INITIAL ASSESSMENT AND RESUSCITATION OF THE SEVERELY BURNED PEDIATRIC PATIENT

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DISCLOSURES

• I have no disclosures to make
OBJECTIVES

- Discuss the initial evaluation of the pediatric burn patient (ABCDE)
- Outline fluid resuscitation of the pediatric burn patient
- Outline the initial wound management of the pediatric burn
- Discuss the initial evaluation of electrical injuries
INCIDENCE OF PEDIATRIC BURNS

- 440,000/year
- 20,000 require hospital admission
- 1,000/year die in house fires
- 2/3 deaths under age 4
MORTALITY

103 children with burns >80%
• 69 survived (mortality 33%)
• Predictors of mortality
  age < 2 (55%)
  burn >95% (65%)
  delay to IV access
  inhalation injury
INITIAL APPROACH

• ABC’s of ATLS
• initial fluid management
• monitoring of resuscitation
• topical agents
• indications for escharotomies
AIRWAY

Inhalation Injury

• anoxic injury – oxygen content of closed room with fire may be as low as 10%; anoxic injury can occur within minutes

• carbon monoxide/cyanide – systemic poisons absorbed through the lungs

• smoke inhalation – chemical byproducts of combustion that create chemical burns to the airway
AIRWAY

- all victims of closed space fires should be placed on 100% oxygen by mask until an arterial blood gas (ABG) with carboxyhemaglobin (COHgb) level is obtained

- carbonaceous sputum, soot in the nasopharyngeal cavity, and facial burns are signs of inhalation injury; they are not absolute indications for endotracheal intubation. The majority of children with mild to moderate smoke inhalation do not require intubation
AIRWAY

Indications for Intubation

- inability to protect airway
- respiratory distress
- hoarseness/stridor
- dyspnea
- third degree facial burns
- CO poisoning
AIRWAY

- hoarseness/stridor – indicates laryngeal edema. This edema will increase for 24-36 hours during the fluid resuscitation and delay in intubation may result in loss of the airway resulting in urgent tracheostomy
- wheezing – does not indicate laryngeal edema. Bronchospasm is common after smoke inhalation and usually responds to bronchodilators/asthma protocols
CARBON MONOXIDE

- byproduct of combustion
- history: exposed to closed space fires
- signs: decreased alertness – burns are painful and decreased mental status suggests anoxic injury, CO poisoning, or associated closed head injury
- cutaneous pulse ox monitors interpret COHgb as oxygen saturated hemoglobin and cannot be used to rule out CO poisoning – an ABG with direct measurement of COHgb is required
CARBON MONOXIDE

Half-life

room air : 240 minutes
100% oxygen : 30-45 minutes
Hyperbaric oxygen : 15-20 minutes

Pathophysiology

impaired oxygen delivery (COHgb)
impaired oxygen utilization (cytochrome oxidase)
CARBON MONOXIDE

Treatment

• intubate/100% oxygen:
  COHgb > 20 if symptomatic
  COHgb > 30

• wean oxygen when:
  COHgb < 10 and serum bicarbonate > 20
  Resolution of metabolic acidosis signifies CO has cleared the mitochondrial cytochrome oxidase system
• Hyperbaric oxygen use in carbon monoxide poisoning remains controversial

• ACEP (2008): “no clinical variables, including COHgb levels” , identify a subgroup of CO-poisoned patients for whom HBO is most likely to provide benefit or harm”
PEARL

- Cochrane Review (2011): “existing randomised trials (seven reviewed) do not establish whether the administration of HBO to burn patients with CO-poisoning reduces the incidence of adverse neurologic outcomes”.
PEARL

- ABA: “HBO is not recommended in burn patients with CO-poisoning requiring active resuscitation”. 
385 patients with transient loss of consciousness (group A-179) or coma (group B-86) were randomized.

• Group A: no statistical difference in status at one month

• Group B: all received HBO. The longer the dive, the worse the outcome
SMOKE INHALATION INJURY

- determinants of mortality: age, burn size, presence of inhalation injury
- leading cause of death at scene: anoxic injury
- leading cause of death in burn unit: pneumonia in patients with pre-existing inhalation injury
SMOKE INHALATION INJURY

- diagnosis: bronchoscopy
- treatment: intubation for significant injury
  - PEEP/low volume ventilation
  - bronchodilators
  - pulmonary toilet
  - high frequency ventilation
RULE OF NINES

- head 9%
- each arm 9%
- anterior trunk 18%
- posterior trunk 18%
- each leg 18%
- genitalia 1%
- palm of patient’s hand including fingers is approximately 1%
Burn Size (3 of 3)

Blood volume in child 80 ml/kg (adult 70 ml/kg)

- Evaporative loss in 10 kg child with 20% burn is 475 ml (50% of circulating volume)
- Evaporative loss in 70 kg adult with 20% burn is 1,100 ml (22% of circulating volume)
FLUID RESUSCITATION

• Parkland formula (4 ml/kg/%burn) will under-resuscitate small children
• Galveston: 2000 ml/m² TBSA + 5000 ml/m² BSA burn
• Brooke: maintenance rate + 3 ml/kg% burn (1/2 in first 8 hours)
FLUID RESUSCITATION

• only 2\textsuperscript{nd}/3\textsuperscript{rd} degree burns are used in the calculation; pink 1\textsuperscript{st} degree burns with intact skin are not counted
• Ringer’s Lactate – large volumes of normal saline will result in hyperchloremic acidosis
• IV access may be through burn but should not be distal to circumferential burns
Resuscitation formulas are an estimate and IV rates must be adjusted.

Under-resuscitation results in hypoperfusion of organs and the burn wound; hypoperfusion of the wound increases cellular death and may convert partial thickness burns to full thickness injury.
MONITORING

• Over-resuscitation exacerbates any co-existing pulmonary injury and increases edema. Increased edema results in hypoperfusion of the wound and conversion of partial thickness burn to full thickness injury.
• Both under- and over-resuscitation are detrimental to the wound.
• Determining the adequacy of resuscitation remains one of the most difficult aspects of burn care.
MONITORING

• goal is urine output of 0.5 ml/kg/hr (1.0 ml/kg/hr in infants)
• urine outputs greater than 1.0 ml/kg/hr are to be avoided
• the initial metabolic acidosis should be improving after 12 hours
• lactic acid levels are unreliable indicators of resuscitation in the burn patient. Full thickness skin death results in release of lactic acid that will not resolve until the burn is excised
WOUND CARE

Immediate transfer
• clean, dry or lubricated dressing
• tetanus
• no systemic antibiotics

Transfer delay > 6 hours
• topical agents
TOPICAL AGENTS

• Systemic antibiotics do not penetrate the dead surface tissue of the burn wound and cannot prevent infection of the necrotic tissue. There is no role for prophylactic antibiotic use in burn patients.

• Topical antibiotics decrease surface colonization and decrease the incidence of invasive infections.
TOPICAL AGENTS

- An antibacterial ointment is acceptable for most superficial second degree burns
- It should be covered by a nonadherent dressing
- It must be applied 2-3 times/daily to keep wound moist
TOPICAL AGENTS

Silver Sulfadiazine

- poor penetration
- allergies – Sulfa drug
- transient neutropenia – WBC of < 1,500 are not uncommon
- resistant Pseudomonas strains exist
- inhibits epithelial healing – should not be used in superficial second degree burns which have minimal risks of infection
- advantage: soothing
  overall excellent coverage
ADJUNCTS

- NG tube for burns >20% - gastric ileus is common
- tetanus
- fluorescein eyes if facial burns present
- avoid hypothermia
- beware associated injuries – patients found “down” in fires may have associated injuries from the fall