Anesthesia and Analgesia for the Child with Major Thermal Injury and Smoke Inhalation

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Burn related injuries account for over 1 million emergency room visits, 50,000 acute admissions and 4000 deaths in the US annually. Children account for approximately a third of burn admissions and burn deaths. 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 and frequently involve inhalation injury. Although burns directly affect the skin, large burns alter the physiologic function of almost all other body organs and create increased risk of infection and death directly related in magnitude to burn size.

Pathophysiology of thermal injury and smoke inhalation

The primary determinants of severity of burn injury are the size and depth of burn area. However, patient age, the body part burned, presence of pre-existing disease, and associated non-burn injuries also have an important impact on the outcome. Children, especially those younger than 2 years of age, have a high surface area to body weight ratio, very thin skin, and minimal physiologic reserves causing higher morbidities and mortalities than in the older age groups.

Classification of burn depth: First-degree burns affect only the epidermis and are characterized by erythema and edema of the burned areas. 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.

Cardiovascular Changes: 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. Damaged skin is no longer able to retain heat and H₂O; consequently large evaporative losses ensue. Combination of these mechanisms results in hypovolemic shock acutely following burn injury. Increased capillary permeability is seen in the burned tissue for > 72 hours and in the nonburned tissue for up to 24 h. Tumor necrosis factor, oxygen free radicals and, endothelin-1 exert a negative inotropic effect and reduce the cardiac output acutely. The cardiovascular response to both endogenous and exogenous catecholamines is attenuated due to decreased adrenergic receptor affinity and decreased production of second messenger. Systemic vascular resistance increases in the initial post burn period. Following successful resuscitation, in the hypermetabolic phase, the cardiovascular response is an increased cardiac output and reduced systemic vascular resistance.

Metabolic complications: Metabolic complications are directly related to extent of burn. Thermal injury leads to hypermetabolism and protein hypercatabolic state. Up to a 10-fold increase in circulating levels of catecholamines has been demonstrated following severe burn injury. These, along with wound released mediators, hormones and bacterial products from the gut and wound result in a systemic inflammatory
response syndrome (SIRS), manifested as hyperdynamic circulation and large increases in basal energy expenditure \((\text{hypermetabolic response})\). The secretion of glucagon and cortisol are increased and together with postinjury insulin resistance, result in the use of amino acids to fuel production, with consequent muscle wasting and nitrogen imbalance. The supraphysiologic thermogenesis is associated with resetting of the core temperature to higher levels, proportional to the size of the burns. Concomitant sepsis can greatly exaggerate these metabolic demands.

**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.\(^1\) 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 sepsis 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 favor the diagnosis of infection.

**Renal Function:** The incidence of acute renal failure in burn patients ranges from 0.5% to 38%, depending on the severity of burns. In the early postburn period, the renal blood flow is reduced as a result of hypovolemia and decreased cardiac output. In addition, increased levels of catecholamines, angiotensin, vasopressin, and aldosterone contribute to renal vasoconstriction. Myoglobinuria and sepsis can also aggravate renal dysfunction.

**Hematologic changes:** Hematologic and coagulation factor changes following burn injury depend on the magnitude of burn injury and time from injury. Hematocrit is typically maintained early in the postburn period, but drops during the weeks of care and erythrocyte half-life is reduced. Platelet count diminishes as a result of formation of microaggregates in the skin and smoke-damaged lung, though this is rarely a clinical problem. Both the thrombotic and fibrinolytic mechanisms are activated after major burns.

**Pharmacologic changes:** Burn injury also affects the pharmacodynamic and pharmacokinetic properties of many drugs. Decreased levels of serum albumin in these patients leads to increased free fraction of acidic drugs such as thiopentone or diazepam, while increased levels of \(\alpha\) acid glycoprotein result in decreased free fraction of basic drugs (with \(\text{pKa}>8\)) such as lidocaine or propranolol. Renal and hepatic functions may be impaired in patients with large burns and this may impair the elimination of some drugs. Pharmacokinetics of morphine are unchanged following burn injury. The response to muscle relaxants is altered and is discussed later under perioperative management.

**Inhalation Injury:** Most injuries occur from inhalation of smoke although rarely superheated air or steam produces direct thermal injury. Unless steam is involved, direct heat injury to airway is supraglottic. The pharyngeal mucosal lining acts as a heat reservoir and the vocal cords close reflexly in response to sudden exposure to hot air, thus limiting the physical effects of heat to the upper airway. The natural history of upper airway inhalation injury is edema that narrows the airway over the initial 12 –24 h. Presence of stridor, wheeze, or voice changes is indicative of airway swelling and compromise. Circumferential burns to the neck can result in tight eschar formation that combined with inhalation injury-induced pharyngeal edema can exacerbate upper airway compromise.
Smoke inhalation is a combination of direct pulmonary injury and systemic and metabolic toxicity. The severity of smoke lung injury depends on fuels, intensity, duration, and confinement. Gas phase constituents of smoke include carbon monoxide (CO), cyanide, acid and aldehyde gases and, oxidants. These can cause direct damage to muco-ciliary function, bronchial vessel permeability, alveolar destruction and secondary edema. Smoke exposure causes inactivation of surfactant and immediate atelectasis. Bronchial blood flow increases manifold and lung macrophages and neutrophils are activated. The ensuing release of inflammatory mediators, oxygen derived free radicals, nitric oxide causes a large increase in the vascular permeability of the pulmonary circulation. The resultant airway edema, when combined with sloughing of necrotic epithelial mucosa and thick, viscid secretions, produces airway obstruction at various levels of the bronchial tree. Concomitant cutaneous burn injury aggravates the lung damage by releasing pro-inflammatory mediators and causing hydrostatic pulmonary edema. The end result is a mismatched V/Q ratio and hypoxemia. Mechanical ventilation can cause or worsen lung damage (Ventilator Induced Lung Injury, VILI).

Carbon monoxide (CO) poisoning is a leading cause of death in major burns with inhalation injury. CO has a 250-fold affinity for haemoglobin (Hb) as compared to O₂. CO displaces O₂ from Hb and also shifts the O₂-Hb dissociation curve to the left resulting in impairment of delivery of O₂ to tissues. CO also inhibits cytochrome oxidase a3 complex at the tissue level and thus interferes with aerobic cellular metabolism. Binding of CO to cardiac and skeletal muscles results in direct toxicity and impaired function while central nervous demyelination can occur by a poorly understood mechanism. CO poisoning is diagnosed by co-oximetric estimations of COHb and HbO₂. Pulse oximetry is unreliable in the presence of carbon monoxide poisoning, as it can not detect COHb, and a falsely elevated oxygen saturation reading will be obtained. Treatment of CO poisoning relies on administration of oxygen. 100% oxygen administration results in 5-fold reduction of half life of COHb from 2.5 hr to approximately 40 min. Hyperbaric oxygen therapy is fraught with practical difficulties of transporting critically ill patients into hyperbaric chambers.

**Fluid Resuscitation of child with major burns:** Children older than 2 years with more than 20% BSA burns and all younger children regardless of burn size require intravenous fluids for optimal management. The objective of resuscitation is to replace fluid losses and restore euvoolemia with the minimal amount of fluid required to maintain organ function. The fluid requirements may be calculated using several different formulae, all of which achieve good results. There is no conclusive evidence re: what kind of fluid; when (how fast) to give; and how much to give. Early fluid resuscitation (within 2 hours of burn injury) helps in limiting the morbidity and mortality in pediatric burn victims. The Parkland (Baxter) formula provides a simple, easily remembered basis for resuscitation (4 mL Ringer's lactate [RL]/kg/percent BSA burned; one half to be given during the first 8 hours after injury and the rest in the next 16 hours).

The fluid volume determined by a burn formula is only the starting point of resuscitation. Exact volumes are individually adjusted based on clinical response. It is widely believed that the Parkland formula tends to underestimate resuscitation volumes, especially in children. Studies using invasive monitoring in adults have reported use of significantly higher volumes (4.5 – 9.2 ml/kg/TBSA/24h) for adequate resuscitation, particularly in the presence of concomitant inhalation injury. Goals of resuscitation include stable hemodynamics, urine output > 1ml/kg/hr, and normal mentation. Serial lactate levels and base deficit can also be used to guide the adequacy of fluids. Isotonic fluids are currently preferred for resuscitation of burn victims. Dextrose containing fluids and free water (in the form of 0.25 normal saline for maintenance fluid) may be needed in infants under 6 months of age.
**Inhalation Injury and Airway Management:** Inhalation injury is best divided into upper airway obstruction and lower airway injury.

*Upper airway injury* is usually an indirect heat injury causing swelling of the posterior pharynx and supraglottic regions, leading to upper airway obstruction. It is typically seen in patients with burns of the face and neck. The onset of acute obstruction may be sudden without any warning signs or symptoms. Tracheal intubation serves both as prophylaxis and therapy and should be performed when there is a suspicion of airway injury. Early tracheostomy is rarely required and when performed in children is safe, effective and *not* associated with increased risk of infection.\(^4\) Translaryngeal intubation for periods over 10 days may be associated with increased incidence of subglottic stenosis.\(^5\) Steroids *do not* help in preventing the development of edema associated with burn injuries but may increase the risk of infection.

*Lower airway or pulmonary parenchymal damage* usually manifests 12 to 48 hours after the injury. The clinical picture and radiographic findings are identical to that of acute respiratory distress syndrome. 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 obstruction by secretions. Use of as large an endotracheal tube as possible allows easy suctioning and fiberoptic bronchoscopy. Most large pediatric burn centers in North America frequently use cuffed endotracheal tubes for ventilating burned children in ICU.

**Perioperative management of pediatric burn patients**

**Preoperative Preparation** – The preoperative evaluation of burn patients should take into account the continuum of pathophysiologic changes due to burns. Patient age, % body surface area burned, depth of burns, time after injury, sites and extent of planned excision and donor areas, presence of infection, other injuries (especially inhalation injury), presence and extent of co-morbidities should all be assessed.

Careful assessment of airway should be made using the usual bedside tests. Mallampati class, thyromental distance, head, neck and jaw mobility, presence of facial or airway burns (or edema), contractures of face and neck should be looked for and used to plan the perioperative airway management technique. When there is potential for airway complications, a difficult airway cart containing a range of various size endotracheal tubes, Eschmann stylet, laryngeal mask airways (LMA), Fastrach LMA, fiberoptic bronchoscope, fiberoptic stylets should be available.

Warm OR (ambient temp of 80-90 F), warm IV fluids, warm irrigation fluids and Pitkin solution, overbed radiant warmers, blood products should all be ready prior to patient arrival in the OR.

**Fasting requirements:** 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).\(^6\) 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.\(^7\) 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.

**Types of Surgical Procedures** - Burn patients could present for five types of surgical procedures:
1. decompression procedures such as escharotomy or laparotomy,
2. excision and biologic closure of burn wounds,
3. definitive closure procedures,
4. burn reconstructive procedures, or
5. general supportive procedures like gastostomy or line placement.

**Excision and Grafting:** The need and timing for excision and grafting surgery is determined primarily by the size of injury. The objective is to identify, excise and achieve biologic closure of all full thickness burns. Extensive burns may need staged excision to limit the physiologic insult of one massive surgery and to allow autologous skin grafts to be available. Excision and grafting involves ‘tangential excision’ of the second-degree 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 or allogeneic skin from cadavers or skin substitutes such as Integra. Excision of third-degree burns requires ‘fascial excision’, where the overlying burned skin and subcutaneous fat are excised down to muscle fascia.

**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. Succinylcholine administration to patients >24 h after burn injury is unsafe, due to the risk of hyperkalemic ventricular dysrhythmias. Patients who have been bedridden because of severity of illness or concomitant disease or injury, or those receiving prolonged muscle relaxant therapy to facilitate mechanical ventilation may be particularly vulnerable. The exact period of risk is unknown, but a duration of 6 months can be considered the absolute minimum. This is because the burn injury causes proliferation and spread of acetylcholine receptors (AChR) throughout the skeletal muscle membrane under the burn and at sites distant from the burn injury. The upregulation of acetylcholine receptors, along with altered protein binding, especially to \( \alpha_1 \)-glycoprotein, makes patients with thermal injury resistant to the action of nondepolarizing muscle relaxants (NDMR). In these patients, larger doses of NDMR may be required to achieve a given degree of neuromuscular blockade, the onset of paralysis may take longer and the duration of paralysis may be shorter. The resistance is usually seen in patients with greater than 30% TBSA burns, develops after the 1\(^{st}\) week of injury and peaks at 5-6 weeks postinjury. Mivacurium may be immune to this resistance, possibly as a result of decreased metabolism of the drug from depressed pseudocholinesterase activity in burn patients.

**Airway management:** Airway management in burn patients can be challenging. Mask ventilation may be a problem with facial burns. Successful use of laryngeal mask airway (LMA) for burn surgery has been reported. However, major procedures in critically ill patients, with frequent intraoperative changes in patient position are best done with endotracheal intubation. Awake fiberoptic intubation may be indicated if difficulties for intubation and/or ventilation are identified preoperatively. Inhalation induction, maintenance of spontaneous respirations, and intubation with fiberoptic guidance or Fastrach laryngeal mask airway may be advocated in uncooperative patients.

Location of burns and donor skin sites indicate the need for special positioning, for repositioning the patient during operation, or both. Adequate fixation of the endotracheal tube is essential. Unfortunately, facial burns can make fixation of the tube extremely difficult. Close proximity of topical burn wound agents and fluid exuding through a facial burn often cause failure of the usual methods of fixation. Umbilical tape secured to the tube and passed around the head provides rapid fixation, as does wiring of the tube to firmly intact primary or secondary teeth, or stitching it to the nares. We commonly use dental floss to tie the tube to the teeth or tie the tube to a oro-nasal or nasal septal loop of rubber catheter. 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, especially in older children. 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.

Depending on the age of the burns, edema, scarring or contractures may narrow the mouth opening and limit the neck movements. Surgical release of neck contractures to facilitate intubation has been described in both elective and emergency settings.

**Monitoring:** Various problems can be encountered in perioperative monitoring of a child with burns. Standard electrocardiogram electrodes may not adhere to burned surfaces. Needle electrodes (used for SSEP) or alligator clips attached to skin staples may be effective alternatives. If skin sites for pulse oximetry monitoring are limited, ear, nose, tongue, penis can be used with standard probes. Reflectance oximetry is another alternative. Extensive debridement and excision or critically ill patient may demand placement of invasive lines. In addition to continuous blood pressure monitoring, arterial line placement allows repeated analysis of blood gases and other laboratory parameters including hematocrit, blood sugar and coagulation panel. A central venous catheter provides long term access besides being a guide for perioperative fluid administration. Urine output monitoring is vital to guide fluid therapy. Core temperature should always be monitored and normothermia aimed for.

**Blood loss and transfusion requirements:** Burn excisions are bloody operations. Blood loss can be sudden, rapid and severe during tangential excision of full thickness burns. Up to 3% of the BV can be lost for every 1% of the BSA excised and grafted. Presence of infection increases the amount of blood loss. Blood products should be readily available in the OR before excision begins.

Various methods that are commonly employed to reduce the blood loss include staged excision, compression dressings, performing all extremity excisions under pneumatic tourniquet, subcutaneous injection and/or topical administration of epinephrine to the burn and donor sites, and use of thrombin spray, fibrin sealant or platelet gel.

Quantification of blood loss is not possible during burn excision as it may be hidden in the drapes, sponges, gowns and on the floor. Transfusion is best guided by serial hematocrit estimations. If blood loss is excessive, it is prudent to rule out coagulation abnormalities. Platelets or coagulation factors may need to be replaced as guided by the coagulation profile.

**Analgesia:** Severe pain is an inevitable consequence of a major burn injury and analgesic requirements are frequently underestimated, especially so in the perioperative period. Anxiety and depression are common components in a major burn and can further decrease the pain threshold. High-dose opioids are commonly used to manage perioperative pain and morphine, fentanyl or methadone is our common choices. There is an inter-individual variation in response to morphine, so ‘titration to effect’ is important. Furthermore, most burned patients rapidly develop tolerance to opioids. Donor area pain may be alleviated using topical local anesthetic, regional blocks and intravenous lidocaine.

For background analgesia in the postoperative period, analgesics such as acetaminophen can be used for their opioid-sparing effect, and combined with generous administration of oral or parenteral opioids. Nonsteroidal anti-inflammatory 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. Breakthrough pain can be treated either with boluses of opiates using patient controlled analgesia (PCA) or nurse controlled analgesia (NCA) devices as dictated by the patient age or using rapidly acting transmucosal fentanyl lozenges.12

**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 opioid analgesics (IV, oral or transmucosal) alone or in combination with benzodiazepine anxiolysis is the typical regimen. However, when wound care procedures are extensive, more potent anesthetic agents may be of benefit. Ketamine offers the advantage of stable hemodynamics and analgesia. It has been used extensively as the primary agent for both general anesthesia and analgesia for burn dressing changes. Nitrous oxide with oxygen (Entonox) has also been used successfully 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 also carries a risk of inducing 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. Music therapy, hypnotherapy, massage, a number of cognitive and behavioral techniques and more recently, virtual reality techniques have been successfully used to reduce pain during debridement and wound care.13 Opioids and benzodiazepines can be used together for both background and procedural sedation, as anxiety is a common component of burn pain associated with wound care procedures. Patient monitoring must be appropriate to the level of sedation.

**References:**

Further Reading