Chapter 23: Trauma and Special Emergencies

Cathie Tingey Jones

INTRODUCTION

FOCUS POINTS

1. Trauma is the leading cause of pediatric mortality and these patients are best served at facilities equipped to treat children.
2. Injuries not in character with the play patterns of children should raise concerns for nonaccidental trauma.
3. The primary survey for pediatric patients is ABCDE: Airway, Breathing, Circulation, Disability, and Exposure.
4. It is important to prevent hypothermia in trauma patients to avoid the triad of acidosis, coagulopathy, and death.
5. Burn patients are in a hypermetabolic state and have altered pharmacodynamics of many drugs, including neuromuscular blockers. They can also become very tolerant to pain medications.
6. If inhalational injury is suspected in a burn patient, intubation should happen early.

Traumatic injuries are an important public health concern in pediatrics. Accidents are the leading cause of death in children over the age of 1. Unintentional injury was responsible for 17,603 deaths in the United States in patients ages 19 years and under in 2017.[1] Twenty-five children in the United States die from injuries every day.[2] Accidental injury also places a large cost burden on the health care system, exceeding $20 billion annually in costs.[3]

TYPES OF INJURY

Falls are the leading cause of nonfatal injuries in children from birth to age 14. Motor vehicle accidents are the leading cause of death for 5- to 19-year-olds. Drowning is the most common cause of death in children ages 1 to 4. Burns are also common causes of mortality from ages 1 to 9. Other types of unintentional injuries common in children are suffocation, poisoning, burns, and sports or recreation injuries.[2,4]

Non-Accidental Trauma

While many injuries are unintentional, child abuse, or non-accidental trauma, is a wide spread concern. Over 3 million reports of child abuse are filed annually in the United States. However, it is likely under-reported and under-detected. The majority of fatalities from child abuse in the United States are in children under 3 years of age, with the greatest percentage under 1 year of age.[5] For the protection of the child, it is important to consider early if the injury pattern is suggestive of intentional injury is suggestive of intentional injury.

Some injuries reflect normal childhood behavior and experiences. Toddlers learning to walk and climb are prone to falling. School-aged children are prone to playground accidents. Normal patterns of injury include shin bruises and forehead bumps for toddlers or forearm and elbow fractures from playground accidents. Patterns of injury that should raise concerns include bruising in areas that aren't usually bumped when walking or running (eg, thigh bruises), rib fractures, fracture of base or vault of skull, eye contusions, intracranial bleeding, multiple burns, and age younger than 1 year. Fractures of large bones, such as a femur, should also raise concern due to the amount of force needed for the fracture. Ribs are difficult to break due to the cartilaginous components of a child's chest. In accidental trauma, a child is more likely to have a pulmonary contusion without an overlying rib

fracture.

Injury Prevention

Primary prevention in trauma aims to prevent the accident from ever happening. This includes improving traffic barriers or changing a dangerous intersection. Secondary prevention attempts to decrease the seriousness of the injury with aids such as car seats, seat belts, and air bags. Education to raise awareness about car seat safety, seat belt use, bicycle helmets, and household dangers is important to treat this problem. In the United States, the rate of death from unintentional injury has decreased due to implementation of injury prevention programs. Other injuries are unfortunately inflicted by family members or those caring for children. Parenting classes, childcare education, and stress reduction programs may be helpful in decreasing the incidence of these injuries as well.[2]

Tertiary prevention aims to minimize the problems and deterioration when the first two means fail. This includes appropriately identifying children with major trauma before hospital arrival. Many hospitals may not be equipped to deal with pediatric trauma. This allows referral to hospitals with the best resources to treat them and to activate transport teams if needed. Children have better outcomes when treated at centers equipped for pediatric trauma and should be preferentially triaged to pediatric trauma centers where available.[6]

PRESENTATION TO HOSPITAL

Before patients reach the emergency department, they usually have initial triage by Emergency Medical Systems (EMS). Appropriate triage is critical to identify seriously injured patients. The initial field triage considers Glasgow Coma Score, systolic blood pressure, and respiratory rate. This is the first point where a decision may be made to proceed to a designated trauma center. The second step looks at penetrating injuries, extremity, pelvic fractures, and chest wall instability. Steps 3 and 4 in field triage consider the mode of injury (fall from height >10 feet in children or high-risk auto crash) as well as if they are of extremes of age or have burns or other considerations.[7]

PRIMARY SURVEY

On presentation to the trauma center the primary survey, established from Advanced Trauma Life Support (ATLS), is key to establishing the patient's status in an organized manner. It has a simple mnemonic **ABCDE**: **A**irway, **B**reathing, **C**irculation, **D**isability, and **E**xposure. All of these assessments should be accomplished in the first 5 minutes after presentation.[8,9]

Some hospitals use aids to assist with the care of children. Luten and Broselow designed a system to help guide medical staff. Their system takes the length of the child, measured by the Broselow Tape, and approximates weight of the child. On the card, the appropriate equipment and dosing of common medications are listed. Some centers have carts stocked which correspond to the colors on the Broselow Tape and corresponding color-coded card.[10–12]

Many emergency departments are using ultrasound as part of their initial trauma assessment. A Focused Assessment with Sonography for Trauma (FAST) exam should be done immediately after the primary survey per ATLS guidelines. This can be done at the bedside in the emergency department. It looks at the four quadrants of the abdomen as well as assessing for the presence of hemothorax or pneumothorax checking pericardial fluid for signs tamponade. An extended FAST exam, E-FAST, adds the views to evaluate for pneumothorax.[13–16]

Airway Maintenance with Cervical Spine Protection

The first step in the ATLS primary survey is ensuring a patent airway. Many EMS providers may not have experience or equipment for intubating a pediatric patient. The child may present to the emergency department with bag-valve-mask ventilation ensuing. Intubation is indicated if the patient has respiratory, circulatory, or neurological compromise.[8,9,17]

While cervical spine injuries are uncommon in children, it is important to maintain a neutral neck position during mask ventilation and intubation. In-line stabilization should be done before laryngoscopy to prevent the head from moving side to side and prevent flexion and extension.[16] Video laryngoscopy (VL) can be used but is not part of ATLS. VL has not been shown to improve mortality in trauma situations.[18]

Breathing and Ventilation

Children presenting with breathing problems may have nasal flaring, grunting, or retractions. While assessing for adequacy of respirations, it is important to assess for intrathoracic pathology, such as a hemothorax or pneumothorax. This can be done with physical exam, portable x-ray, or bedside sonography. If a tension pneumothorax or hemothorax is found, a needle decompression or chest tube placement should occur in the trauma bay. A tension pneumothorax is more likely to cause hemodynamic instability in children and the institution of positive pressure ventilation may worsen vital signs.[9]

Circulation with Hemorrhage Control

Adequacy of circulation should be determined at this step. Vascular access, if not already established in the field, should be obtained. Fluid resuscitation should be started or assessed (if underway) and laboratory studies obtained. External hemorrhage, if present, should be attempted to be controlled. If cardiac tamponade is present on FAST exam, a pericardiocentesis should be considered. If the cardiac arrest was witnessed after trauma, a thoracotomy should be considered. If there is no pulse, cardiac compressions should be started. The pelvis should also be examined for possible fracture or potential occult site of blood loss.[9]

Disability/Neurological Assessment

The patient's neurological status needs to be established. The Glasgow Coma Scale (GCS) is the most widely established way to communicate this, though it is modified slightly for young children (Table 23-1).

Table 23-1

Modification of the Glasgow Coma Scale for Pediatric Patients

Type of Response	Score*	Age-Related Responses		
		>1 Year	<1 Year	
Eye-opening response	4	Spontaneous	Spontaneous	
	3	To verbal command	To shout	
	2	To pain	To pain	
1	None	None		
		>1 Year	<1 Year	
Motor response	6	Obeys commands	Spontaneous	
	5	Localizes pain	Localizes pain	
	4	Withdraws to pain	Withdraws to pain	
	3	Abnormal flexion to pain (decorticate)	Abnormal flexion to pain (decorticate)	
	2	Abnormal extension to pain (decorticate)	Abnormal extension to pain (decorticate)	
	1	None	None	
		>5 Years	2–5 Years	0–2 Years
Verbal response	5	Oriented and converses	Appropriate words, phrases	Babbles, coos appropriately
Verbal response	5	Oriented and converses	Appropriate words, phrases	Babbles, coos appropriately
	4	Confused conversation	Inappropriate words	Cries but is consolable
	3	Inappropriate words	Persistent crying or screaming to pain	Persistent crying or screaming to pain
	2	Incomprehensive sounds	Grunts or moans to pain	Grunts or moans to pain
	1	None	None	None

*Scoring: severe, <9; moderate, 9–12; mild, 13–15.

Source: Modified with permission, from James HE, Anas NG, Perkin RM, eds. *Brain Insults in Infants and Children: Pathophysiology and Management.* 1985. Copyright © Grune and Stratton Inc. All rights reserved.

If the GCS is less than 8 or rapidly declining, intubation is indicated. The patient should be assessed for signs of potential herniation or spinal cord

injury. If there is concern for herniation, moderate hyperventilation can be started, as well as osmotic agents if the patient is normotensive to temporize. The head of the patient's bed can also be elevated.

Exposure and Environmental Control

During the primary survey, the patient's clothing needs to be removed to fully evaluate the extent of his or her injury. If the patient is hypothermic, rewarming should be started.

SECONDARY SURVEY

After the primary survey, if the patient is stable, a secondary survey is done. It is a head to toe evaluation which includes a history and comprehensive physical and additional studies. If the patient is unstable and needs rapid operative care, it is important for this survey to be done after the patient is stabilized.

Injuries by System

Blunt trauma is the most common kind of traumatic injury in children. When a blunt force is applied to a child's body, multisystem trauma often occurs. Penetrating trauma typically comprises only 10% of injuries in children. While penetrating trauma is uncommon in pediatrics, it can lead to significant injury and disability. It is more likely to lead to operative intervention than blunt trauma.

A surgeon should be consulted early in presentation if there are gunshot or stab wounds to the head, neck, chest, or abdomen. Gunshot wounds (GSWs) are classified by the velocity of the weapon: low, medium, and high. Low-velocity GSWs take an erratic path through soft tissue as they follow tissue planes and rarely penetrate bone. Medium- and high-velocity weapons make a more direct path through the body and cause injury when they enter and exit the body.

Head

Traumatic brain injury (TBI) is a significant public health concern. It is the most common cause of death and long-term disability from injury regardless of age. Most pediatric head trauma is minor and does not need operative intervention or hospitalization. Observation is still important, as signs of TBI may arise after discharge. In children with head injury, systemic blood pressure needs to be maintained in order to maintain cerebral perfusion pressure. While most children present with a systolic blood pressure (SBP) greater than 90 mm Hg, children who present with SBP less than 90 mm Hg had three times greater mortality than those who present with a SBP greater than 90 mm Hg.[19,20]

Neck

Children are less likely to have cervical spine fractures than adults. Their spines are more cartilaginous and the vertebrae are not completely ossified. When injury occurs from a substantial force, such as from a motor vehicle accident or fall, a C-spine injury is more likely. Cervical spine injury in children is typically in a more cephalad location: C3 or above. If a C-spine injury is suspected, or if patient's mental status is decreased or altered, the C-spine should be immobilized until further studies can be done. Fear, pain, age, and stranger anxiety can be confounding factors when examining for cervical spine injury. In nonverbal children, the examiner has to rely on facial expression, guarding & other less reliable inputs.[21,22]

It can be difficult to rule out cervical spine injury in children based on x-rays alone. There is a phenomenon called **S**pinal **C**ord **I**njury **W**ith**O**ut **R**adiographic **A**bnormality (SCIWORA). This may occur in 25% to 50% of children with spinal cord injuries. According to ATLS guidelines, a CT scan of the neck, to evaluate for fracture, may replace x-rays. While this has increased the number of fractures found, ligamentous injuries cannot be found on x-ray or CT. If there is concern for a ligamentous injury or SCIWORA, an MRI should be obtained when the patient is stable.[23,24]

Pseudosubluxation of the cervical spine is also a common finding in children. It appears as an anterior displacement of C2 on C3. The person examining the child needs to elucidate if it is a true subluxation or benign pseudosubluxation. The appearance of pseudosubluxation on x-ray can be reduced by placing the child's head in sniffing position when the film is taken. This should only be done after consultation with the surgeon due to the risk of spinal cord injury. Older children who can cooperate can have an open mouth film taken. This allows evaluation of the more cephalad vertebrae and helps rule out a fracture and accounts for the potential pseudosubluxation.

Some children with genetic syndromes have predispositions to cervical spine injury, such as Down and Klippel–Feil syndromes. If there are findings, either clinical or radiographic, suggestive of injury, consultation with a specialist is recommended.[21–24]

Chest

Thoracic trauma is the second leading cause of traumatic death in children. Patients with abnormal vital signs, significant bony tenderness, or abnormal breath or heart sounds may have intrathoracic injury, and warrant additional evaluation. Due to a more cartilaginous thorax, internal injuries may be masked due to the lack of fractures. If a child has a rib fracture, without an underlying pulmonary contusion, it should raise the clinical suspicions for non-accidental trauma. Patients with rib fractures may require aggressive pain treatment to breathe adequately.

Abdominal

Children are more prone to abdominal trauma than adults. With a more compact torso, there is a smaller area to disperse the force. The organs are often larger, relative to the patient's size, and may project below the costal margin. They also have less abdominal fat to cushion them from the trauma. Approximately 5% to 10% of pediatric blunt trauma have intraabdominal injury.[25] Mortality after blunt trauma is generally low but increases as injury to organs and vessels are injured.

Children that have intraabdominal injuries and are hemodynamically unstable, despite fluid resuscitation, warrant operative intervention. Signs of hemorrhagic shock include tachycardia, narrow pulse pressures, prolonged capillary refill time, pallor, altered mental status, and decreased urine output. Other external signs to look for are ecchymoses, abrasions, tire marks, seat belt signs, abdominal tenderness or distension, and absent bowel sounds.[26]

Extremities

Some injuries to extremities may require immediate operative intervention. These include situations when there is neurovascular compromise to the extremity. The extremities should be examined for swelling, contusions, deformities, tenderness, and presence of pulses. If the patient is able to participate in the exam, sensation in extremities should be checked.

Anesthetic Care and Planning

When the patient is stable and time allows, a full preoperative evaluation should be conducted. Along with the traumatic presentation, this includes past medical and surgical history, family history of problems with anesthesia, review of systems, and time of last meal. If surgery needs to proceed rapidly, the preop assessment can be truncated to include many salient points.

NPO

When planning for the trauma anesthetic, if nil per os (NPO) status is unknown, it should be assumed that the patient has a full stomach. Gastric residual volume is greater in children undergoing emergency surgery versus elective surgery. Precautions should be taken to reduce the chance of aspiration. These may be pharmacological or mechanical, such as elevating the head of the bed during induction. A rapid sequence induction may be considered.[27]

Vascular Access

Adequate intravascular access is essential to post-trauma resuscitation. In pediatrics, the patient may have been uncooperative with intravenous (IV) catheter placement by EMS. Placing an IV catheter in a moving vehicle presents an extra level of difficulty, and decreased intravascular volume may increase the challenge. If the child arrives at the hospital without a peripheral intravenous (PIV) line, and a PIV line is not easily obtainable, an intraosseous (IO) line should be considered. Urgent life-saving care should not be delayed for placement of a central venous line without attempting an IO line. An IO line can be placed quickly with minimal discomfort for the patient. It is traditionally placed below the tibial tuberosity in children. The humerus and sternum have also been used.

Local anesthesia is typically not used for IO placement as the patient is often in extremis. After sterile preparation of the skin, the needle is inserted at

90 degrees to the skin and tibia. The needle is advanced using constant pressure and a rotating motion until a “give” is felt. This suggests that the needle has gone through the outer cortex of the bone and into the marrow. The stylet is removed from the needle and a syringe attached. Using the syringe, marrow or blood should be aspirated and can be sent for lab tests if needed. If no marrow is obtained, fluid should freely flow to gravity through the needle. The surrounding area can be palpated for extravasation of fluid. Fluid may flow slowly, so medication should be followed by a saline flush. An ultrasound can confirm placement of the IO needle. It is important not to insert the needle too far in small children as it can come out the backside of the bone. IO needles should be replaced as soon as more definitive access can be obtained.[28,29]

Central venous access can be obtained via the Seldinger technique. The femoral vein can be easily accessed in an awake child. Placing the patient in a cervical collar, if there is concern for head and neck injury, can also impede central venous access. If an internal jugular or subclavian location is preferred the patient may require some sedation. Ultrasound guidance can be used to facilitate visualization of the vessel and placement of the central line. Nonpediatric centers may be limited in what catheters they have for central access in pediatric patients.

Airway

If the patient has not had his or her airway secured prior to arrival in the operating room, adequate equipment should be available. Personnel should also be available to maintain in-line stabilization to protect the cervical spine if it has not been cleared. Additional equipment and personnel should be ready and available if there are concerns that the airway will be difficult to manage.

Blood and Fluid Resuscitation

Massive hemorrhage is uncommon in the pediatric population. When it occurs, it is typically the result of injury to solid viscera or major vascular structures. Traumatic injury severe enough to cause exsanguination typically results in death at the scene of the accident. In both adults and children, approximately 25% of trauma patients receive blood transfusions.

Children typically maintain their blood pressure for a longer period than adults following blood loss. The first sign of blood loss in a child may be tachycardia, which may appear when 10% to 20% of the patient’s blood volume is lost. Hypotension typically does not present until 25% or more of the patient’s blood volume has been lost. The presence of hypotension due to hypovolemia is a concerning sign and may signal impending cardiovascular collapse.

The primary goal of resuscitation is to maintain the patient’s intravascular volume. Massive transfusion and fluid resuscitation after injury may result in a coagulopathy. Massive transfusion in children is defined as replacement of 1 blood volume in 24 hours or 50% of the blood volume in 3 hours.[30] The coagulopathy is usually from the dilution of clotting factors and thrombocytopenia. Hypothermia may also contribute to clotting problems, and should be actively treated. Fibrinolysis or disseminated intravascular coagulation (DIC) may also occur after a traumatic injury, but like hypothermia, they are not the most likely cause of coagulopathy. The “triad of death” consists of coagulopathy, hypothermia, and acidosis.[30–32]

It is important to know your institution’s ability to handle a massive transfusion. Whenever possible, pediatric patients with anticipated need for transfusion should be triaged to a center with this capability. The blood bank should be notified as soon as it is anticipated that large volumes of blood may be needed to allow time for preparation. Additional personnel such as additional anesthesia providers, nurses, or other operating room staff may be needed to check blood products as well as get the blood components from the blood bank.[7]

Figure 23-1

Massive hemorrhage critical events checklist. (Data from Society for Pediatric Anesthesia. *Critical Events Checklist*. 2018. https://www.pedsanesthesia.org/critical-events-checklist/. Copyright © Society for Pediatric Anesthesia. All rights reserved.)

Massive Hemorrhage

Defined: Hemodynamic instability with blood loss > ~ 40 mL/kg or expecting needing to replace patient's total blood volume over 24 hours or less

- Contact Blood Bank to activate pediatric massive transfusion protocol (MTP)
- Send blood sample for type & cross
- If typed blood is not available, use uncrossmatched O negative PRBCs and AB+ plasma until type specific blood is available
- Recommend transfusing at ratio of 1 PRBC: 1FFP: 1 platelet unit
- Treat for Hemoglobin (hgb) <7, Platelet <50,000, INR>1.5, Fibrinogen <100 mg/dL
  - PRBC 10-20 mL/kg (4 mL/kg increases Hgb by 1)
  - FFP 10-15 mL/kg
  - Platelets 2-3 mL/kg or 1unit/10 kg
  - Cryoprecipitate 5 mL/kg or 1U/5 kg
- Maintain normothermia
- Check labs frequently to monitor CBC, Electrolytes, ABG, PT/PTT/INR, Fibrinogen & lactate
- Monitor for hyperkalemia, give calcium gluconate 50-100 mg/kg or calcium chloride 20 mg/kg if needed
- Watch for signs of citrate toxicity: hypocalcemia and hypomagnesemia
  - Calcium: CaGluconate 30-50 mg/kg or CaChloride10-20 mg/kg
  - Magnesium: if Mg<1.5 mg/dL, Mg Sulfate 50 mg/kg
- When bleeding is under control, contact blood bank to stop MTP

Source: Herodotos Ellinas, Kai Matthes, Walid Alrayashi, Aykut Bilge: *Clinical Pediatric Anesthesiology*

There is not a fixed formula for when to transfuse packed red blood cells (PRBCs), platelets, or clotting factors in pediatric trauma. Most of the studies have been carried out in adults and extrapolated to pediatrics. Transfusion exclusively of PRBCs is known to dilute clotting factors and platelets. The adult trauma literature suggests that transfusing plasma and platelets early in trauma care can prevent some of the acute coagulopathy.34,35 Many adult centers use a 6:6:1 transfusion strategy (6 units PRBCs:6 units FFP:1 unit of pooled platelets) for trauma patients expected to receive a large volume of blood. There is one pediatric study in combat trauma patients that showed higher mortality in patients receiving balanced component transfusion.[36] However, other pediatric studies support the balanced approach.[37,38] There is also some evidence to support the use of antifibrinolytics such as tranexamic acid in trauma patients, including pediatric trauma. It has been shown to be associated with decreased mortality in combat patients; however, this may not extrapolate to blunt trauma patients.[39–42]

Other problems with massive transfusion are hypothermia and hyperkalemia. Warming the blood products prior to transfusing prevents the blood products from contributing to hypothermia. Hyperkalemia is more common when older blood products are transfused, due to the higher amount of lysed RBCs in the unit. This potassium load can be diminished by transfusing the freshest blood possible. Another option to lower the potassium is washing the blood cells prior to transfusion. As the RBCs need time to be washed, this is typically not something that can be done in the setting of rapid or massive transfusion.

Temperature Regulation

Operating room temperature is the most important factor influencing heat loss in surgical patients. The average surgical patient will maintain his or her core temperature if the room temperature is 23°C for adults or 26 °C for infants. Maintaining normothermia is important to help with peripheral perfusion, as well as preventing worsening coagulation from hypothermia.

Depending on the nature and extent of injuries, not all patients can have all of their definitive treatment on their initial trip to the operating room. The concept of "damage control surgery" has evolved in adults and pediatrics. The initial, life- and limb-threatening issues are treated surgically in the operating room. Then, if needed, the abdomen or other injured parts of the body are packed or a wound-vacuum system is used, and the patient is taken to the intensive care unit for further management of critical medical issues. This may require diuresis, stabilization of acid-base status, or completion of the secondary survey before the patient is ready to return to operating room to finish his or her surgical care.[43,44]

SPECIAL EMERGENCIES

Burns

Burns are the fifth leading cause of death in children in the United States.[2] While mortality has declined due to advances in burn care and prevention, burn injury requires intensive treatment and has long-term disability. It is important to identify and treat injuries early, resuscitate appropriately, and refer to a burn center early in care in children with major burns.

Scald burns represent 65% of burns in children under age of five in the United States. Family education should occur whenever a child presents with scald burns. Turning down the temperature of the water heater to less than 120°F and turning in pan handles on the stove are simple measures that can decrease the incidence of these burns.[2] Scald injuries, although common, can be representative of non-accidental trauma to the child. Signs suggestive of non-accidental scald burns are injury inconsistent with the story, clear edges of the burn (as if the patient were submerged in the hot water), and lack of splash marks.[44–46]

Older children are more likely to be injured from actual fire and flames. Fireworks can cause severe burns to the hands, face, and eyes of older children. Electrical burns cause direct thermal damage. Chemical burns vary based on the substance and the duration of contact.[47]

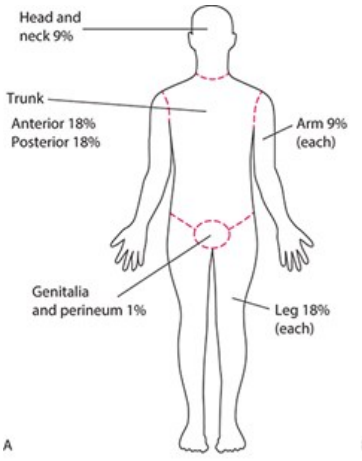
Burns can be superficial, superficial partial-thickness, deep partial-thickness, or full-thickness. *Superficial* burns involve the epidermis. They are painful but heal quickly and without scarring. *Superficial partial-thickness* burns involve the epidermis and portions of the dermis. They typically form blisters. *Deep partial-thickness* burns extend into the deeper dermis. They damage hair follicles and glandular tissue; they can be difficult to distinguish from full-thickness burns. *Full-thickness* burns destroy the dermis and extend into the subcutaneous tissue. They are often white or black on the surface. As nerves are damaged in full-thickness burns, they are less painful than partial-thickness burns.[45]

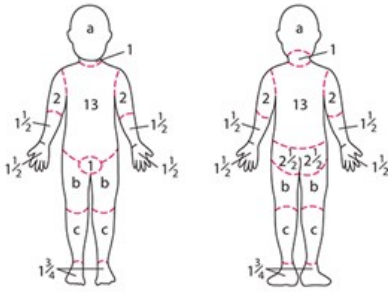
Rule of 9s

The amount of body surface burned is typically estimated by the "Rule of 9s." A reference chart (see Figure 23-2) can be used to help estimate the portion of the body surface area burned. Palm size represents approximately 1% of a patient's total body surface area. Small children do not have the same rule of 9s as adults. Their head is larger relative to the rest of their body and represents a larger percent of total body surface area (TBSA) (see Figure 23-2), though the overall process of estimation is the same.

Figure 23-2

Rule of Nines for calculating Body Surface Area (BSA) percentage burned. (A) Shows the BSA percentages for an adult using the Rule of Nines. (B) The table and associated drawing show how the Rule of Nines changes with age as a child's head is a large portion of their BSA when they are an infant but as they age their legs become a larger portion of their BSA. (Redrawn with permission, from Artz CP, JA Moncrief: *The Treatment of Burns.* 2nd ed. 1969. Copyright © WB Saunders Company. All rights reserved.)





Relative percentage of body surface area (% BSA) affected by growth

	Age				
Body part	0 yr	1 yr	5 yr	10 yr	15 yr
a = 1/2 of head	9 1/2	8 1/2	6 1/2	5 1/2	4 1/2
b = 1/2 of 1 thigh	2 3/4	3 1/4	4	4 1/4	4 1/2
c = 1/2 of 1 lower leg	2 1/2	2 1/2	2 3/4	3	3 1/4

Source: Herodotos Ellinas, Kai Matthes, Walid Alrayashi, Aykut Bilge: *Clinical Pediatric Anesthesiology*

Severity of the burn is impacted by depth, TBSA, location of the injury, and the presence or absence of inhalation injury.[46]

Fluid Resuscitation

The Parkland formula represents the approximate crystalloid fluid needs over the initial 24 hours in a burn patient. It is meant as a guide for adequate fluid resuscitation so patients are not under-resuscitated. The formula is as follows: 4 mL × weight in kg × TBSA (%) burned plus normal maintenance fluid requirements. Half of the fluid should be given in the first 8 hours, with the remaining half given over the subsequent 16 hours. Urine output (UO) needs to be maintained at 1 mL/kg/h as it acts as a marker of organ perfusion. If the UO falls, additional fluids should be given. Children under 30 kg need to maintain UO of 1 to 2 mL/kg/h, while children 30 kg and greater need to maintain 0.5 to 1 mL/kg/h.

While the Parkland formula is most commonly used, it is not the only formula for burn victim's fluid resuscitation. Another formula, the Brooke formula, uses less fluid overall. Originally, the Brooke formula employed the use of colloids. The modified Brooke formula uses 2 mL × weight in kg × TBSA (%) burned. Additionally, it also titrates fluids to urine output. The Parkland formula is more commonly used in children as they have relatively higher daily fluid requirements than adults. Both formulas provide an estimate, and volume status should be monitored carefully.[44]

The use of colloids is controversial in burn patients. Some centers employ their use after 24 hours of resuscitation, others use them only when a patient's albumin levels decrease. As significant capillary leakage occurs in burned tissue, some experts feel colloids do not carry a significant benefit. There is currently no consensus on colloid use.[47–49]

The first 48 hours of a burn injury are considered the acute phase. During the acute phase the patient typically loses a large amount of protein and is hypovolemic. In the subsequent phase, after 48 hours, the patient enters a hypermetabolic phase. Early burn excision and grafting decreases the metabolic demands of the burn patient. As the patient's metabolic demands are highest in the first few days following a burn, early excision and grafting will help with healing and improve the patient's physiology.

Inhalational Injury and Intubation

Inhalational injury from smoke to the tracheobronchial tree is a concern whenever burn patients present. This is especially true with fires in enclosed spaces, such as house fires. Combustion of materials in a closed space can consume significant amounts of oxygen, leaving the patient in a hypoxic environment. Though the number of patients with true inhalational injury is small, when it does occur there is a high risk of morbidity and mortality. Patients with a high likelihood of inhalational injury should be intubated early.

Thermal damage, asphyxiation, and pulmonary irritation are the main mechanisms that lead to smoke inhalation injury. Thermal damage is typically limited to the oropharynx as heat typically dissipates by the time the air reaches the trachea. External signs of inhalational injury include facial burns, blistering/edema of the oropharynx, mucosal lesions, scorched nasal hairs, carbonaceous sputum, sooty secretions, singed eyebrows, hoarseness, and stridor.

The incidence of inhalational injury increases as the TBSA percentage increases. Intubation should be strongly considered when the TBSA is 30% or greater. In burns that exceed 30% of the TBSA, if bronchopulmonary injury is present, mortality increases to 70%. Along with swelling that may come from fluid resuscitation, patients which a large TBSA often develop progressive upper airway edema in the first 48 hours, even if there was no inhalational component. This can make delayed intubation difficult and even impossible. If a patient needs to be transferred to a higher eschelon of care, consideration should be given to securing his or her airway prior to transport.[50–54]

Carbon Monoxide

Carbon monoxide (CO) is an odorless, colorless gas and is a major component of smoke in fires. Burns from enclosed spaces place the patient at risk for CO poisoning. CO binds hemoglobin with a greater affinity than oxygen. It also causes a left shift of the oxyhemoglobin dissociation curve. This reduces the ability of the red blood cells to off-load oxygen and can lead to hypoxia in the tissues. CO can also bind to heme molecules in myoglobin and decrease oxygen diffusion into muscle, including in the heart.[55,56]

CO and oxyhemoglobin absorb the same wavelength (660 nM) of light. This will falsely elevate standard pulse oximetry as carboxyhemoglobin is misread as oxyhemoglobin. The amount of CO in the blood is likely best detected by co-oximetry. Co-oximeters measure absorption at several wavelengths of light and can distinguish carboxyhemoglobin from oxyhemoglobin. Affected patients who may be able to be treated with 100% oxygen should improve with time. If they are severely affected and have CO encephalopathy, they may require hyperbaric oxygen treatment.[44,55,56]

Cyanide (CN) poisoning can also occur after a fire due to incomplete combustion of nitrogen-containing materials. CN is a colorless gas with a bitter almond odor. It is produced by the combustion of plastic, polyurethane, wool, silk, nitriles, rubber, and paper products. CN stimulates the chemoreceptors in the carotid and aortic bodies, which can lead to hyperpnea. It interferes with metabolism on the cellular level by inhibiting cytochrome *c* oxidase and affecting the mitochondria. This converts the cell to anaerobic metabolism and lactic acidosis ensues. One of the ways it can be diagnosed is with a high mixed venous oxygen saturation. CN toxicity can be treated by sodium thiosulfate or hydroxocobalamin.[44,57]

Eschars

Eschars, which are from the dead and denatured dermis, often form over burned tissue and may form rapidly. They can form at any location in the body. Eschars that form over circumferential burns over the abdomen or thorax may impede the patient's ability to breathe. As edema increases in the first 24 hours, eschars may become tourniquet-like. They may act like restrictive lung disease if there is thoracic wall involvement or lead to abdominal compartment syndrome, with decreased cardiac output, venous return, and urinary output.[58]

Extremities should also be monitored for signs of ischemia from circumferential eschars as well. Warnings signs showing an escharotomy is needed are as follows: (5 Ps)— pain, pallor, paresthesia, paralysis, and pulselessness. If these signs are ignored and an emergent escharotomy is not undertaken it can lead to limb loss.[44]

Thermoregulation

When patients are burned, burns disrupt their integumentary system. The patient's ability to regulate and maintain his or her temperature is decreased due to large raw surfaces. Core temperatures are higher in burn patients (38 to 39°C). Heat loss is proportional to the TBSA burned. Denervated areas can no longer vasoconstrict in response to cold, further complicating the issue. Maintaining normothermia is especially important as heat production adds to already high metabolic demands. Ultimately skin grafting helps prevent the fluid and heat loss at the burned sites.

Anesthesia leads to thermoregulatory vasodilation, which causes a redistribution of body heat from the core to the patient's periphery. Burn patients maintain temperature best with an ambient temperature of 30 to 31°C. This minimizes radiant heat loss. Convective warming, while very effective in most patients, is often difficult to use in burn patients. It can be challenging to use a convective warmer on a patient who needs large areas of skin exposed to debride the wound, harvest skin, and place the skin grafts. Warming IV fluids can prevent further heat loss but is not an effective way to warm patients. For each 1 L of fluid warmed, it prevents the body temperature from losing 0.25°C. The inability to warm fluids significantly above the patient's body temperature is a barrier to this technology.[44]

Nutrition

Burn injury patients require good nutrition to promote healing. Burn injuries create a hypermetabolic state; the larger the area burned, the higher the metabolic rate. Enteral feeding is important to introduce calories, maintain gastrointestinal motility, and protect the mucosa. Without enteral feeds, the risk of infection and sepsis increases. While parenteral feeds can be used, they do not help maintain the integrity of the mucosal barrier in the gut. The mucosal barrier helps prevent bacterial translocation. Feeding can also help decrease the incidence of stress ulcers following a burn. Gastroduodenal ulcers are a common problem, unless the patient is getting enteral feeds or is treated with preventative measures such as proton pump inhibitor (PPI), histamine-2 receptor antagonists, or neutralizing agents.[59–61]

If the patient is unable to take food by mouth, a feeding tube should be placed early to help with caloric intake as well as the other benefits of enteral feeding. A post-pyloric feeding tube is helpful in maintaining round-the-clock feeding in patients with large burns. There is no consensus if post-pyloric feeds should be stopped before anesthesia or sedation. If the patient is not hemodynamically stable and on vasoactive agents, enteral feeding may be held until the patient is more stable. Some sources advocate enteral feeding via a feeding tube in patients with a 30% to 40% TBSA burn as it will be difficult to meet their caloric requirements by oral feeding. For children the estimated protein requirements are 3 g/kg/day after a burn.[44]

Other Changes

Acute Phase

The acute phase is immediately after the burn. As there are systemic release of vasoactive substances and cytokines, the cardiac output is transiently low. The myocardial function is depressed, the blood is viscous, systemic vascular resistance (SVR) is high, and intravascular volume is low. There can be airway obstruction and edema (presenting as bronchospasm and laryngospasm), along with potential carbon monoxide poisoning. The kidneys usually have a low glomerular filtration rate (GFR). In patients with either electrical burns or crush injuries, they may develop myoglobinuria. The liver is often poorly perfused at this point manifested with increasing liver enzymes. There may be hemoconcentration as fluids leave the intravascular space. There may be mucosal damage in the gastrointestinal tract, release of endotoxins, and neurological changes secondary to cerebral edema and increased intracranial pressure.[44,46,62]

Late Phase

After the acute phase passes, the patient enters into hypermetabolic phase and things again change throughout the body. The cardiac output increases and SVR decreases. Patients may be persistently tachycardic and hypertensive. They may develop tracheal stenosis, pneumonia, or tracheobronchitis. There can also be significant sloughing of the mucosa in the airway. The kidneys will see an increase in GFR, tubular dysfunction, as well as breakdown of glucose, lipids, and muscle. The patients may develop anemia, and have stress ulcers and ileus. Their neurological changes may include a personality change, delirium, seizures, or even coma.[44,46]

Neuromuscular Blocking Drugs (NMBDs)

Burn injury can lead to the upregulation of fetal and mature acetylcholine receptors. This usually leads to a resistance to nondepolarizing muscle relaxants (NMBDs) and an increased sensitivity to depolarizing muscle relaxants such as succinylcholine. This is noted to occur between 24 and 72 after injury. This also occurs after stroke, spinal cord injury, prolonged immobility, prolonged exposure to neuromuscular blockers, multiple sclerosis, and Guillain-Barré syndrome. While burn injury upregulates the receptors, they are downregulated in myasthenia gravis, anticholinesterase poisoning, and organophosphate poisoning.[63–66]

Resistance to nondepolarizing NMBDs is typically seen in patients with at least 25% burns. It may take months to years for the receptors to return to normal. Potassium has been noted to markedly increase in burn patients following succinylcholine (SCh) use. SCh has been safely administered in the first 24 hours of a burn injury. After this period, the muscle receptors are likely already altered and SCh should be avoided. Over time, as normal skin regrows, normal acetylcholine receptors return. While it is not known exactly how long to avoid SCh, a conservative estimate avoids the use from 24 to 48 hours after injury and for the next 1 to 2 years.[67,68]

PAIN MANAGEMENT AFTER BURNS

Proper pain treatment minimizes the burn patient's metabolic demands, which aids healing. It is important to manage the patient's analgesic and anti-anxiety medications well. The patient undergoes multiple procedures: burn dressing changes, debridement, skin grafting, physical therapy, and scar tissue resections or releases. Pain tends to be the most intense from the freshly harvested donor sites as they have the greatest number of nerve endings. A tumescent solution can be used by the surgeons intraoperatively to help decrease the pain.[68–70]

Burn patients often need rapid escalations of their doses of analgesics. Some of this is likely from tolerance; however, the opiate receptors likely undergo thermal-injury changes which cause the doses to rapidly escalate. The nerve endings may also change from burn and grafting procedures. Once the skin wound is closed, the need for opiates rapidly decreases. Opiates and anxiolytics should be slowly titrated down to minimize withdrawal symptoms. Alpha-2 agonists have also been used to prevent withdrawal.[46,71]

Many analgesics can be used to treat the pain from burns. Fentanyl, morphine, and hydromorphone are options. Methadone and ketamine, with their *N*-methyl-d-aspartate (NMDA) antagonist activity, may decrease the hyperalgesia and minimize opioid tolerance. Dexmedetomidine is also being used for anxiolysis and pain treatment in burn patients. Nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen can also be considered but the patients need to closely be watched for renal or hepatic problems. Remifentanil may also contribute to hyperalgesia and should likely be avoided due to that reason.[72–75]

Pain is better treated in burn patients on a regular standing schedule, rather than as needed (PRN). Boluses of pain medications should be given prior to therapies and dressing changes; however, they should not be limited to those times. Stool softeners should be started to prevent side effects from

the opiates. Diphenhydramine should be considered in patients, especially children, who have healing burns. Shear forces from itching or scratching at the graft site can cause graft failure. Pruritus may result from medication effects or from granulation tissue growth.[44]

CONCLUSION

Trauma and burns are both significant public health concerns to children today. They both are prime examples of areas where education as well as team-based care can have great opportunities for positive impacts on the health of children. Knowing about the physiological concerns, as well as the concerns for different health care systems, can improve the care delivered to this group of patients.

REFERENCES

1. Center for Disease Control and Prevention. 10 Leading causes of death by age group, United States—2017. Available at https://www.cdc.gov/injury/images/lc-charts/leading_causes_of_death_by_age_group_2017_1100w850h.jpg.

2. Center for Disease Control and Prevention. National Action Plan for Child Injury Prevention. Available at https://www.cdc.gov/safechild/pdf/cdc-childhoodinjury.pdf. Updated 2012.

3. Center for Disease Control and Prevention. *Data & Statistics (WISQARS): Cost of Injury Reports*. Available at https://wisqars.cdc.gov:8443/costT. Updated 2015. Available at http://www.cdc.gov/injury/wisqars/index.html.

4. Centers for Disease Control and Prevention (CDC) MMWR. Vital signs: unintentional injury deaths among persons aged 0–19 years—United States, 2000–2009. *Morb Mortal Wkly Rep.* 2012;61:270.

5. U.S. Department of Health and Human Services, Administration for Children and Families, Administration on Children, Youth, and Families, Children's Bureau. Child maltreatment 2014. Available at https://www.acf.hhs.gov/cb/resource/child-maltreatment-2014. Accessed January 9, 2017.

6. American Academy of Pediatics. Management of pediatric trauma. *Pediatrics*. 2008;121:849–854. [PubMed: 18381551]

7. *Guidelines for Field Triage of Injured Patients: Recommendations of the National Expert Panel on Field Triage* (2011). Available at https://www.cdc.gov/mmwr/pdf/rr/rr6101.pdf. Accessed October 10, 2016.

8. Kortbeek JB, Al Turki SA, Ali J, et al. Advanced trauma life support, 8th edition: the evidence for change. *J Trauma*. 2008;64:1638–1650. [PubMed: 18545134]

9. American College of Surgeons, Committee on Trauma: Advanced trauma life support (ATLS). 8th ed 2008 American College of Surgeons Chicago.

10. Luten R. Error and time delay in pediatric trauma resuscitation: addressing the problem with color-coded resuscitation aids. *Surg Clin North Am.* 2002;82:303–314. [PubMed: 12113367]

11. Agarwal S, Swanson S, Murphy A, et al. Comparing the utility of a standard pediatric resuscitation cart with a pediatric resuscitation cart based on the Broselow tape: a randomized, controlled, crossover trial involving simulated resuscitation scenarios. *Pediatrics*. 2005;116:e326–e333. [PubMed: 16061568]

12. Rosenberg M, Greenberger S, Rawal A, et al. Comparison of Broselow tape measurements versus physician estimations of pediatric weights. *Am J Emerg Med.* 2011;29:482–488. [PubMed: 20825816]

13. Schonfeld D, Lee LK. Blunt abdominal trauma in children. *Curr Opin Pediatr.* 2012;24(3):314–318. [PubMed: 22450250]

14. Blackbourne LH, Soffer D, McKenney M, et al. Secondary ultrasound examination increases the sensitivity of the FAST exam in blunt trauma. *J Trauma*. 2004;57(5):934–938. [PubMed: 15580013]

15. Heller K, Reardon R, Joing S. Ultrasound use in trauma: the FAST exam. *Acad Emerg Med.* 2007 Jun;14(6):525.

16. Warner KJ, Carlbom D, Cooke CR. Paramedic training for proficient prehospital endotracheal intubation. *Prehosp Emerg Care*. 2010;14:103–108. [PubMed: 19947874]

17. Vanderhave KL, Chiravuri S, Caird MS, et al. Cervical spine trauma in children and adults: perioperative considerations. *J Am Acad Orthop Surg.* 2011;19:319–327. [PubMed: 21628643]

18. Yeatts DJ, Dutton RP, Hu PF et al. Effect of video laryngoscopy on trauma patient survival: a randomized controlled trial. *J Trauma Acute Care Surg.* 2013;75(2):212–219. [PubMed: 23823612]

19. Koepsell TD, Rivara FP, Vavilala MS, et al. Incidence and descriptive epidemiologic features of traumatic brain injury in King County, Washington. *Pediatrics*. 2011;128:946–954. [PubMed: 21969286]

20. Savitsky E, Eastridge B. *Combat Casualty Care: Lessons Learned from OEF and OIF.* Fort Detrick, MD: Borden Institute; 2012:565–576.

21. Parent S, Mac-Thiong JM, Roy-Beaudry M, et al. Spinal cord injury in the pediatric population: a systematic review of the literature. *J Neurotrauma*. 2011;28:1515–1524. [PubMed: 21501096]

22. Easter JS, Barkin R, Rosen CL, et al. Cervical spine injuries in children, part II: management and special considerations. *J Emerg Med.* 2011;41:252–256. [PubMed: 20493656]

23. Pang D. Spinal cord injury without radiographic abnormality in children, 2 decades later. *Neurosurgery*. 2004;55:1325–1342. [PubMed: 15574214]

24. Yucesoy K, Yuksel KZ. SCIWORA in MRI era. *Clin Neurol Neurosurg.* 2008;110:429–433. [PubMed: 18353536]

25. Holmes, Lillis K, Monroe D, et al. Identifying children at very low risk of clinically important blunt abdominal injuries. *Ann Emerg Med.* 2013;62(2):107. [PubMed: 23375510]

26. Marwan A, Harmon CM, Georgeson KE, et al. Use of laparoscopy in the management of pediatric abdominal trauma. *J Trauma*. 2010;69:761–764. [PubMed: 20173653]

27. Sagarin MJ, Chiang V, Sakles JC, et al. Rapid sequence intubation for pediatric emergency airway management. *Pediatr Emerg Care*. 2002;18:417–423. [PubMed: 12488834]

28. Tobias JD, Ross AK. Intraosseous infusions: a review for the anesthesiologist with a focus on pediatric use. *Anesth Analg.* 2010;110(2):391–401. [PubMed: 19897801]

29. Guy J, Haley K, Zuspan SJ. Use of intraosseous infusion in the pediatric trauma patient. *J Pediatr Surg.* 1993;28(2):158–161. [PubMed: 8437069]

30. Maw G, Furyk C. Pediatric massive transfusion: a systematic review. *Pediatr Emerg Care*. 2018;34(8):594–598. [PubMed: 30080793]

31. Holcomb JB, et al. Damage control resuscitation: directly addressing the early coagulopathy of trauma. *J Trauma*. 2007;62:307–310. [PubMed: 17297317]

32. Dehmer JJ, Adamson WT. Massive transfusion and blood product use in the pediatric trauma patient. *Semin Pediatr Surg.* 2010;19:286–291. [PubMed: 20889085]

33. Massive hemorrhage. In: Pedi Crisis: Critical Events Checklists. Available at https://www.pedsanesthesia.org/wp-content/uploads/2018/08/SPAPediCrisisChecklistsJuly2018.pdf. Accessed March 29.

34. Holcomb JB, Tilley BC, Baraniuk S, et al. Transfusion of plasma, platelets, and red blood cells in a 1:1:1 vs a 1:1:2 ratio and mortality in patients with severe trauma: the PROPPR randomized clinical trial. *JAMA.* 2015; 313:471–482. [PubMed: 25647203]

35. Fraga GP, Bansal V, Coimbra R. Transfusion of blood products in trauma: an update. *J Emerg Med.* 2010;39:253–260. [PubMed: 19345046]

36. Edwards ME, Lustik MB, Clark ME, et al. The effects of balanced blood component resuscitation and crystalloid administration in pediatric trauma patients requiring transfusion in Afghanistan and Iraq 2002 to 2012. *J Trauma Acute Care Surg.* 2015;78:330–335. [PubMed: 25757119]

37. Hwu RS, Spinella PC, Keller MS, et al. The effect of massive transfusion protocol implementation on pediatric trauma care. *Transfusion*. 2016;56:2712–2719. [PubMed: 27572499]

38. Chidster SJ, Williams N, Wang W, et al. A pediatric massive transfusion protocol. *J Trauma Acute Care Surg.* 2012:73:1273–1277. [PubMed: 23064608]

39. Eckert MJ, Wertin TM, Tyner SD, et al. Tranexamic acid administration to the pediatric trauma patients in a combat setting: the pediatric trauma and tranexamic acid study (PED-TRAX). *J Trauma Acut Care Surg.* 2014;77:852–858.

40. The CRASH-2 Collaborators. The importance of early treatment with tranexamic acid in bleeding trauma patients: an exploratory analysis of the CRASH-2 randomised controlled trial. *Lancet.* 2011;377(9771):1096–1101. [PubMed: 21439633]

41. The CRASH-2 Collaborators. Effects of tranexamic acid on death, vascular occlusive events, and blood transfusion in trauma patients with significant haemorrhage (CRASH-2): a randomised, placebo-controlled trial. *Lancet.* 2010;376(9734):23–32. [PubMed: 20554319]

42. Leeper CM, Neal MD, McKenna CJ, et al. Trending fibrinolytic dysregulation: fibrinolysis shutdown in the days after injury is associated with poor outcome in severely injured children. *Ann Surg.* 2017;266:508–515. [PubMed: 28650356]

43. Fabian TC. Damage control in trauma: laparotomy wound management acute to chronic. *Surg Clin North Am.* 2007;87(1):73–93, vi. [PubMed: 17127124]

44. Savitsky E, Eastridge B. *Combat Casualty Care: Lessons Learned from OEF and OIF.* Fort Detrick, MD: Borden Institute; 2012:596–631.

45. Souza AL, Nelson NG, McKenzie LB. Pediatric burn injuries treated in US emergency departments between 1990 and 2006. *Pediatrics*. 2009;124:1424–1430. [PubMed: 19805456]

46. Fuzaylov G, Fidkowski CW. Anesthetic considerations for major burn injury in pediatric patients. *Paediatr Anaesth.* 2009;19:202–211. [PubMed: 19187044]

47. Lawrence A, Faraklas I, Watkins H, et al. Colloid administration normalizes resuscitation ratio and ameliorates “fluid creep.” *J Burn Care Res.* 2010;31:40–47. [PubMed: 20061836]

48. Faraklas I, Lam U, Cochran A, Stoddard G, Saffle J. Colloid normalizes resuscitation ratio in pediatric burns. *J Burn Care Res.* 2011;32:91–97. [PubMed: 21131844]

49. Lawrence A, Faraklas I, Watkins H, et al. Colloid administration normalizes resuscitation ratio and ameliorates “fluid creep.” *J Burn Care Res.* 2010;31:40–47. [PubMed: 20061836]

50. Tredget EE, Shankowsky HA, Taerum TV, Moysa GL, Alton JD. The role of inhalation injury in burn trauma: a Canadian experience. *Ann Surg.* 1990;212:720–727. [PubMed: 2256764]

51. Fein A, Leff A, Hopewell PC. Pathophysiology and management of the complications resulting from fire and the inhaled products of combustion: review of the literature. *Crit Care Med.* 1980;8:94–98. [PubMed: 7353393]

52. Barillo DJ, Goode R, Esch V. Cyanide poisoning in victims of fire: analysis of 364 cases and review of the literature. *J Burn Care Rehabil.* 1994;15:46–57. [PubMed: 8150843]

53. McCall JE, Cahill TJ. Respiratory care of the burn patient. *J Burn Care Rehabil.* 2005;26:200–206. [PubMed: 15879741]

54. Palmieri TL, Warner P, Mlcak RP, et al. Inhalation injury in children: a 10 year experience at Shriners Hospitals for Children. *J Burn Care Res.* 2009;30:206–208. [PubMed: 19060756]

55. Walker AR. Emergency department management of house fire burns and carbon monoxide poisoning in children. *Curr Opin Pediatr.* 1996;8:239–242. [PubMed: 8814401]

56. Barker SJ, Tremper KK. The effect of carbon monoxide inhalation on pulse oximetry and transcutaneous PO2. *Anesthesiology*. 1987;66:677–679. [PubMed: 3578881]

57. Barker SJ, Tremper KK, Hyatt J. Effects of methemoglobinemia on pulse oximetry and mixed venous oximetry. *Anesthesiology*. 1989;70:112–117. [PubMed: 2912291]

58. Quinby WC Jr. Restrictive effects of thoracic burns in children. *J Trauma*. 1972;12:646–655. [PubMed: 4560086]

59. Deitch EA, Rutan R, Waymack JP. Trauma, shock, and gut translocation. *New Horiz.* 1996;4:289–299. [PubMed: 8774803]

60. Khorasani EN, Mansouri F. Effect of early enteral nutrition on morbidity and mortality in children with burns. *Burns*. 2010;36:1067–1071. [PubMed: 20403667]

61. Andel D, Kamolz LP, Donner A, et al. Impact of intraoperative duodenal feeding on the oxygen balance of the splanchnic region in severely burned patients. *Burns*. 2005;31:302–305. [PubMed: 15774284]

62. Gupta KL, Kumar R, Sekhar MS, Sakhuja V, Chugh KS. Myoglobinuric acute renal failure following electrical injury. *Ren Fail*. 1991;13:23–25. [PubMed: 1924913]

63. Martyn JA, Fukushima Y, Chon JY, Yang HS. Muscle relaxants in burns, trauma, and critical illness. *Int Anesthesiol Clin.* 2006;44:123–143. [PubMed: 16849960]

64. Martyn J, Goldhill DR, Goudsouzian NG. Clinical pharmacology of muscle relaxants in patients with burns. *J Clin Pharmacol.* 1986;26:680–685. [PubMed: 2947935]

65. Han T, Kim H, Bae J, Kim K, Martyn JA. Neuromuscular pharmacodynamics of rocuronium in patients with major burns. *Anesth Analg.* 2004;99:386–392. [PubMed: 15271712]

66. Gronert GA, Theye RA. Pathophysiology of hyperkalemia induced by succinylcholine. *Anesthesiology*. 1975;43:89–99. [PubMed: 1147311]

67. Martyn JA, Richtsfeld M. Succinylcholine-induced hyperkalemia in acquired pathologic states: etiologic factors and molecular mechanisms. *Anesthesiology*. 2006;104:158–169. [PubMed: 16394702]

68. Stoddard FJ, Ronfeldt H, Kagan J, et al. Young burned children: the course of acute stress and physiological and behavioral responses. *Am J Psychiatry*. 2006;163:1084–1090. [PubMed: 16741210]

69. Stoddard FJ, Saxe G, Ronfeldt H, et al. Acute stress symptoms in young children with burns. *J Am Acad Child Adolesc Psychiatry*. 2006;45:87–93. [PubMed: 16327585]

70. Kavanagh C. Psychological intervention with the severely burned child: report of an experimental comparison of two approaches and their effects

on psychological sequelae. *J Am Acad Child Psychiatry*. 1983;22:145–156. [PubMed: 6841835]

71. Angst MS, Clark JD. Opioid-induced hyperalgesia: a qualitative systematic review. *Anesthesiology*. 2006;104:570–587. [PubMed: 16508405]

72. Owens VF, Palmieri TL, Comroe CM, et al. Ketamine: a safe and effective agent for painful procedures in the pediatric burn patient. *J Burn Care Res.* 2006;27:211–216. [PubMed: 16566568]

73. Walker J, Maccallum M, Fischer C, et al. Sedation using dexmedetomidine in pediatric burn patients. *J Burn Care Res.* 2006;27:206–210. [PubMed: 16566567]

74. Potts AL, Anderson BJ, Warman GR, et al. Dexmedetomidine pharmacokinetics in pediatric intensive care: a pooled analysis. *Paediatr Anaesth.* 2009;19:1119–1129. [PubMed: 19708909]

75. Potts AL, Warman GR, Anderson BJ. Dexmedetomidine disposition in children: a population analysis. *Paediatr Anaesth.* 2008;18:722–730. [PubMed: 18613931]

76. Kirkpatrick AW1, Simons RK, Brown R, et al. The hand-held FAST: experience with hand-held trauma sonography in a level-I urban trauma center. *Injury*. 2002;33(4):303–308. [PubMed: 12091025]