The Neonatal Cardiovascular System and Congenital Heart Disease

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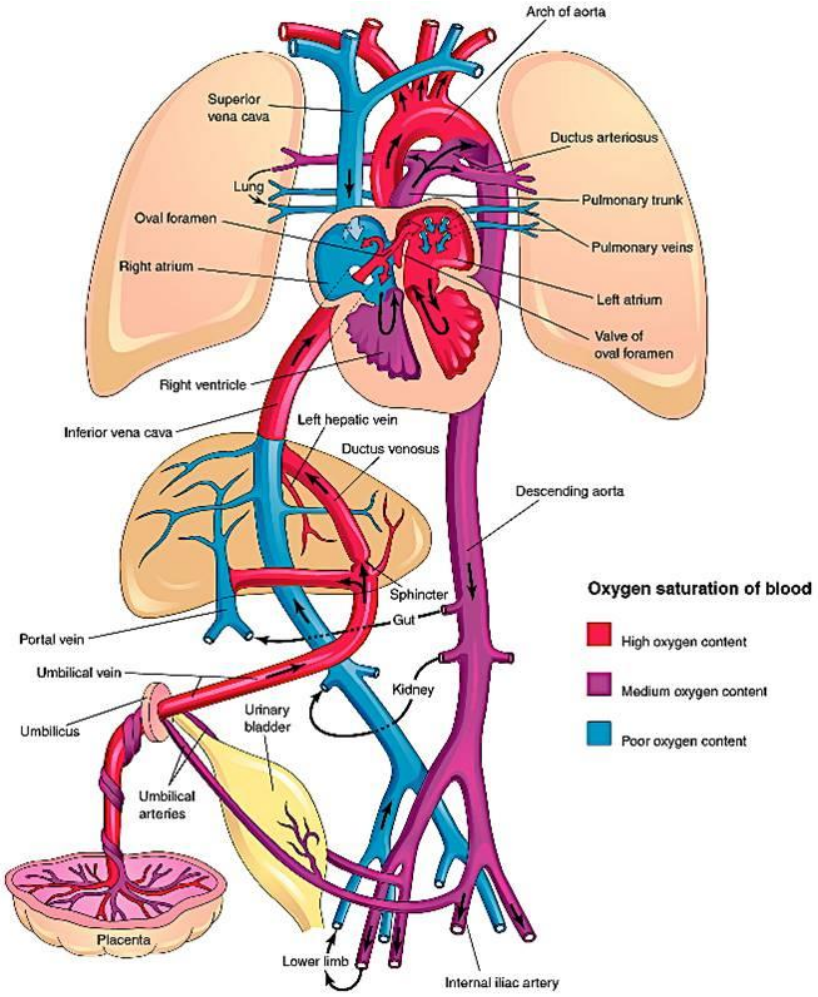
Fetal Circulation



Fetal Circulation

- Differs from adults in many ways
  - Gas exchange occurs at placenta not lungs
  - Blood with highest O_2 content is directed to heart and brain
  - Circulation relies on 3 shunts
  - Circulation is in parallel not series
  - Right-to-left shunting across FO and DA
  - PVR is high not low
  - SVR is low not high

Fetal Blood Gases

Umbilical Vein

- A single umbilical vein connects the placenta to the fetus to deliver oxygen
- Umbilical vein blood gas: pH= 7.35 PaO2= 30 PaCO2= 40

Umbilical arteries

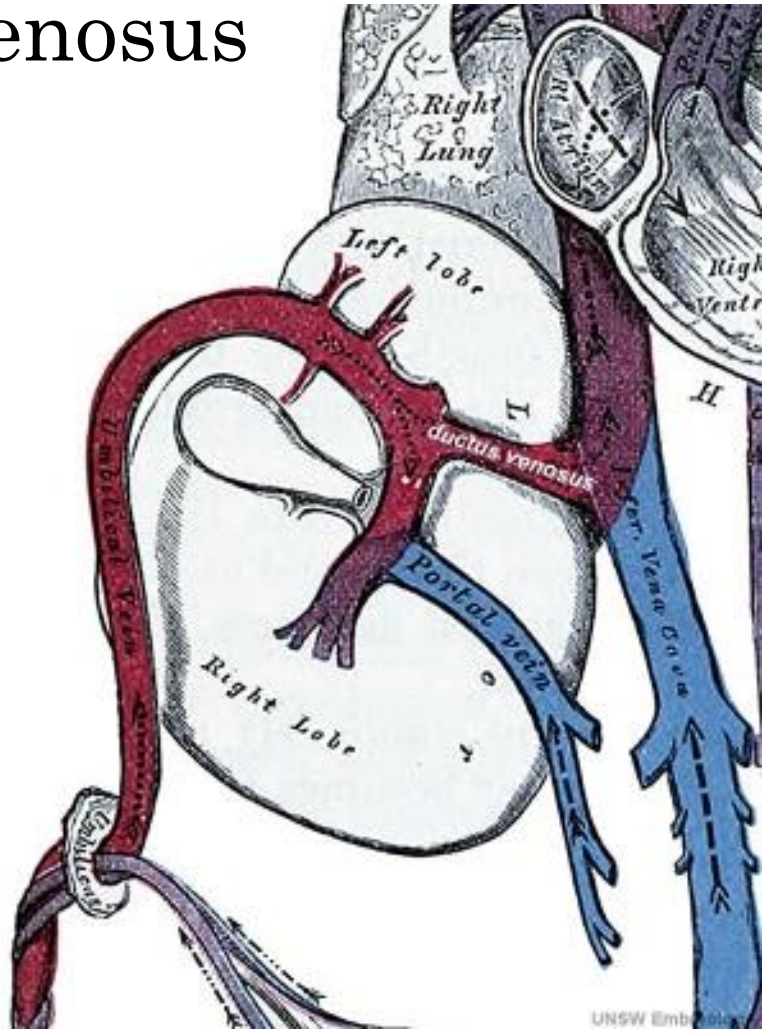
- Two umbilical arteries connect the fetus back to the placenta to return carbon dioxide and deoxygenated blood
- Umbilical arteries blood gas: pH= 7.30 PaO2= 20 PaCO2= 50

Fetal Circulation

- The 3 shunts explained
  - Ductus venosus
    - Connects the left portal vein to the left hepatic vein at junction with IVC
    - 50% of oxygenated blood from placenta is able to bypass the hepatic sinuses
  - Foramen ovale
    - Found between right and left atrium directs O_2 rich blood to LA
  - Ductus arteriosus
    - Connects PA to descending aorta, lower O_2 blood is directed to PA, bypasses lungs, then to lower body

Fetal Circulation

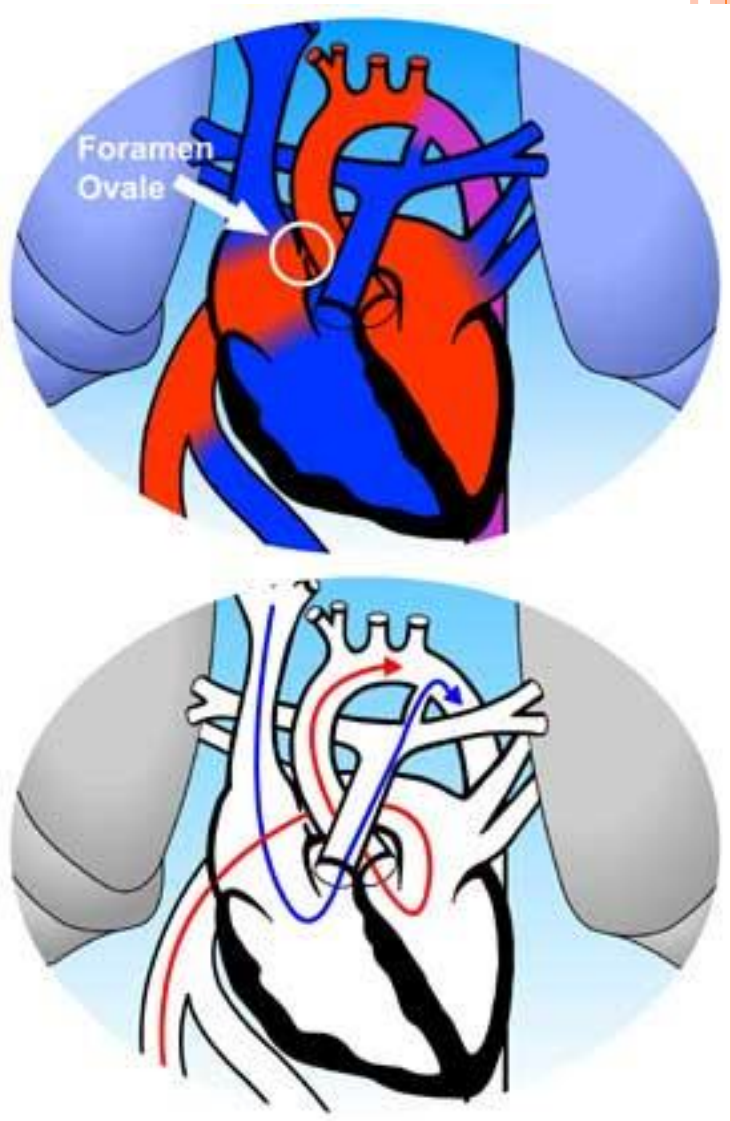
Ductus Venosus



Fetal Circulation

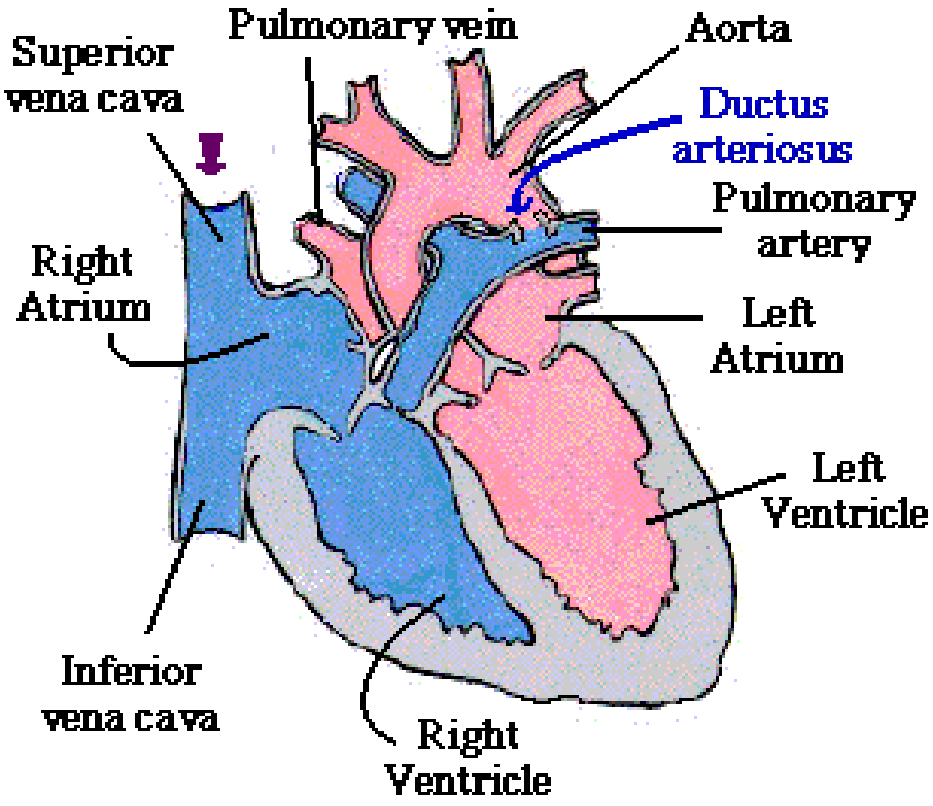
- Foramen ovale

- O_2 rich to LA, brain & heart (red pathway)
- Low O_2 blood to RV, PA and bypasses lungs (blue pathway)



Fetal Circulation

- Ductus arteriosus



Fetal Circulation

1. The umbilical vein carries oxygen rich blood (pao2 = 30 mmhg) into the portal venous system
2. The ductus venosus allows 50% of oxygenated blood to bypass the liver, directing it to the IVC in favor of perfusing the brain and heart
3. Oxygentated and deoxygenated blood in IVC move at different velocities
4. Higher velocity oxygentated blood from ductus venosus moves to IVC → RA → FO → LA → Aorta → Brain/Heart

Fetal Circulation

5. The Eustachian valve is a flap of tissue that diverts the oxygenated blood across the PFO into the LA
6. Lower velocity deoxygentated blood from lower body moves into SVC/IVC → RA→ RV → PA → bypasses lungs → DA → descending aorta
7. This deoxygenated blood perfuses the lower body and returns to placenta via 2 umbilical arteries (pao2= 16mmhg)

Transition After Birth

- The first breath leads to ↑ PaO_2, ↓ $PaCO_{2,}$ ↓ PVR
- Umbilical cord clamping ↓ area for blood to circulate and ↑SVR
- Right to left shunt shifts to left to right (SVR>PVR)
- This reversed blood flow closes the flap valve (FO)
- The lungs open up (↓ PVR) and reverses flow across ductus arteriosus (DA) from aorta to PA
- Oxygenated blood flows over DA and leads to closure
- Cord clamping decreases prostaglandin (PGE1) and also closes DA

Shunt Closures

- Functional versus Permanent Anatomic Closure
- Fetal shunts may not close in the presence of
  - Acidosis
  - Sepsis
  - Hypothermia
  - hypoxia
  - hypercarbia

Shunt Closures

- Ductus Venosus
  - Functional closure via loss of umbilical venous blood flow over the first week of life
  - Physically closed by three months of age
    - The remnant is the ligamentum venosum
- Foramen Ovale
  - Functional closure at cord clamping
    - Decreased right atrial pressure related to loss of venous return from umbilical vein
    - Increased left atrial pressure related to increased pulmonary blood flow
  - Physical closure quickly follows functional
    - Usually by 3 days
    - A PFO may be present in 30% of adults and 50% of children less than 5 years

Shunt Closures

- Ductus Arteriosus
  - Functional closure at first day of life via increased PO2, decreased prostaglandin, and increased SVR/decreased PVR
    - Functional closure may be reversed with PGE1 infusion if defect is ductal dependent
  - Physical closure at 2-3 weeks old via fibrosis
  - Pre-term neonates are at risk for prolonged ductal closure
    - They have increased PGE1 and a reduced response to oxygen-driven smooth muscle constriction
    - Indomethacin, a prostaglandin inhibitor, may promote closure
    - Device closure or ligation may be required

Neonatal Cardiovascular system

- Neonatal Cardiovascular System
  - Reduced Ventricular compliance
    - Due to a lack of elastic elements
  - Reduced contractility
    - Reduced contractile elements, mitochondria, and sarcoplasmic reticulum
    - Poorly developed T-tubules
      - Increased reliance on extracellular calcium
  - Increased intraventricular dependence
    - Due to a lack of ventricular compliance
    - Ventricular filling is easily affected by opposite ventricle end-diastolic filling pressure
    - RV and LV size and thickness are equal at birth
      - The increased afterload on the LV will aid to double its mass relative to the RV after several months of life

Neonatal Cardiovascular system

- Neonatal Cardiovascular system
  - Afterload mismatch
    - Stroke volume quickly declines with increased afterload
  - Poor preload reserve
  - Parasympathetic NS dominates, incomplete sympathetic innervation
    - Reduced response to chronotropic/inotropic support
  - Poor baroreceptor response to hypotension
  - Increased dependence on anaerobic metabolism
    - Myocytes have ↑ glycogen storage/ anaerobic glycolysis allowing for buffer for ischemic insult
    - Bradycardia often a LATE sign of hypoxia
  - Overall cardiac output **is heart rate** dependent

Pediatric CV system

Normal range of resting heart rate and blood pressure in children

Age	Heart Rate (bpm)	Blood Pressure (mm Hg)
0-3 mos	100-150	65-85/45-55
3-6 mos	90-120	70-90/50-65
6-12 mos	80-120	80-100/55-65
1-3 yrs	79-110	90-105/55-70
3-6 yrs	65-110	95-110/60-75
6-12 yrs	60-96	100-120/60-75
>12 yrs	55-85	110-135/65-85

Congenital Heart Disease

- Incidence
  - 7-10 per 1,000 live births
- 9 lesions account for 80% of all CHD
  - VSD, ASD, PDA, PS, AS, Coarc of aorta, AVSD, TOF, TGA
- Infant signs and symptoms of CHD
  - Tachypnea, poor feeding, ↓ weight gain, HR > 200 bpm
  - Hear murmur, CHF, cyanosis

CHD Overview

- Acyanotic congenital heart lesions
  - **Atrial Septal Defect (ASD)**
  - **Ventricular Septal Defect (VSD)**
  - **Patent Ductus Arteriosus (PDA)**
  - **AV Canal**
  - Aortic Stenosis (AS)
  - Pulmonary Stenosis (PS)
  - **Coarctation of the Aorta**

CHD Overview

- Cyanotic congenital heart lesions
  - **Tetralogy of Fallot (TOF)**
  - **Transposition of the great arteries (TGA)**
  - Tricuspid valve abnormality (Ebstein's anomaly)
  - **Truncus arteriosus**
  - Total anomalous pulmonary venous connection
  - **Single ventricle physiology**
  - Eisenmenger syndrome

CHD Diagnosis

- Chest x-ray
- Barium esophogram
  - limited use
- Echocardiogram
  - gold standard for initial evaluation and serial assessment
- Cardiac MRI
  - beneficial for assessment of complex disease and to guide surgical intervention
- Cardiac CT
  - Helpful for evaluation of aortic arch anomalies, coronary abnormalities, airway pathologies, and defining systemic and pulmonary venous returns
- Cardiac cath
  - Pressure and resistance data, shunt ratios, anatomic definition, EP treatment, and catheter based interventions

CHD Important Concepts

- Vascular Resistance
  - What increases PVR?
  - What decreases PVR?
  - What increases SVR?
  - What decreases SVR?
- Physiological Shunting
  - It is the recirculation of blood
  - A L-R shunt is the recirculation of pulmonary venous blood
  - A R-L shunt is the recirculation of systemic venous blood
  - Physiological shunts are commonly due to anatomic shunts

CHD Important Concepts

- Quantifying Shunt Volumes
- Pulmonary-to-Systemic Blood Flow Ratio
  - Qp/Qs ratio 1:1 is normal and *typically* suggests no shunting is present
  - Qp/Qs ratio > 1:1 defines a left to right shunt in which pulmonary blood flow is greater than systemic blood flow
  - Qp/Qs ratio < 1:1defines a right to left shunt in which pulmonary blood flow is less than systemic blood flow
  - Bidirectional shunting can occur in a single patient, and if the degree of left to right shunting is similar to the degree of right to left shunting, Qp:Qs ratio may equal 1:1

CHD Important Concepts

- Effects of Intracardiac Shunts on Anesthetic Induction
- Right to Left Shunts
  - Rapid IV induction
  - Slower inhalation induction
- Left to Right Shunts
  - Little difference on IV induction
  - Little difference on inhalation induction

Acyanotic Congenital Heart Lesions

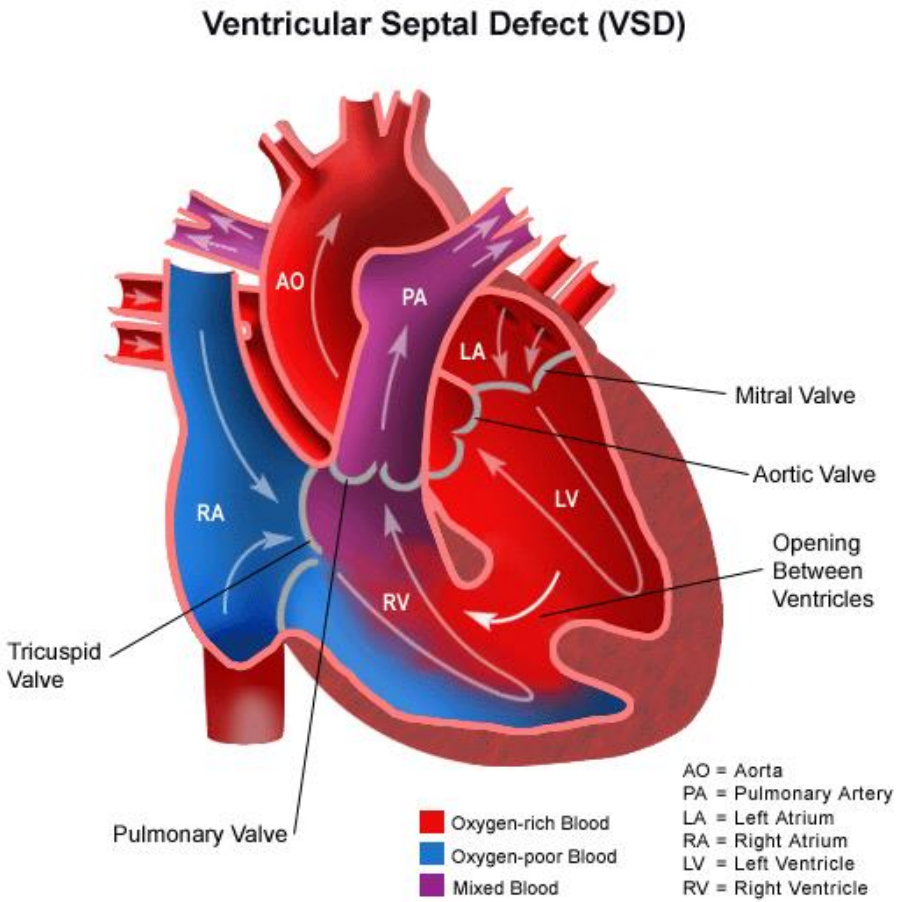
Left to Right Shunts

- Acyanotic heart disease (pink baby)
- VSD, ASD, PDA, AV Canal

- Increased pulmonary circulation ➔ pulmonary hypertension ➔ RV hypertrophy ➔ CHF

Left to Right Shunts

- Ventricular Septal Defect



Left to Right Shunts

- Ventricular Septal Defect
  - The most common CHD lesion accounting for greater than 20% of CHD cases
  - Many close spontaneously by the age of 2
  - Associated with Trisomy 13,18, and 21
  - Classified by location the septum
    - Most common is **perimembranous VSD**

Left to Right Shunts

- Ventricular Septal Defect
- Signs and Symptoms
  - If VSD is small, pulmonary blood flow is only slightly increased
  - If VSD is large, direction/magnitude of shunt is determined by PVR and SVR
  - Large VSDs also predispose patients to pulmonary artery hypertension
  - Symptoms of Congestive Heart Failure
  - Can convert to R-L shunt

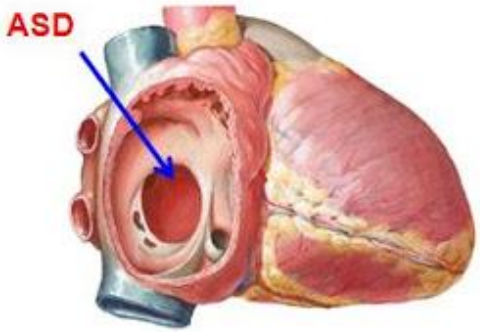
    (Eisenmenger Syndrome) from pulmonary vascular disease

Left to Right Shunts

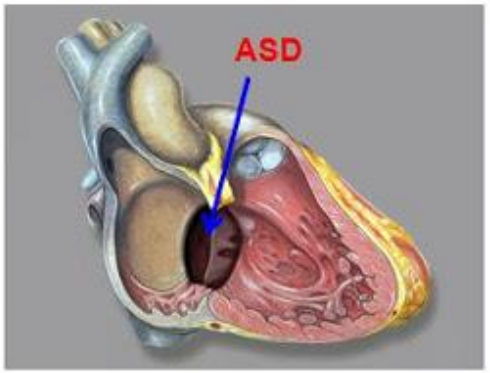
- Ventricular Septal Defect
- Treatment
  - Observation
  - Medical management
  - Closure via open heart surgery or transcatheter device closure
- Anesthetic management
  - Inhalation induction is reasonable if no CHF
  - Avoid drugs that increase SVR
  - Volatiles and positive pressure ventilation ↓ SVR and ↑ PVR thus decrease the left to right shunting
  - Reduce FIO2 and avoid hyperventilation to maintain PVR
  - Avoid air in the IV tubing
  - Post bypass, reduce PVR and provide inotropic support to RV if needed
  - If stable, can often attempt early extubation

Left to Right Shunts

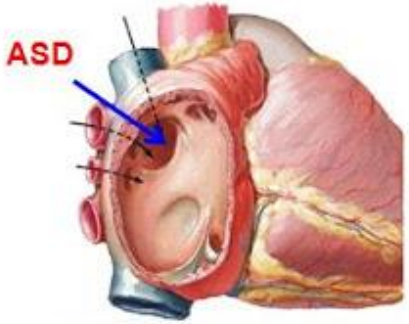
- Atrial Septal Defect



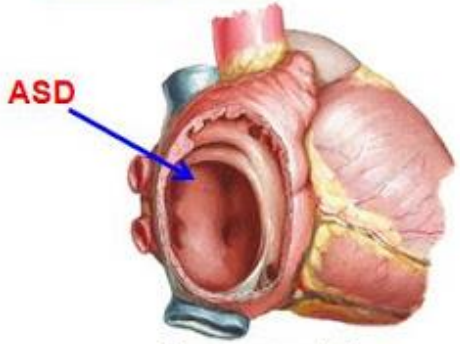
Ostium secundum defect



Ostium primum defect



Sinus venosus defect



Common atrium

Left to Right Shunts

- Atrial Septal Defect
  - The second most common CHD lesion
  - Pressure gradient is typically low
  - When > 1-2 cm, L-R shunting occurs because of ventricular compliances, PVR, and SVR
  - Net effect is increased pulmonary circulation
  - A heart murmur can be detected at age 6-8 weeks

Left to Right Shunts

- Atrial Septal Defect
- Signs and Symptoms
  - RV volume overload and increased pulmonary blood flow
  - Poor exercise tolerance/poor feeding
  - Poor weight gain
  - Paradoxical embolism
  - Recurrent pulmonary infections
  - Atrial flutter/fibrillation (late)
  - Congestive heart failure (late)

Left to Right Shunts

- Atrial Septal Defect
- Treatment
  - Observation
  - Medical Management
  - Closure via open heart surgery or transcatheter device closure
- Anesthesia management
  - Avoid drugs that increase SVR
  - Volatiles and positive pressure ventilation ↓ SVR and ↑ PVR thus decrease the left to right shunting
  - Reduce FIO2 and avoid hyperventilation to maintain PVR
  - Avoid air in the IV tubing
  - Post bypass, reduce PVR and provide inotropic support to RV if needed
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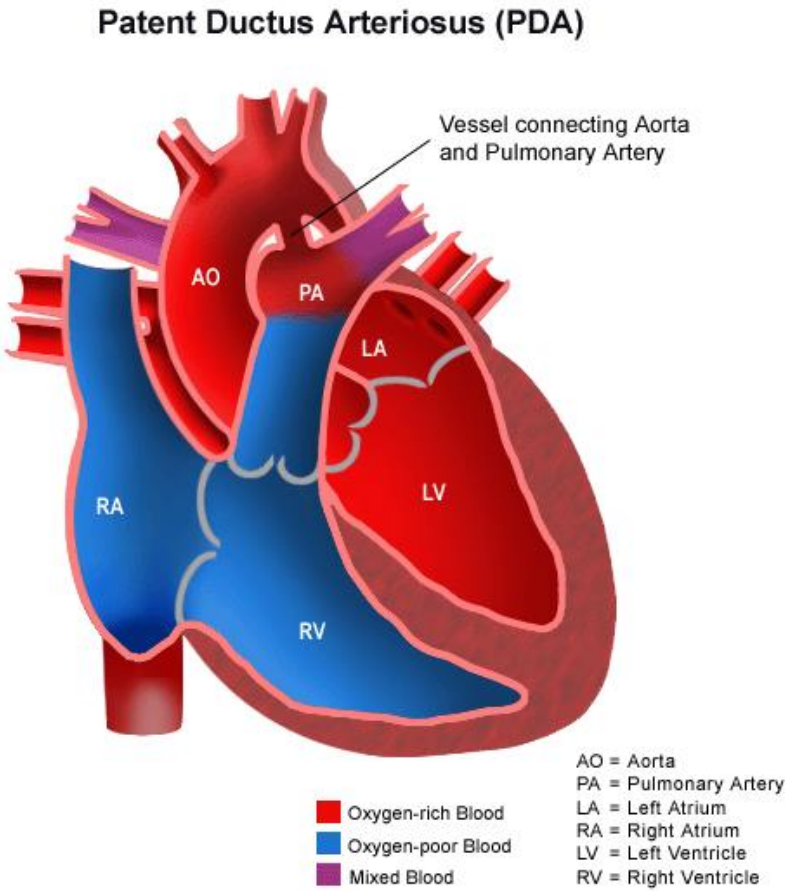
Left to Right Shunts

- Patent Ductus Arteriosus



Left to Right Shunts

- Patent Ductus Arteriosus
  - Accounts for 10% of CHDs
  - Normal closure at birth (physical closure via fibrosis over first several weeks of life)
  - Stays open in 20-30% of premature infants
  - Results in continuous flow of blood from aorta to PA
  - Shunting dependent on PVR and SVR

Left to Right Shunts

- Patent Ductus Arteriosus
- Signs and Symptoms
  - Premature infants with respiratory distress
  - May be older child with isolated PDA
  - Continuous systolic and diastolic murmur
  - If L to R shunt is large → left ventricular hypertrophy on ECG and chest x-ray
  - Diastolic run-off from proximal aorta to PA
    - Can decrease end-organ perfusion
  - If left untreated can lead to CHF, pulmonary HTN, Eisenmenger's syndrome, poor growth, infective endocarditis, aneurysm of ductus, and ductal calcification

Left to Right Shunts

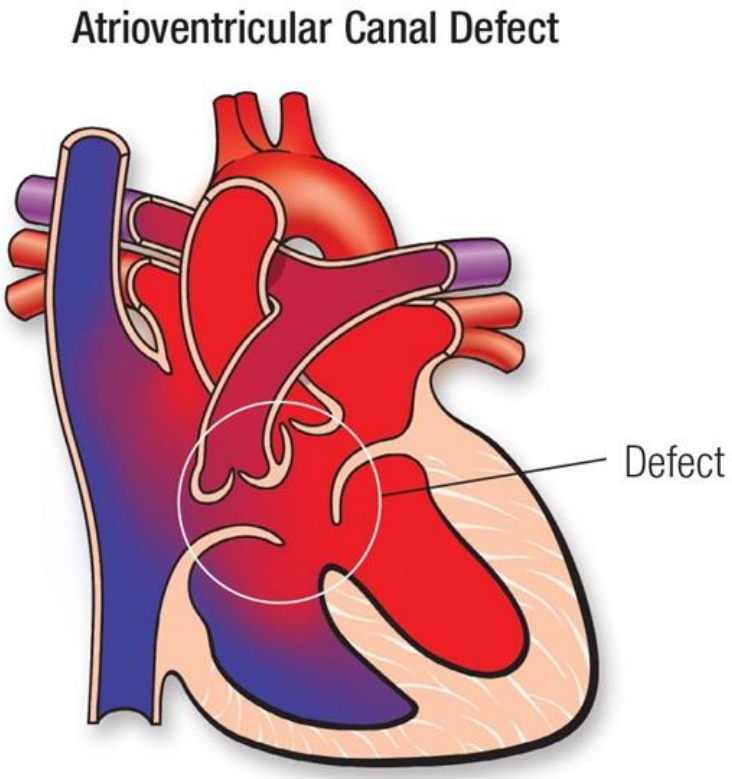
- Patent Ductus Arteriosus
- Treatment
  - Observation
  - Closed via medical closure, transcatheter device closure, or PDA ligation
- Medical closure involves cox-1 or cox-2 inhibitors
  - Indomethacin has reduced the need for surgery by 60% and is first line therapy
  - Adverse effects include decreased mesenteric, renal, and cerebral blood flow
- Surgical closure involves thoracotomy off cardiac bypass

Left to Right Shunts

- Patent Ductus Arteriosus
- Anesthetic Management
  - Maintain heart rate, contractility, and preload
  - Avoid air bubbles in IV tubing
  - Volatile anesthetics and positive pressure ventilation are desirable due to ↓ left to right shunt
  - Be prepared for high FIO2 requirements with left lung retraction with thoracotomy approach
  - After PDA ligation you see ↑ SVR and can see hypertension
  - Compare upper and lower extremity blood pressures to ensure coarctation was not created

Left to Right Shunts

- Atrioventricular Septal Defect (AVSD)



Left to Right Shunts

- Atrioventricular Septal Defect (AVSD)
  - Accounts for 5% of all CHD cases
  - Most common in infants with down syndrome
  - Described by the lack of production of the endocardial cushion
  - The endcordial cushion includes:
    1. The lower portion of the atrial septum
    2. The ventricular septum
    3. The separation of the mitral and tricuspid valves

Left to Right Shunts

- Complete AVSD
  - Defect in the atrial septum, ventricular septum, and common AV valve
- Partial AVSD
  - Atrial septal defect
  - Mitral valve cleft
  - Ventricular septum has filled in and AV valves are separated
- Transitional AVSD
  - Similar to complete AVSD structurally, but functionally has two separate AV valves

Left to Right Shunts

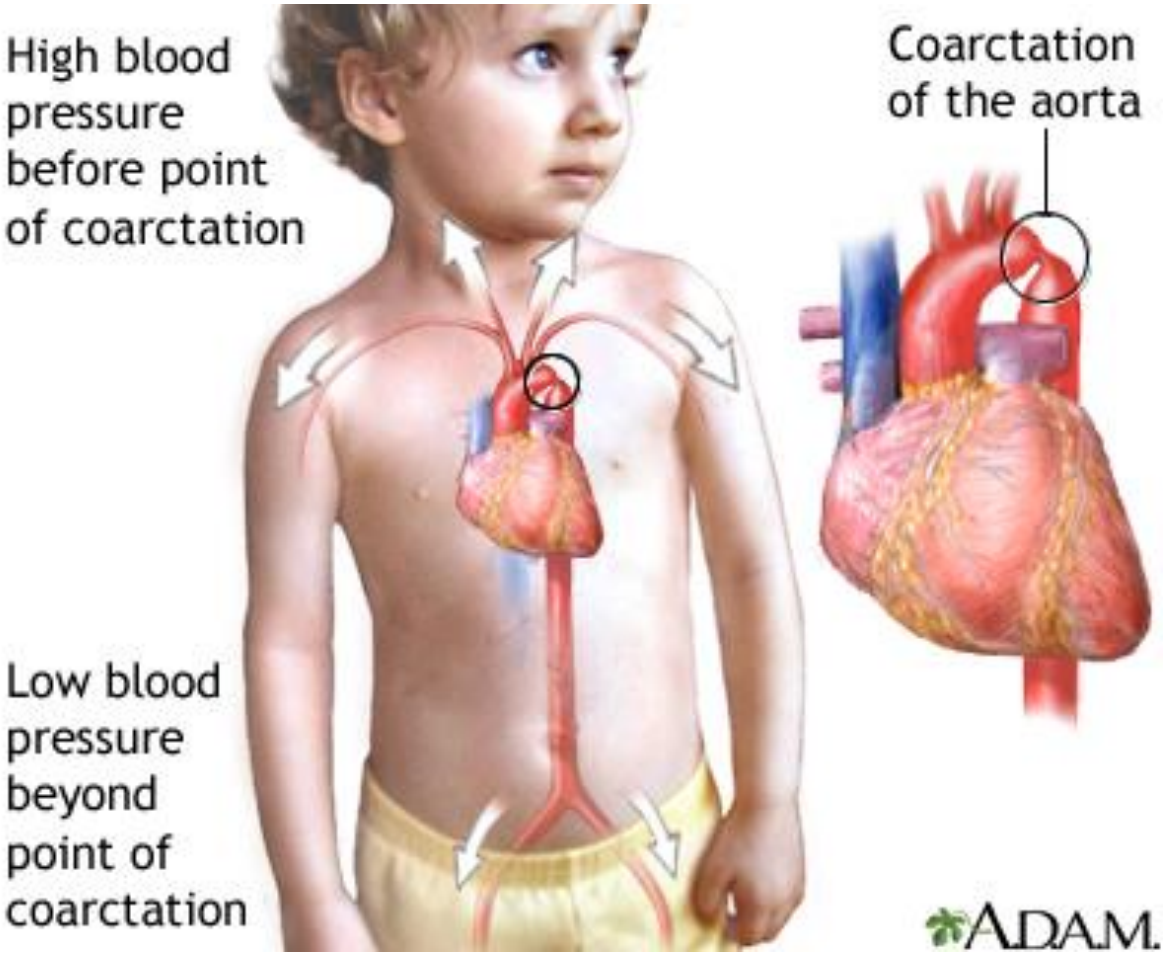
- Atrioventricular Septal Defect (AVSD)
- Signs and Symptoms
  - Left to right shunt that increases pulmonary artery pressure and can lead to pulmonary edema
  - Increased blood volume in the left ventricle posing risk for CHF
  - Tachypnea, sweating, poor feeding, poor weight gain
  - Heart murmur
  - Symptoms usually develop gradually over first few months of life

Left to Right Shunts

- Atrioventricular Septal Defect (AVSD)
- Treatment
  - Medical management
    - Often require diuretics/ace inhibitors for CHF symptoms
  - Complete AVSD surgical repair occurs at 3-6 months
  - Incomplete AVSD surgical repair occurs at 6-18 months
  - Repair includes closure of the atrial/ventricular septums with patches and separation of the common AV valve
- Anesthetic Management
  - Prior to repair, maintain PVR
  - Post-repair treat pulmonary hypertension with tactics that reduce PVR
    - Nitrix oxide, 100% FIO2, hyperventilation, deep anesthetic
  - **Be prepared for conduction disturbances**
    - Rhythm and rate control agents
    - Epicardial pacer wires

Obstructive Lesions

- Coarctation of the Aorta



Obstructive Lesions

- Coarctation of the Aorta
  - Represents 5-10% of CHD cases
  - Associated with Turner syndrome
  - Causes LV pressure overload and reduced perfusion to lower body
  - Most common at the juxtaductal portion of aorta
    - Portion where ductus arteriosus connected

- Presentation
  - Preductal → proximal to left subclavian artery, less common, presents in infants
  - Postductal → distal to left subclavian artery at site of ligamentum arteriosum, presents in young adults
    - Blood pressure should be attempted to be taken on the upper extremities, preferably the right arm

Obstructive Lesions

- Coarctation of the Aorta
- Signs and Symptoms
  - Harsh systolic ejection murmur
    - Loudest in the back where aorta is located
  - Systolic BP is higher in the arms than the legs but diastolic BP is similar
  - Weak and delayed femoral pulses
  - Systemic hypertension due to injecting LV stroke volume into fixed resistance of narrowed aorta
  - Headache, dizziness, epistaxis, and palpitations

Obstructive Lesions

- Coarctation of the Aorta
- Severe obstruction in the infant
  - Blood flow to lower body depends on PDA
  - Closure of the PDA can cause acute decompensation
  - Patency maintained with Prostaglandin E_1
  - Surgical correction is performed soon after stabilization
- Mild to moderate obstruction in older child
  - Body forms many collaterals through internal thoracic, intercostal, scapular, and subclavian arteries
  - Posterior rib notching visible on chest x-ray

Obstructive Lesions

- Coarctation of the Aorta
- Treatment
  - Surgical correction involves left thoracotomy incision off bypass
  - Resection with end-to-end anastomosis (most common)
  - Arch Advancement
    - Aorta distal to constriction is anastomosed to ascending aorta
  - Synthetic patch aortoplasty
  - Left subclavian flap aortoplasty
  - Balloon angioplasty +/- stent placement

Obstructive Lesions

- Coarctation of the Aorta
- Anesthetic Management
  - Must have RUE arterial line for monitoring during aortic clamp
  - Monitor blood gases as left thoracotomy poses risk for lung retraction
  - Permissive hypothermia for spinal cord protection related to cross-clamp
  - Prepare for hypertension during aortic clamp
  - Prepare for hypotension prior to the removal of aortic clamp
    - Decrease sevoflurane and fluid resuscitation
  - Rebound hypertension post-repair up to 1 week
    - Esmolol or nitroprusside

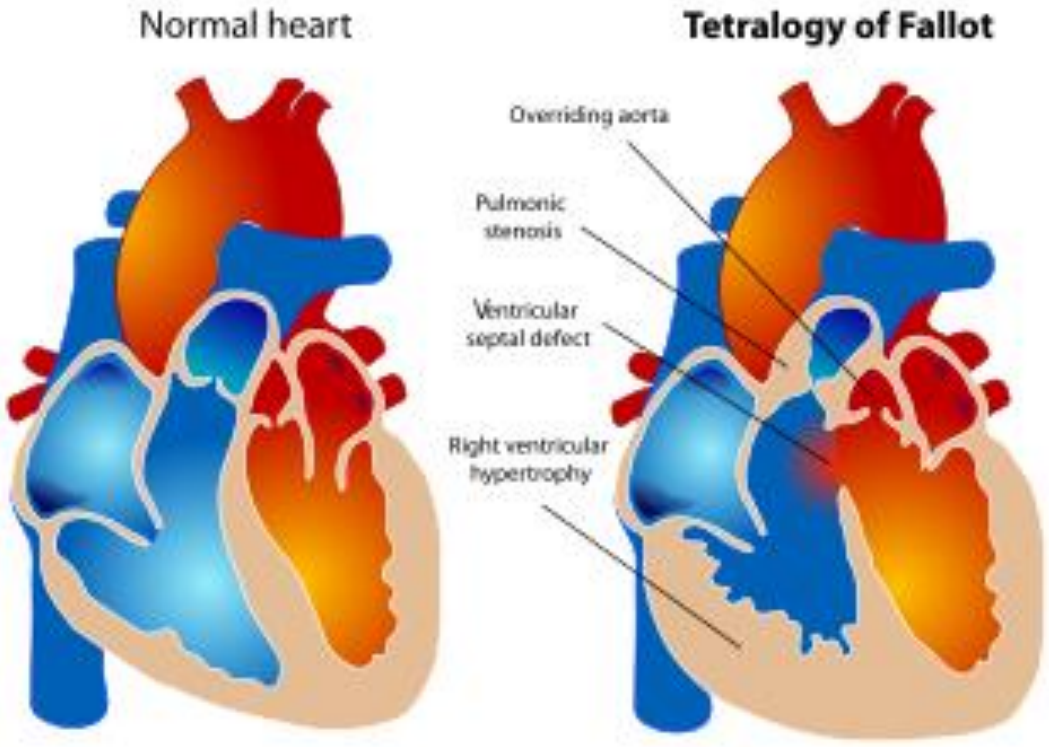
Cyanotic Congenital Heart Lesions

Right to Left Shunts

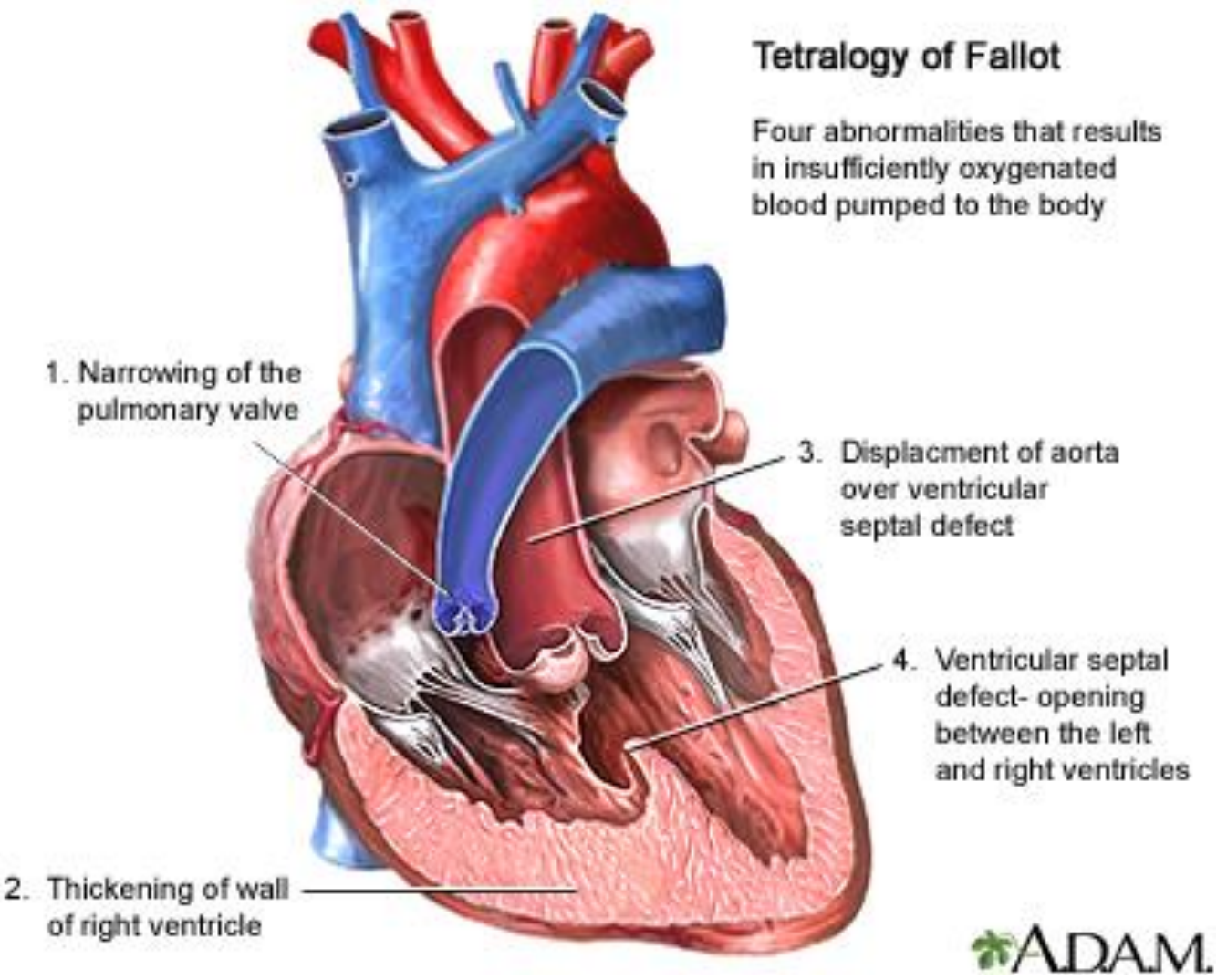
- The 5 T's
  - **Tetralogy of Fallot**
  - Tricuspid Atresia
  - **Transposition of the Great Arteries**
  - Total Anomalous Pulmonary Venous Connection
  - **Truncus Arteriousus**
- Characteristics
  - Decreased pulmonary blood flow
  - Arterial hypoxemia (blue baby)

Right to Left Shunts

- Tetralogy of Fallot
  - Most common cyanotic CHD (7-10% of all CHD)



Right to Left Shunts



Right to Left Shunts

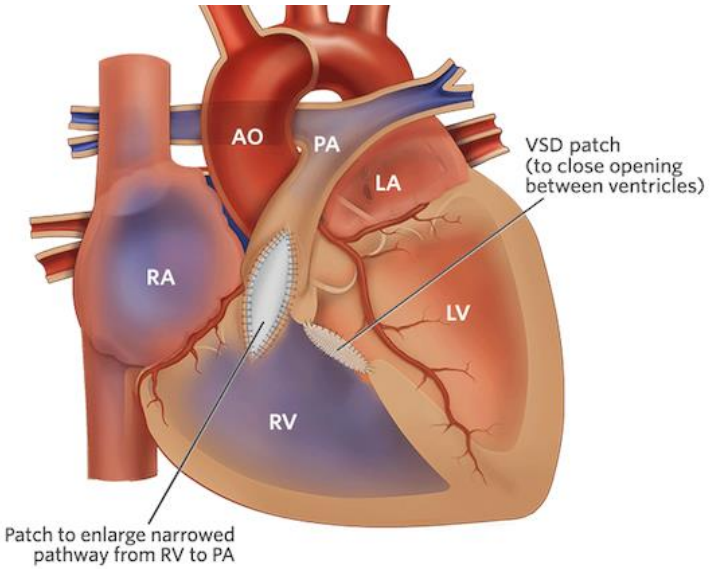
- Tetralogy of Fallot
- How does increased PVR influence intracardiac shunting?
- How does decreased SVR influence intracardiac shunting?
- Squatting or phenylephrine increase SVR, what does this do?

Right to Left Shunts

- Tetralogy of Fallot
- Signs and symptoms
  - Cyanosis develops between ages 2 and 6 months
  - Systolic ejection murmur at left sternal border
  - Boot shaped heart on chest radiograph
  - PaO_2 is usually <50 mm Hg
  - Squatting is common in children with TOF
  - "Tet spells" hypercyanotic attacks triggered by crying, defecation, feeding, or exercise

Right to Left Shunts

- Tetralogy of Fallot
- Treatment
  - Patch closure of VSD which allows blood to flow only from LV to aorta
  - RVOT is augmented by cutting away obstructive RV muscle and enlarging the path with a patch



Right to Left Shunts

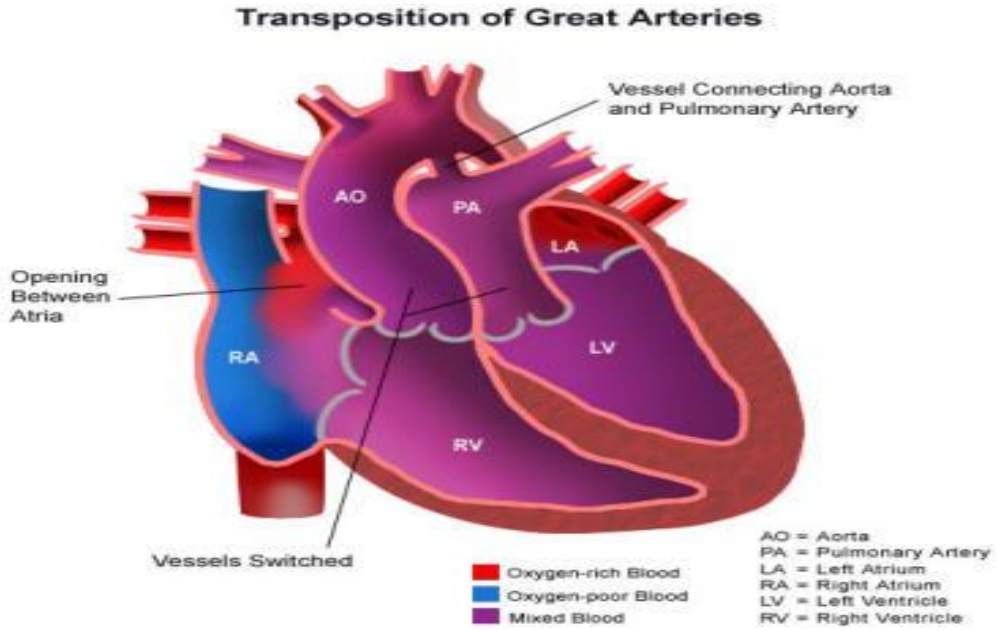
- Tetralogy of Fallot
- Anesthetic Management
  - Right to Left shunt increased by
    - ↓ SVR (volatiles, histamine, α-blockers)
    - ↑ PVR (positive airway pressure, PEEP)
    - ↑ myocardial contractility
    - Loss of (–) intrapleural pressure
  - Slow careful sevoflurane inductions
  - Initiate volume expansion immediately
  - Phenylephrine available to treat decreases in SVR
  - Inotropes to support the RV
  - Pacing for possibility of JET

Right to Left Shunts

- Tetralogy of Fallot
- Tet Spells
  - Most common during induction, just prior to surgical stimulation (lack of SNS tone), and during surgical manipulation
- Tet Spell Treatment
  - 100% FIO2
  - Fluid boluses
  - Phenylephrine 5-10mcg/kg
  - Abdominal compression or trendelenburg position
  - Esmolol 50 mcg/kg to reduce infundibular spasm
  - Aortic compression by surgeon

Right to Left Shunts

- Transposition of the Great Arteries
  - Accounts for 4%-8% of all CHD
  - Also classified as an admixture lesion
  - The most frequently encountered cause of cyanosis in the first week of life



Right to Left Shunts

- Transposition of the Great Arteries
- Flow Pattern
  - Flow is in parallel instead of the normal series
  - Right flow → RA to RV to Aorta to systemic
  - Left flow → LA to LV to PA to lungs
- Survival
  - Communication through ASD, VSD, or PDA

Right to Left Shunts

- Transposition of the Great Arteries
- Signs and Symptoms
  - Symptoms are based on anatomy (VSD, pulm. stenosis)
  - Cyanosis
  - CHF
  - Chest Radiograph is egg shaped with narrow stalk

Right to Left Shunts

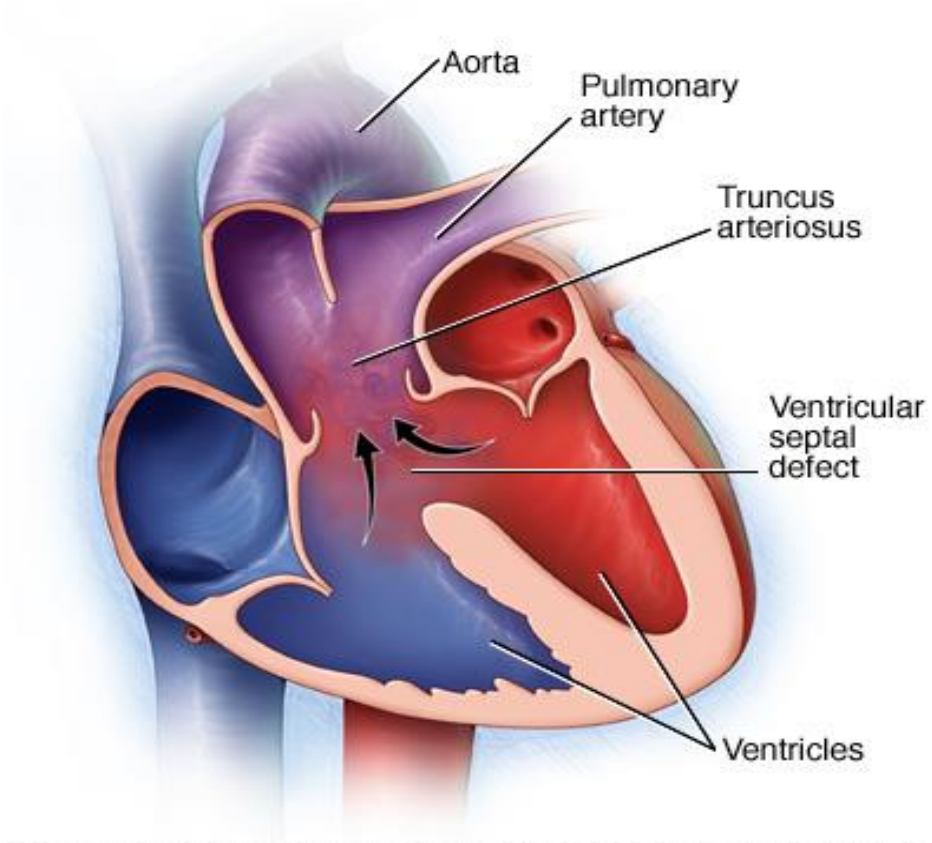
- Transposition of the Great Arteries
- Treatment (temporary vs. permanent)
  - Temporary
    - Prostaglandin E_1 infusion to keep ductus arteriosus patent
    - Balloon atrial septostomy (Rashkind's procedure)
  - Permanent
    - Arterial switch operation

Right to Left Shunts

- Transposition of the Great Arteries
- Anesthetic Management
  - Maintain heart rate, contractility, and preload
  - Maintain PGE if ductal-dependent
  - Prudent balance of SVR and PVR
    - High PVR can decrease pulmonary blood flow and reduce mixing
  - Tight blood pressure parameters to avoid pressure on arterial sutures
    - Bleeding is great risk
      - Anticipate requiring platelets and cryoprecipitate
      - May leave chest open
  - Monitor for myocardial ischemia that may be related to the re-implanted coronaries or left ventricle
    - Support left ventricle with inotropes and afterload reduction

Right to Left Shunts

- Truncus Arteriosus
  - Also classified as an admixture lesion



Right to Left Shunts

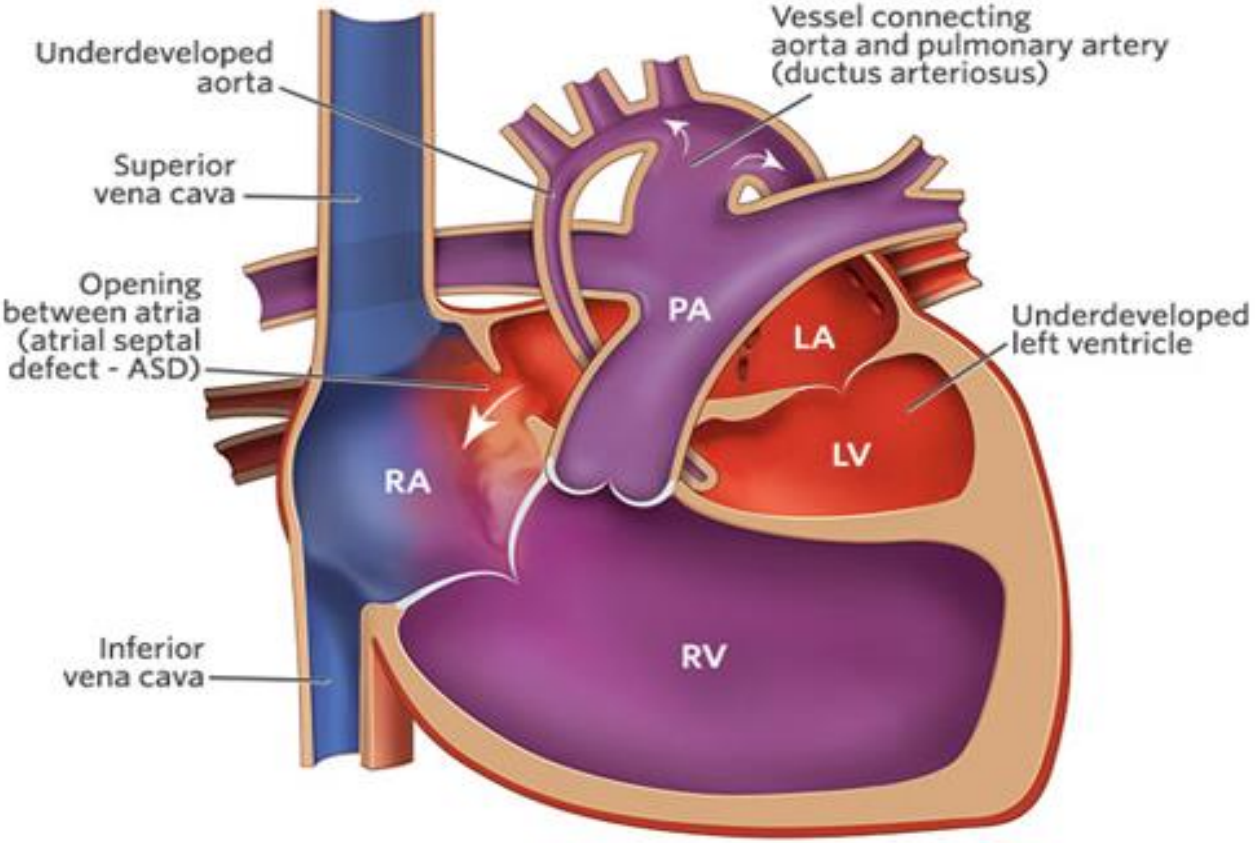
- Truncus Arteriosus
  - Represents 1-4% of all CHD
- Signs and symptoms
  - Unrestrictive left to right shunting (admixture lesion)
  - Pulmonary overcirculation
  - Cyanosis and arterial hypoxemia
  - Failure to thrive
  - CHF
  - Cardiomegaly on chest x-ray

Right to Left Shunts

- Truncus Arteriosus
- Treatment
  - Medical management
    - diuretics to control CHF symptoms
  - Surgical Treatment
    - Separation of pulmonary arteries from main truncus
    - Patch closure of VSD (to isolate LV output to truncus)
    - Dacron conduit with valve from RV to PA
- Anesthetic Management
  - Maintain heart rate, contractility, and preload
  - Minimize pulmonary overcirculation by maintaining PVR with positive airway pressure, PEEP, low FiO_2
    - Phenylephrine and fluids to reverse myocardial ischemia in the setting of "steal" from the systemic circulation
  - Inotropic support for CHF

Admixture Lesions

- Single Ventricle Lesions (HLHS)



Admixture Lesions

- Single Ventricle Lesions
  - Double inlet left ventricle (single LV)
  - Hypoplastic Left Heart Syndrome (single RV)
  - Double outlet right ventricle (single RV)
  - Parallel circulation
  - **Balance between SVR and PVR is critical for survival**

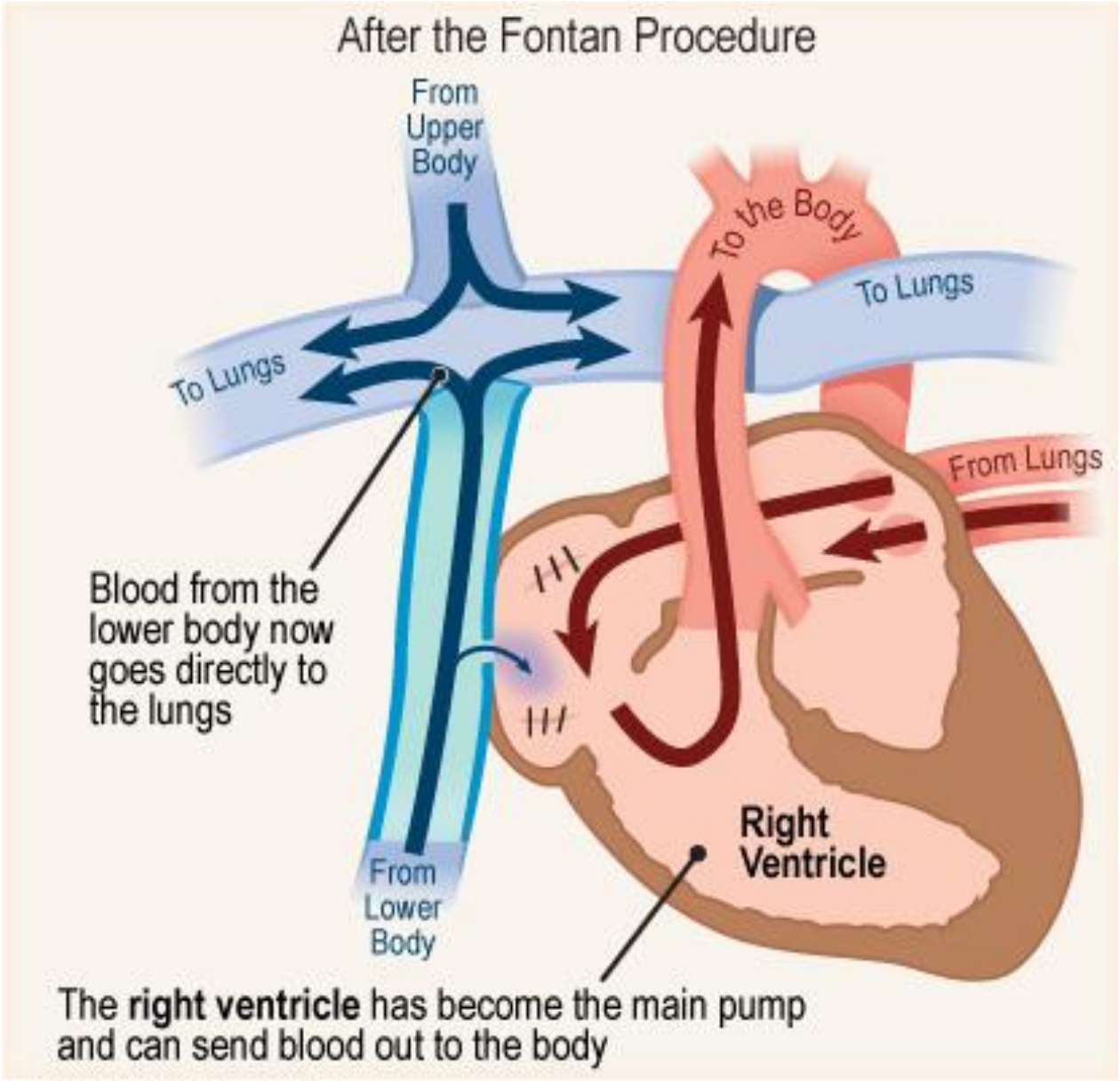
Admixture Lesions

- Single Ventricle Lesions
- Treatment
  - Three stage process

- *Neonatal*: aortic reconstruction, systemic-pulmonary shunt, or PA band (Norwood Procedure)
- *Age 3-6 months*: Superior cavopulmonary connection (Glenn Procedure)
- *Age 2-4 years*: inferior cavopulmonary connection (Fontan Procedure)

Admixture Lesions

- Single Ventricle Lesions
- Life and physiology after Fontan Procedure
  - Perfusing the lungs relies on passive venous return from SVC and IVC
  - RV becomes main pump for systemic blood flow
  - **It is important to keep negative intrathoracic pressure and adequate preload**
  - **Avoid positive pressure ventilation or hypovolemia**


After the Fontan Procedure
From Upper Body
To the Body
To Lungs
To Lungs
From Lungs
Blood from the lower body now goes directly to the lungs
Right Ventricle
From Lower Body
The right ventricle has become the main pump and can send blood out to the body
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Heart Transplant

- Pediatric patients account for 13% of all heart transplants
  - Cardiomypoathies, CHD, retransplantations
- Anesthetic Management
  - Patient's underlying defect/repairs should guide management
  - Induce to blunt SNS response to intubation, but prevent myocardial depression and hypotension
    - Etomidate, high fentanyl dosing, low sevoflurane
  - Allow time for drugs to circulate in low ejection fraction state
  - Preparing for termination of cardio-pulmonary bypass
    - Impaired systolic function
    - Right ventricular afterload mismatch
    - SA and AV node dysfunction
    - Reduced diastolic dysfunction

Heart Transplant

- Non-Cardiac Surgery after Transplant
- Denervated heart
  - Dependent on intact Frank-Starling mechanism
  - Dependent on circulating cathecholamines
  - Increased resting heart rate (loss of vagal tone)
  - Reduced response to exercise and stress
- Altered response to medications
  - No response to atropine, glyco, nor digitalis
  - Possible severe bradycardia with neostigmine
  - Exaggerated response to direct acting sympathetic agents
  - Exaggerated response to calcium channel blockers, beta blockers, and adenosine
  - Reduced response to indirect acting agents including dopamine and ephedrine

SBE Prophylaxis

AHA Recommendations:

- Patients antibiotics are required for
  - If prosthetic valve is in place
  - History of infective endocarditis
  - Unrepaired cyanotic CHD
  - Repaired CHD in last 6 months with prosthetic material
  - Repaired CHD with residual defects
  - Heart transplant recipient with valvuloplasty
- Procedures antibiotics are required for
  - Dental procedures with manipulation of gingival tissue
  - Respiratory tract procedures
  - Procedures on infected skin, skin structures, or musculoskeletal procedures
  - NOT required for GI/GU procedures if no active infection

Pediatric Vs Adult CPB Systems

Table 6.1 Differences between adult and pediatric cardiopulmonary bypass systems.

	Adult	Pediatric
Estimated blood volume	65 ml kg^{-1}	<10 kg: 85 ml kg^{-1}
Dilution effects on blood volume	25–33%	Up to 100–200%
Addition of whole blood or packed red blood cells to prime	Rarely	Usually
Oxygen consumption	2–3 ml kg^{-1} min^{-1}	6–8 ml kg^{-1} min^{-1}
Full CPB flow at 37 °C	50–75 ml kg^{-1} min^{-1}	150–200 ml kg^{-1} min^{-1}
Minimum CPB temperature	Rarely <30 °C	Commonly <30 °C
Use of total circulatory arrest or regional cerebral perfusion	Rare	Common for some defects; HLHS, Hypoplastic aortic arch
Perfusion pressure	50–80 mmHg	30–50 mmHg
Acid–base management	Primarily alpha-stat	Primarily pH-stat ≤30 °C
Measured $PaCO_2$ differences	30–45 mmHg	20–80 mmHg

- Chart taken from Nasr, V. G. & Dinardo, J. A. (2017) pg. 53.

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