without a major burn. Blood cyanide level greater than 0.2 mg/L is toxic (lethal at about 1 mg/mL). Cyanide toxicity is measured by using a spectrophotometric assay using methemoglobin. Children who sustain cyanide poisoning as a result of inhalational injury may succumb from asphyxiation and should be treated with sodium

thiosulfate or sodium nitrite if seizures, cardiopulmonary failure, or persistent lactic acidosis occurs.

How Will You Manage This Patient's Body Temperature?

Paradoxically, many patients seem adequately warm on presentation due to the nature of the injury. However, these patients can rapidly become hypothermic due to large exposed surface areas and lack of intact skin. Hypothermia can cause arrhythmias, coagulopathy, and decreased wound healing. Hypothermia in these patients is a major source of morbidity and mortality. Therefore, the clinician should treat the normothermic patient as if they are hypothermic.

Steps of therapy include room warming, blankets to unaffected areas (including the head in young children), warmed intravenous fluids, and underbody warmers whenever available.

How Will You Manage This Patient's Fluid Status?

During the acute phase of a burn, patients have a transiently decreased cardiac output due to depressed myocardial function, increased blood viscosity, and increased systemic vascular resistance from the release of vasoactive substances. Burn shock can occur due to hypovolemia from the translocation of intravascular volume to extravascular space. During the hypermetabolic phase, tissue and organ blood flow are increased due to increased cardiac output and decreased systemic vascular resistance.

Administration of IV fluids is indicated when the burn is >10% BSA. Several formulas have been proposed to estimate fluid requirements in children. The Parkland formula describes use of Lactated Ringer's solution, which is administered at the child's maintenance rate plus 4 mL/kg per 1% BSA burn for the initial 24 hours. At some institutions, half the total fluid is given over the first 8 hours with the other half given over the next 16 hours. Fluid requirements are greatest during the first 24 hours after injury.

Established formulae should be used as guides only; fluid administration should be based on parameters of adequate tissue perfusion. The rate of urine output should be maintained at a minimum of 0.5–1.0 mL/kg/h. Oliguria in burned patients is usually the result of inadequate fluid resuscitation and not renal insufficiency.

After the first day, fluid losses occur by evaporation through denuded skin and as a component of the burn exudate. Maximum weight gain due to edema occurs on the second or third day and is followed by a diuresis and return to normal weight by day 14. Protein loss through the burn exudate occurs until all wounds are grafted and healed. During graft harvesting, the surgeon may infuse crystalloid solution under the donor skin to facilitate harvesting. The amount infused can sometimes be large and should be accounted for in the total volume of fluids given. The use of colloids as a component of fluid resuscitation is controversial. In children, albumin administration increases the serum albumin level, but no data exist to support its routine administration.

What Are the Pharmacological Implications of Administering Anesthesia Drugs to Burn Patients?

Any IV induction agent is acceptable, although if significant hypovolemia is suspected, ketamine or etomidate may preserve blood pressure the best. The choice of neuromuscular blocker depends on the time since the burn. Following the initial 24-hour post-burn period, administration of succinylcholine can cause symptomatic hyperkalemia secondary to up-regulation of extrajunctional acetylcholine receptors on injured or burned muscle. This risk peaks between five days and three months and may persist for up to two years after the initial injury or until the patient has regained adequate muscle function. Burn patients may demonstrate resistance to the nondepolarizing neuromuscular blockers up to 1–2 months after the initial burn; thus, relatively larger doses are required to achieve relaxation, especially in patients with major burns. This resistance is thought to be due to changes in post-junctional acetylcholine receptors. Maintenance of general anesthesia can be accomplished using a balanced technique with an inhalational anesthetic agent. Burn patients have greater requirements for opioids due to both pharmacokinetic changes and development of tolerance. Because of the pharmacologic changes with burn injury, all drugs should be titrated to clinical effect and appropriate hemodynamic and oxygenation monitoring are required to detect opioid-induced respiratory depression. Large opioid needs during the intraoperative and postoperative periods are common.

How Much Blood Loss Is Expected during Burn Debridement?

Burn debridement commonly results in large blood losses because of the relatively large surface area of exposed skin from donor and graft sites. Once the burn eschar is excised and the large capillary bed is exposed, bandages soaked in epinephrine are often placed over the wound to decrease bleeding; this often manifests as tachycardia and hypertension. Consequently, since blood loss may be underestimated, early transfusion is often warranted during these procedures to avoid sudden hypovolemia.

Red blood cell transfusion is indicated when oxygen-carrying capacity is inadequate to meet cellular metabolic demands. The maximum allowable blood loss is determined using the estimated blood volume (EBV) of the child, which varies with age. There is no absolute hematocrit below which all patients require blood transfusion; most previously healthy children tolerate a hematocrit lower than 30% without adverse sequelae. In the trauma setting, the hematocrit is often unknown and estimates of EBV, blood loss, and blood needs are empiric. In life-threatening emergencies, immediate transfusion with type O blood is warranted. Administration of large amounts of blood to small children entails risks that include hyperkalemia from lysis of stored red cells, hypocalcemia from citrate toxicity, hypothermia, and coagulopathy that results from dilution of platelets and clotting factors.

What Are the Unique Considerations for Monitoring Children with Burns?

Standard monitoring can be challenging in the burned child. Blood pressure cuffs may have to be placed over a burned area, and needle electrodes may have to be used for the ECG. Pulse oximetry readings may not be reliable with extensive burn injury, low blood pressure, or hypothermia. In this case, pulse oximetry probes may be placed on the ear lobe, buccal mucosa, or tongue. Children that require major burn surgery will require invasive arterial blood pressure monitoring, and possibly central venous pressure monitoring to accurately track volume status acutely. Temperature measurement and maintenance of normothermia is essential since burned children are susceptible to hypothermia. The operating room and IV fluids should be warmed, inspired gases

should be heated and humidified, and all exposed body surfaces covered.

What Organ System Changes Occur after a Burn?

During the acute phase of injury, the glomerular filtration rate is decreased due to decreased cardiac output and increased vascular tone from circulating catecholamines, vasopressin, and activation of the renin-angiotensin-aldosterone system. The child with a major burn is at higher risk of renal failure secondary to rhabdomyolysis, myoglobinuria, and hypotension. Neurologic dysfunction can result from hypoxia, hyperthermia, electrolyte imbalance, carbon monoxide, and hypertension. Hematologic abnormalities may include anemia from red cell hemolysis and decreased red cell survival, and thrombocytopenia due to platelet aggregation at wound sites and at damaged microvasculature. A hypercoagulable state with possible DIC can develop from increasing clotting factor production. The burned child is susceptible to development of sepsis secondary to the loss of skin and intestinal mucosal barriers, and an impaired immune response. Early enteral nutrition minimizes the intestinal mucosal barrier damage and decreases endotoxemia. Liberal application of topical antibiotic ointment has decreased the mortality from infection. Children with major burns are at risk of hypothermia from evaporative losses through large areas of exposed skin. Duodenal and gastric stress ulcers may cause chronic occult bleeding; therefore, H₂-antagonists are routinely administered. Nearly all patients will develop an ileus and will require gastric decompression with a nasogastric tube.

Children with approximately 30% BSA burn or more will demonstrate a hypermetabolic response that consists of release of stress hormones, including catecholamines, antidiuretic hormone, renin, angiotensin, aldosterone, glucagon, and cortisol. This hypermetabolic state lasts until all wounds are healed (possibly many months), and generally declines during the first 9–12 months post-injury. The degree of hypermetabolism depends on the severity of the burn. The mechanisms involved in this response are complex and may include increased adrenergic output, gut-induced endotoxemia, and endogenous resetting of energy production. The hypermetabolic state manifests as increased catabolism, nitrogen wasting, hyperthermia, hyperglycemia, increased

CO_2 production, and increased oxygen utilization. Approximately eight hours after the burn, the hypothalamic-pituitary thermoregulatory set point resets to a higher than normal body temperature. During the hypermetabolic phase, high caloric nutritional support will help prevent protein breakdown and promote wound healing. Since hypermetabolism increases oxygen consumption and CO_2 production, minute ventilation should be increased, and a warm ambient temperature ($\geq 30^\circ\text{C}$) should be maintained to help prevent catabolism due to hypothermia.

How Is Pain Management Accomplished in the Burned Child?

Pain is a major problem in burned children. In the acute period, pain can be categorized as background pain, procedural pain, and postoperative pain. Background pain is proportional to the size of the burn and the mainstay of therapy is with oral or IV opioids. Because of rapidly developing tolerance, opioid doses need to be adjusted daily. Adjuvant analgesics such as acetaminophen, ketamine and clonidine should also be included. Procedural pain is controlled with regional anesthesia when possible, as well as opioids, benzodiazepines (for procedure-related anxiolysis), and ketamine.

Suggested Reading

Battle CE, Evans V, James K, et al.
Epidemiology of burns and scalds in children presenting to the emergency department of a regional burns unit: a 7-year retrospective study. *Burns Trauma*. 2016;4:19. PMID: 27574688.

Fuzaylov G, Fidkowski CW. Anesthetic considerations for major burn injury in pediatric patients. *Paediatr Anaesth*. 2009;19(3):202–11. PMID: 19187044.

Hoffman HG, Chambers GT, Meyer WJ, III, Arceneaux LL, Russell WJ, Seibel EJ, et al. Virtual reality as an

adjunctive non-pharmacologic analgesic for acute burn pain during medical procedures. *Ann Behav Med*. 2011;41(2):183–91. PMID: 21264690.

What Are the Analgesic and Sedative Options for Postoperative Wound Care?

The adequate provision of anxiolysis and analgesia for daily wound care can be very challenging for providers, patients, and families. Opioid-based techniques are most commonly used, and the route of administration depends on the availability of IV access and the expected number of dressing changes over time, as well as the child's level of anxiety. Inhaled nitrous oxide is often effective for mildly painful procedures. More painful procedures may be amenable to oral or IV ketamine, in conjunction with a benzodiazepine and a topical local anesthetic. Epidural analgesia is useful for repeated wound care of the perineum or lower extremities. In some cases, administration of general anesthesia (by facemask or supraglottic device) may be required. Non-pharmacologic techniques such as hypnosis and distraction therapy have proven very useful in some children. Child-life specialists should be intimately involved with the patient and their family. Using the same medical and nursing personnel in a familiar environment with expectant management for repeated wound care lessens patient and family stress and improves provider conditions.