**Title:** Pediatric Burns and Inhalation Injury - Anesthetic Management and Analgesia for Procedural Sedation

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**Learning Objectives:**
At the end of this educational session, the participants will be able to

1. List the pathophysiologic changes in pediatric burn victims

2. Discuss the anesthetic implications of pediatric burn surgery with special emphasis on airway management, analgesia, fluid and volume management and, temperature control

3. Debate the anesthetic management for procedural sedation in these patients.

**Stem Case and Key Questions:**
A 2 year old, 15 kg toddler is scheduled for excision and grafting of burns. She was the victim of an accidental house-fire 5 days ago. She suffered second and third degree burns to 52% of her total body surface area (TBSA) involving most of the anterior chest, abdomen, both arms, and parts of her face and lower extremities. She also suffered smoke inhalation injury. She was responsive, but drowsy at the scene of the accident. She was brought to the hospital where she was intubated and mechanically ventilated in view of her smoke inhalation injury. Her initial Carboxyhemoglobin level was 18%. She was extubated 72 hours later.

She was appropriately volume resuscitated at admission, initially via an intraosseus access, and a double lumen CVP line was later placed in her left femoral vein. High calorie enteral feeds via a naso-jejunal tube were initiated on the day of admission. She was on a continuous infusion of morphine and round the clock acetaminophen.

Her post-extubation hematocrit was 22% and carboxyhemoglobin levels were down to 1%. Serum albumin was 25 gms%. The rest of the labs were unremarkable. Her femoral line fell out yesterday evening and multiple attempts to secure an IV were unsuccessful. She was switched to oral morphine. At the surgeons orders, tube feeds were continued till 6AM on the day of surgery.

She is scheduled to have excision and grafting to all the burn areas at 8 AM. You see the patient in the preop waiting area, with her mother at 7. 15 AM. The patient is crying and difficult to calm down. She has had her dose of oral morphine at 5 AM. The child was previously healthy, has no known allergies. She has never needed any medicines in the past. When she cries, she opens her mouth to about >3 cm. She does not allow you to auscultate her chest, but the cry is loud enough to suggest good air entry. SpO2 on 1 lpm oxygen by nasal canulae is 94% and HR is 148. Her tympanic temperature is 38° C.

**Key Questions:**

1. What are the pathophysiologic alterations induced by burns? How is temperature regulation affected? How does burn affect the metabolic and caloric requirements? How do we handle the issue of continuous enteral feeds in the perioperative period?

2. What additional preoperative information would you like to have before proceeding with the anesthetic? How would you like to premedicate this child?

The child is premedicated with oral midazolam and intranasal Fentanyl. Mother heaves a sigh of relief and thanks you for calming her down. She has a dry cough and auscultation of her lungs reveals a few crackles. SpO2 now reads 91% and HR is 136.
Key Questions:

3. Do you want to proceed with the anesthetic? What are your concerns (if there are any)?

4. How would you like to induce and maintain anesthesia? What vascular access and monitoring is appropriate for this child?

You proceed with an inhalational induction with sevoflurane in oxygen. IV access is obtained with a 16 G angiocath in the left femoral vein. The child is then intubated with an uncuffed 4.5 endotracheal tube. Bilateral breath sounds and end-tidal CO₂ is confirmed. A 22 G arterial line is sited in the left posterior tibial artery. A sample is sent for blood gas estimation, electrolytes, hematocrit, and crossmatch. The surgical team opens the dressings, inspects the wounds and decides to harvest the split thickness skin graft from the back and the scalp. They would like to use Pitkin solution with bupivacaine and epinephrine added to the solution. The initial ABG shows pH 7.35, pCO₂ 48 mm Hg, pO₂ 345 mm Hg, Na⁺ 137 meq/L, K⁺ 4.5 meq/L, Hct 21%, lactate 1.1 mmol/L.

Key Questions:

5. What blood products do you order? What is your transfusion trigger for this child? Would you like any additional information before you proceed?

6. What intraoperative analgesia would be optimal for this child?

7. How would you manage the fluid balance? Is your choice of IV access appropriate?

8. What is Pitkin solution? What are the implications of using Pitkin solution? How safe and effective is tumescent anesthesia for this procedure? What is the allowable concentration and dose of local anesthetics and epinephrine?

The patient is turned prone. Increments of morphine are titrated to HR and BP. Split thickness skin grafts are harvested from her back. The donor areas are covered with epinephrine soaked gauze. There is some bleeding, but it is impossible to quantify the blood loss. As the surgeons are preparing the scalp for harvesting, Spo₂ drops to 87% suddenly. The patient is on 100% oxygen. End tidal CO₂ is reading 18. You start hand ventilation and confirm bilateral air entry. Spo₂ rises to 97% and you allow the surgeons to proceed. Just as they begin injecting Pitkin solution into the scalp, Spo₂ drops to 85% and now it is difficult to ventilate the patient. End tidal CO₂ is still reading 20.

Key Questions:

9. What is the differential diagnosis? How do you approach this problem?

The surgery is stopped. Suctioning the tracheal tube does not reveal anything. Hand ventilation is now impossible. Patient is turned supine and the tracheal tube changed with improvement in oxygenation and ventilation. The patient is returned to prone position and the graft harvested from the scalp. The patients vital now read HR 168, BP 78/42, Spo₂ 99%, temp 35.8°C, Hct is 18%.

The surgeons are now ready to excise the burn areas.

Key Questions:

10. What is the normothermic level in this patient? How would you achieve it?

11. What is the anticipated blood loss for burn excision surgery? How would you monitor the blood loss in this patient?
Excision and grafting is completed over the next 3 hours. She received 2 adult units of PRBCs and the hematocrit is 20 at the end of surgery. Her vitals read HR 155, BP 70/34, SpO2 98%, temp 35.2°C; urine output is 60 cc over the 5 hour procedure.

**Key Questions:**

12. What criteria would you use to extubate this patient?

13. What postoperative analgesia would you use and why?

The patient is scheduled for removal of staples and change of dressings 5 days later in the procedure room on the burn floor. Since you are familiar with the patient, you are scheduled to provide sedation for the procedure.

**Key Questions:**

14. What are your choices for procedural sedation? What are the anesthetic implications of such procedures?

**Model Discussion:**

**Epidemiology:** Burn related injuries contribute to more than 1 million emergency room visits, 50,000 acute admissions and 4000 deaths in the US annually. Nearly one third of burn admissions and burn deaths occur in children. Burns are second only to motor vehicle crashes as the leading cause of death in children older than 1 year. Flame burns account for about a third of pediatric burns, are often more severe, and frequently involve concomitant inhalation injury.

**Pathophysiology of burns:** The skin is the largest organ of the body and although burns directly affect the skin, large burns (>20% TBSA) alter the physiologic function of virtually all other body organs, as well as create increased risk of infection and death directly related in magnitude to burn size. The primary determinants of severity of burn injury are the size and depth of burn area. However, patient age, body part burned, pre-existing disease, and associated non-burn injuries have an important impact on the outcome. Children younger than 2 years of age have high surface area to body mass ratios, extremely thin skin, and minimal physiologic reserves, causing higher morbidities and mortalities than in the older age groups. Severe burn injury results in vasoactive mediator release throughout the body to increase capillary permeability and third spacing of fluid in tissues both surrounding the burn, and distant from the burn (larger burn sizes). Damaged skin is no longer able to retain heat and H2O2; consequently large evaporative losses ensue. Combination of these mechanisms results in hypovolemic shock acutely following burn injury.

**Metabolic changes:** Metabolic complications are directly related to extent of burn. The core temperature in these patients is reset to 38.5°C. Thermal maintenance is critical in young children, especially those with burns of more than 10% TBSA. Body temperature is best maintained by a thermoneutral environment (room temperature of 28 -32°C) with the additional use of an overbed warming shield and warming of IV fluids. Dry-air warmers used directly over the burn wound can cause tissue desiccation. Also, forced air warming devices are less effective in these patients because of the significant area of burned and donor skin sites that must remain exposed.

Thermal injury leads to hypermetabolism and protein hypercatabolic state. Blunting of immune response, with consequent sepsis can exaggerate these metabolic demands. Adequate pain control, alleviation of anxiety, a thermoneutral environment, and treatment of infection are important steps in limiting catecholamine secretion and thus hypermetabolism. Early postpyloric enteral feeding, which can be continued in the perioperative period, is recommended by the evidence based guidelines group of the American Burn Association (ABA). Early enteral feeding decreases infections and sepsis, improves wound healing and nitrogen balance and, reduces stress ulceration and duration of hospitalization. The safety and advantages of perioperative enteral feeds have been reported by Jenkins et al in 1994. Early excision and grafting of the wounds help to reduce the metabolic requirements.
**Fluid resuscitation in major pediatric burns:** The disproportionate ratio of head to body size makes the rule of 9s (to estimate TBSA) not applicable in small children. Lund-Browder or Berkow burn chart divides TBSA into smaller units and makes age-appropriate corrections. A copy of the Berkow burn chart (which estimates the head area to contribute to 19% TBSA at 1 year, 17% TBSA at 2-4 years, 13% TBSA at 5-9 years, 11% TBSA at 9-14 years etc) will be distributed to the attendees at the PBLD session.

The widely quoted Baxter (Parkland) formula for fluid resuscitation of burn victims is 4 mL Ringers lactate \([RL]/kg\) body weight/percent TBSA burned; one half to be given during the first 8 hours after injury and the rest in the next 16 hours. Hypertonic saline may be useful in early shock and colloids are most effective when used in the 12-24 hour period of resuscitation. It is widely believed that the Parkland formula underestimates resuscitation volumes, particularly when concomitant smoke inhalation is present. Allowances should be made for daily maintenance fluids in infants and toddlers. Adequate resuscitation is reflected by normal mentation, stable vital signs, and a urine output of 1-2 mL/kg/hr. However, several recent studies have shown advantages to invasive hemodynamic monitoring (PA catheter) in adults with serious burns who do not respond as expected to fluid resuscitation. Blood sugar should be monitored, and glucose-containing solutions added as necessary, in infants.

Aggressive and overzealous fluid resuscitation can result in complications such as pulmonary edema, ARDS, abdominal and extremity compartment syndromes, etc (Phenomenon described as fluid creep in burn literature). Increased intra-abdominal pressure can lead to decreased splanchnic blood flow, decreased renal perfusion, difficulty in ventilation and diminished venous return (hence cardiac output). Bladder pressure measurement and monitoring is used to diagnose increasing intra-abdominal pressures. Bladder pressures in the 12-20 mm Hg may be considered intra-abdominal hypertension while sustained pressures over 20 mm Hg with evidence of organ dysfunction or failure suggest abdominal compartment syndrome, which needs immediate treatment with abdominal decompression!

**Inhalation injury:** Most airway inhalation injuries are due to inhalation of smoke. The severity of inhalation injury depends on the fuels burned, intensity of combustion, duration of exposure, and confinement. Unless steam is involved, heat injury to the airway is supraglottic, causing swelling of the posterior pharynx and supraglottic regions, leading to potential upper airway obstruction. The natural history of upper airway inhalation injury is edema formation that narrows the airway over the initial 12 - 24 h. Intubation is recommended in patients who present with stridor, wheeze, or voice changes. Burns to the face and neck can result in tight eschar formation that combined with pharyngeal edema, can cause difficult airway management.

Lower airway or pulmonary parenchymal damage results from inhalation of the chemical constituents of smoke, usually becoming apparent 24 to 48 hours after the injury. Findings include dyspnea, rales, rhonchi, and wheezing. The clinical picture is identical to that of acute respiratory distress syndrome and is caused by chemical irritation of the terminal bronchiolar tree. Bronchoscopy reveals carbonaceous endobronchial debris and/ or mucosal ulceration.

Meticulous pulmonary toilet is the cornerstone of early care. Tracheal secretions are often very viscous. The small internal diameter of pediatric endotracheal tubes increases the risk of tube obstruction by these secretions.

**Carbon monoxide and cyanide poisoning:** Gas phase constituents of smoke include carbon monoxide (CO), cyanide, hydrochloric acid, aldehyde gases, and oxidants. These can cause direct damage to mucociliary function and bronchial vessel permeability, as well as produce alveolar destruction and pulmonary edema. CO has a high affinity for hemoglobin (250 times more than oxygen), and can interfere with oxygen delivery to the tissues at higher concentrations. Administration of 100% oxygen reduces the half-life of carboxyhemoglobin from 2.5 hours to 40 minutes and facilitates the elimination of CO. Hyperbaric oxygen therapy has limited indications due to the logistical challenges presented by transport of patient with concomitant burns to such chambers. Cyanide causes tissue hypoxia by uncoupling oxidative
phosphorylation in mitochondria. Treatment should be considered for cyanide poisoning in patients with unexplained severe metabolic acidosis associated with elevated central venous O\textsubscript{2} (therefore patients are clinically not cyanotic), normal arterial O\textsubscript{2} content and low carboxyhemoglobin.

**Clinical course of cutaneous burns:** First-degree burns affect only the epidermis and are characterized by erythema and edema of the burned areas without blistering or desquamation. Second-degree or partial thickness burns involve the epidermis and a portion of the dermis. In most cases, these wounds can be expected to spontaneously heal in 7 to 28 days, although surgical treatment may be necessary for extensive or deep second-degree burns. Pain is characteristic of partial thickness burns. Third-degree or full-thickness burns extend entirely through both the epidermis and dermis and will not heal spontaneously.

**Excision and grafting surgery:** Excision and grafting involves tangential excision of the burn wound, in which the eschar is shaved off from the burn until a plane of viable tissue is reached, followed by covering the excised wound with a split thickness skin graft. Excision of third-degree burns requires fascial excision, where the overlying burned skin and subcutaneous fat are excised down to muscle fascia.

**Fasting requirements:** Metabolic complications in burn patients are directly related to extent of burn. Thermal injury leads to hypermetabolism and protein hypercatabolic state. Early post-pyloric enteral feeding, which can be continued in the perioperative period, is recommended by the evidence based guidelines of the American Burn Association (ABA). Early enteral feeding in these patients decreases infections and sepsis, improves wound healing and nitrogen balance and, reduces stress ulceration and duration of hospitalization. Gastric emptying may not be delayed in burn patients and gastric acid production may actually be reduced in the early postburn period. The safety and advantages of perioperative enteral feeds have been reported by Jenkins et al. At our institution, we continue enteral feeds throughout the perioperative period in patients who come to the operating room intubated. In nonintubated patients, shorter fasting times, typically 2-4 hours may be acceptable.

**Airway management:** Airway management in pediatric burn patients can be challenging. Mask ventilation may be a problem with facial burns. Depending on the age of the burns, edema, scarring or contractures may narrow the mouth opening and limit the neck movements. Location of burns and donor skin sites indicate the need for special positioning, for repositioning the patient during operation, or both. Fixing the endotracheal tube for prone positioning in the presence of facial burns is best achieved by wiring it to the teeth or stitching it to the nares. We commonly use dental floss to tie the tube to the teeth (if present) or tie the tube to a oro-nasal loop of rubber catheter.

In critically ill children requiring high inspiratory pressures during mechanical ventilation, a cuffed endotracheal tube may be a better choice. Frequent suctioning helps in clearing the mucus and debris from the tracheal tree and a high index of suspicion should be maintained for plugging of the tracheal tube. A combination of prolonged prone positioning and relatively high fluid volume administration may cause significant airway swelling. It is best to wait until an air leak is present around the endotracheal tube before tracheal extubation, because this indicates resolution of edema. If there is still no air leak and the patient is deemed ready for tracheal extubation, direct laryngoscopy may be necessary to determine the extent of residual edema. Once extubated, the patient should be closely monitored for progressive airway obstruction during the subsequent 24-48 h.

**Blood loss:** Burn excision can result in massive and sudden blood loss. Quantifying blood loss is typically difficult in pediatric patients and transfusion is best guided by serial hematocrit estimations. Adequate venous access is a prerequisite to burn excision and grafting procedures. At least two intravenous access routes should be established (peripheral or central) and blood products should be available in the room before excision begins. Housinger et al reported a mean blood loss of 2.8% of a patients blood volume for each %TBSA excised.

Intraoperative tourniquet use on burned extremities reduces overall blood loss. Pitkin solution is subcutaneous crystalloid injected in generous amounts to facilitate donor skin harvesting and reduce blood
loss. Post-excision compression dressings and topical epinephrine have been used to reduce blood loss during excision and grafting procedures. Application of bandages soaked in 1:10,000 epinephrine after excision of burned skin is effective in producing a bloodless surface for placement of skin grafts. Although extremely high levels of catecholamines in the blood have been measured with this technique, complications such as dysrhythmias are uncommon. We prefer to keep the hematocrit between 20 and 25% to help with high metabolic demands for oxygen. If blood loss is excessive, it is prudent to rule out coagulation abnormalities. Platelets or coagulation factors may need to be replaced.

**Anesthetic technique:** General anesthesia with the combination of an opioid, muscle relaxant, and a volatile agent is the most widely used technique for burn excision and grafting. Tumescent local anesthesia with maximum dose of 7 mg/kg lidocaine has been shown to be safe and the sole possible effective locoregional anesthesia technique for the surgical treatment of pediatric burns.

Succinylcholine administration to patients >24 h after burn injury is unsafe, due to the risk of hyperkalemic ventricular dysrythmias. By the same mechanism, patients with thermal injury are resistant to the action of nondepolarizing muscle relaxants. This is because the burn injury causes acetylcholine receptors in muscle to proliferate under the burn and at sites distant from the burn injury.

**Regional Anesthesia:** Regional anesthesia alone, or in combination with general anesthesia can be used in patients with small burns or for reconstructive procedures. For procedures on lower extremities, lumbar epidural or caudal catheters can be used to provide intra- and post-operative analgesia. The greatest limitation to the use of regional techniques is the extent of surgical field; most patients with major burns have a wide distribution of injuries and/or need skin harvesting from areas too large to be blocked by a regional technique. Presence of a coagulopathy or, systemic or local infection may also contraindicate regional anesthetic techniques in these patients.

**Temperature homeostasis:** Hypothermia is a common complication of excision and grafting and often delays extubation. Body temperature is best maintained by a thermoneutral environment (operating room temperature of 28 -32°C) with the additional use of an overbed warming shield and warming of IV fluids. Dry-air warmers used directly over the burn wound can cause tissue desiccation. Forced air warming devices are less effective in these patients because of the significant area of burned and donor skin sites that must remain exposed. Use of “space blankets” (aluminum foil coverings on nonexposed areas), plastic sheets over the head and face, heat and moisture exchangers (HME) in the breathing system and low fresh gas flow with circle absorber can also help to reduce the heat loss.

**Analgesia:** Severe pain is an inevitable consequence of a major burn injury and perioperative analgesic requirements are frequently underestimated. Anxiety and depression are common components in a major burn and can further decrease the pain threshold. Pain management should be based on an understanding of the types of burn pain (acute, or procedure-related pain versus background, or baseline pain), frequent patient assessment by an acute pain service team, and the development of protocols to address problems such as breakthrough pain. High-dose opioids are needed to manage pain associated with burn procedures, and morphine is currently the most widely used drug. There is an interindividual variation in response to morphine, so titration to effectand frequent reassessment are important. Furthermore, most burned patients rapidly develop tolerance to opioids.

**Infection Prevention/control:** Loss of barrier function of skin and blunting of immune response result in increased susceptibility to infection and bacterial overgrowth within the eschar. Sepsis is a leading cause of death in patients who survive the acute burn injury. Cutaneous burn toxin, a toxic lipid protein isolated from burned skin, is 1000 times more immunosuppressive than endotoxins. Bowel permeability is increased in burn patients, leading to translocation of bacteria and absorption of endotoxins into the bloodstream. Burn wound infection, intravenous catheter associated septicaemia and ventilator-associated pneumonia are particularly common in burned children. Pyrexia, tachycardia, leukocytosis are almost universal in burn patients and cannot be considered signs of sepsis and blood cultures may be negative in up to half of septic patients. Hypotension, lactic acidosis, intolerance of enteral feeds in the non-acute phase favour the
diagnosis of infection. Quantitative cultures of wound biopsy help in identifying the offending pathogens and appropriately treating the infection. Systemic antibiotics should be reserved for treatment of proven infection and in the perioperative period.

**Procedural sedation:** Procedures such as dressing changes and wound care frequently require sedation and analgesia in pediatric burn patients. These procedures are often performed on a daily basis on the burn ward, making anesthesiologist involvement impractical. Nurse administered opioids (IV, oral or transmucosal) alone or in combination with benzodiazepine anxiolysis is the typical regimen. However, when wound care procedures are extensive, particularly in children, more potent anesthetic agents may be of benefit. Ketamine offers the advantage of stable hemodynamics and analgesia and has been used extensively as the primary agent for both general anesthesia and analgesia for burn dressing changes. Nitrous oxide with oxygen has been used effectively for analgesia during burn wound dressing changes. However, scavenging of the gas when administered outside of an operating room is problematic.

Combination of nitrous oxide with opioids may induce a state of general anesthesia with profound respiratory depression. The efficacy of general anesthesia administered by an anesthesiologist for procedures on a burn intensive care unit has been well documented. One of the commonly used anesthetic techniques at our institution involves the use of a mixture of alfentanil and propofol administered as an infusion. The details will be discussed at the PBLD session. The use of alpha-2 agonist, dexmedetomidine, administered as an infusion, is also gaining popularity.

For background analgesia, analgesics such as acetaminophen can be used for their opioid-sparing effect, and are combined with generous administration of oral opioids. Nonsteroidal antiinflammatory drugs have antiplatelet effects and may not be appropriate for patients who require extensive excision and grafting procedures. In addition, burn patients can also manifest the nephrotoxic effects of nonsteroidal anti-inflammatory drugs. Opioids and benzodiazepines can be used successfully together if patients are anxious. Patient monitoring must be appropriate to the level of sedation, as required by the JCAHO and described by the ASA guidelines for sedation monitoring.

**References:**
4. Practice guidelines for burn care. Published by the American Burn Association, J Burn Care Rehabil, 2001: 22:S1-S69