ADVANCED NICU FELLOWSHIP CARDIAC

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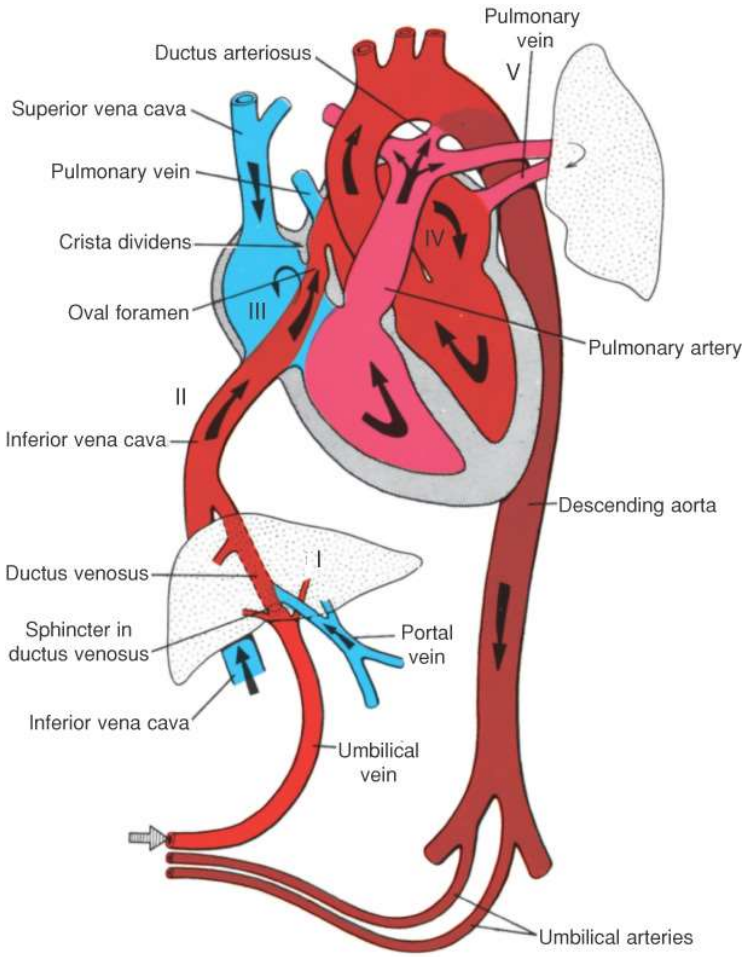
FETAL CIRCULATION

- The placenta provides gas exchange for the fetus (not the lungs)
- The ductus venosus & the foramen ovale close
- Decrease in pulmonary vascular resistance occurs at birth and continues for 6-8 weeks



3 MAJOR FETAL SHUNTS

Ductus arteriosus – shunts blood from pulmonary artery into aorta

- Functionally closes shortly after birth
- Becomes the ligamentum venosum

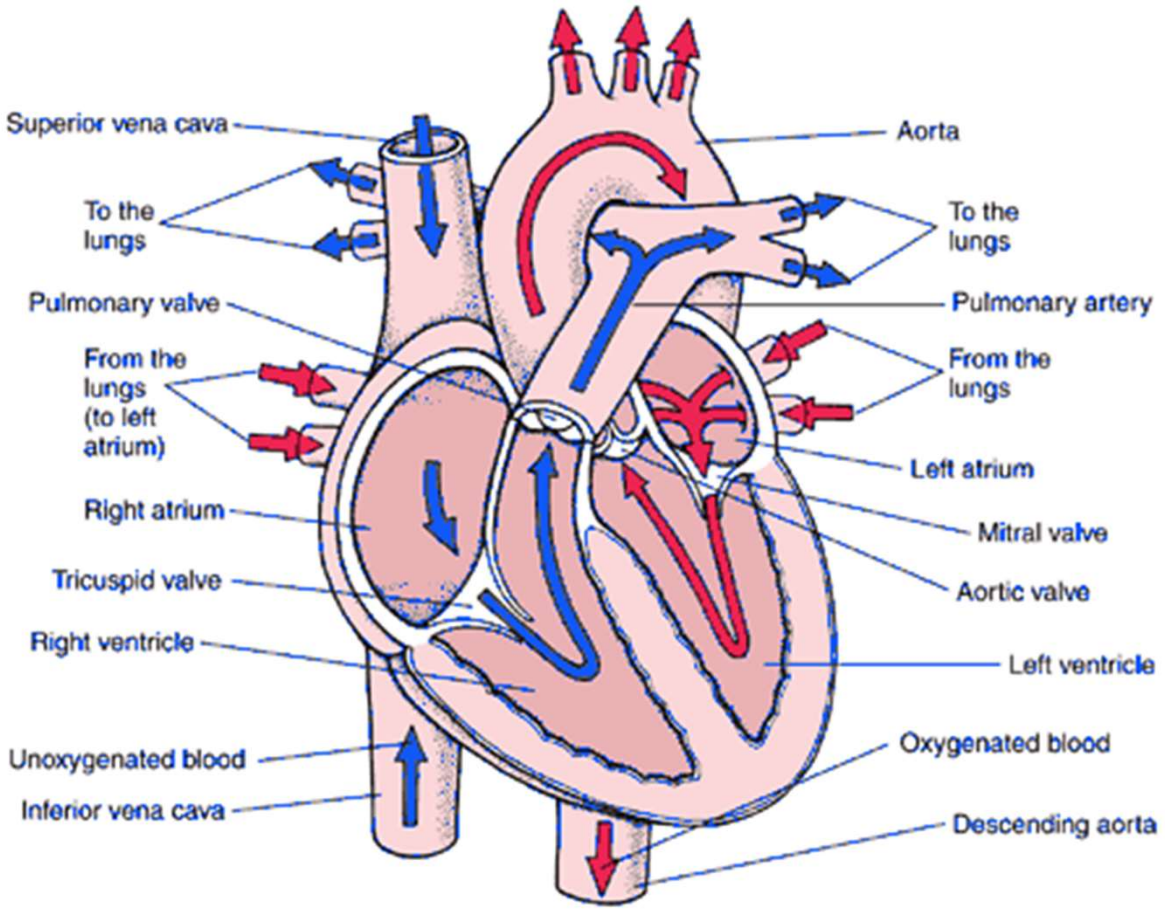
Foramen ovale – permits oxygenated blood from right atrium directly into left atrium

- Functionally closes soon after birth
- Seals between 6 months – 1 year
- Age appropriate PFO

Ductus venous – shunts most of the oxygenated blood from placenta

- Disappears within 2 weeks of birth

BLOOD FLOW


Superior vena cava
To the lungs
Pulmonary valve
From the lungs (to left atrium)
Right atrium
Tricuspid valve
Right ventricle
Unoxygenated blood
Inferior vena cava
Aorta
To the lungs
Pulmonary artery
From the lungs
Left atrium
Mitral valve
Aortic valve
Left ventricle
Oxygenated blood
Descending aorta

CARDIAC OUTPUT

Cardiac Output (CO) = Heart rate (HR) X Stroke volume (SV)

3 components of Stroke volume

- Preload: volume of blood in the ventricles before contraction
- Contractility: speed of ventricular contraction
- Afterload: the resistance to blood leaving the ventricles

CARDIAC OUTPUT

- Heart rate is most important in determining cardiac output in the neonate & fetus.
- The neonate has a decreased ability the increase SV due to the fetal myocardium having few contractile elements.
- The fetal myocardium is poorly innervated by the sympathetic nervous system.
- In the neonate, the parasympathetic nervous system predominates.

BLOOD PRESSURE

- In the first few hours of life, blood pressure can be affected by the type of delivery, placental transfusion, and birth asphyxia.
- Blood pressure in neonates is affected by temperature, activity, and behavioral state.
- Pulse pressure is the difference between the systolic and diastolic blood pressure.
- Wide pulse pressure can be a sign of large aortic runoff, as seen with PDA.
- Narrow pulse pressures are seen with neonates with peripheral vasoconstriction, heart failure, or low cardiac output.

HYPERTENSION

- Systolic BP > 90
- Systolic BP > 95th percentile for age and sex on 3 occasions
- Systolic BP > 90 and diastolic BP > 60 in full term infants
- Systolic BP > 80 and diastolic BP > 50 in preterm infants
- Rare

HYPERTENSION

- BP cuff should be 2/3 the length of the extremity
- Small cuff yields falsely ⬆ BP
- BP ⬆ when infant is feeding, sucking, or in an upright position.
- BP can be transiently elevated when infant is in pain, crying, agitated, or being suctioned

CAUSES OF HYPERTENSION

- Unknown etiology
- Renal artery thrombosis commonly related to UAC (very common cause)
- Aortic thrombosis
- Obstructive uropathy
- Infantile polycystic kidneys
- Renal failure
- Meds (corticosteroids, pancuronium, doxapram)
- Fluid overload
- Renal artery stenosis (accounts for 20%, infant will be hypertensive at birth)
- Pain/agitation
- BPD (up to 40% infants with BPD)
- Drug withdrawal
- Coarctation of the aorta (pressure lower in legs than arms could indicate coarctation)

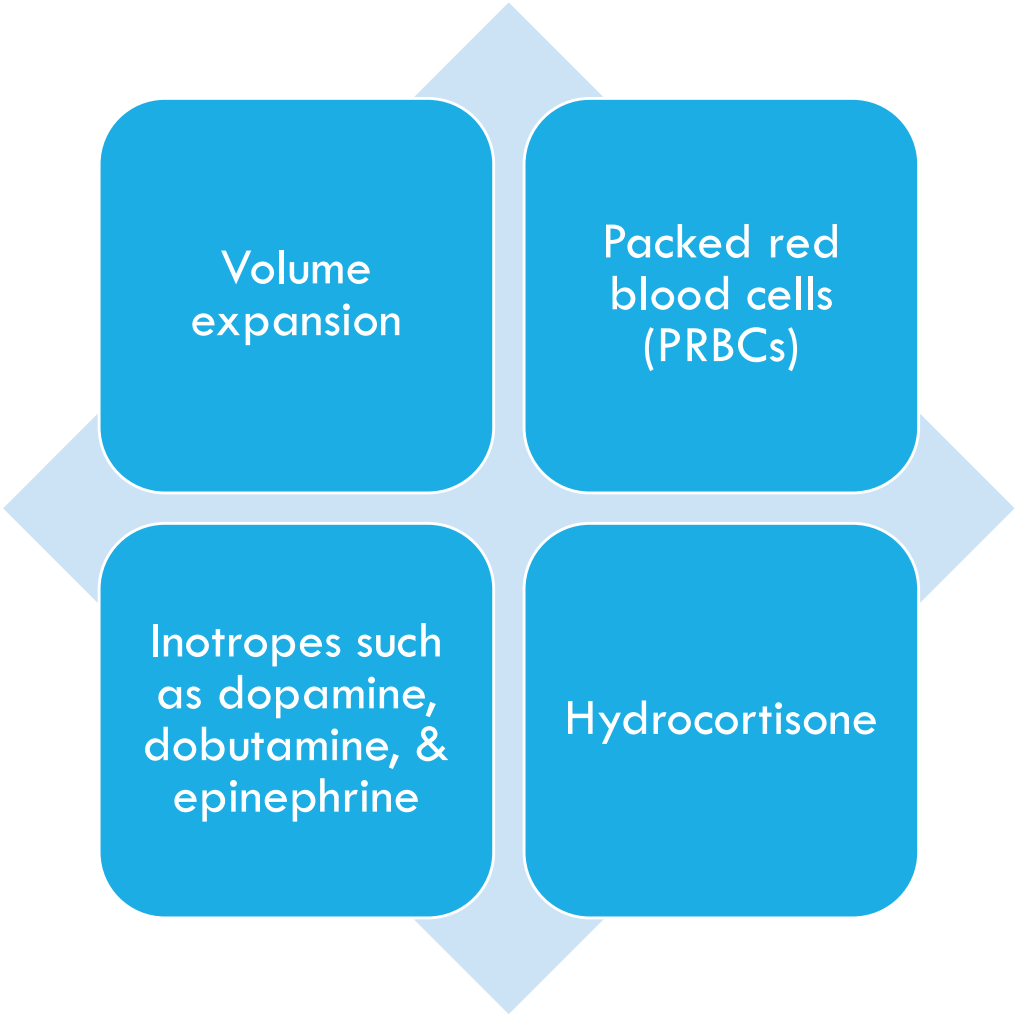
TREATMENT OF HYPERTENSION

Treatment depends on severity

- Asymptomatic patients with mild hypertension can be monitored initially
- Mild hypertension can be treated with diuretics and Angiotensin-converting enzymes (ACE) inhibitors if needed
- Moderate to severe hypertension is treated with diuretics and ACE inhibitors (Captopril)
- Life-threatening hypertension is treated with IV drugs (labetalol or hydralazine)

HYPOTENSION

- Infants < 30 weeks: BP should be at least the same as gestational age or greater
- If BP cuff is too large, readings will be falsely ↓
- Monitor for symptoms of shock
- ↓ urine output could be a sign of decreased renal perfusion
- Causes could include birth asphyxia, maternal bleeding, or extreme prematurity



TREATMENT OF HYPOTENSION

TREATMENT OF HYPOTENSION

Dopamine

- Improve CO, blood pressure and urine output
- 2 – 20 mcg/kg/min via continuous IV infusion
- Can cause tachycardia and arrhythmias
- Observe IV site for blanching and infiltration

TREATMENT OF HYPOTENSION

Dobutamine

- Treatment of hypoperfusion and hypotension, especially related to myocardial dysfunction
- Increases myocardial oxygen consumption
- 2 – 25 mcg/kg/minute
- Adverse effects:
  - Hypotension can occur in the presence of hypovolemia
  - Tachycardia at high dose
  - Arrhythmias
- Observe IV site—tissue ischemia can occur with infiltration

TREATMENT OF HYPOTENSION

Epinephrine

- Acute cardiovascular collapse, short-term use for systemic hypotension
- 0.1 mcg/kg/min
- More likely to cause hyperglycemia, tachycardia, elevations in serum lactate, and cardiac arrhythmias (PVC, V-tach)
- Observe IV site—tissue ischemia and necrosis can occur with infiltration

HYDROCORTISONE

Maybe helpful in extreme preterm infants with hypotension

Stabilizes BP

Response usually happens in 2 hours after administration

3 mg/kg/day for 3-5 days

Do not use in infants receiving Indocin

TREATMENT OF HYPOTENSION

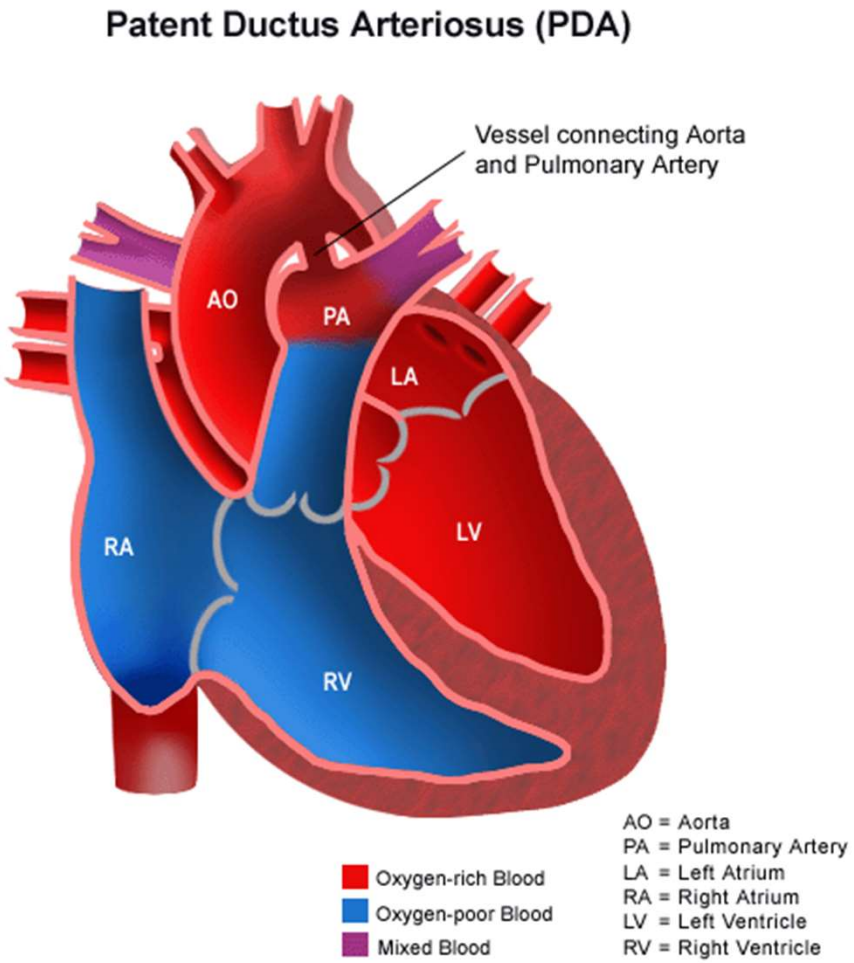
Hydrocortisone

- Hypotensive babies who are cortisol deficient are likely to respond (BP will increase approximately 2 hours after initial dose)
- 20 - 30 mg/m2 per day IV (given in 2 – 3 doses)

or approximately 1 mg/kg per dose ever 8 hours

- Monitor for hyperglycemia, hypertension, sodium retention, increased risk of GI perforations when given indomethacin, increased risk of disseminated Candida infections

PDA


Patent Ductus Arteriosus (PDA)
Vessel connecting Aorta and Pulmonary Artery
AO
PA
LA
LV
RA
RV
Oxygen-rich Blood
Oxygen-poor Blood
Mixed Blood
AO = Aorta
PA = Pulmonary Artery
LA = Left Atrium
RA = Right Atrium
LV = Left Ventricle
RV = Right Ventricle

PDA

- Incidence is inversely related to gestational age
- In utero patency of the ductus arteriosus is functional, diverting blood to the placenta for oxygen gas exchange
- Improved oxygenation decreases PVR
- Premature infants have immature response to O2
- Lack of ductal smooth muscle in preterm infants prolongs patency

SYMPTOMS OF PDA

Inability to wean from vent or increased support

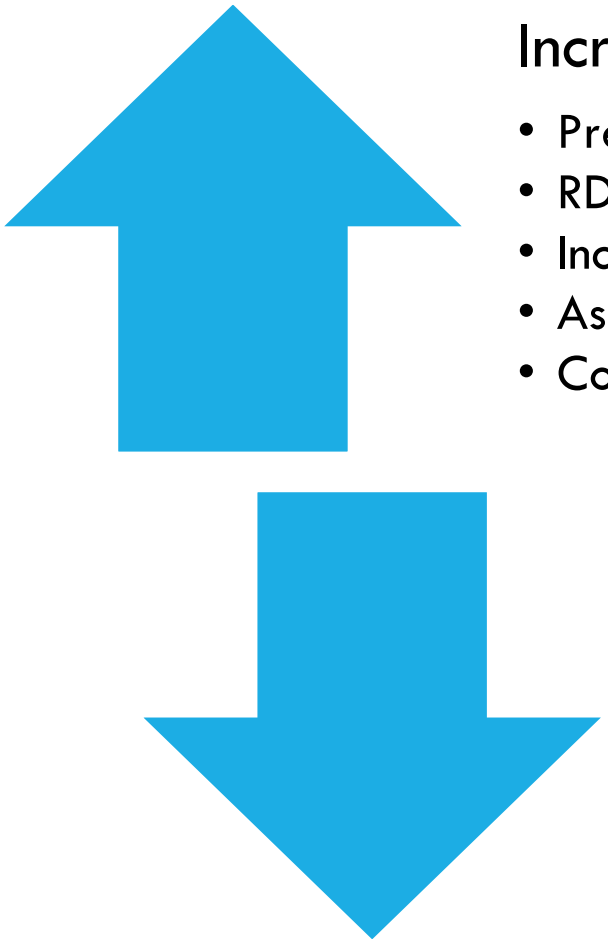
Bounding peripheral pulses and hyperactive precordium

Widening pulse pressure (> 20mm Hg)

Continuous murmur heard in upper left sternal border (may not be present even if significant shunt)

X-ray with normal heart size or cardiomegaly, pulmonary edema, and increased pulmonary vascularity

RISKS FACTORS OF PDA



Increased risk:

- Prematurity
- RDS and surfactant treatment
- Increased fluid load
- Asphyxia
- Congenital Heart Disease

Decreased risk:

- Antenatal steroid administration
- Intrauterine growth restriction (IUGR)
- Prolonged rupture of membranes (PROM)

ASSOCIATED RISKS

IUGR infants with PDAs are at greater risk for:

- Pulmonary hemorrhage
- IVH
- NEC
- Renal failure

TREATMENT OF PDA

- Ventilatory support
- Fluid restriction
- Increasing Hct
- Ibuprofen
- Indocin
- Acetaminophen
- Surgery

TREATMENT OF PDA

Indocin

- 0.2 mg/kg 1st dose, then 0.1 mg/kg 2nd and 3rd dose (repeat q 8 hours)
- Total of 3 doses
- Less effective if administered after 7 days of life (most likely no effect after14 days)
- Monitor urine output
- Contraindications: renal impairment, active bleeding in the CNS or GI tract, and NEC

TREATMENT OF PDA

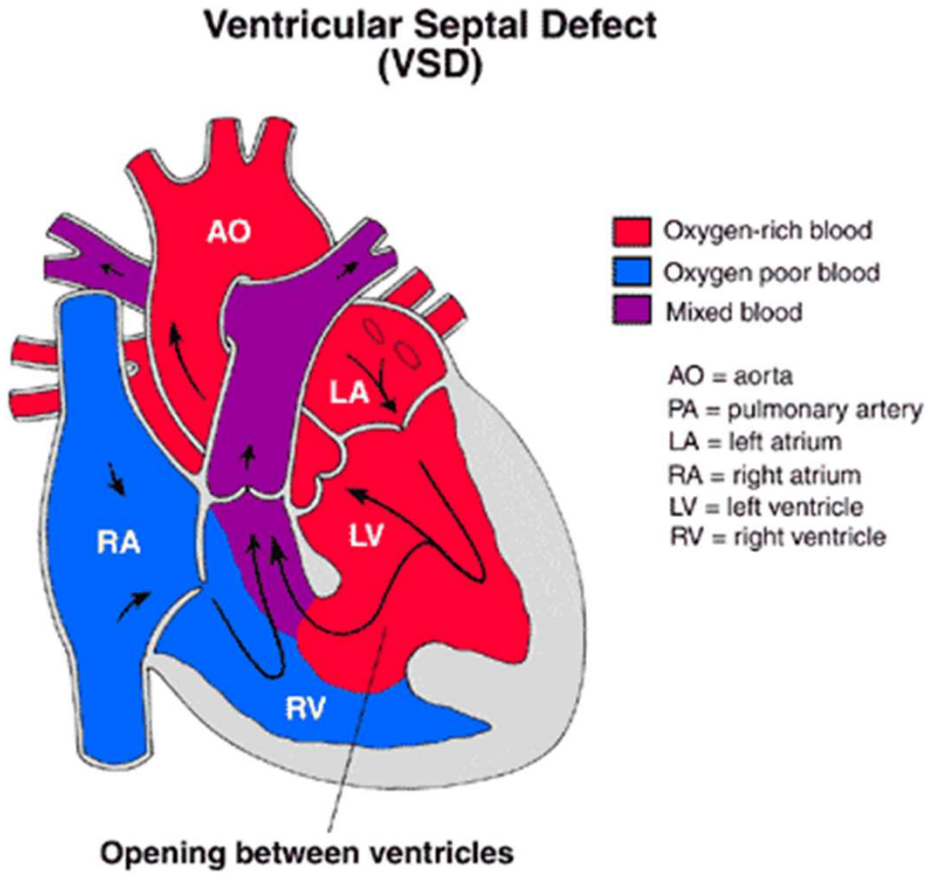
Ibuprofen

- 20 mg/kg X 1 dose (q 24 hours) oral unless NPO
- 10 mg/kg X 2 doses (q 24 hours) oral unless NPO
- Most effective in first 3 days of life
- Monitor urine output

Tylenol

- 15 mg/kg q6h X 7 days
- Used for refractory cases in which Ibuprofen did not work, or is contraindicated
- Must monitor LFTs

VSD


Ventricular Septal Defect
(VSD)
AO
LA
RA
LV
RV
Opening between ventricles
Oxygen-rich blood
Oxygen poor blood
Mixed blood
AO = aorta
PA = pulmonary artery
LA = left atrium
RA = right atrium
LV = left ventricle
RV = right ventricle

VSD

Most common CHD

Opening in the septum between the left and right ventricles

Size ranges from pinhole to near absence of ventricular septum

PVR < SVR = L to R shunt, leading to increased pulmonary blood, resulting in decreased pulmonary compliance and pulmonary edema

VSD MANAGMENT

Small defect:

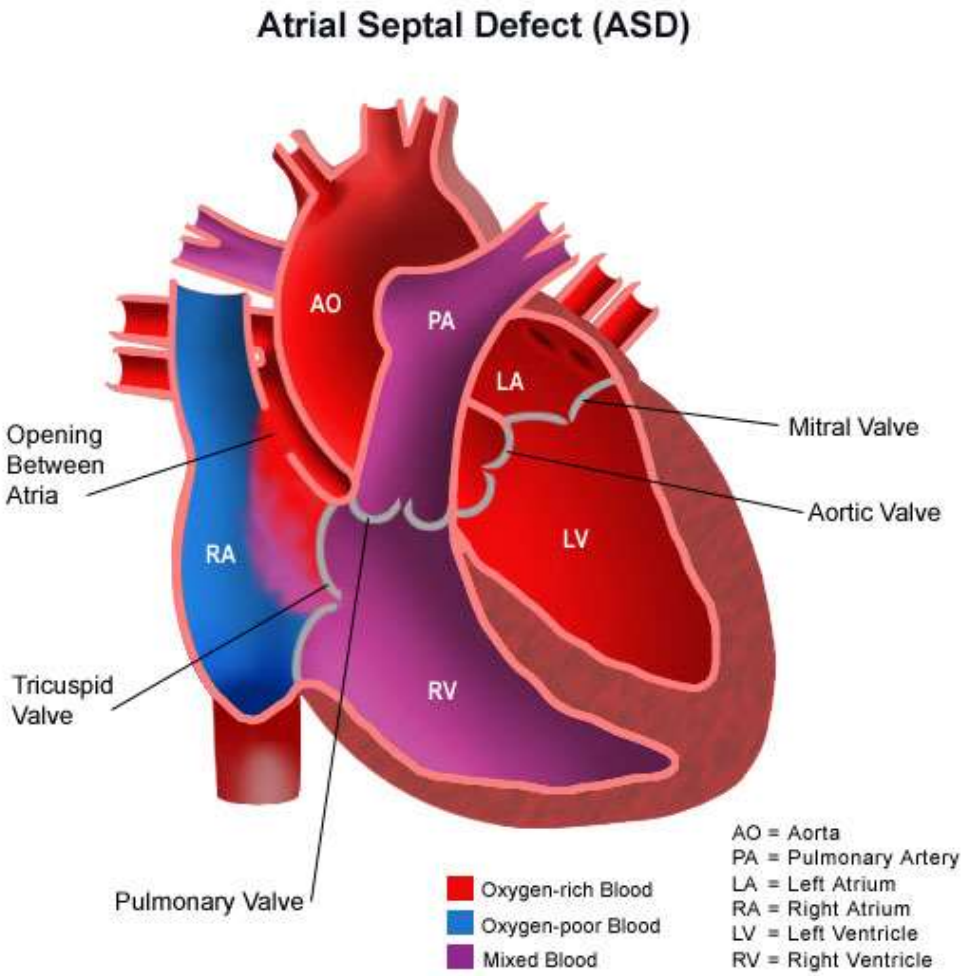
50-75% close spontaneously

Large defect:

20% become smaller or close

Surgery indicated when infant develops CHF or failure to thrive

ASD


Atrial Septal Defect (ASD)
AO
PA
LA
RA
LV
RV
Opening Between Atria
Tricuspid Valve
Pulmonary Valve
Mitral Valve
Aortic Valve
Oxygen-rich Blood
Oxygen-poor Blood
Mixed Blood
AO = Aorta
PA = Pulmonary Artery
LA = Left Atrium
RA = Right Atrium
LV = Left Ventricle
RV = Right Ventricle

ASD

Opening between left and right atria

Ostium primum defect, ostium secundum defect (most common), or partial endocardial cushion defect

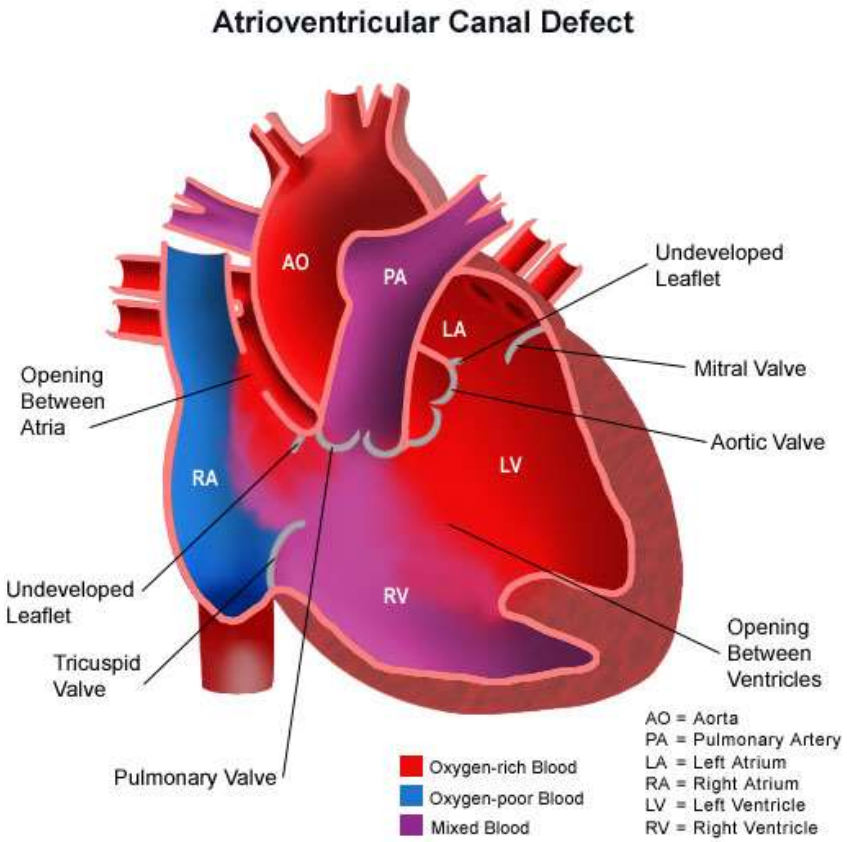
R ventricle pressure > L ventricle pressure after birth, therefore, no shunt or small L to R shunt

PVR decreases, L to R shunt develops with accompanying R ventricular overload and hypertrophy

ASD MANAGEMENT

- Small ASD followed clinically, may spontaneously close
- Treat CHF to delay surgical repair
- If significant CHF, early surgical repair
- Spontaneous closure occurs in up to 40% during first 5 years of life

ENDOCARDIAL CUSHION DEFECT (ATRIOVENTRICULAR CANAL)



ENDOCARDIAL CUSHION DEFECT (AV CANAL)

Most common heart defect found in infants with Trisomy 21

Endocardial cushions form the lower portion of the atrial septum, the upper portion of the ventricular septum, and septal portions of the mitral and tricuspid valves

Wide range of defects, from simple cleft of mitral and/or tricuspid valves to complete absence of the lower atrial and upper ventricular septa with common atrioventricular valve (AV canal)

ENDOCARDIAL CUSHION DEFECT (AV CANAL)

Since PVR < SVR, blood shunts
L to R via ASD and VSD

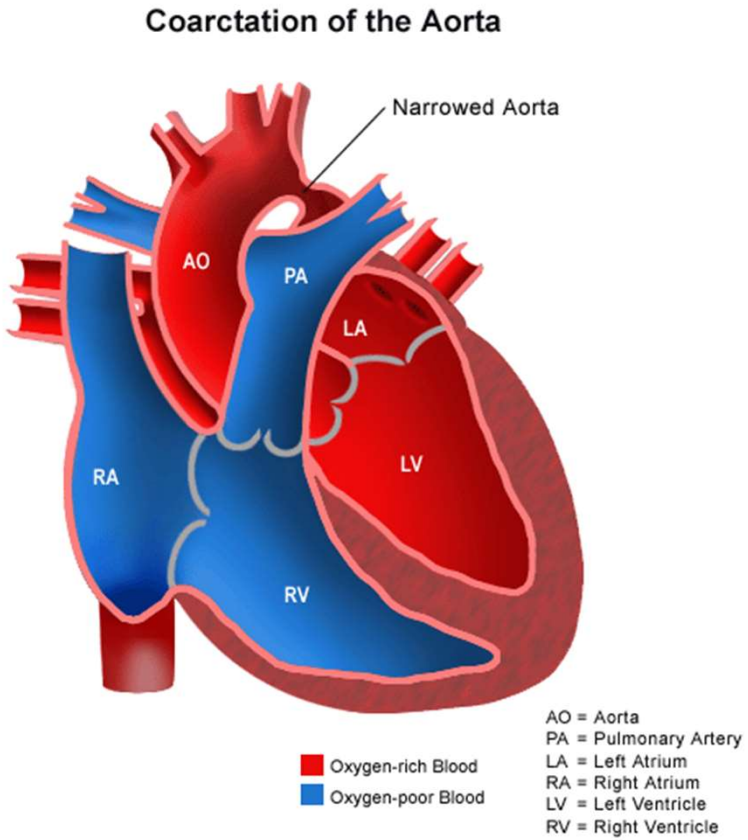
High pressure in VSD creates
L to R shunting via AV valve
(AV valve regurg)

Blood flows from L ventricle → mitral portion of AV valve → L atrium to ASD → R atrium

ENDOCARDIAL CUSHION DEFECT MANAGEMENT

- Monitor CHF
- Diuretics and digoxin
- Palliative PA banding to decrease pulmonary overload
- Primary repair closure of atrial and ventricular septal defects and mitral and tricuspid valve repair—usually at 6 months to 2 years of age

COARCTATION OF AORTA



COARCTATION OF AORTA

Constriction of aorta at the junction or the transverse aortic arch or the vicinity of ductus arteriosus

Turner's syndrome (30%)

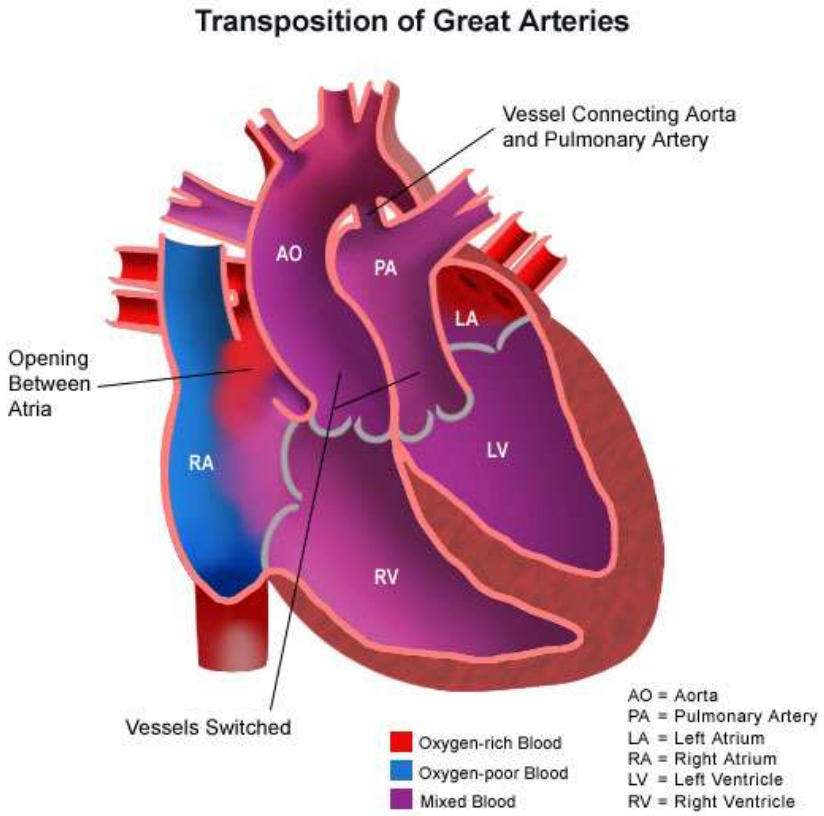
Male dominance 2:1

Higher BP in upper extremities (> 15mm Hg)

Decreased or absent pulses in lower extremities

Most common heart defect presenting in 2nd week of life

TRANSPOSITION OF THE GREAT VESSELS



TRANSPOSITION OF THE GREAT VESSELS

Position of great arteries are reversed

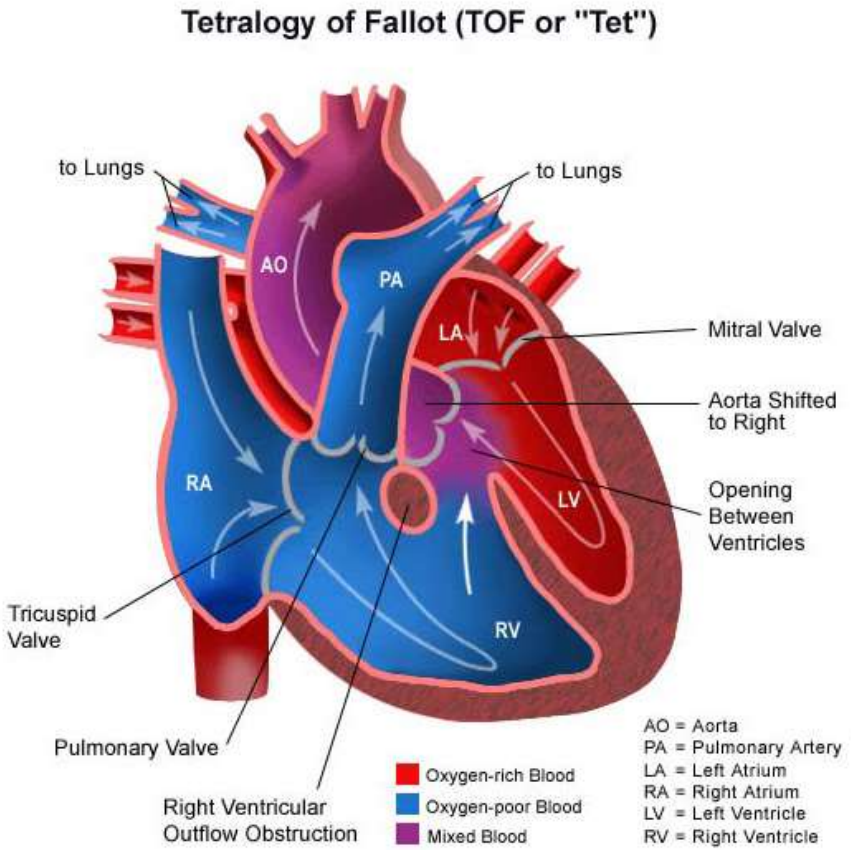
Without other intracardiac defects (ie: VSD, ASD), an independent parallel circuit exists

Mixing is needed for survival

Most common cardiac cause of cyanosis in the neonate

OTHER CHD

- Tetralogy of Fallot
- Pulmonary atresia
- Truncus arteriosis
- Tricuspid atresia
- Ebstein's anomaly
- Single ventricle
- Total anomalous pulmonary venous return
- Double outlet right ventricle
- HLHS



ALPROSTADIL (PROSTAGLANDIN E1)

- Used to promote dilation of ductus arteriosus in infants with CHD dependent on ductal shunting for oxygenation/perfusion
- Assess infant for improvement in oxygenation
- Can cause apnea
- Monitor temperature
- Need reliable IV access (duration effect is short)

MURMURS

- Caused by turbulent blood flow
- Systolic murmurs occur after S1
- Diastolic murmurs occur after S2
- Continuous murmurs are heard throughout systole and diastole

MURMURS

1. Grade I: Barely audible
2. Grade II: Soft, audible after careful auscultation
3. Grade III: Moderate intensity
4. Grade IV: Louder
5. Grade V: Very loud, may be heard with stethoscope barely on chest
6. Grade VI: Extremely loud, may be heard with stethoscope just slightly off chest

MURMURS

- Many infants have innocent murmurs in the first 48 hours of life due to decreasing PVR
- Many CHD murmurs do not present until 3 days, 1 week or 4-6 weeks due to changing PVR
- Absence of murmur does not rule out CHD
- Up to 20% of infants that die within the first month of life due to CHD do not have heart murmurs

SUPRAVENTRICULAR TACHYCARDIA (SVT)

Ventricular rate of 180-300 beats/min

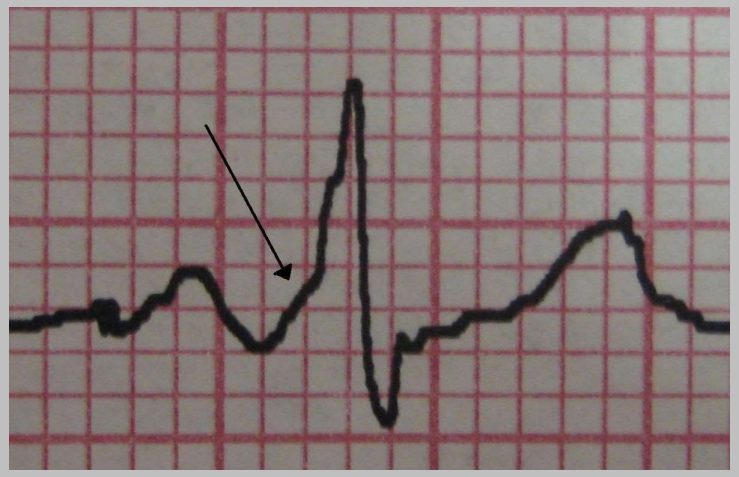
No change in HR with activity or crying

Abnormal P wave or PR interval

Most common type of cardiac arrhythmia seen in the neonate

Increased risk in infants with CHD (Ebstein's anomaly, L-TGA), medications (i.e. caffeine, epinephrine), cardiomyopathy, myocarditis, cardiac tumors, fever, and hyperthyroidism

SVT



Wolf-Parkinson-White (WPW)—a type of SVT

Prolonged QRS, shortened PR interval, and initial slurring of QRS

The abnormal electrical pathway directly connects the atria and ventricles and bypasses the AV node

Can be associated with Ebstein's or L-TGA, however, is most commonly associated with a structurally normal heart

MANAGEMENT OF SVT

- Assess if hemodynamically stable
- If unstable, synchronized cardioversion
- If stable, vagal maneuvers (ice to face), then IV adenosine if no change
- Repeat EKG afterwards to assess if underlying rhythm is normal
- Long term therapy—propranolol (first line for WPW) or digoxin

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