

# Critical Care Pediatrics

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# Importance of Early Recognition of System Failure

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- Outcomes in pediatric cardiopulmonary arrest are generally poorer than in adults.

# Projected Impending Existing Failure

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- Respiratory Failure
- Circulatory Failure
- CNS Failure
- Organ Failure

# Respiratory Failure

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- **Inadequate oxygenation and CO<sub>2</sub> excretion**
- **Respiratory distress is not always present**

Respiratory failure causes most of the cardiopulmonary arrests and involves most of the critical pediatric arrests. The respiratory system needs to maintain oxygenation and excrete carbon dioxide. Hypoxemia may be occult. It takes a high degree of hypoxemia before the respiratory center is stimulated to generate the kind of ventilatory response that will identify clinically. So there are some babies whose oxygen saturation is in the low 90's or the high 80's who may have no symptoms as a result of the hypoxemia. And therefore we try to keep a high index of suspicion for respiratory disease and for hypoxemia, especially in young infants.

# Causes of Airway Obstruction

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- Congenital Lesions
- Acquired Lesions
- Infections
  - Epiglottitis
  - Laryngotracheobronchitis (croup)
  - Retropharyngeal Abscess
  - Bacterial Tracheitis

Now when we talk about respiratory failure I think it makes sense to start with the upper airway and then work our way down through the respiratory tree to the alveoli. An airway obstruction can be caused by any number of different lesions. Congenital lesions are numerous. Take for instance a child born with bilateral choanal atresia. The child has severe respiratory distress, which may be caused by bilateral choanal atresia. Newborn infants are almost exclusively obligate nose-breathers, and, therefore, bilateral choanal atresia would be an example of a congenital lesion that would cause upper airway obstruction. There are various acquired lesions. They could be mass lesions or infectious lesions, which cause inflammatory conditions that obstruct the airway.

There's always a lot of focus on epiglottitis. We've seen one case of epiglottitis in our emergency department that treats 50,000 kids a year. One case of epiglottitis in the last four years. But as a model for upper airway obstruction I think all that you learned about epiglottitis pertains to other inflammatory conditions.

Laryngotracheobronchitis, or croup, is a much more common cause of upper airway obstruction. On rare occasions viral laryngotracheobronchitis can be life-threatening. And in those cases they may need critical care intervention to maintain the airway. Steroids or epinephrine may be required to reduce the inflammatory obstruction of the upper airway.

This is a child who comes in with difficulty swallowing and fever. He demonstrates a fever and swelling of the pharyngeal soft tissues, consistent with a retropharyngeal abscess. Retropharyngeal abscesses usually occur in younger children because of lymphatic drainage. Older kids with perforating injuries also can develop soft tissue infections which in some cases can obstruct the upper airway.

# Causes of Acquired Airway Obstruction

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- Neurologic Dysfunction - loss of pharyngeal tone
- Trauma
- Foreign Body Aspiration
- Mass Lesion

Airway obstruction can also be caused by a loss of the pharyngeal tone. There is a very complex, integrated neural network by which on the start of inspiration the upper airway dilates and thereby keeps the airway patent for expired air. If the integrated neural network of dilation of the upper airway on inspiration is not functioning, the mandibular soft tissue will fall back on the posterior pharynx, leading to airway obstruction. Neurologic problems can cause loss of pharyngeal tone and airway obstruction from a number of causes. Occasionally there is acute onset, instantaneous onset of severe respiratory distress from foreign body aspiration in the upper airway. It is more common for foreign bodies to lodge in the lower airways, in which case the presentation may be a little less dramatic. And certainly when you hear a child has instantaneous onset of severe respiratory distress, it's a foreign body in a central area.

Mass lesions tend to develop and lead to a chronic, progressive upper airway obstruction. Sometimes the signs or symptoms can be pushed backwards to the past few weeks. In think one example of that is adenoid-tonsil hypertrophy where nighttime airway obstruction can be reported by parents.

# Management of Airway Obstruction

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## ■ Positioning

- Head Tilt
- Chin Lift
  - Lifts base of tongue off posterior pharyngeal wall
- Clear Secretions

## ■ Bag-Valve-Mask Ventilation

- Self-inflating bags
- Anesthesia bags

## ■ Intubation

- Suction
- Oxygen
- Bag-Valve-Mask
- Laryngoscope
- Blades
  - Straight
  - Curved
  - Endotracheal Tubes: 16 + age (yrs)

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Now, what do we do for a child who has upper airway obstruction? Responding to any emergency we want to immediately open the airway and maintain airway patency even before an artificial airway is inserted. In the delivery room and in the emergency departments and in severely ill children who show up in the office, you want to position the child in an optimal way, and that can be either by tipping the head back or pulling the tongue forward, or both. This helps to move the tongue off the posterior pharynx. A small movement, like positioning a child in the manner in which the shoulders are elevated, can make a big difference in terms of the ability of that child to maintain a patent airway. You want to clear any secretions with suction, and we want to use bag-valve-mask ventilation if necessary.

Children who have severe airway obstruction, more severe with respiratory distress and respiratory failure, may require intubation. Intubation is something that should be done in as controlled a manner as is possible. If you are not working in a hospital and there are more experienced intubators in the building, I think it makes sense to get the most experienced person there when possible. You want to have suction, oxygen available, and bag and masked ventilation available. It takes some time to properly put in an endotracheal tube. So you want the functional residual capacity of the child, that reservoir of oxygen in their lungs that maintains oxygenation despite a period of apnea or inadequate ventilation, you want the FRC to be full of 100% oxygen. You want to pre-oxygenate with bag-valve-masks. You want to make sure the laryngoscope works before you begin intubation.

Now there are different blade types. Most pediatricians like myself, are more comfortable with a straight blade. In older children, the curved blades in some ways are easier, where the curved blade is replacing the vallecula in the elevation because the epiglottis is somewhat rigid. It lifts up and lifts off the pressure on the trachea at intubation.

In the 16+ age in years, half a tube and a half size smaller and a half size larger for routine or emergent intubation. If the child has signs of airway obstruction, especially in the case of something like epiglottitis or perhaps croup, you may want to have a tube a whole size smaller or even one and a half sizes smaller, recognizing that there may be a narrowed area, making the predicted tube size too large.

# Causes of Lower Pulmonary Disease and Respiratory Failure

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- Bronchiolitis
- Asthma
- Pneumonia
- Acute Respiratory Distress Syndrome
- Aspiration/Near Drowning

Now as we move down the respiratory tree, there are a number of other causes of respiratory failure. And most of the causes of respiratory failure, both upper and lower, are more common in young infants. In fact if you look at all cardiopulmonary arrests, infants far outnumber any other age range. And so treat all these conditions, certainly bronchiolitis and asthma and cardiacs, with great caution.

# Respiratory Pump Dysfunction

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- The chest wall is much more compliant in infants
- Paradoxical respirations or thoracoabdominal asynchrony occurs when airway resistance becomes increased

In addition to the problems with the respiratory tree and the alveoli, critically ill children may have problems with their respiratory pump, the bellows of their chest. The chest wall, especially in younger infants, is so compliant that there are problems when there is high airway resistance. And so with upper or lower obstruction, often we have to ventilate the child with very high intrathoracic pressures to entrain air into the lungs. And if the chest wall itself is very pliant, you may have paradoxical respirations.

# Chest Trauma

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- Chest trauma is most commonly caused by a blunt mechanism, such as a motor vehicle accident.
- Chest trauma is usually associated with other injuries.
- Chest trauma is more likely to injure both intrathoracic and abdominal organs.

Chest trauma. Most chest trauma in children is blunt trauma. And again, because of the compliance of the chest wall, force is more likely to be transmitted to the internal organs. And the good news is that the compliant chest wall seldom results in fractured ribs. Fracturing a young infant's ribs takes considerable force, but internal injuries to the thorax are more common because the chest wall doesn't serve to protect the thorax as well.

There is an intrathoracic component to the abdominal structures, so the liver and the spleen are really tucked up into the thorax. After chest trauma in a small patient, one needs to be concerned about injuries to abdominal structures that are within the thorax. Pulmonary contusion is one of the more common, significant chest injuries that occur. It occurs frequently without rib fractures, again because of the compliance of the chest wall. Occasionally there will be tachypnea or hypoxemia without a definite abnormality on chest exam. Perhaps there are decreased breath sounds. The chest x-ray may lag behind these clinical findings, so we need a high index of suspicion of pulmonary contusion in a young child who had suffered blunt trauma to the chest. In addition to pulmonary contusion, occasionally there will be rib fractures. Most rib fractures in young infants are inflicted. Fracture of the lower ribs are often associated with abdominal visceral injuries. And occasionally there are so many rib fractures that the bellows function of the chest is disrupted. With multiple rib fractures, which is quite common in young infants, you may have a problem with bellows function.

Pneumothoraces are not that common, especially in very young children. As children get older they become somewhat more common. They may occur without a rib fracture, but it must be excluded in patients with significant chest trauma. Tension pneumothorax develops because of the tension, and shifts the mediastinum over. There is respiratory failure associated with living off one depressed lung and there is also a circulatory failure associated with the obstruction of the superior and inferior vena cava and inflow to the heart. So the tension physiology needs to be relieved as soon as possible. If there's tension in the thorax, a chest tube or a needle should be placed in the chest.

Hemothoraces are uncommon in younger kids. Often shock is the presenting sign, rather than respiratory failure from blood loss.

# Chest Injuries

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## ■ Pulmonary Contusion

- Chest wall findings may be absent
- Tachypnea, asymmetric breath sounds, and hypoxemia may be present
- Symptoms and radiographic changes may be delayed

Cardiac injuries are quite rare. In children we seldom see clinically significant cardiac contusions, but they can occur. *Commotio Cordis* or cardiac concussion. I like to think of it as an analogies to what we used to do with adult arrests was deliver a precordial thump. With a precordial thump, more than an amp of electrical energy is discharged to the heart. In the fibrillation situation, a precordial thump may defibrillate the patient. It works probably infrequently, but there are anecdotal reports of it working. Similarly a precordial thump say from a pitched baseball that hits right on the sternum, may deliver a small electrical burst of energy which can trigger fibrillation and sudden death in young children. And so the story of a child being hit by a baseball in the chest, dropping unconscious and dying from immediate fibrillation after trauma, is what we call *Commotio Cordis*, and it is probably by a similar mechanism.

## Rib Fractures

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- Rib fractures in infants are usually intentionally inflicted by another person
- Severe intrathoracic injury may occur without rib fractures
- Fractures of the lower ribs are often associated with abdominal visceral injuries
- Multiple fractures can cause a flail chest, resulting in ineffective respiratory mechanics

Chest trauma causes problems with bellows function. Obviously children with neuromuscular disease can have respiratory failure. Children with simple hypoventilation need to have assisted ventilation, obviously, or they will arrest. And children with spinal cord injuries or any of these other conditions may have a central cause of their hypoventilation.

# Pneumothorax

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- Most occur occurs in the absence of a rib fracture
- Untreated tension pneumothorax may be rapidly fatal

# Hemothorax

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- Hemothorax is usually caused by penetrating trauma
- Shock usually occurs before respiratory failure

# Cardiac Injuries

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- Cardiac injuries are rare
  - Commotio cordis
  - Cardiac contusion

Circulatory failure. Children do not have a lot of inotropic reserve. Their cardiac output increases mostly through an increase in the heart rate. And so when children have circulatory failure, tachycardia is the most reliable symptom to look for. Although absence of tachycardia doesn't absolutely exclude the possibility of circulatory failure. Shock can be compensated or uncompensated. And clearly children who present with shock and low blood pressure do very poorly. So our role in emergency medicine or critical care is to identify kids with shock before blood pressure drops. We need to identify shock early, looking for tachycardia and decreased perfusion of the skin. That's capillary refill, color and temperature of the peripheral extremities. Altered sensorium and decreased urine output is obviously critical in a child with projected, impending or existing circulatory failure.

# Neuromuscular Respiratory Muscle Failure

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- Central Hypoventilation
- Spinal Cord Injury
- Tetanus
- Poliomyelitis
- Guillain Barre
- Myasthenia Gravis
- Botulism

# Circulatory Failure

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- Cardiac Output is rate dependent in infants
- Compensated vs. uncompensated shock
  - Recognize shock before blood pressure drops

## Clinical Manifestations of Circulatory Failure

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- Tachycardia
- Decreased perfusion of skin (temperature, color, capillary refill)
- Altered sensorium
- Decreased urine output

# Vascular Access

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- Peripheral
- Central
- Intraosseous
- Endotracheal

Much more fluid can go through 18 gauge IV and a 20 gauge IV, over a given period of time. Similarly the length is also of importance, so very long central lines, even if they are large bore, have some added resistance by virtue of their length. So larger IV's are better for rapid fluid administration. We know now, and this is not new, is that the bone marrow can be used for vascular access when the peripheral veins are difficult to get, and spinal needles, or hypodermic needle or a bone marrow needle preferably placed in the bone marrow very rapidly gains access to the central circulation. Fluid or medication administered to the bone marrow is more like administering it to a central line than to a peripheral line. Even a child with no arms and legs can have vascular access by putting a bone marrow needle in the iliac region of the hip.

# Causes of Circulatory Failure

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## ■ Inadequate Blood Volume - Hypovolemic Shock

- Dehydration
- Hemorrhage
- Third Spacing

## ■ Inadequate Pump Function - Cardiogenic Shock

- Cardiomyopathy
- Dysrhythmia
- Tamponade

## ■ Inadequate Vascular Resistance - Distributive Shock

- Drug Overdose
- Toxic Shock
- Septic Shock

Circulatory failure takes many forms, hypovolemia and in particular, dehydration is something we deal with on a daily basis. There is a lot of overlap between an ill-appearing child and dry child. Certain tenting of abdominal skin has been used for a sign. In addition to hypovolemia, cardiogenic shock, as appears in cardiomyopathy, a rhythm disturbance or tamponade, are also causes of cardiogenic shock. This is a child with a large heart and some fluffy infiltrates in the beginning of congestive heart failure.

# Septic Shock

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- Early "warm" shock is caused by decreased vascular resistance from cytokine release
- Late "cold" shock is caused by hypovolemia from capillary leak, acidosis, myocardial depression

# Central Nervous System Failure

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- Asphyxia
- Trauma
- Toxic/Metabolic
- Status Epilepticus

# Head Trauma

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- Primary vs. Secondary Injury
- Outcome is usually related to concurrent hypoxemia hypercapnia, duration and degree of coma (Glasgow Coma Scale)

Head trauma causes primary injury that occurs instantaneously and then a secondary injury. Secondary injury after head trauma occurs from swelling and poor perfusion and poor airway management. The outcome in many cases is delayed swelling, hypoxemia, hypercapnia. This is just taken from one study showing head trauma plus hypotension, hypoxemia, hypercapnia. These have a very high mortality rate. Head trauma alone would seem to have a much lower mortality rate. Physiology of head trauma is important. Intracranial volume is a constant so anything that increases in the brain, something else has to decrease or the pressure will go up dramatically. And perfusion of the brain is really a difference between a arterial pressure and the intracranial pressure. All increases in intracranial pressure therefore reduce cerebral perfusion pressure. But the arterial reductions can also reduce cerebral perfusion factor. So injured children need to maintain the arterial pressure and do it because it minimizes the intracranial pressure.

# Head Trauma

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- Pathophysiology
  - Intracranial volume is constant
  - Intracranial compliance curve is exponential
  - Cerebral Perfusion Pressure = Mean arterial pressure - ICP
  - Edema peaks 48 - 72 hrs after injury

# Head Trauma

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## ■ Signs and Symptoms of Brain Injury

- History of loss of consciousness
- Retrograde amnesia
- Prolonged vomiting
- Subtle alterations in consciousness are important
- Glasgow Coma Score
- Herniation/Brainstem reflexes

Any child with a loss of consciousness we will obtain a CT-scan. We are not talking about a couple of seconds of being dazed and confused, but a true loss of consciousness, which actually is quite, quite significant. Other signs of concussion are defined here. And we certainly, on the severely head injured children, look for signs of herniation.

# Brainstem Reflexes

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- Pupillary Response
- Oculocephalic Reflex (Doll's eyes)
  - Positive if eyes lag behind movement of the head
- Oculovestibular Reflex (cold calorics)
  - Positive if cold water causes sustained deviation of eyes toward the irrigated ear

The herniation that is most importantly identified is uncal herniation where the third nerve gets depressed. You look for a pupillary response. Once we have cleared the neck we look for an oculocephalic or doll's eyes reflex. And when we talk about doll's eyes we are talking about a normal child, if the brain stem is intact, the eyes will stay in position as the head moves and so the eyes will match the plane of the movement of the head. And a child who has a nonfunctioning brain stem, the eyes will be painted on the face and they will move with the turning of the head. So doll's eyes is positive if the eyes move relative to the head as opposed to staying fixed in position. And then a squirt of cold water in the ear should cause the eyes to deviate towards the cold water in a sustained fashion and occasionally have a fast phase away.

# Head Trauma Management

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- Airway management is crucial
- Treat elevated intracranial pressure before Cushing's Triad or uncal herniation occur
- Elevate head
- Hyperventilate to maintain a  $p\text{CO}_2$  of 25-30 mmHg
- Mannitol - osmotic diuretic

Airway management in head injuries is crucial. You want to treat intracranial pressure before Cushing's Triad--hypotension, bradycardia--occurs and we want to prevent uncal herniation whenever possible. Elevate the head, hyperventilate between  $p\text{CO}_2$  25 and 30. Extreme hyperventilation causes too much vasoconstriction and that can cause poor perfusion in the brain.

# Organ Failure

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## ■ Renal Failure

- Prerenal
- Intrarenal
- Postrenal

## ■ Hepatic Failure

- Causes
  - Infectious (viral)
  - Toxic (acetaminophen, iron)
  - Ischemic

Organ failure. When you are dealing with kidney failure, the patient usually can get critical, and you want to decide whether it's prerenal, which is characterized usually by hypervolemia and high ratio of BUN to creatinine. Intrarenal where there may have been an ischemic insult. Postrenal failure can usually be defined through an imaging study.

# Signs and Symptoms of Hepatic Failure

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- Protracted vomiting
  - Behavior change - euphoria, belligerence, confusion
  - Bruising
  - Edema, Ascites
  - Asterixis
  - Coma. The depth of encephalopathy correlates with the severity of hepatic failure

For liver failure, the causes include viral hepatitis, toxic conditions, especially acetaminophen and iron. And as part of a toxic ischemic event. It's important to recognize that this type of failure can have a solo presentation of vomiting and a solo presentation of unusual behavior change. Other findings include easy bruisability, edema and ascites. Asterixis occurs in the severe phase, which is the flapping of the hands and coma. Often the severity of the failure, as related to the depth of encephalopathy, correlates with the severity of the hepatic failure and is somewhat related to the height of the pneumonia.

# Hepatic Failure

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## ■ Laboratory Tests

- The height of the transaminase elevation does not correlate with severity of disease
- Elevated conjugated and unconjugated bilirubin, prolonged prothrombin time and decreased albumin are indicators of liver dysfunction
- Decreasing transaminases with increasing bilirubin and PT suggest liver failure
- Increased arterial ammonia correlates with hepatic coma

Recognize that high transaminase is liver enzymes. They are not truly liver function tests; these are enzymes that leak from the hepatocytes. So very high transaminase levels doesn't necessarily correlate with very severe disease. We get worried with the pre-liver function tests. The way they function is to conjugate bilirubin and synthesize various proteins. When we see elevated conjugated and unconjugated bilirubin, prolonged coagulation times and low albumin, which really reflects liver synthesis function. Arterial ammonia results are very reasonable for documenting the severity of the hepatic coma.