Critical/Emergency Care Hour

Myke Federman, MD
Associate Professor
Pediatric Critical Care
Mattel Children’s Hospital UCLA
OUTLINE

• Shock
• Life support – CPR
• Environmental exposures/injuries
  – Near drowning, foreign body aspiration, burns, carbon monoxide poisoning, bites and stings, heat exhaustion/stroke
• Trauma
• Acute abdomen
• Brain death

** Indicates items mentioned in content specifications
SHOCK
Shock

• Definition: Physiologic state of systemic reduction in tissue perfusion resulting in decreased tissue oxygen delivery
  – Supply does not meet demand
  – Signs of poor perfusion include mental status changes, poor urine output, cool extremities, delayed capillary refill**
Shock

• Compensated
  – Blood pressure maintained**, tachycardia, vasoconstriction

• Decompensated
  – Hypotension (<5th percentile)

• Irreversible
  – Progressive end organ dysfunction leading to irreversible organ damage and death
Patterns of Shock

• Hypovolemic – 7 month old with diarrhea, vomiting, decreased urine output
  – Decreased preload due to fluid loss (diarrhea, vomiting, osmotic diuresis, hemorrhage)**
  – Tachycardia, poor perfusion**
Patterns of Shock

• Cardiogenic – *8 day old with poor feeding, tachypnea, mottled*
  – Cardiomyopathies, arrhythmias, obstructive disorders (coarctation, tamponade)
  – Large heart, gallop, murmur, hepatomegaly, jugular venous distension**
Patterns of Shock

• Septic – 14 day old with fever, lethargy, extreme tachycardia
  – Different etiologies
  – Tachypnea, grunting, tachycardia, warm extremities, bounding pulses
Patterns of Shock

• Distributive - 4 year old with peanut allergy who went to the circus
  – Anaphylaxis, neurogenic (spinal cord injury)
  – Tachycardia, warm extremities, stridor/wheezing with anaphylaxis
  – Bradycardia and hypotension with spinal cord injuries
Shock - Therapy

- A,B,Cs
- Vascular access – use of intraosseous early** (5 minute attempt at IV access)
- Fluid administration 20 ml/kg crystalloid, repeat as necessary (60-200 ml/kg)
- Recognize no or poor response to fluid with cardiogenic shock
Shock - Therapy

• Vasoactive medications:
  – Dopamine
  – Epinephrine for cold shock
  – Norepinephrine for warm shock
  – Milrinone for some types of cardiogenic shock
  – Steroids for refractory shock in patients with risk of adrenal insufficiency or for neurogenic shock after spinal cord injury
LIFE SUPPORT / CPR

**
General concepts

• Sudden cardiac arrest in children is rare
• Progressive respiratory failure more common
• During CPR:
  – Push hard and fast (100/min)
  – 15 compressions : 2 breaths until intubated
Bradycardia with pulse

- Heart rate < 60/min
- ABCs, oxygen, CPR
- Epinephrine IV/IO 0.01 mg/kg (1:10000), ETT 0.1 mg/kg (1:1000)
- Atropine 0.02 mg/kg (minimum dose 0.1 mg)
Tachycardia with poor perfusion

- ABCs, oxygen
- Narrow vs wide QRS
- Sinus tachycardia: P waves, variable rate
- Supraventricular tachycardia: P waves abnormal, rate not variable
  - Vagal maneuvers, adenosine, synchronized cardioversion (0.5-1 J/kg)
Tachycardia with poor perfusion

- Ventricular tachycardia: synchronized cardioversion, may increase to 2J/kg if not effective
Pulseless arrest

- ABCs, oxygen, CPR
- Shockable (VF/VT) vs not shockable (asystole, PEA)
- VF/VT: 2-4 J/kg, epinephrine, amiodarone, lidocaine, magnesium for torsades de pointes
- Asystole/PEA: epinephrine
NEAR-DROWNING
A 2 year old boy is brought to the emergency room after he is found unconscious in a swimming pool. Which of the following is a risk factor for poor prognosis?

1. Age less than 3 years
2. Water temperature < 10° celsius
3. Chlorinated pool
4. Male sex
5. Water depth > 3 meters
Near drowning

- Drowning leads to ~ 4000 fatalities/year in US; approximately ½ in children
- Near drowning defined as, at least, temporary survival after submersion event
- Bimodal incidence peaks: age less than 5 and between ages 15 and 25 (usually males, alcohol related)
- Most common cause of death for patients with epilepsy
- Children with developmental/behavioral disorders at increased risk
Near drowning

• Hypoxemia occurs by either aspiration or reflex laryngospasm

• Multiple complications of hypoxemia:
  – Pulmonary (ARDS, pulmonary edema)
  – Neurologic (edema, increased ICP – most likely cause of death)
  – Cardiovascular (arrhythmia, asystole)
  – Renal (renal failure, ATN)
  – Metabolic (acidosis, hypernatremia in the Dead Sea)
Near drowning

- Care is supportive**
- Spinal cord injuries are uncommon, unless obvious mechanism
- Must observe minimum 8-12 hours
- Poor prognosis with age < 3 y, submersion > 10 minutes, resuscitation > 25 mins, resuscitation in ER, water temperature > 10 C**
- 75% survival
Hypothermia

• Generally protective – attenuates effects of hypoxemia and activates diving reflex
• Careful assessment of pulses as difficult to palpate – bradycardia and a-fib do not necessarily require chest compressions
• Continue resuscitative efforts until temperature > 30 deg, even if hours
Near drowning

- Residential swimming pools are the site of 90% of submersion events
- Bathtub, bucket drowning common in toddlers
- Prevention: 5 ft fences that isolate the pool from house and yard, self closing gates, appropriate supervision, CPR education, ?swim lessons
FOREIGN BODY ASPIRATION
Foreign body aspiration

- 80% in children under 3 years
- Food items in infants and toddlers, non food items in older children (coins, pins)
- Balloons most common in fatal cases
- Bronchi > trachea > larynx
- Presentation varies – medical emergency vs delayed diagnosis
Foreign body aspiration

• Life threatening:
  – Complete airway obstruction
  – Unable to speak or cough

• Suspected:
  – Generalized wheezing
  – Regional variation in aeration
  – History of choking in 80-90%
Foreign body aspiration

- Life threatening: back blows/chest compressions in infants, Heimlich maneuver in older children
- Suspected FBA:
  - CXR often negative
  - Rigid bronchoscopy standard of care
  - Infection if delayed presentation
BURNS
<table>
<thead>
<tr>
<th>Degree</th>
<th>Depth</th>
<th>Symptoms</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>FIRST</td>
<td>superficial</td>
<td>pain, redness</td>
<td>pain control</td>
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<tr>
<td>SECOND</td>
<td>partial thickness</td>
<td>superficial – pain, blisters</td>
<td>pain and fluid management</td>
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<tr>
<td></td>
<td></td>
<td>deep – white, leathery</td>
<td></td>
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<tr>
<td>THIRD</td>
<td>full thickness</td>
<td>well demarcated, black, leathery, painless, no blistering</td>
<td>skin grafting</td>
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<tr>
<td>FOURTH</td>
<td>full thickness plus adjacent structures</td>
<td>includes fascia, muscle, bone</td>
<td>reconstructive surgery</td>
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Treatment

• Major hemodynamic changes occur with severe burns
  – Hypovolemia, metabolic, autonomic instability
• Evaporative fluid losses significant
• Protein losses from exudative fluid loss
Treatment

• First 24 hours**
  – (4mL/kg x %BSA) + maintenance OR
  – 2000 mL/m² BSA + 5000 mL/m² TBSA
  – ½ over 1st 8 hours, rest over 16 hours
  – Crystalloid (first 24 hours) vs colloid (later)
  – Target urine output > 1 mL/kg/h
Electrical Burns

• Power source (lightning vs electrical), voltage (high vs low), current (alternating vs direct)
  - High voltage AC: conductive object touching overhead power line
  - Low voltage AC: children biting electrical cords, adults grounded while touching appliances
  - Direct current: third rail of electrical train system
Electrical Burn Sequelae

- High voltage AC burns cause devastating thermal injuries, not loss of consciousness or cardiac arrest.
- Low voltage AC burns can cause significant skin/oral injury particularly if strong enough to cause tetanic muscle contraction (16-20 milliamps):
  - Respiratory muscle paralysis (20-50 milliamps), ventricular fibrillation (50-120 milliamps).
Electrical Burn Sequelae**

- **DC burns** cause single muscle contraction that throws victim away from source.
- **Lightning injury** (neurologic effects)
  - **Mild**: superficial burns, loss of consciousness, amnesia, non specific neuro symptoms
  - **Moderate**: seizures, respiratory arrest, cardiac standstill that “self resolves”, superficial burns
  - **Severe**: cardiac arrest, long delay in CPR, high mortality
Management – Electrical Burns

• Same fluid management goals with focus on improving clearance of myoglobin (diuretics, urine alkalinization)
• Orthopedics/plastic surgery for wound care/fasciotomy
• Oral surgery for children for high frequency several oral trauma
• Supportive care
CARBON MONOXIDE POISONING
A 9 year old boy is brought via ambulance to the emergency room after being found unconscious in the basement of his house which was on fire. He has no visible burns and pulse oximetry shows a saturation of 95% on nasal cannula. Which of the following is true?

1. He does not have carbon monoxide poisoning since his saturation is over 90%.
2. Carbon monoxide increases the P50 of hemoglobin so hemoglobin will only release oxygen at much lower PO2.
3. Using a nonrebreather mask with carbon monoxide poisoning will decrease the half life of carbon monoxide to 90 minutes.
4. The lack of cherry red lips or skin rules out carbon monoxide poisoning.
5. Hyperbaric oxygen should be used since this patient was found unconscious.
Carbon Monoxide

- Colorless, odorless, tasteless, non irritating gas
- Binds to iron moiety of heme with 240 times the affinity of oxygen
- Diminishes the ability of the other three oxygen binding sites to release oxygen
- Causes deformation and leftward shift of oxyhemoglobin dissociation curve
Carbon Monoxide

- Spectrum of presenting symptoms: headache, malaise, mental status changes, myocardial ischemia, ventricular arrhythmias, profound lactic acidosis
- “Cherry red” lips and skin are insensitive signs
- Standard pulse oximetry will be normal
- Need co-oximetry
Carbon Monoxide

• Half-life of CO is 300 minutes breathing room air, 90 minutes with high flow non rebreather mask
• Hyperbaric oxygen for COHb levels > 25% (half-life decreases to 30 minutes)
• Prevention – home CO monitors
BITES AND STINGS
Human Bites

• 3rd leading cause of bites seen in ED
• Peak incidence 10-34 years of age
• Clenched fist injury (fist to mouth): small wound on dorsal metacarpophalangeal joint with risk for joint infection
• Occlusive bites that break skin: fingers and hands
• 10-15% rate of infection: eikenella corroden, staph, strep, corynebacterium, can transmit Hep B, HIV (rare)
Management – human bites**

• Minimize soft tissue deformity, especially bites to ear/nose (poor vascular supply)
• Antibiotics for high risk (bone/tendon/hand) vs all bites
  – Amoxicillin/clavulanate or ampicillin/sulbactam
  – Penicillin allergic: trimethoprim-sulfamethoxazole or quinolone and clindamycin
An 11 year old boy was on a camping trip and woke up to find a bat in his tent. You are called by his parents and asked if there is anything they should do. You:

1. Tell them to monitor him very closely for any signs of illness at which point he may need rabies prophylaxis.
2. Tell them to bring him to an ER immediately where he will be given rabies immunoglobulin and rabies vaccine.
3. Tell them to bring him to your office where you will arrange to administer rabies immunoglobulin if you see signs of a bite.
4. Tell them never to go camping again.
Rabies

• Highest case fatality rate of any infectious disease (i.e. everyone dies)
• 30,000-70,000 deaths per year worldwide
  – In developing countries, dogs account for 90% of cases transmitted to humans
• Almost all cases reported in the US are due to bat exposure
  – Other reservoirs in the US are raccoons, skunks and foxes
Rabies

• Number of different species of neurotropic RNA viruses
  – Enter local motor and sensory nerves from inoculation site
  – Migrate to spinal cord and then brain
• Progressively worsening encephalopathy
  – Encephalitic type, paralytic type, atypical type (often described with bat bites)
Milwaukee Protocol

• Two documented survivors of rabies infection (both children)
  – Drug-induced coma with ketamine and midazolam
  – Antiviral treatment with amantadine and ribavirin
Rabies**

- Post-exposure prophylaxis depends on:
  - Rabies epidemiology in region
  - Type of exposure
  - Provoked or unprovoked
  - Species and vaccination status of animal

- Consists of rabies immunoglobulin (passive immunization) into the wound or IM and rabies vaccine (active immunization) and good wound care
Domestic Animal Rabies

- Prophylaxis immediately if animal is rabid or if bite is to the head/neck region
- If animal is healthy, observe animal for 10 days and start prophylaxis if animal becomes rabid
- If animal is unavailable, consider prophylaxis if rabies common in region
Bats, raccoons, skunks, foxes

- Should be considered rabid unless available for testing
- Begin postexposure prophylaxis immediately after any bite, but especially for ANY contact with a bat (unless absolutely sure no bite or scratch occurred)
Snake bites

- 99% of all venomous bites in U.S. involve pit vipers (rattlesnakes, copperheads, water moccasins/cottonmouth)
- Release enzymes and spreading factors that start digestion from within (Mohave rattlesnake also releases neurotoxin)
- Rapid, intense local pain
- Perioral numbness, metallic taste
- Swelling, ecchymoses, vesicles in 8 hours
Snake bites

- Local necrosis, coagulopathy, rhabdomyolysis, nephrotoxicity, neurotoxicity if antivenom not given
- Crotalid antivenom – sheep immunoglobulin (Fab portion)
- Antivenom should be given if moderate to severe symptoms or bite to face or neck**
- Same dose for children and adults
- 6-8% incidence hypersensitivity reaction
A 2 year old boy is brought in to the emergency room after receiving a spider bite to his leg. The father describes the spider as black and shiny with red markings. The boy is crying and clearly in pain and there is a white patch on his leg that is surrounded by a warm, erythematous perimeter. He is refusing to eat or drink. The most appropriate next step in his care is:

1. Discharge the patient with close follow up
2. Admit the patient for pain control and IV hydration
3. Administer antivenom immediately
4. Begin antibiotics for infection of the bite site
5. Obtain surgical consultation for possible excision of tissue surrounding the bite
Spider Bites

• Brown recluse spider – brown with dark violin shaped mark on back, most accurately identified by six eyes vs eight eyes in other spiders
• Endemic in limited areas in South, West and Midwest
• Found mostly inside homes – basements, attics, behind bookshelves
• Rarely outdoors
BROWN RECLUSE SPIDER
• Venom contains sphyingomyelinase D, which can lead to necrosis

• Acute local reaction
  – Bite usually painless
  – Two puncture marks with surrounding erythema – becomes red plaque
  – Pain increases over 8 hour period
  – Lesion and pain resolve in 1 week
• Systemic symptoms – rare, small children more susceptible
  – Appears several days after bite
  – Malaise, nausea, vomiting, fever, myalgia
  – **Rarely** hemolytic anemia, DIC, rhabdomyolysis, renal failure, coma death
Necrosis

- Minority of lesions become necrotic over several days
- Original plaque becomes dusky red or blue with dry, depressed center
- Usually 1-2 cm in diameter
- Stop extending after 10 days, heal over several weeks usually without surgical intervention
3 days after brown recluse bite

4 weeks after brown recluse bite

From emedicinehealth.com
Management**

- Wound care, ice packs, pain control, possible tetanus prophylaxis
- Antibiotics only if appears infected
- Dapsone sometimes use for prevention and treatment of necrotic lesions (inhibits neutrophil chemotaxis and free radical generation)
- Early surgical intervention potentially harmful, evaluate after lesion has demarcated and stabilized
- Antivenom not available in U.S.
Widow spiders

• 30 species of widow spiders – female widows responsible for most significant bites

• Most adult widows are shiny black with red markings
  – American subtype has red hourglass or anvil shape on abdomen
  – Brown widow spiders also common in US, generally only cause pain w/o systemic sx's
BLACK WIDOW SPIDER
Black widow spiders

- Mostly in warm climates – very abundant in southwestern US
- Typically live outdoors surrounding homes
- Bites used to occur predominantly on the genitalia before indoor plumbing existed “There’s a redback on the toilet seat”
- Now bites mostly on peripheral limbs – reaching into pots, putting on gloves or boots
Local symptoms

• Clinical manifestation of a bite = latrodectism
• Painless bite or local pain
• Blanched patch with erythematous perimeter
• More generalized symptoms occur in 20-120 minutes
Generalized symptoms

- PAIN – abdominal, musculoskeletal, back
- Nausea/vomiting
- Generalized or local (limb of the bite) diaphoresis
- Headache
- Hypertension/tachycardia
- Rarely ileus, hematuria, compartment syndrome, rhabdomyolysis
- Death is rare, even in children
Venom properties

• Contains neurotoxins that causes massive exocytosis from presynaptic nerve terminals

• Acetylcholine, norepinephrine, dopamine, glutamate are all susceptible
Treatment**

- Most widow bites can be managed with supportive care – local wound care, pain management (benzodiazepines) and tetanus prophylaxis if indicated
- Antibiotics only if appears infected (direct towards skin flora)
- In the US, antivenom is reserved for severe envenomation that does not respond to other measures
- In Australia, almost all patients receive antivenom (1000/year)
Antivenom

- Recent reports of death due to allergic reaction to antivenom (in patients with asthma and other allergies)
- Give with caution and in consultation with poison control
- If antivenom doesn’t lead to death, pain and symptoms resolves in minutes to hours
- “what doesn’t kill you, makes you stronger”
Scorpion stings

• Lead to 10x as many deaths worldwide than snake bites (1/3 in Mexico)
• 25% of children < 5 years old die if not treated
• Venom with multiple toxins including very potent neurotoxin
• Mydriasis, nystagmus, hypersalivation, dysphagia
• Treat with local care, alpha and beta blockers, antivenom**
Jellyfish stings

• Portuguese man-o-war, box jellyfish
• Don’t pee on your friend or patient— use vinegar, coca cola, old wine**
• Treat anaphylaxis**
• Box jellyfish antivenom
HEAT STROKE AND MALIGNANT HYPERTHERMIA
Heat stroke

• Core body temperature > 40°C with central nervous system dysfunction
• Body’s ability to dissipate heat is overwhelmed
• Life-threatening emergency
• Non-exertional: underlying medical condition that impairs thermoregulation or prevents removal from a hot environment
Heat stroke

- Exertional heat stroke: athletes, military recruits

- Pathophysiology:
  - Hyperthermia directly induces cellular injury
  - Enzymatic function deteriorates
  - Uncoupling oxidative phosphorylation
  - Anaerobic metabolism predominates
Clinical findings

- Neurologic dysfunction is cardinal feature
  - Delirium, syncope, seizures, coma
- ARDS in later stages (poor prognosis)
- Hypotension, tachycardia, hypovolemic shock
- Rhabdomyolysis and renal failure
- Liver injury
- Disseminated intravascular coagulation
- Bacteremia and bacteruria after 24 hours
Treatment

• Care is supportive
• Maintenance of organ system perfusion/function and rapid cooling
  – Fluid resuscitation, “wet and windy”, internal cooling
Risk factors

- Lack of acclimatization
- Obesity
- Poor fitness
- Recent fever
- Sunburn
A 2 year old patient is admitted to your ICU after routine tonsillectomy complicated by acute vital sign changes in the operating room. Which of the following statements is FALSE regarding a diagnosis of malignant hyperthermia?

1. It can be triggered by local anesthetics
2. It is inherited in an autosomal dominant manner.
3. It is associated with central core myopathy.
4. Symptoms include mixed acidosis and hyperkalemia.
5. It is treated by administering dantrolene.
Malignant Hyperthermia

- Skeletal muscle hypermetabolism
- Triggered by succinylcholine and volatile anesthetics (sevoflurane)**
- Autosomal dominant inheritance of abnormality in skeletal muscle receptors
- Certain muscle disorders have similar abnormal receptors and those patients are MH susceptible – central core myopathy
MH Symptoms

- Early: tachycardia, muscle rigidity, mixed acidosis (rising ETCO2), hyperkalemia
- Late: hyperthermia, myoglobinuria, multiorgan failure
MH Treatment and Prevention

• Therapy is dantrolene plus cessation of triggering agents
• Avoidance in MH susceptible patients: clean anesthesia machines, have dantrolene available
• No good screening test for general public
TRAUMA
A 14 year old boy presents after being hit by a car on his way home from school on his bicycle. He was not wearing a helmet. There was a 2 minute loss of consciousness at the scene, but he is now alert and appropriate. There is tenderness and a hematoma over the left parietal region of his head. Thirty minutes later, he is lethargic and confused. What is the most likely diagnosis?

1. Epidural hematoma
2. Subdural hematoma
3. Subarachnoid hematoma
4. Concussion
5. Stroke
Primary Survey

• Rapid identification of potentially fatal conditions

• Child should be immobilized if not done in the field (cervical spine precautions and backboard)

• ABCDE
Airway

• Maintain airway patency with chin lift or jaw thrust maneuvers or with oral airway

• Endotracheal intubation for respiratory failure or impending airway compromise in head injured children
  – Uncuffed endotracheal tube (age in years/4) + 4**
  – Cuffed endotracheal tube (age in years/4) + 3**

• Premedication to blunt rise in intracranial pressure (barbiturate, lidocaine)
Breathing

• 100% oxygen should be delivered
• Assess respiratory rate, work of breathing, symmetry
• Suspect pneumothorax or hemothorax with decreased breath sounds or shifting of mediastinum
  – Place chest tube or needle emergently if unstable
  – Drain hemothorax cautiously
Breathing

• Flail chest: Multiple rib fractures lead to loss of stability of chest wall**

• Flail segment moves paradoxically:
  – Retracts with inspiration and bulges with expiration
  – Responds to changes in intrathoracic pressure vs respiratory muscle action (must use positive pressure ventilation)
Circulation

- Assess peripheral perfusion and rate and quality of pulse
- Acute volume loss is common cause of circulatory collapse
- Two large bore IVs, intraosseous early
- Aggressive fluid resuscitation: 2-5 20 mL/kg crystalloid boluses
- 10 mL/kg packed red blood cells if still unstable
Disability

• General assessment of neurologic function and level of consciousness

• AVPU
  – Alert
  – Responds to voice
  – Responds to pain
  – Unresponsive
Glasgow Coma Scale

- 3-15 point scale
- Eye opening (4 points)
- Verbal (5 points)
- Motor (6 points)
- Coma = GCS < 8, intubation indicated
Environment/Exposure

- Removal of clothing
- Warming lights/blankets if necessary
Secondary Survey

• Head: basilar skull fracture**
  – Battles sign, raccoons eyes, hemotympanum, CSF leak

• Abdomen: liver/spleen laceration**
  – Clinically stable children with Grade I-IV injuries (not shattered/ruptured) managed non-operatively with serial hematocrits
  – Children who require surgery declare themselves within 12 hours (blood loss, instability); emergent laparotomy with/without CT is indicated
Imaging for abdominal trauma

• Abdominal CT with IV contrast is preferred diagnostic imaging modality if patient is stable, indicated for:
  – Elevated transaminases
  – Gross/microscopic hematuria
  – Positive FAST exam
  – Declining hematocrit
  – Inability to perform serial exams (other injury, OR etc)
Intracranial hemorrhage

• Epidural hematoma**
  – Middle meningeal artery
  – Initial loss of consciousness, lucid interval

• Subdural hematoma**
  – Bridging veins
  – Slow deterioration

• Subarachnoid bleed
  – Severe brain injury, blood in CSF
Epidural hematoma

Subdural hematoma
A CUTE ABDOMEN
Errrrrr…..ACUTE ABDOMEN
Appendicitis

• Most common indication for emergent abdominal surgery in children
• Most frequent in second decade
• Risk of perforation higher < 4 yrs of age
• Caused by obstruction of appendiceal lumen
Clinical findings**

• Fever, vomiting, anorexia
• Periumbilical pain then right lower quadrant pain
• Physical exam:
  – Lying quietly
  – One or both hips flexed
  – Generalized abdominal pain with rigidity
  – Local tenderness at McBurney’s point
Clinical findings

• Classic signs:
  – Rovsing’s sign: pain RLQ on palpation of left side
  – Obturator sign: pain on internal rotation of right hip (appendix in pelvis)
  – Iliopsoas sign: pain on extension of right hip (retrocecal appendix)
Labs

• Elevated WBC with left shift
• RUA to rule out pyelonephritis (7-25% with appendicitis have pyuria)
• Pregnancy test
• CRP?
Scoring systems

- Pediatric appendicitis score (PAS) and Alvarado score use history, physical exam and laboratory results to predict appendicitis
- PAS shows variable results and Alvarado only used for adults for now
Imaging

- For children with typical presenting signs, obtain surgical consultation before imaging
- If unlikely to have appendicitis (no nausea/vomiting/anorexia, no RLQ pain, neutrophil count < 6750/mm3), can be managed without imaging
- For atypical presentations, imaging may be helpful
Imaging

• Ultrasound
  – First for non-obese children

• CT
  – First for obese children
  – If ultrasound cannot visualize appendicitis
  – If ultrasound normal, but clinical suspicion high
Treatment

• Pre-operative: fluid resuscitation, pain management, antibiotics
• Minimal delay in surgical intervention
• Post-operative: antibiotics, pain control, nutrition
Perforation

- Highest risk in children < 4 years
- Common with delayed presentation, especially after 72 hours
- Early appendectomy after initial stabilization and antibiotics
- 7-10 days of IV/PO antibiotics post operatively
Intussusception

- Most common abdominal emergency in children < 2 years
- Slight male predominance
- Occurs most often near ileocecal junction
- More proximal intussusceptum telescopes into distal intussuscepiens
- Venous and lymphatic congestion → intestinal edema → ischemia/perforation
Intussusception

- 75% idiopathic, up to 30% have preceding viral illness
- Lead point more common if > 5 years
  - Small bowel lymphoma, Meckel diverticulum, Henoch Schonlein purpura, cystic fibrosis
Clinical presentation

• Sudden, intermittent (15-20 minutes), severe, crampy pain
• Gross or occult blood in 70% (currant jelly = blood and mucous)
• Sausage shaped mass on right side
• In infants, can present as lethargy only
Diagnosis

• High index of suspicion – may not need additional radiologic studies
• Plain films: target sign, crescent sign, obstruction, dilated bowel
• Ultrasound: “bull’s eye”, lack of perfusion with duplex
Treatment

- Radiologic reduction with air contrast vs barium or water soluble contrast
- Air more successful with less complications
- Surgery for failed radiologic reduction or with perforation
- Risk of recurrence 1% after non-operative reduction
Midgut volvulus

- 50% of children with malrotation present within the 1st month with volvulus
- Small bowel twists around superior mesenteric artery
- Emesis most common presenting sign**
- Quickly leads to shock
Diagnosis

• Plain films: gasless abdomen most typical and nonspecific, “double-bubble” sign signifies duodenal obstruction

• Upper GI series: gold standard for diagnosis
  – Misplaced duodenum
  – Corkscrew sign

Radiologic evaluation should not delay intervention in severe cases
Treatment

- Surgery (Ladd procedure)
- Mortality rate 3-9%
- Recurrence rate 2-8%
Differential diagnosis - emergencies

• Ovarian torsion – female predominance
  – Dx with pelvis ultrasound
  – Palpable mass
  – Ovary often not salvageable, act quickly

• Testicular torsion – male predominance
  – Referred abdominal pain
  – Should examine scrotum

• Hemolytic uremic syndrome
Differential diagnosis

• Pancreatitis
  – Blunt abdominal trauma, gallstones in adolescent girls
  – Pain radiates to back, emesis
  – Lipase more specific than amylase

• Nephrolithiasis
  – Uncommon
  – Episodic pain

• Henoch Schoenlein purpura
  – Purpuric rash over legs and buttocks
  – Episodic pain with emesis
  – Rarely associated with intussusception
Brain Death
You are caring for a 9 year old girl who was involved in a severe motor vehicle accident. Based on her clinical status, you are beginning a brain death evaluation. Her parents have many questions about the diagnosis and process of declaring brain death. Which of the following is TRUE regarding the declaration of brain death in children?

1. The declaration of brain death for this patient requires two exams performed by different physicians with a minimal observation period of 24 hours between exams.
2. An ancillary study, like EEG or cerebral blood flow exam, is required for the declaration of brain death.
3. For an apnea test to be consistent with brain death, the PCO2 must rise more than 20 mmHg above baseline and be greater than 60 mmHg with no respiratory effort noted.
4. A patient must be normotensive with a temperature over 34 degrees Celsius to be declared brain dead.
5. The first brain death exam may be performed 12 hours after severe brain injury.
Brain death in children

• Possible contributing factors must be corrected:
  – Core temp over 35 degrees Celsius
  – Systolic BP or MAP in acceptable range for age (not less than 2 SD below norm)
  – Sedative/analgesic drug effect excluded
  – Metabolic intoxication excluded
  – Neuromuscular blockade excluded
Brain death exam

- First exam may be performed 24 hours after CPR or other severe brain injury
- 2 exams + apnea test performed by different physicians (apnea test can be performed by same physician)
  - 24 hour observation period if 37 weeks – 30d
  - 12 hour observation period if 30d-18 year
Apnea test

• Provide oxygen, do not extubate
• Consistent with brain death if:
  – No spontaneous respiratory efforts noted
  – Final PaCO2 > 60mmHg
  – Final PaCO2 >20mmHg increase over baseline
Ancillary testing**

- EEG or radionuclide cerebral blood flow study
- Required if apnea test contraindicated or cannot be completed due to hypoxia or hemodynamic instability
- Required if uncertainty about exam
- Required if medication effect may be present
- Optional to reduce inter-examination period
Finally…. 

• “Assess ABC’s” is always a good answer 
• “Give 20 cc/kg NS” is almost always a good answer 
• “Get ophtho consult” is almost always a good answer 
• Pay attention to all information given 
• GOOD LUCK!