Children's
HOSPITAL & MEDICAL CENTER

We know children.

Neonatal Emergencies and Transport

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Relative Anatomy and Physiology

Physiology of Thermoregulation

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- Neonate at significant risk of hypothermia
  - Ratio of neonatal body surface area to volume is four times that of an adult
  - Neonate has less adipose tissue than adult
  - Thermogenesis in neonate only one and a half as high as adult
  - Muscle tone is immature in neonate
  - Neonate cannot shiver effectively enough to generate heat

Heat Loss in the Neonate

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- Results from:
  - Evaporation
    - Most of heat loss, especially in moments immediately after birth
  - Convection
    - Depends on birthing environment
    - When care providers are comfortable in the room, it is too cold for the neonate
  - Conduction
  - Radiation
    - Room's ambient temperature should be as close to core temperature as possible

Heat Loss in the Neonate

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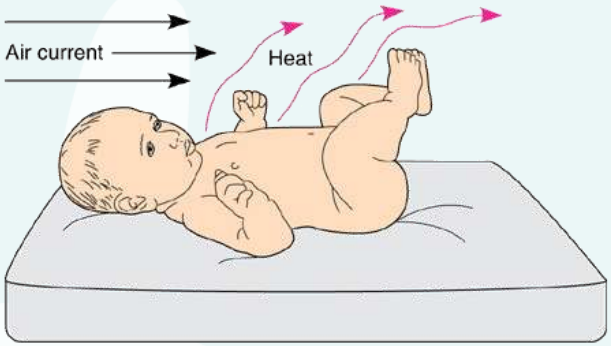
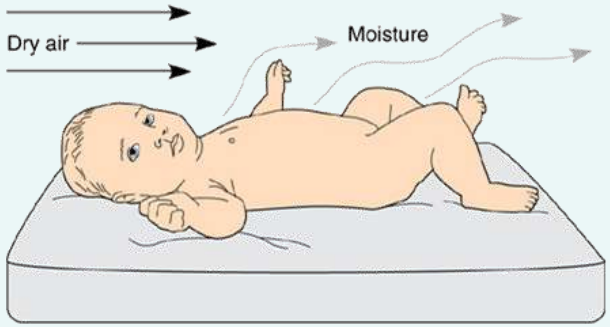
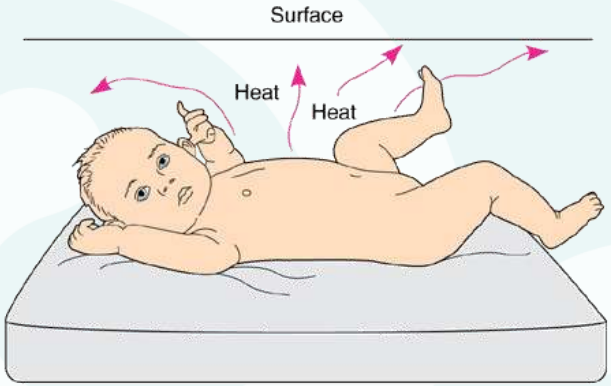


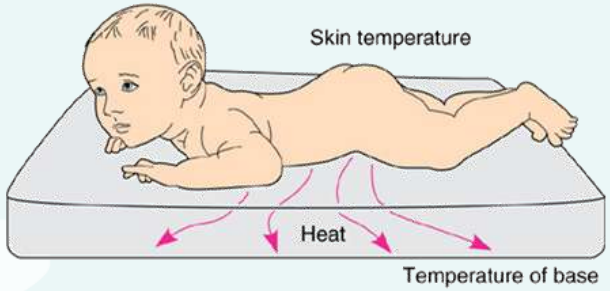
Convection



Evaporation



Radiation



Conduction

Glucose Requirements

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- Newborns at significant risk of acute hypoglycemia due to:
  - Poor glucose stores
  - Inability to stimulate the immature neonatal liver to release glucose
  - Increased metabolism that uses large quantities of available glucose
    - Assess neonatal glucose levels within 1 to 2 hours after birth
      - Reassess every 30 minutes to 1 hour thereafter until glucose levels are normal
    - Neonate blood glucose levels (BGLs) should be maintained above 70–80 mg/dL

Signs and Symptoms of Hypoglycemia

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- Twitching, seizure activity, eye rolling
- Muscular hypotonia (limpness)
- High-pitched cry
- Respiratory apnea, irregular respirations

Management of Hypoglycemia

- Administer 10 percent dextrose as needed at 80ml/kg/day

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Airway Anatomy and Physiology

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We know children.

- Unique differences between neonatal and adult airway anatomy and physiology
  - Neonatal tongue larger compared to the oropharynx
  - Little room for airway edema
  - Increased likelihood of airway obstruction in depressed neonate
  - Neonatal trachea more pliable, narrow
  - Airway obstruction from:
    - Hyperextension, hyperflexion kinking
    - Edema

Airway Anatomy and Physiology

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We know children.

- Neonatal epiglottis is large and more U-shaped or oblong, floppy from incomplete cartilaginous support
- Use of straight versus curved blade during laryngoscopy
- Neonatal larynx more cephalad, anterior
- Level of first or second cervical vertebrae
- Harder to achieve single plane view needed for optimal orotracheal intubation conditions

Pulmonary Anatomy and Physiology

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We know children.

- Many differences in neonatal pulmonary anatomy and physiology compared to the adult
- Bones in neonatal thoracic cavity not fully calcified
  - Flexible
- Neonatal ribs are more horizontal than they are rounded
  - Little leverage to increase the anterior and posterior diameter of the chest
  - Inability to provide the degree of lift needed to increase the volume of the chest cavity upon inspiration

Pulmonary Anatomy and Physiology

- Poorly developed accessory muscles
  - Cause diaphragmatic breathing
- Neonatal sternum very pliable
  - Contributes to inability to create a strong negative intrathoracic pressure
  - Inhibits efficiency of inspiratory effort

Pulmonary Anatomy and Physiology

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We know children.

- Neonates have diminished pulmonary reserve capacity
  - Heart larger, ribs and sternum fail to adequately support the lungs
  - Less space for lung expansion compared to adults
  - More rapid development of hypoxemia and hypercapnia
- Neonates are primarily abdominal breathers
  - Rely heavily on diaphragmatic motion to breathe
  - Overcrowding of the neonatal abdominal cavity a significant problem
  - Negatively affects the neonate's compensatory ventilation mechanisms
  - Limits diaphragmatic excursion secondary to increased abdominal pressure

Pulmonary Anatomy and Physiology

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We know children.

- Neonates consume twice the oxygen of adults
  - Lower pulmonary reserve capacity coupled with a higher metabolic demand for oxygen predisposes the neonate to hypoxemia

Cardiovascular Anatomy and Physiology

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We know children.

- Several differences between adult and neonatal cardiovascular systems
- While still in utero, the fetus receives its oxygen through the placenta
  - Disturbances to alveolar ventilation and gas exchange following birth must be dealt with immediately

Cardiovascular Anatomy and Physiology

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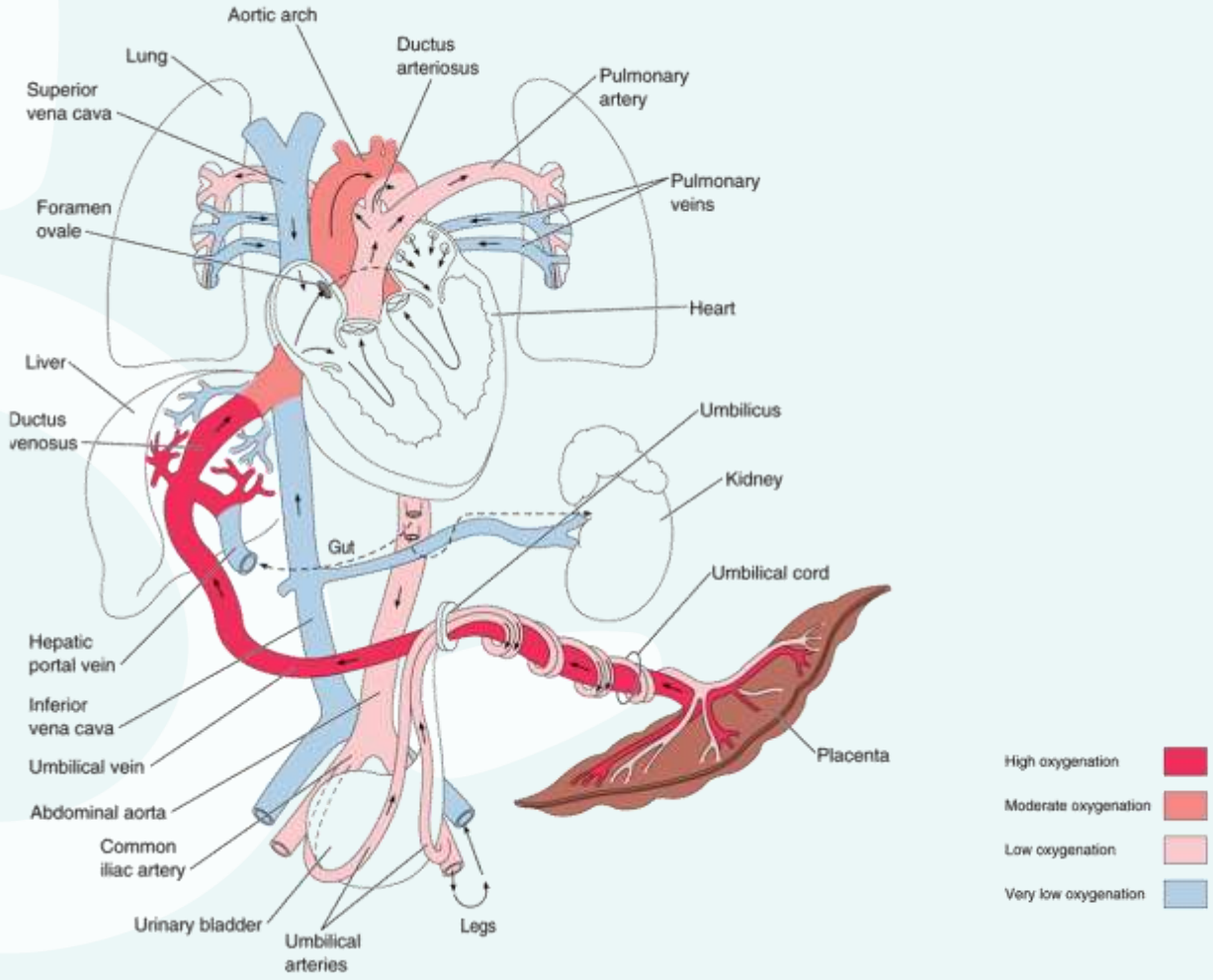

Cardiovascular Anatomy and Physiology

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We know children.

- Neonatal heart can usually only increase rate to improve cardiac output
  - Cannot increase contractile force
  - Cardiac output drastically reduced with bradycardia

Cardiovascular Anatomy and Physiology

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We know children.

- Most of physiologic change that occurs with the shift from intrauterine to extrauterine life occurs in the first few minutes after delivery
  - Clamping of umbilical cord moves circulation from placenta to pulmonary system
  - Interruption of low-resistance, placental blood flow from the umbilical cord increases systemic vascular resistance (SVR)
  - Increased SVR closes the ductus venosus
  - Closure of ductus venosus causes renal perfusion

Cardiovascular Anatomy and Physiology

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We know children.

- Neonate's first breaths expands the lungs
  - Lung expansion reduces pulmonary vascular resistance
  - Reduced pulmonary vascular resistance:
    - Increases pulmonary blood flow
    - Reduces pulmonary artery pressures
      - Left side of heart assumes higher pressures than right
    - Closes the foramen ovale
    - Closes the ductus arteriosis
      - Occurs in first hours to weeks after birth

General Pathophysiology: Pulmonary

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- Assessment of respiratory distress
  - Etiology of respiratory compromise may not be readily identifiable
    - First goal is to replace any lost function of the airway or breathing components
    - Once airway or breathing insult is corrected, can identify potential causes of the hemodynamic and/or respiratory compromise
      - Goals in managing respiratory compromise in the critical care environment are to:
        - » Identify a set of causes and
        - » Treat the patient based on the most likely etiology

Respiratory Distress, Failure, and Arrest

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We know children.

- Must use precise terms when describing respiratory distress, respiratory failure, and respiratory arrest
  - Distinction between the three dictates the management of the acutely ill neonate
  - Respiratory distress
    - Maintains the ability to compensate
  - Respiratory failure
    - Has exhausted compensatory mechanisms
  - Respiratory arrest
    - Patient is apneic

Persistent Pulmonary Hypertension of the Newborn

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We know children.

- Clinical syndrome in which pulmonary vascular resistance is elevated in the presence of changes in pulmonary vessel reactivity
  - Results in sustained fetal circulation
  - Ductus arteriosus and foramen ovale remain open

Persistent Pulmonary Hypertension of the Newborn

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We know children.

- Commonly associated with severe hypoxia, meconium aspiration syndrome, and congenital diaphragmatic hernia
- Clinical presentation mirrors many of the signs and symptoms of congenital heart diseases
  - May be difficult to assess in the aeromedical or ground transport environment

Persistent Pulmonary Hypertension of the Newborn

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We know children.

- Management
  - Maintain oxygenation
  - Give nitric oxide
    - Promotes pulmonary vascular dilation
    - Keeps pulmonary perfusion pressures closer to normal
    - Closes vascular structures
  - Use adenosine, magnesium sulfate as pulmonary vasodilators

Meconium Aspiration Syndrome

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We know children.

- Meconium expelled prematurely in 10 to 15 percent of all deliveries
  - Only 2 to 10 percent will aspirate meconium into lower airways
- Meconium aspiration can obstruct airway and/or may contribute to inactivation of alveolar surfactant
- No known prevention strategies
  - Nasopharyngeal and endotracheal suctioning before delivery of the thoracic cavity may limit meconium aspiration into the lower airways

Meconium Aspiration Syndrome

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We know children.

Following delivery

After delivery of the infant, if a great deal of meconium is present, the trachea should be intubated and any residual meconium removed from the lower airway.

Transient Tachypnea of the Newborn (TTN)

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We know children.

- Also known as “wet lung” or “Type II Respiratory Distress Syndrome”
- Self-limiting process
  - Auto-resolves within 48–72 hours from birth
  - Caused by delayed clearing of fluids in the lungs
- Management
  - Ensure adequate oxygenation
  - Give antibiotic therapy until sepsis, pneumonia ruled out

Infant Respiratory Distress Syndrome (IRDS)

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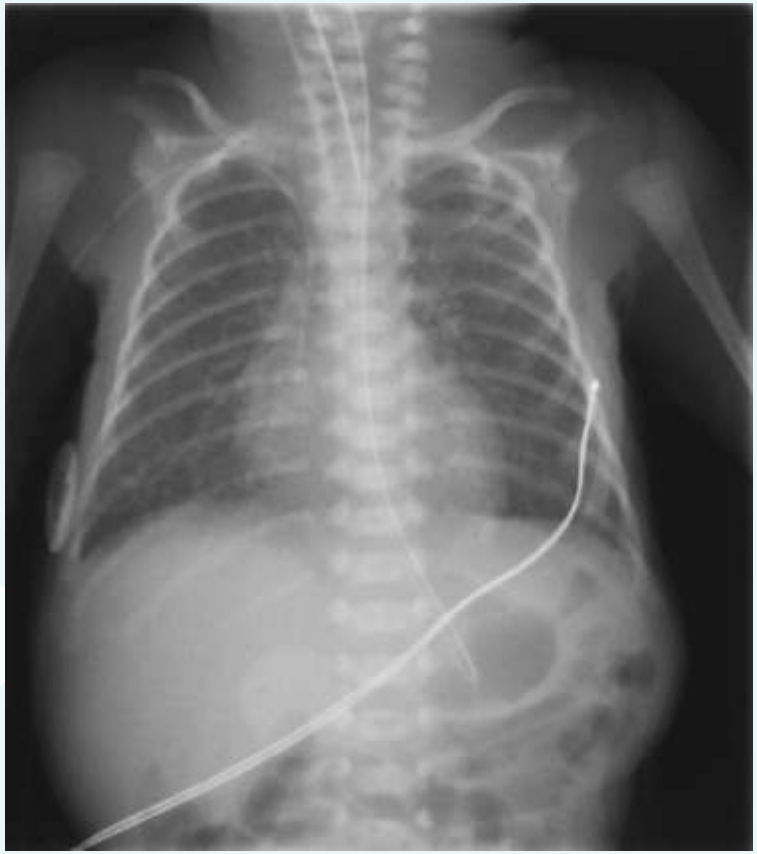
We know children.

- Affects about 10 percent of all preterm infants
  - Rarely seen in full-term infants
- Result of lack of pulmonary surfactant
  - Causes atelectasis
  - Increased work of breathing
  - Ineffective gas exchange
  - Hypoxia, hypercapnia



Courtesy of Carol Harrigan, RNC, MSN, NNP

Infant Respiratory Distress Syndrome (IRDS)

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We know children.

- Signs and symptoms include:
  - Tachypnea, shortness of breath
  - Accessory muscle use, sternal retractions, grunting, nasal flaring
  - Respiratory arrest from muscle fatigue, hypoxemia, and acidosis
- Management
  - Ensure adequate ventilation and oxygenation
  - Administer exogenous surfactant

Congenital Diaphragmatic Hernia

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We know children.

- Complication in which the bowel protrudes into the thoracic cavity through an interruption of the diaphragm
  - Usually the result of congenital abnormality
  - 85 percent of all congenital diaphragmatic hernias occur on left side
  - Mortality rate between 40 and 60 percent
- Herniated abdominal contents prevent full lung expansion in the affected hemithorax
  - Pulmonary compromise ensues

Congenital Diaphragmatic Hernia

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We know children.

- Signs and symptoms
  - Respiratory distress
  - Unequal lung sounds
  - Scaphoid shaped abdomen
- Management
  - Ensure adequate ventilation and oxygenation
  - Insert NG tube
  - Conduct gastric decompression
  - Repair surgically (definitive treatment)
  - General pathophysiology, cardiovascular

Congenital Heart Disease Overview

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- Incidence of congenital heart disease in the United States is approximately 8 per 1,000 live births
  - About 40,000 neonates born each year with a heart defect
    - Many congenital heart defects are subclinical
    - Defects can cause:
      - Abnormalities in volumes and/or pressures in the atria or ventricles
      - Mixing of venous and arterial blood
      - Inadequate cardiac output and poor systemic perfusion
    - Neonate can have multiple defects at once

Left-to-Right Shunt Defects

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- Condition in which oxygenated blood shifts from left to right side of the heart
- Defect is considered acyanotic
  - Higher pressures on left side of heart prevent unoxygenated blood from right side from entering the aorta and systemic circulation

Atrial Septal Defect (ASD)

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We know children.

- Commonly the result of foramen ovale nonclosure
  - "Patent" foramen ovale
  - Oxygenated blood from pulmonary vein enters left atria
  - Higher left atrial pressure compared to right produces volume shift to right side
  - Eventually causes right atrial and ventricular enlargement

Acyanotic

Atrial Septal Defect (ASD)

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We know children.

- Signs and symptoms
  - Commonly subclinical
  - Clinical significance related to size of defect
  - Rarely, congestive heart failure might develop
- Management
  - Give supportive care
  - Repair surgically (definitive treatment)

Atrial Septal Defect (ASD)

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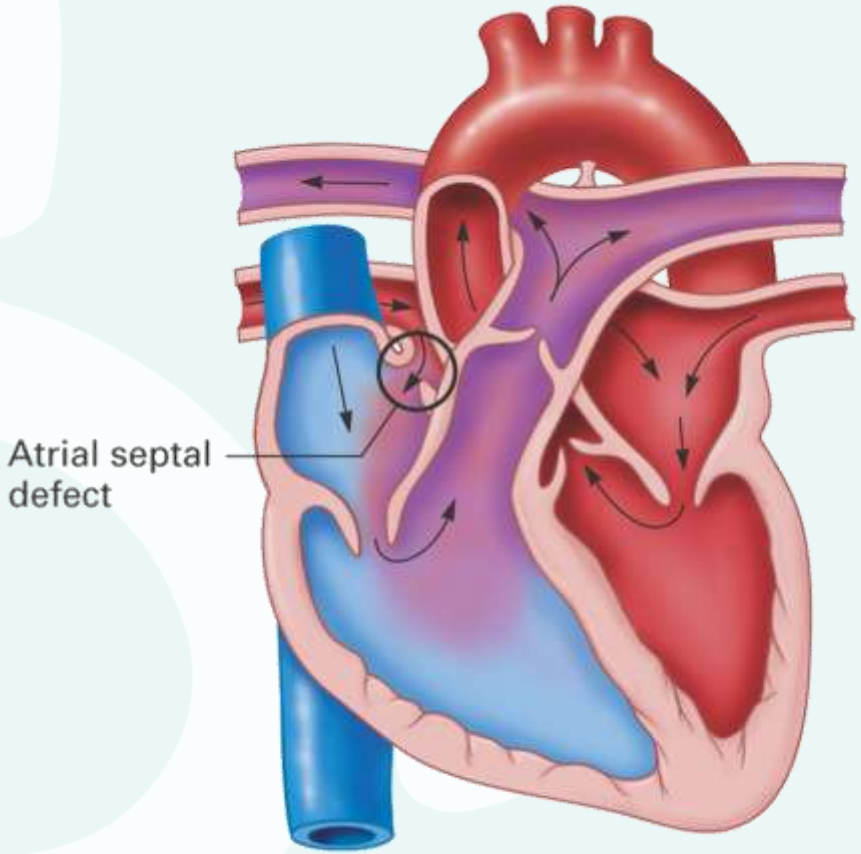
We know children.



Acyanotic

Ventricular Septal Defect (VSD)

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We know children.

- Defect in ventricular septum allows blood flow between ventricles
  - Can cause:
    - Left-to-right shunting of blood
    - Pulmonary hypertension
    - Changes in pulmonary vascular bed
  - Size of defect determines clinical significance

Acyanotic

Small VSD

- Produces a small, left-to-right shunt
- Little pulmonary vascular congestion, chamber enlargement
- More difficult to diagnose

Large VSD

- Pulmonary hypertension develops
- Signs of left ventricular overload, congestive heart failure develop
- Can present early or late
  - Early presentation typified by global ventricular enlargement
  - Late presentation typified by equal left-to-right and right-to-left shunting/mixing of blood
- Result of equal/near-equal PVR and SVR

Acyanotic

Signs and Symptoms of VSD

- Respiratory distress, fatigue, diaphoresis at feedings
- History of poor weight gain or weight loss
- Congestive heart failure

Acyanotic

Management of VSD

- Ensure adequate oxygenation
- Treat congestive heart failure, when present
- Repair surgically (definitive treatment)

VSD

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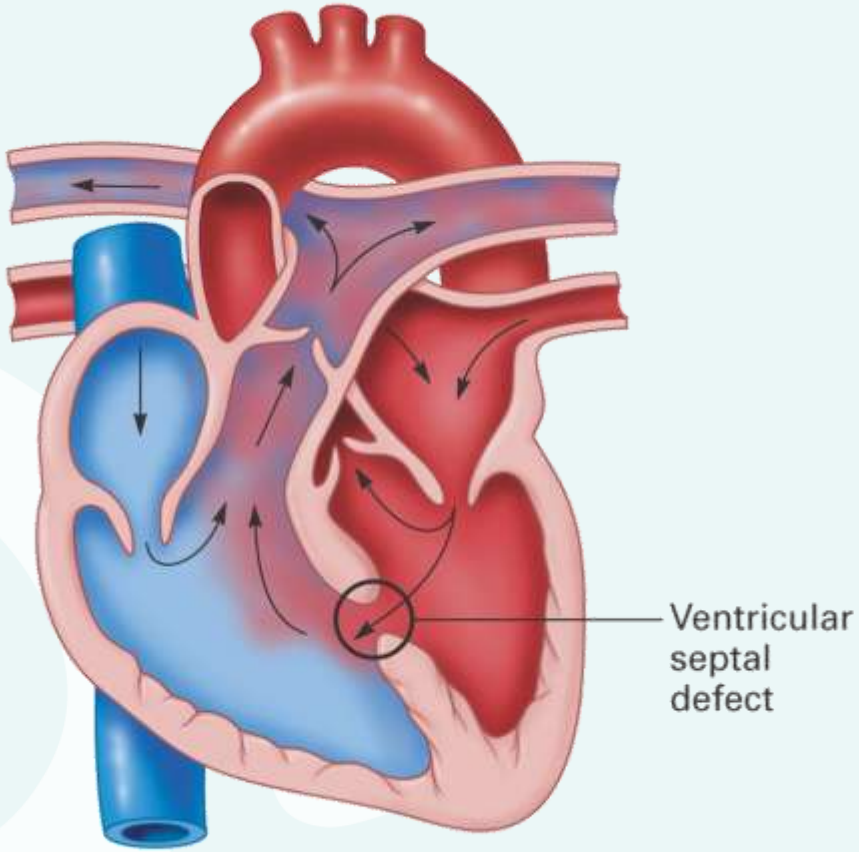

Acyanotic

Patent Ductus Arteriosus (PDA)

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We know children.

- Condition characterized by failure of the ductus arteriosus to close after pulmonary circulation has been established
  - PDA allows for flow of blood from aorta to pulmonary artery
  - Causes:
    - Pulmonary hypertension
    - Myocardial hypertrophy
  - Size of defect, amount of blood flow determines clinical significance

Acyanotic

Patent Ductus Arteriosus (PDA)

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We know children.

- Signs and symptoms
  - Difficulty breathing, tachypnea, tachycardia
  - Bounding pulses, widening pulse pressures, fatigue at feedings
- Management
  - Give supportive care
  - Administer aldomethacin
  - Use prostaglandin inhibitor

Patent Ductus Arteriosus (PDA)

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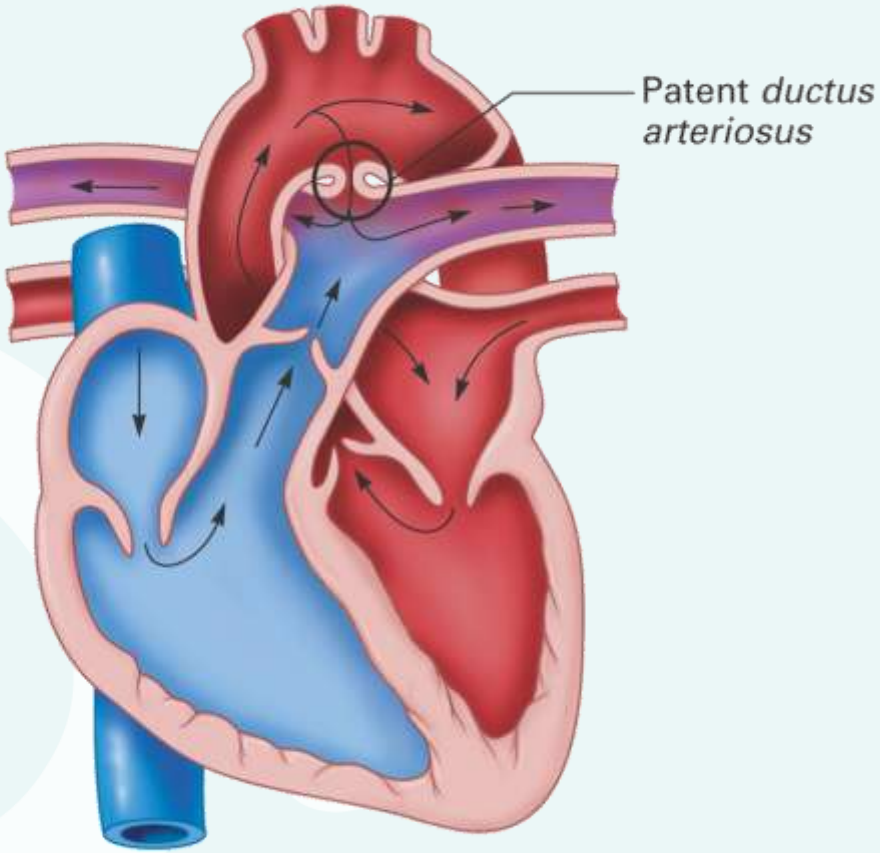
We know children.



Acyanotic

Obstructive Defects

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- Overview
  - Complete or partial blockage of blood flow commonly caused by a structural deformity
  - Signs and symptoms are secondary to the cardiovascular structures involved

Aortic and Pulmonary Stenosis

- Aortic or pulmonary valve narrowed
  - Blood flow impeded
  - Ventricular pressure increased
  - Ventricles enlarged
  - Poststenotic vessel dilation evident
- Signs and symptoms
  - Respiratory distress, tachypnea, tachycardia
  - Weak pulses, hypotension, and fatigue at feedings

Aortic and Pulmonary Stenosis

- Management
  - Give supportive care
  - Conduct oxygenation
  - Proceed with pharmacologic management
  - Undertake balloon angioplasty/valvuloplasty

Aortic and Pulmonary Stenosis

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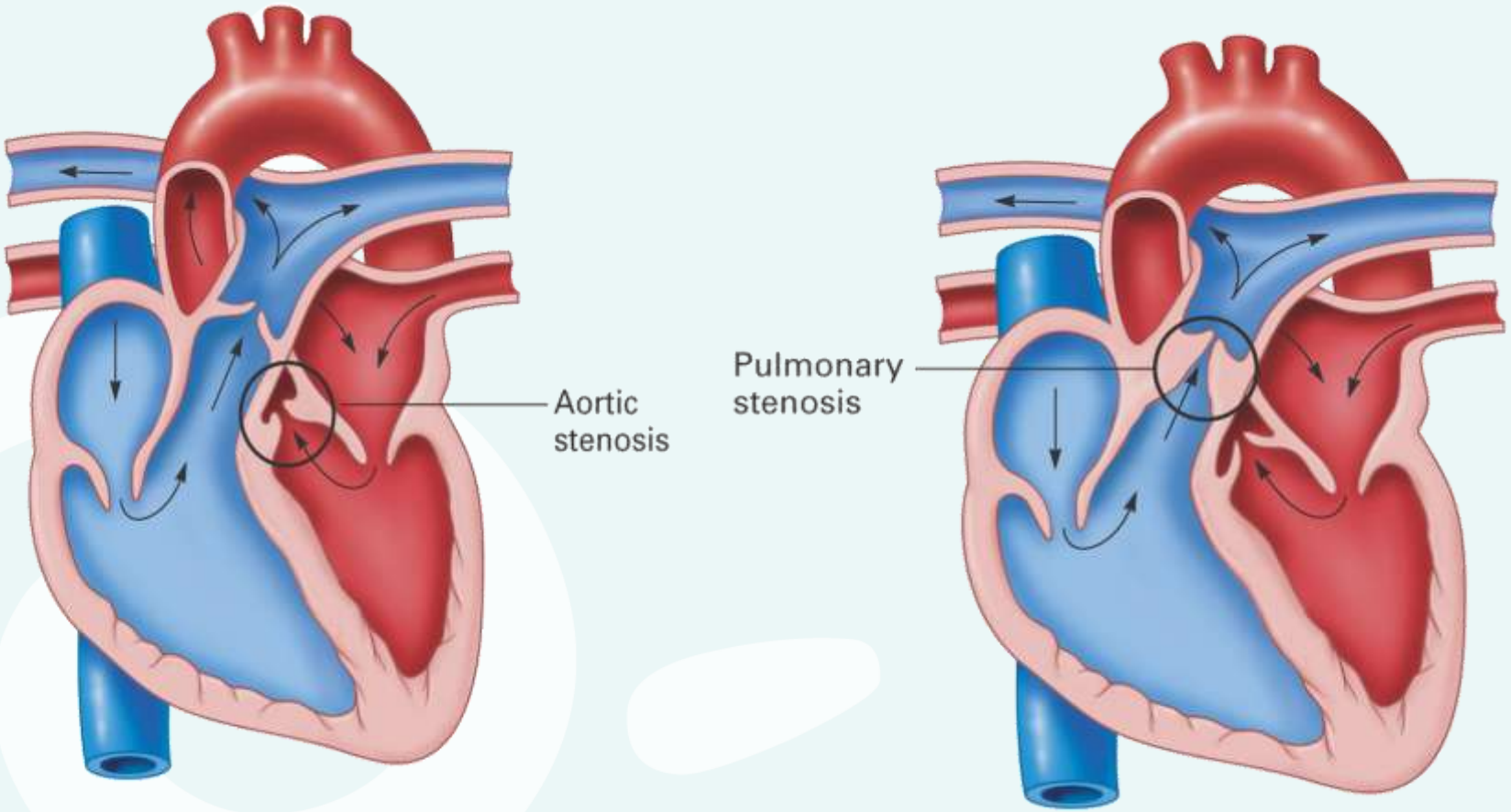

Obstructive

Coarctation of the Aorta

- Characterized by narrowing of the aorta near the distal aspect of the aortic arch
  - Increased left ventricular pressures
  - Increased left ventricular workload
  - Left ventricular hypertrophy
- Signs and symptoms
  - Tachycardia
  - Bounding pulses in the upper extremities with thready or absent pulses in the lower extremities
  - Fatigue at feedings

Coarctation of the Aorta

- Management
  - Give supportive care
  - Administer prostaglandin
  - Treat congestive heart failure, when present
  - Complete balloon angioplasty/surgical resection (definitive treatment)

Coarctation of the Aorta

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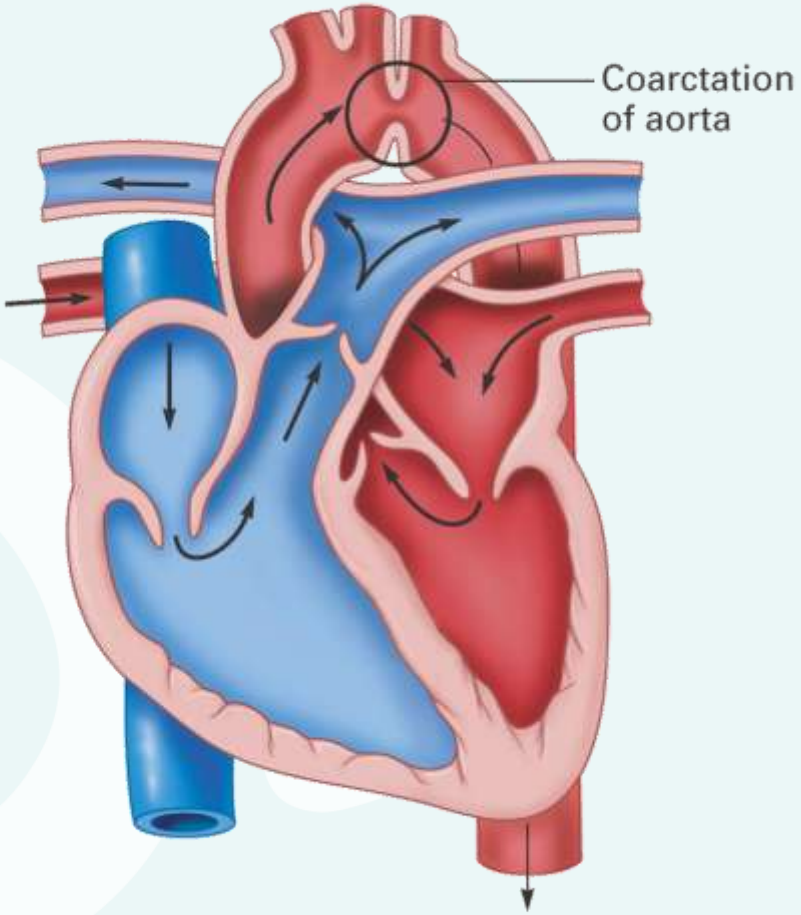

Obstructive

Cyanotic Defects

- Characterized by poor pulmonary blood flow resulting from one or more of the following:
  - Difficulty in pumping blood out the right side of the heart
  - Greater pressure gradient from right to left side of the heart that shunts blood to left side
    - Returns unoxygenated blood to the left side
  - Blockage of pulmonary blood flow or structural deformity

Complete Transposition of the Great Vessels (TGV)

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- Characterized by abnormal positioning of the aorta and pulmonary arteries
  - Pulmonary artery leaves the left ventricle
  - Aorta leaves the right ventricle
  - Creates parallel circulations

Cyanotic

Complete Transposition of the Great Vessels (TGV)

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- Associated with ASD, VSD, and PDA up to 80 percent of the time and has to be for the patient to survive
  - Without these defects, no intracardiac mixing of oxygenated and deoxygenated blood occurs, child dies due to closed parallel circuits
  - Degree of cyanosis/acidosis depends on number and size of intracardiac and extracardiac shunts

Cyanotic

Complete Transposition of the Great Vessels (TGV)

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We know children.

- Signs and symptoms
  - Difficulty breathing, tachypnea, tachycardia
  - Cyanosis
- Management
  - Give supportive care
  - Repair surgically via arterial switch (definitive treatment)

Complete Transposition of the Great Vessels (TGV)

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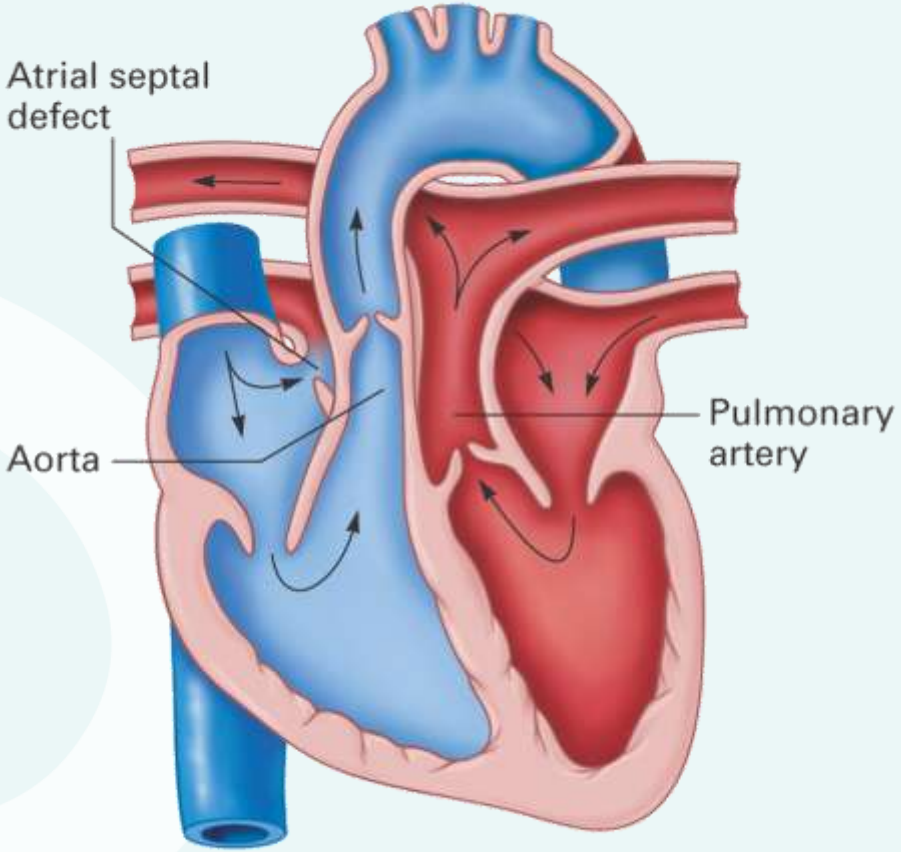

Cyanotic

Tetralogy of Fallot

- Condition characterized by four criteria:
  - VSD
  - Pulmonary stenosis
  - Rightward displacement of aorta
    - Overrides the VSD
  - Right ventricular hypertrophy

Cyanotic

Tetralogy of Fallot

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We know children.

- Degree of cyanosis secondary to mixing of oxygenated/deoxygenated blood determined by degree of pulmonary stenosis
  - Greater the pulmonary stenosis, the greater the right side intraventricular pressure, the greater the right-to-left shunt, the more deoxygenated blood reaching systemic circulation via the aorta

Tetralogy of Fallot

- Signs and symptoms
  - Tachypnea, tachycardia
  - Fatigue at feedings
- Management
  - Give supportive care
  - Ensure adequate oxygenation
  - Administer prostaglandin

Tetralogy of Fallot

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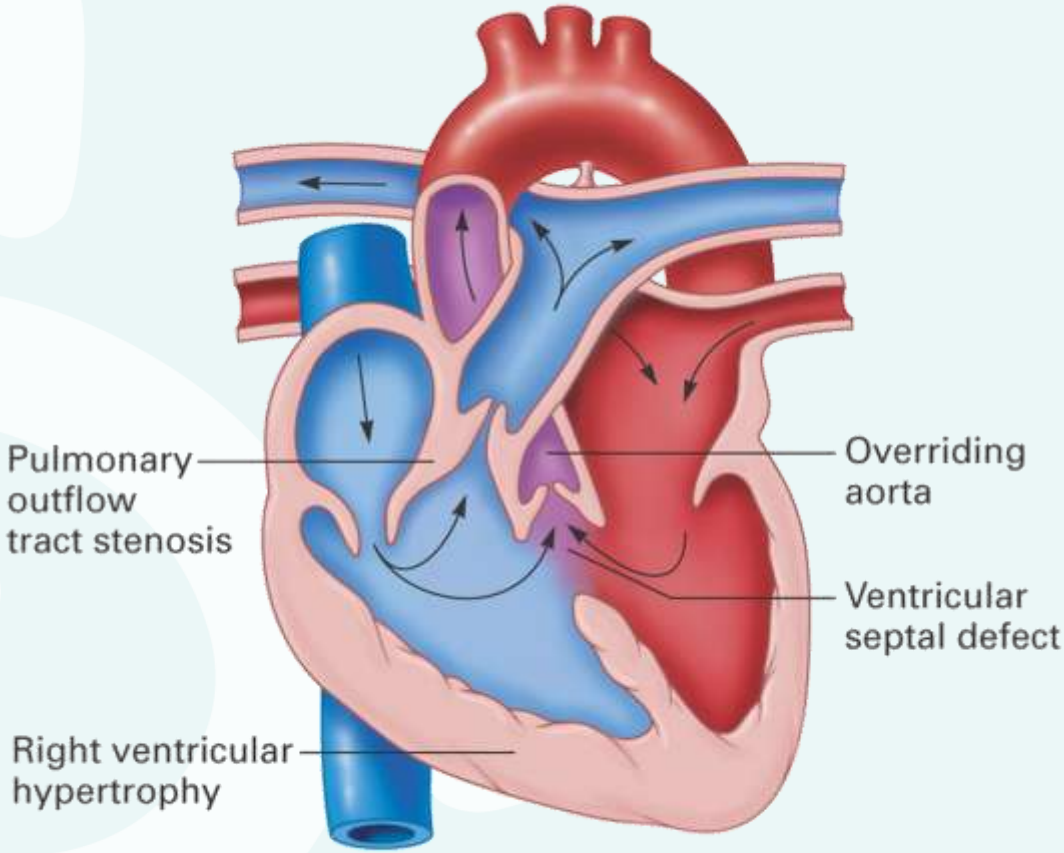

Cyanotic

Transport Guidelines for Congenital Heart Defects

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We know children.

- Ensure patent airway
- Ensure adequate ventilation, oxygenation
- Treat congestive heart failure
- Correct circulatory compromise
  - Conduct fluid volume resuscitation
  - Administer vasopressors
- Keep patient warm

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General Pathophysiology: Other Neonatal Emergencies

Necrotizing Enterocolitis (NEC)

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- Most common serious abdominal emergency in neonates that requires emergency surgical intervention
  - Acute inflammation of the large intestine leading to necrosis of the intestinal mucosa
  - Risk factors include insult to intestinal mucosa and bacterial growth
  - Causative agent has not been identified
  - Risk of sepsis secondary to bowel perforation

Necrotizing Enterocolitis (NEC)

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We know children.

- Signs and symptoms
  - Abdominal distention
  - Decreased or absent bowel sounds
  - Vomiting
  - Bloody diarrhea
  - Lethargy
  - Poor feeding habits
  - Depressed core body temperature

Necrotizing Enterocolitis (NEC)

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We know children.

- Management
  - Give supportive care
  - Keep the patient NPO
  - Insert NG tube and conduct gastric decompression
  - Maintain acid-base and electrolyte balance
  - Maintain IV fluids
  - Administer antibiotic therapy

Sepsis

- Life-threatening infection of the bloodstream resulting in systemic toxicity
  - Often subtle in neonate and may be difficult to distinguish from a noninfectious pathology
  - Maternal gastrointestinal or genital infections are most common etiology
  - Primary site of infection may often be difficult to identify
  - Shock may develop
  - Result of vasodilation secondary to release of bacterial endotoxins
  - Distributive shock

Sepsis

- Signs and symptoms
  - Hypothermia
  - Respiratory distress
  - Pulmonary hypertension
  - Hypoxemia
  - Severe hypoperfusion
  - Disseminated intravascular coagulation (DIC)

Sepsis

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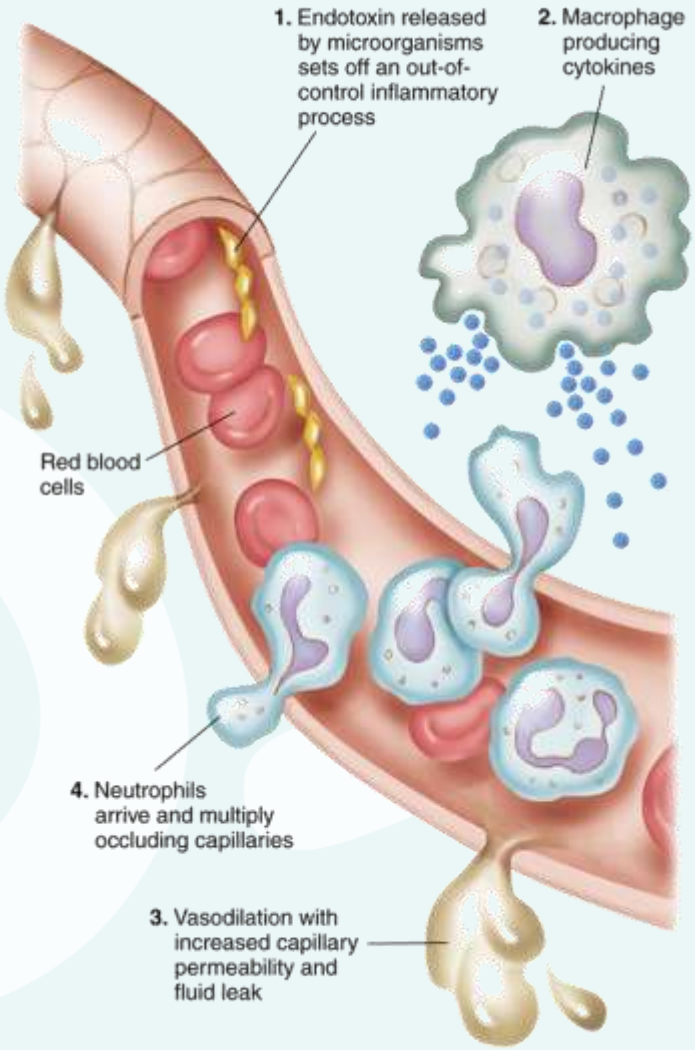

Sepsis

- Management
  - Give supportive care
  - Ensure cardiovascular support
  - Administer antibiotic therapy

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General Neonatal Assessment Findings/Considerations

Skin Color

- Cyanosis commonly found
  - Insignificant when neonate is crying
- Jaundice
  - Result of high serum bilirubin levels
  - Usually resolves without intervention
    - When needed, treat with fluorescent light
    - Blood transfusion needed when fluorescent light treatment fails

Vital Signs

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- Neonatal vital signs variable, deviate from “norm”
- Access to reference material advisable
  - Prdiatric Broselow tape
- In addition to respiratory rate, blood pressure, heart rate, consider blood glucose level a vital sign in neonate
  - 70–80 mg/dL considered nonhypoglycemic

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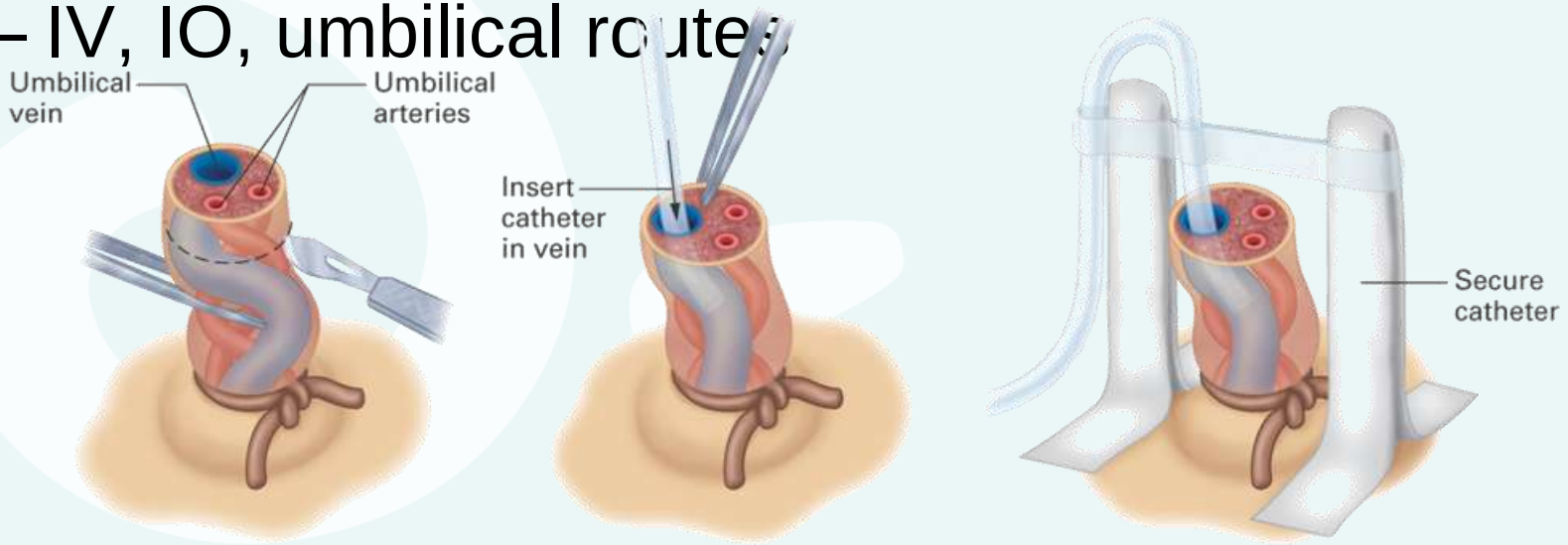
General Neonatal Considerations

Airway

- Should be secured and maintained as soon as possible
  - RSI less common in adults but should be used when needed
- Accidental extubation most frequent respiratory complication
  - Sedation
  - NMBAs
  - C-collars
  - Lateral immobilization

Vascular Access

- Obtaining vascular access can be difficult even for experienced providers
- Multiple access options should be available
  - IV, IO, umbilical routes



Temperature Regulation

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We know children.

- Critical, should be consistently ensured during transport
- Temperature regulation initially provided by preventing heat loss while promoting strategies for aggressiv
  - Before transport use:
    - Radiant warmers
    - Insulated blankets
    - Heated blankets
  - During transport:
    - Transport incubator/isollette

Scott and White Hospital and Clinic

Hypoglycemia

- Hypoglycemia should be managed aggressively
  - Use 10 percent dextrose and water
    - Infuse D10W at 80cc/kg/day
  - D25W, D50W administration contraindicated
  - Can cause significant increases in plasma osmolarity
  - Hypernatremia
    - Cerebral edema

Summary

Children's HOSPITAL & MEDICAL CENTER
We know children.

- Common denominator for unexpected deaths in neonates is hypoxia
  - Via infectious diseases, congenital heart disease, pulmonary compromise, other etiologies
  - Neonates can compensate until they are extremely hypoxic
    - High index of suspicion needed to identify developing hypoxia before decompensation
  - Airway and ventilation highest priority
    - Neonates with high metabolism, high oxygen consumption

Summary

- Manage CHD after addressing airway, breathing, and pulmonary function
  - Transport care for the CHD patient is primarily supportive
  - May require significant intervention
- Ability to diagnose specific defects not top concern
  - Critical care practitioner should know how various defects affect normal perfusion
  - Care provider is responsible for staying abreast of common neonatal emergencies and their current standards of care