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Atrial Septal Defects and Patent Ductus Arteriosus: Diagnosis and Management

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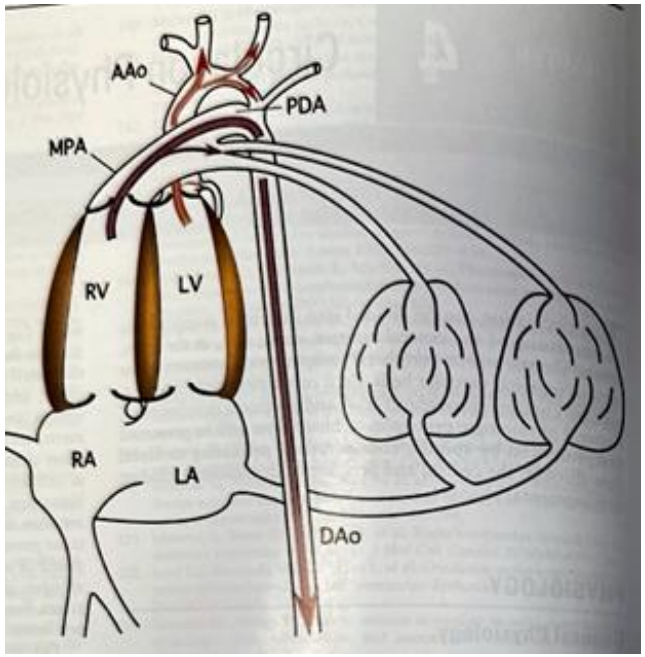
Fetal Circulation and Transition

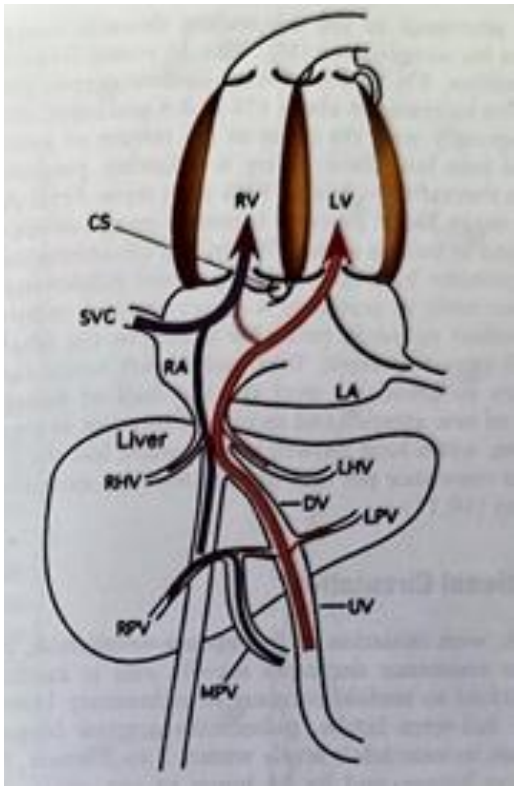
Fetal circulation

Presence of central shunts allows for efficient fetal circulation



PDA

Desaturated blood ejected from the RV crosses the PDA to the descending aorta to the placenta



PFO

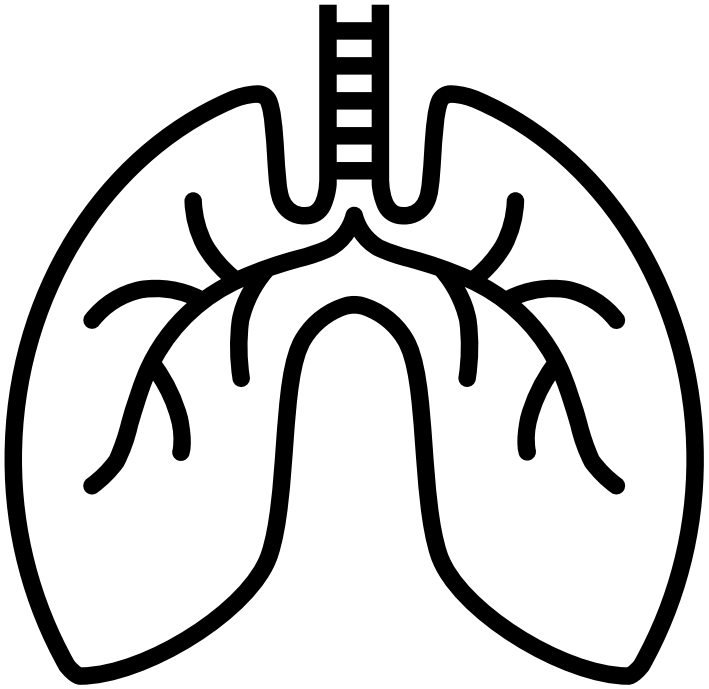
- Desaturated blood from the SVC and coronary sinus are directed toward the tricuspid valve and RV
- Highly saturated blood from the placenta passes through the ductus venosus and is directed toward the PFO into the LA

Moss and Adams 8th ed, figures 4.1 and 4.2

Normal Transition

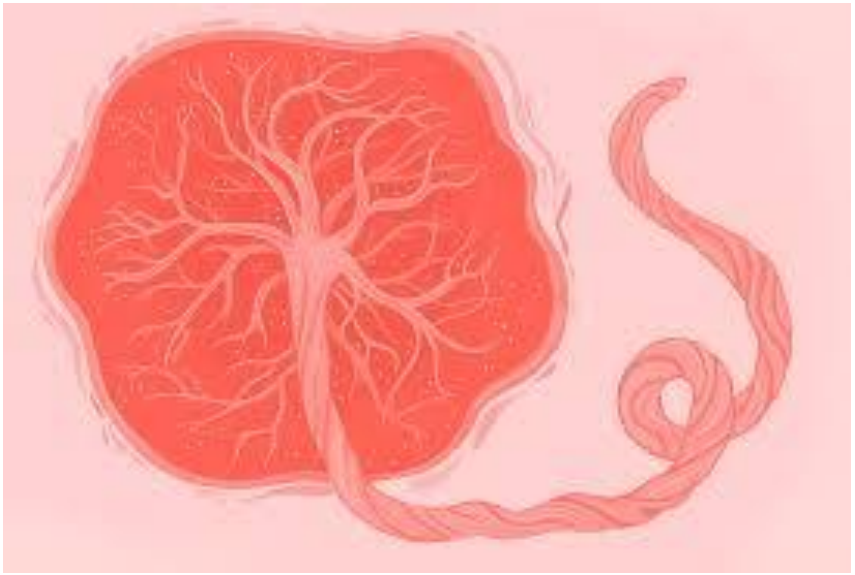
Expansion of the Lungs

1. Rapid and large decrease in PVR →
2. Increased blood flow to the lungs →
3. Increased pulmonary venous drainage to LA →
4. Increased LA pressure →
5. Functional closure of PFO



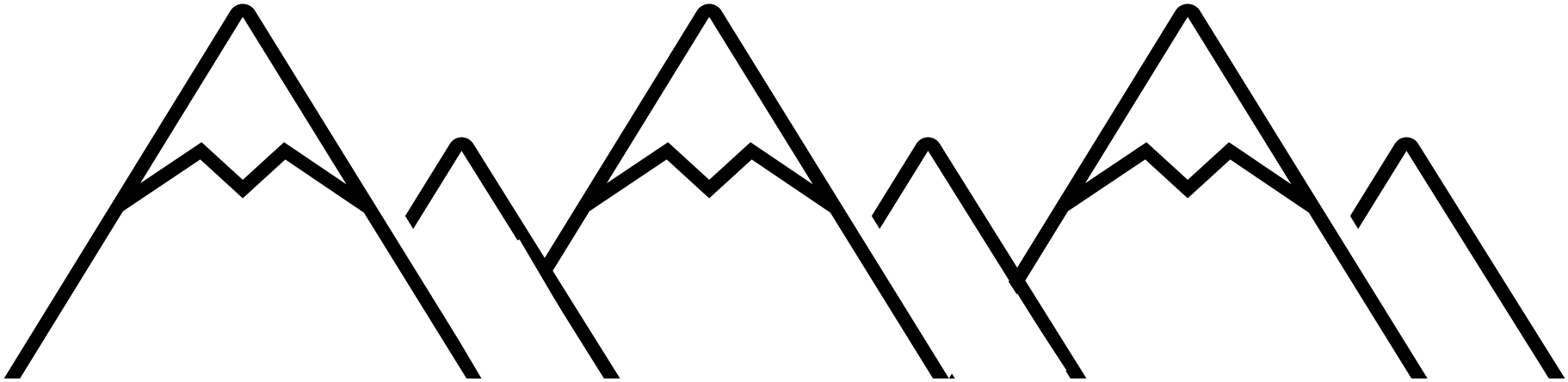
Normal Transition 2

1. Disruption of umbilical-placental circulation →
2. Increase in SVR →
3. PDA reverses direction to left to right →
4. Functional closure of ductus arteriosus by constriction in ~12-15 hours →
5. Hypoxia of the intimal layers →
6. Cell destruction / fibrosis →
7. Permanent closure in ~5-7days



Effects of altitude

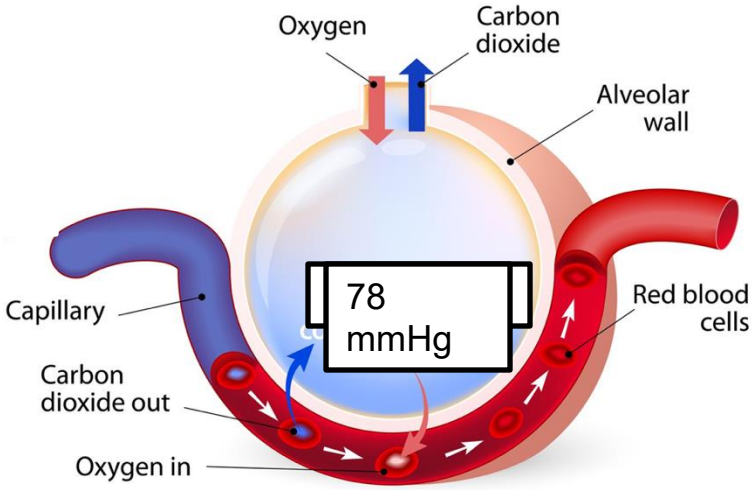
- Evidence to support that exposure to high PO2 and very low PO2 contributes to constriction of the ductus arteriosus in term infants
- Term infants born at high altitudes have greater incidence of PDAs



Effects of altitude 2

Alveolar gas equation

- $P_AO_2=[(P_{atm}-P_{H2O})FiO2]-(P_aCO_2/RQ)$
  - Sea level: P_{atm} = 760 mmHg
    - P_AO_2 =[(760 – 47)0.21] – (40/0.8)
    - P_AO_2 = 100 mmHg
  - Average elevation in Zambia (1200 meters above sea level)
    - P_{atm} = 658 mmHg
    - P_AO_2 =[(658 – 47)0.21] – (40/0.8)
    - P_AO_2 = 78 mmHg
- Normal A-A gradient 5-10 mmHg



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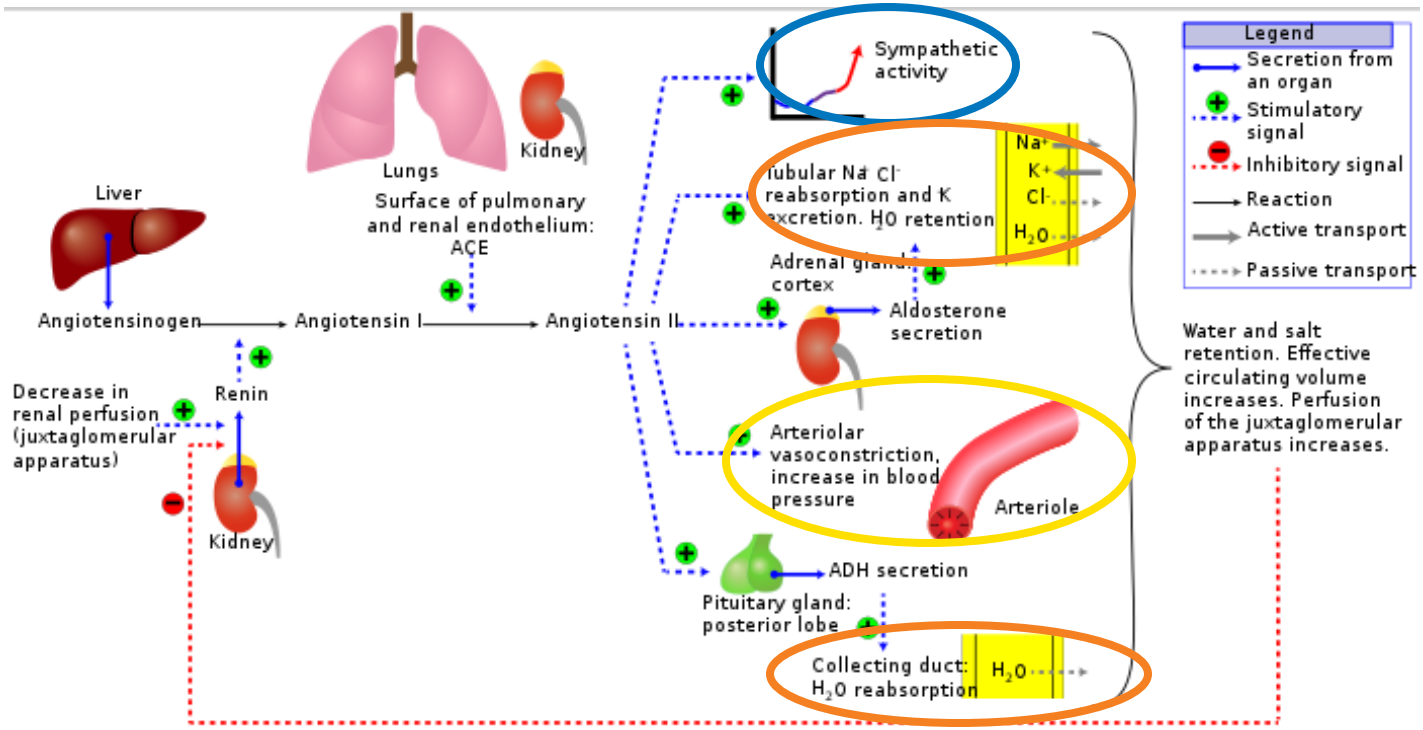
Physiology of left to right shunts

Left-to-right Shunt Lesion

- Normal Physiology (Ao sat > PA sat)
- Inefficient circulation: Highly saturated blood travels to the pulmonary circulation without participating in oxygen delivery
- Pulmonary edema: Qp > Qs
- Volume overload

Renin-angiotensin pathway



1. Increased sympathetic activity
2. Increased water retention
3. Increased SVR

- Increased pulmonary blood flow
- Decreased systemic blood flow → decreased peripheral perfusion
- Activation of renin-angiotensin system

Preload

- Blood in the ventricle at the end of diastole – End-diastolic volume
- Affects the stretch on myocardium just before contraction starts
- Preload is inferred from EDP in clinical practice

Preload in L→R shunts

- Increased water retention – increases blood volume (renin-angiotensin system)
- Pulmonary edema (Qp>Qs)
- Heart chamber dilation

Afterload

- The mass that resists contraction
- The hydraulic load that must be overcome to eject blood
- Any factor that resists ejection of blood from the heart
  - Impedance of the vasculature (SVR)
  - Ejection pressure (end-systolic pressure)
  - Ventricular outflow tract obstruction
  - Ventricular wall stress (law of Laplace and transmural pressure)
  - Inertia

Afterload for L→R shunt

- Ventricular wall stress is higher (Law of Laplace)
- Inertia is higher (the greater the mass, the greater the inertia and therefore the greater the afterload)
- Increase in SVR (renin-angiotensin system)

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Difference between types of shunts

Volume of shunt

Size of the defect

Pressure differential

Volume of shunt 2

PDA

- Tubular structure – resistance of flow depends on length and diameter
- Difference in Aortic pressure and PA pressure drives flow (increases as PVR declines)

ASD

- Primary determinant of flow is the relative compliance of the ventricles

Chamber Dilation

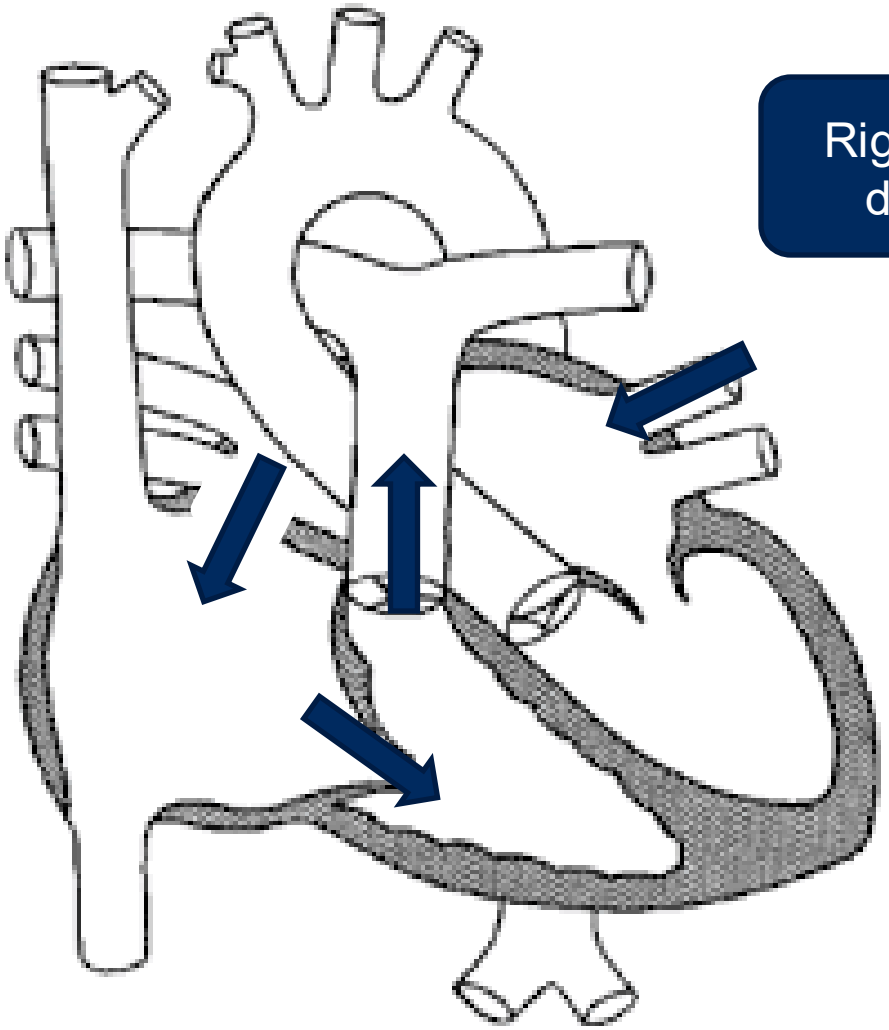
Right Heart

- Hepatic congestion
- Edema
- Atrial arrhythmias

Left Heart

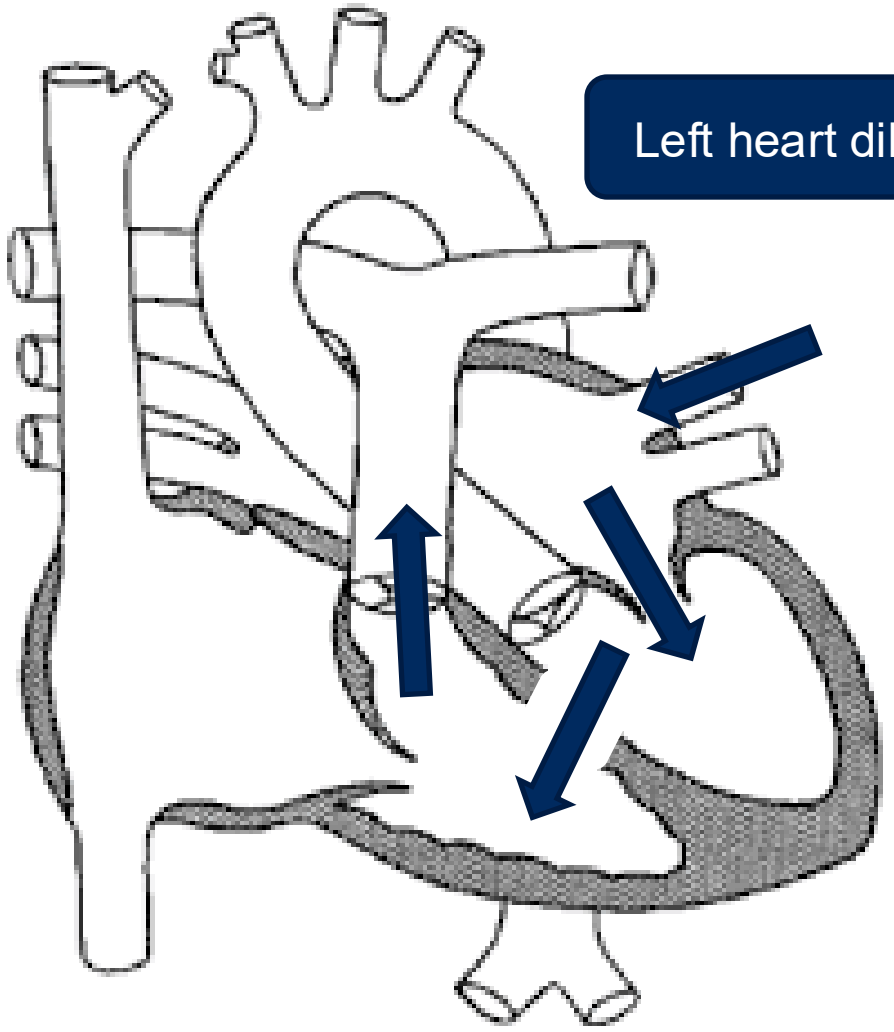
- Pulmonary venous hypertension
- Pulmonary edema
- Can also have right heart volume overload

Atrial Shunt

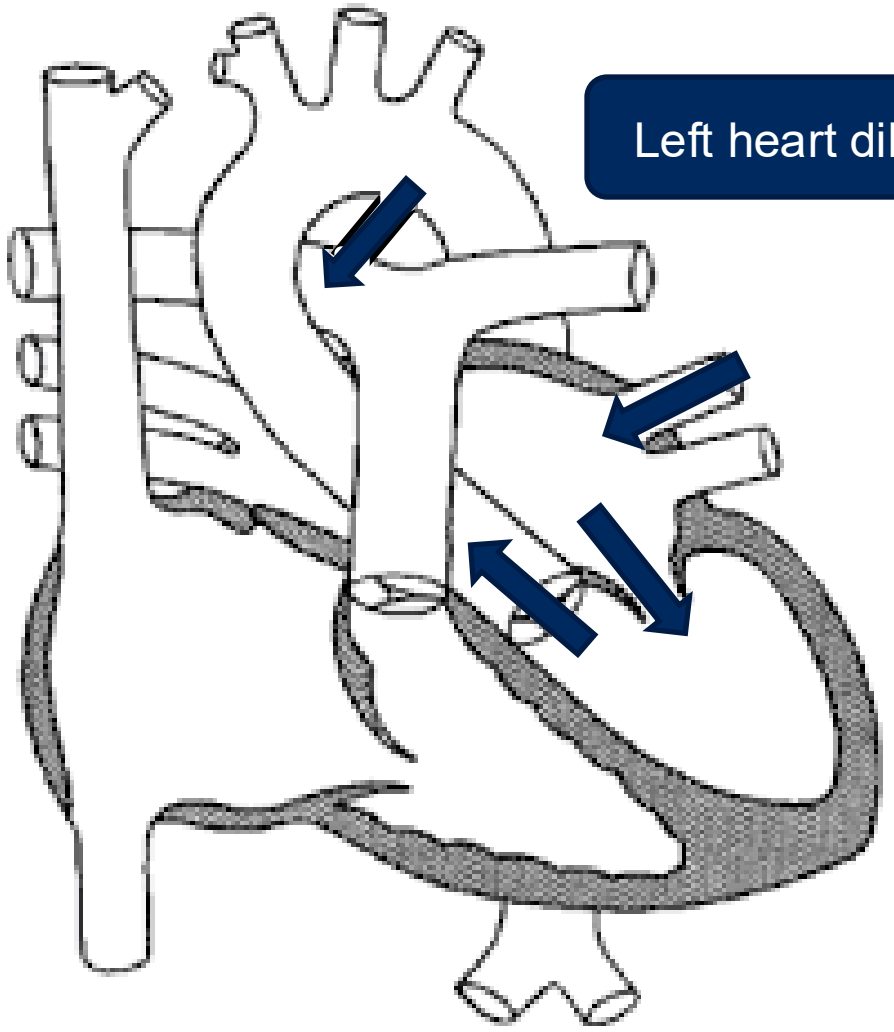


Right heart dilation

Ventricular shunt



Arterial shunt



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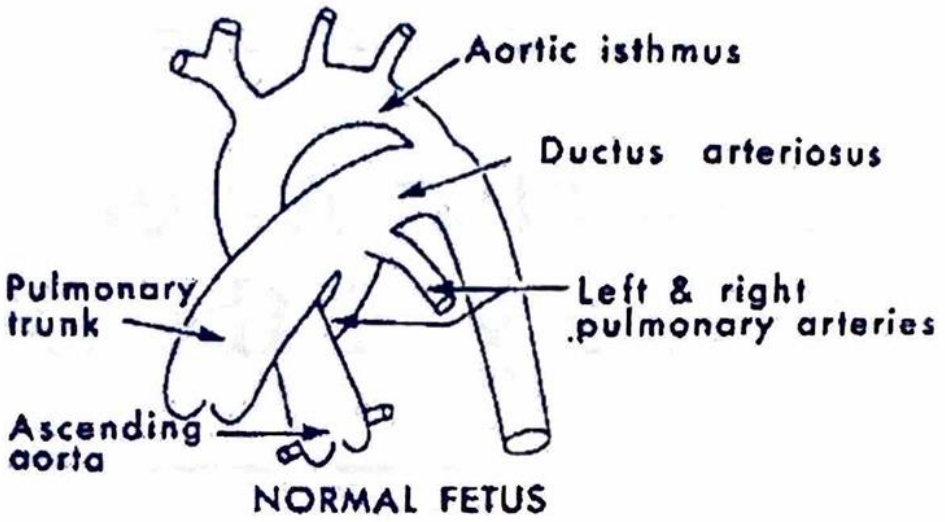
Patent Ductus Arteriosus

Case

- 6-month-old with a PDA and a PFO, 4.5 kg
  - Diagnosed at 3 months of age- difficulties breathing and poor weight gain
  - Furosemide 5 mg 3X/day, Spironolactone 3.125 mg 2X/day
  - Current symptoms: poor weight gain, repeated hospital admissions for chest infections
  - Current exam: no respiratory distress (RR 38 bpm), tachycardia (HR 143 bpm), hyperactive precordium, continuous murmur at left upper sternal border, no hepatomegaly

Anatomy

- In a term fetus: the ductus arteriosus is the same diameter as the descending aorta
- Ductus arteriosus connects:
  - To the aorta ~5-10 mm from the left subclavian artery origin
  - To the MPA at the branching point of the right and left PA
- Recurrent laryngeal nerve, phrenic nerve and thoracic duct are in proximity to the ductus arteriosus



Rudolph, Congenital Disease of the Heart, 2nd ed, figure 5.1

Clinical history

- Small PDA:
  - High resistance → small left to right shunt
  - Do not develop symptoms of LV volume overload
  - Many are asymptomatic

Physical Exam

- Small PDA
  - Continuous flow murmur 2nd left intercostal space

Diagnostic evaluation

- Small PDA
  - ECG and CXR usually normal

Diagnostic Evaluation 2

- Echocardiography
  - Size of the PDA
  - Assessment of shunt: LA and LV dilation, MPA dilation, flow reversal in the descending aorta, diastolic flow in the branch Pas
  - PA pressure estimation
  - Other congenital cardiac abnormalities
- Cardiac Cath:
  - Not typically needed for the diagnosis of PDA
  - Patients undergo cardiac cath if require assessment of pulmonary hypertension prior to closure

Natural History

- Small PDA
  - Typically do well

Premature Infants

Constriction is ineffective in premature infants

The more premature – the more likely the ductus will remain patent

May have earlier and more significant clinical presentation

If PVR is high from lung disease, may not have a significant L→R shunt

Aggressive treatment and closure if signs of respiratory distress, low cardiac output and/or signs of systemic steal (NEC)

Symptom management

Diuretics

Fortification of EBM/formula to increase kcals without increasing volume intake

Respiratory support if significant respiratory distress

Closure

Symptomatic PDAs and asymptomatic PDAs causing LA/LV enlargement should be closed

In asymptomatic infants, can wait until ~9 months of age to see if there is spontaneous closure

Contraindication to closure: Patients with non-reactive pulmonary vascular disease and R→L shunt across PDA

Closure Continued

Medical

- Preterm infants, More likely to be successful at <10 days of life
- NSAIDs: Indomethacin, Ibuprofen
  - Risks: Renal dysfunction, bleeding
- Paracetamol

Surgical ligation

- Left lateral thoracotomy
- May develop low cardiac output – acute increased afterload and decreased preload to the LV
- Risks: injury to structures close to the PDA

Catheter device closure

- Risks: Vascular injury, device embolization, LPA obstruction and coarctation

Follow-up

- Immediately after surgical ligation:
  - Watch for signs of chylothorax (thoracic duct injury), vocal cord paralysis (recurrent laryngeal nerve injury), diaphragm paralysis (phrenic nerve injury)
  - May develop low cardiac output after closure of large PDA which requires support
- After closure: LA and LV should regress to normal size
- SBE ppx for 6 months after catheter device closure

Case 1

- 6-month-old with a PDA and a PFO, 4.5 kg
  - Diagnosed at 3 months of age- difficulties breathing and poor weight gain
  - Furosemide 5 mg 3X/day, Spironolactone 3.125 mg 2X/day
  - Current symptoms: poor weight gain, repeated hospital admissions for chest infections
  - Current exam: no respiratory distress (RR 38 bpm), tachycardia (HR 143 bpm), hyperactive precordium, continuous murmur at left upper sternal border, no hepatomegaly

- What in his history/exam is worrisome?
- What are your next steps in management?
- Is the PDA or the PFO more likely to be causing his symptoms?
  - Echo: 4.7 mm PDA with left to right shunt, small PFO, no AV valve regurgitation, dilated left atrium and left ventricle, normal ventricular function

Case Continued

- 6-month-old with a PDA and a PFO, 4.5 kg
  - Diagnosed at 3 months of age- difficulties breathing and poor weight gain
  - Furosemide 5 mg 3X/day, Spironolactone 3.125 mg 2X/day
  - Current symptoms: poor weight gain, repeated hospital admissions for chest infections
  - Current exam: no respiratory distress (RR 38 bpm), tachycardia (HR 143 bpm), hyperactive precordium, continuous murmur at left upper sternal border, no hepatomegaly

- Echo: 4.7 mm PDA with left to right shunt, small PFO, no AV valve regurgitation, dilated left atrium and left ventricle, normal ventricular function
- Referred for closure of PDA

Patent ductus arteriosus 1

Normal: functionally closes at 10-15 hours
Degree of shunt: size and pressure difference between Ao and PA
Premature infants: aggressive management in symptomtic patients
Term infants: close if symptomatic or asymptomatic with left heart dilation

Pre-op Care:
- Diuretics
- Limit FiO2
- Increase calories

Post-op Care:
- Watch for chylothorax, vocal cord paralysis, diaphragm paralysis

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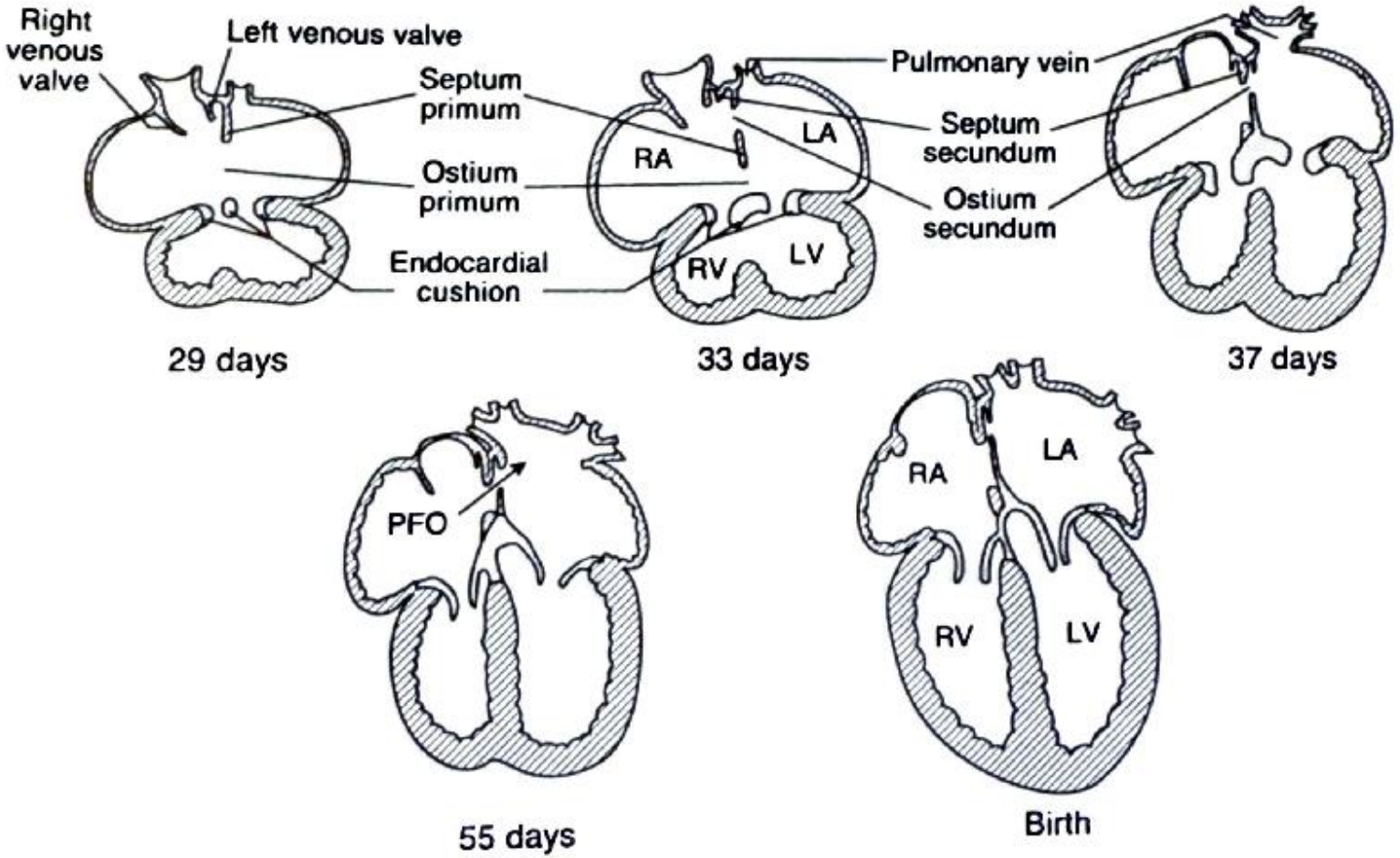
Atrial Septal Defects

Case 2

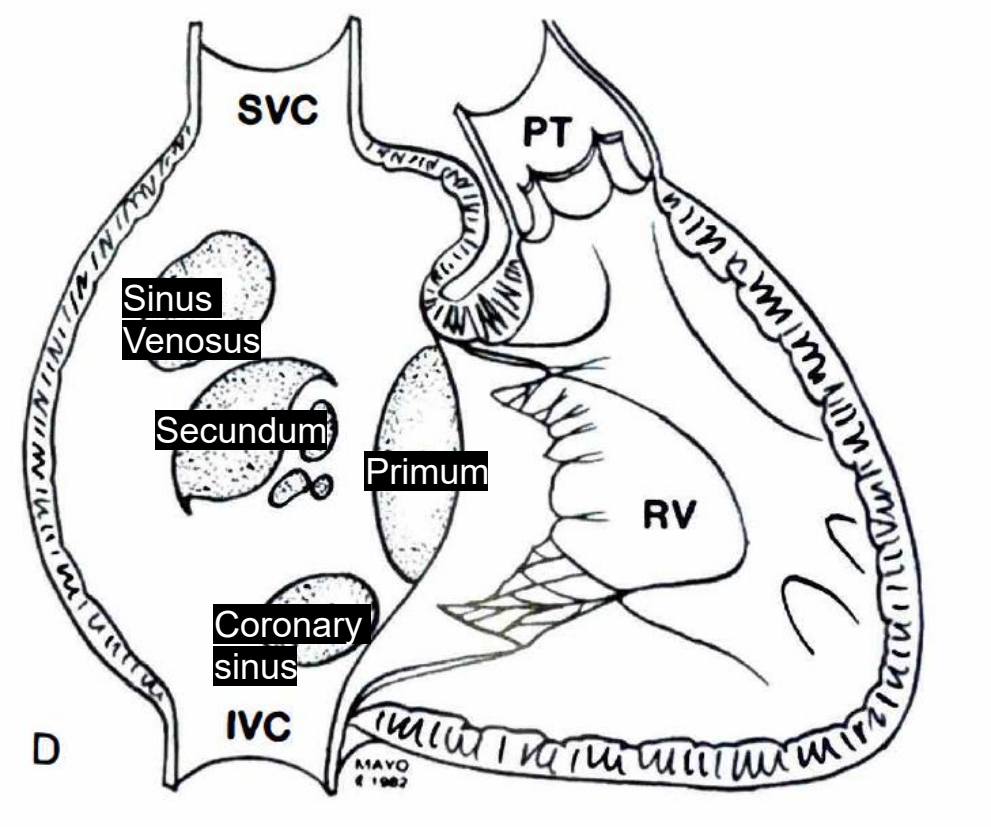
- 14 yo male with ASD repaired at age 3
  - Was the timing of repair appropriate?
  - What are potential complications that must be followed? Does it change based on surgical vs cath based closure?
  - What should follow up look like?

Embryology



Moss and Adams 8th ed, figure 28.1

ASD Anatomy



Moss and Adams 8th ed, figure 28.2

ASD Clinical history

- Most patients with ASD are asymptomatic
- Often diagnosed later in life
- Older children: symptoms of mild fatigue and dyspnea
- If symptomatic early in life – investigate the mitral valve
  - Mitral stenosis and regurgitation can increase L→R shunting across atrial septum
- Associated with syndromes: Holt-Oram syndrome

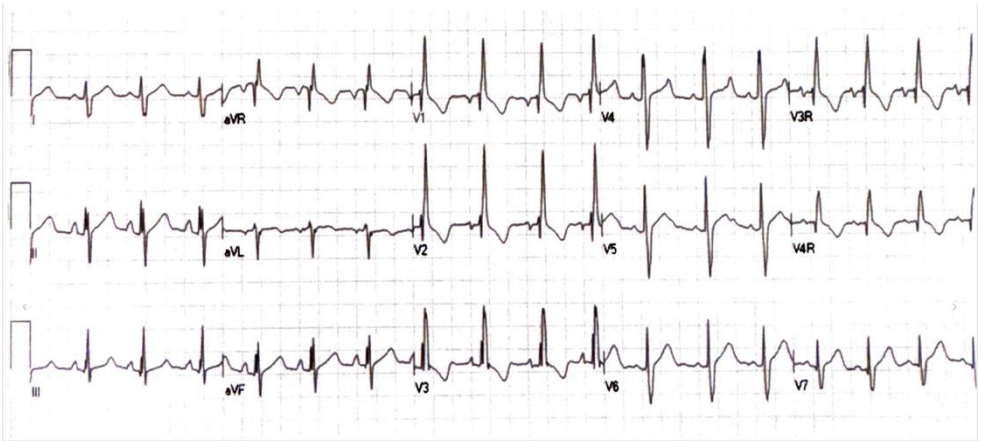
ASD Physical Exam

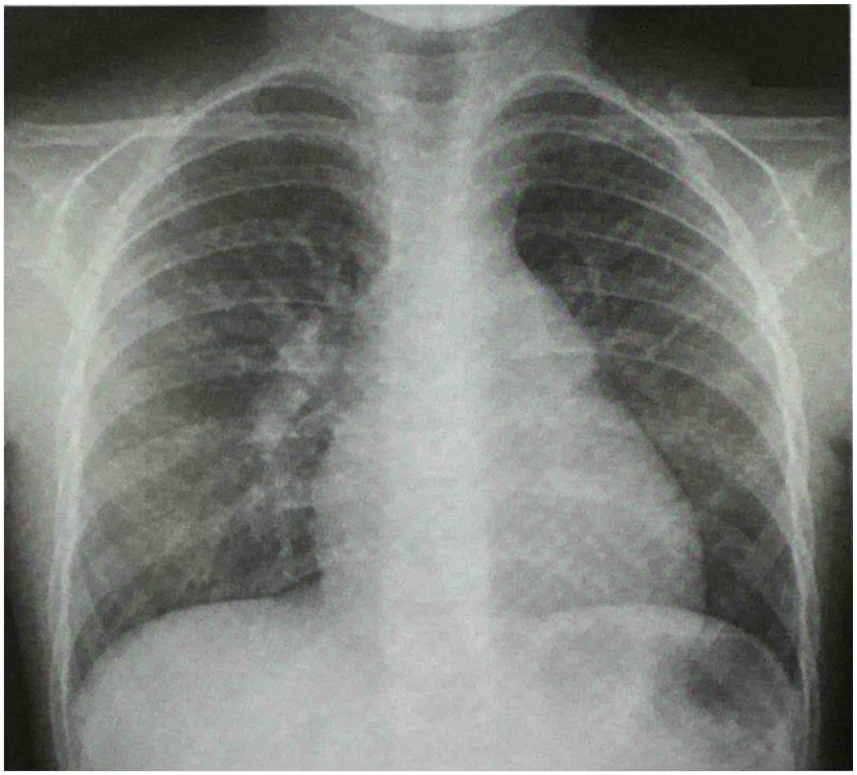
- Prominent RV impulse along lower left sternal border: volume-overloaded RV
- Fixed Split S2: delay in closure of the pulmonary valve due to prolonged emptying of the volume-overloaded RV that does not vary with the respiratory cycle
- Systolic ejection murmur LUSB: increased flow across the pulmonary valve

ASD Diagnostic Evaluation

- ECG
  - Small L→R shunt: ECG can be normal
  - Large L→R shunt:
    - rSR' in right precordial leads (RV volume overload)
    - Right axis deviation (RV volume overload)
    - Tall p waves (RA enlargement)
- CXR
  - Small L→R shunt: normal
  - Large L→R shunt:
    - Cardiomegaly – RA and RV enlargement
    - Increased pulmonary vascular markings
    - Prominent MPA

4 yo with large secundum ASD





Moss and Adams 8th ed, figures 28.6 and 28.7

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Diagnostic Evaluation Continued

- Echocardiogram
  - Define anatomy and size of ASD
  - Degree of atrial shunt
  - Right heart dilation
  - Estimated RV pressure
  - Associated cardiac lesions
- Cardiac Cath
  - Rarely indicated for diagnosis
  - Helpful if concerned for pulmonary vascular disease

ASD Natural history

- Secundum ASDs may close spontaneously
  - <5 mm during infancy – likely to spontaneously close
  - >8 mm – unlikely to spontaneously close
- Primum ASD, sinus venosus defects and coronary sinus defects will not close spontaneously
- Long-term complications:
  - Pulmonary hypertension develops in adulthood in patients with large L→R shunt from ASD (later than other L→R shunts)
  - Risk of atrial arrhythmias due to atrial stretch
  - Paradoxical embolism
  - RV dilation and dysfunction – congestive heart failure

Medical Management

- Most patients do not need medical management
- Diuretics may be indicated

ASD Closure

- Closure indicated: Qp:Qs ≥ 1.5
- Asymptomatic patients: closure recommended at 2-5 years
- Prevention of long-term complications
- If identified later in life:
  - Cardiac cath for pulmonary vascular reactivity prior to closure
  - If irreversible pulmonary hypertension: contraindication for closure

ASD Closure Continued

Surgical closure

- Only option for coronary sinus defects and primum ASDs
- Preferred for sinus venosus defects
- Secundum ASDs that do not have sufficient rims

Catheter device closure

- Option for secundum ASDs
- Sinus venosus defects can be closed in older patients that are not good surgical candidates

Complications

Surgical closure

- Inadvertent attachment of the eustachian valve to atrial septum – cyanosis from right to left shunt
- Residual shunts
- Risks associated with bypass

Catheter device closure

- Fracture or embolization of the device
- Device malalignment
- Residual shunts
- Impingement of adjacent structures
- Late erosion of atrial wall or aorta

Follow up

- Right heart size should regress to normal over time after closure
- Post-pericardiotomy syndrome: risk in first few weeks following surgical repair
- Bacterial endocarditis: antibiotic prophylaxis in the first 6 months after closure
- Pulmonary hypertension: periodic surveillance with echo to estimate PA pressures
- Atrial arrhythmias: After the age of 40, higher risk of atrial arrhythmias compared to general population

ASD Case Continued

- 14 yo male with ASD repaired at age 3
  - Was the timing of repair appropriate?
  - What are potential complications that must be followed? Does it change based on surgical vs cath based closure?
  - What should follow up look like?

- Echo in clinic: no residual ASD, no significant AV valve regurgitation, normal ventricular size and function

Atrial shunt ASD

Indications for closure: Qp:Qs >1.5:1
Fixed splitting of S2 with right heart dilation on echo
Typically repair/close 2-5 years of life
Earlier if signs of significant heart failure

Pre-op Care:
- ?Diuretics

Post-op Care:
- Early extubation
- ?Diuretics

Summary

- Left to right shunt volume depends on size of defect and pressure differential
- ASD: recommend closure between 2-5 years of life, earlier if symptomatic
- PDA: recommend closure for symptomatic premature infants and symptomatic term infants once identified and asymptomatic term infants with left heart dilation at ~9 months of life
- All patients with unrepaired L→R shunts are at risk for developing pulmonary hypertension

Thank you!