Neonatal Emergencies and Transport
Relative Anatomy and Physiology
Physiology of Thermoregulation

- 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

• 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

Convection

Evaporation

Surface

Skin temperature

Radiation

Temperature of base

Conduction
Glucose Requirements

- 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

• 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
Airway Anatomy and Physiology

• 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

- 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

• 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

- 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

• 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

- 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

• 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

• 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

• 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 arteriosus
      – Occurs in first hours to weeks after birth
General Pathophysiology: Pulmonary

• 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

• 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

• 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

• 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

• 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

• 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

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)

- 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)

- 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)

• 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

• 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

• 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

• 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

• 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)

• 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)

- 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)

Acyanotic
Ventricular Septal Defect (VSD)

- 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
Management of VSD

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

Ventricular septal defect

Acyanotic
Patent Ductus Arteriosus (PDA)

- 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)

• 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)
Obstructive Defects

• 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
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

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)

- 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)

- 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
Complete Transposition of the Great Vessels (TGV)

- 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)

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

- 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

Cyanotic
Transport Guidelines for Congenital Heart Defects

- Ensure patent airway
- Ensure adequate ventilation, oxygenation
- Treat congestive heart failure
- Correct circulatory compromise
  - Conduct fluid volume resuscitation
  - Administer vasopressors
- Keep patient warm
General Pathophysiology:
Other Neonatal Emergencies
Necrotizing Enterocolitis (NEC)

- 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)

• Signs and symptoms
  – Abdominal distention
  – Decreased or absent bowel sounds
  – Vomiting
  – Bloody diarrhea
  – Lethargy
  – Poor feeding habits
  – Depressed core body temperature
Necrotizing Enterocolitis (NEC)

- 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

1. Endotoxin released by microorganisms sets off an out-of-control inflammatory process

2. Macrophage producing cytokines

3. Vasodilation with increased capillary permeability and fluid leak

4. Neutrophils arrive and multiply occluding capillaries
Sepsis

• Management
  – Give supportive care
  – Ensure cardiovascular support
  – Administer antibiotic therapy
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

• Neonatal vital signs variable, deviate from “norm”

• Access to reference material advisable
  – Pediatric 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
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

• Critical, should be consistently ensured during transport

• Temperature regulation initially provided by preventing heat loss while promoting strategies for aggressive warming
  – Before transport use:
    • Radiant warmers
    • Insulated blankets
    • Heated blankets
  – During transport:
    • Transport incubator/isollette
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

• 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