

Cardiac Evaluation of the Infant

CHET Education

Dec. 5, 2008

LDF

Congenital Heart Disease

- Most common of all congenital birth defect
- Affects 8 per 1000 live births
- Prompt recognition, stabilization and referral are crucial to outcome
- Due to the complexity of this patient population these kids are most challenging even to the seasoned practitioner

Pulmonary vs. Cardiac

- Pulmonary
 - No murmur
 - + O₂ challenge (inc Sat > 10% with 100%)
 - Changes in PCO₂ (inc or dec)
 - CXR – small heart, parenchymal changes, atelectasis, hyperinflation, etc
 - Quiet precordium
 - Increased respiratory distress, tachypnea, retractions, obvious inc in WOB

Cardiac vs. Pulmonary

- Cardiac
 - +/- murmur
 - - O₂ challenge (< 10% change in Sat in 100%)
 - Quiet tachypnea
 - Minimal changes in PCO₂
 - CXR - +/- large heart/ pulmonary edema
 - Palpation of active precordium
 - More cyanosis with agitation (shunt)

Cardiac Presentation

- Three typical presentations depending on lesion
 - Cyanosis
 - Cardiovascular collapse
 - Congestive heart failure/pulmonary overcirculation

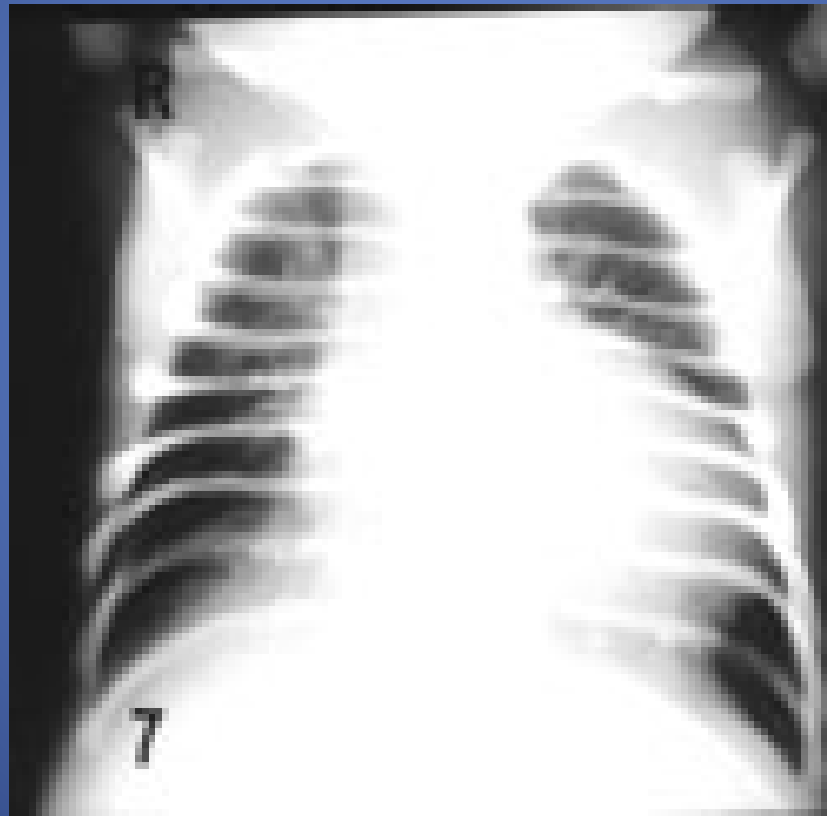
Cyanotic Lesions

- “5 Ts”
- Transposition
- Tetralogy of Fallot with pulm atresia/ stenosis
- Tricuspid atresia
- Truncus arteriosus
- Total anomalous pulmonary venous return (obstructed)

TGA

- Most common cyanotic lesion in newborn period
5% of all CHD
- 90% present in first day of life
- Ao arises from RV; PA from LV; parallel circ
- Must have mixing at Atr/Vent or ductal level
- CXR oval hrt due to anterior Ao and large RV
- PGE₁ life saving to maintain pulm blood flow
- Definitive repair – Switch and septal defect closure

TGA



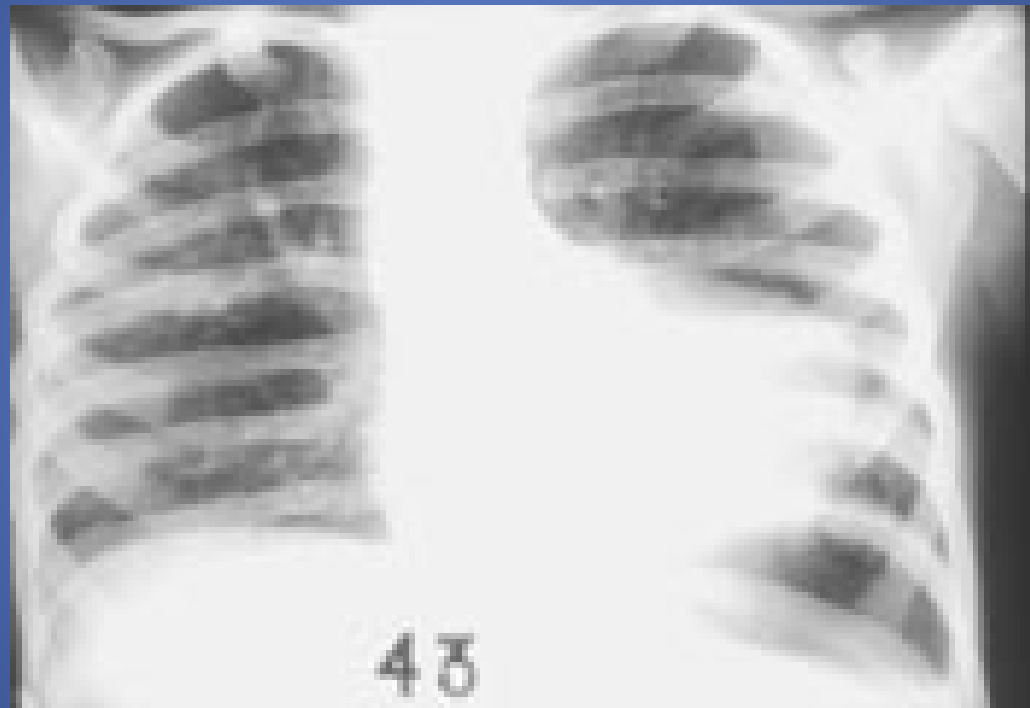
TET

- 6- 10% of congenital heart disease, most common
- Constellation of large VSD, RVOT obst, overriding Ao, RVH
- Degree of cyanosis dependent on RVOT obst
- The more severe the obstruction the earlier the presentation
- Mild obstruction presents later/ “pink” TET

TET

- CXR – boot shape 2nd to RVH
- Severe TET with pulm atresia requires PGE₁ to maintain pulm blood flow (acutely)
- Definitive repair now done as a single stage primary repair (vs. BT shunt then repair)

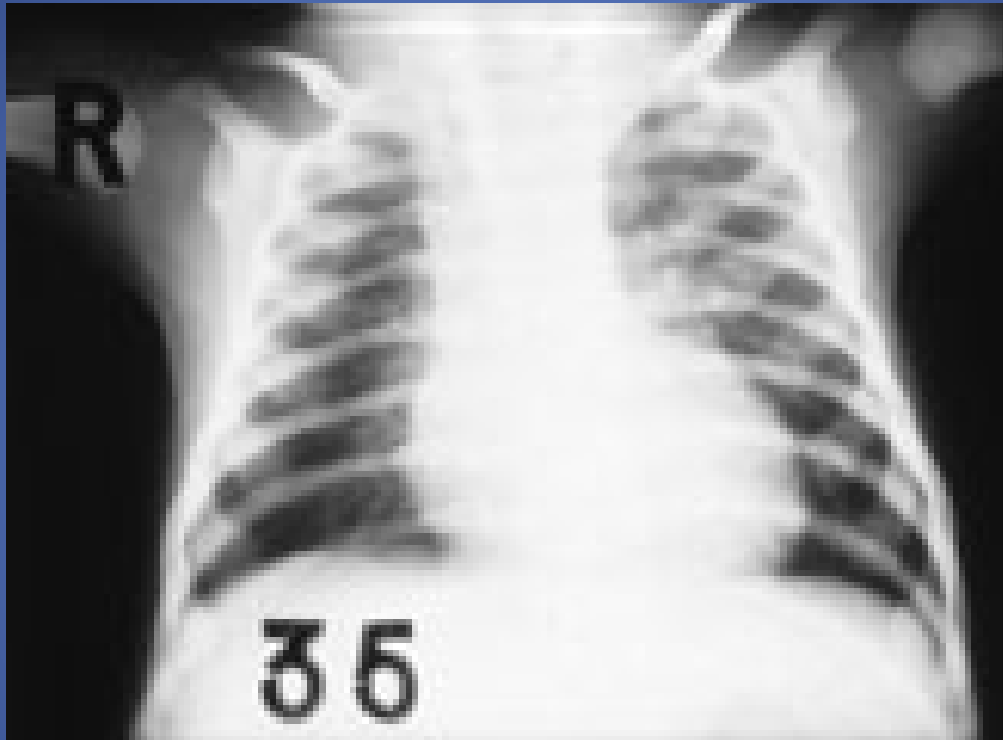
TET



Tricuspid Atresia

- 1% of all CHD, 3rd most common
- No direct connection of RA to RV
- Must have ASD and shunts $R > L$
- RV is underdeveloped
- Ductal dependent/ PGE₁ to maintain pulm blood flow
- Definitive repair BDG -> Fontan

TA



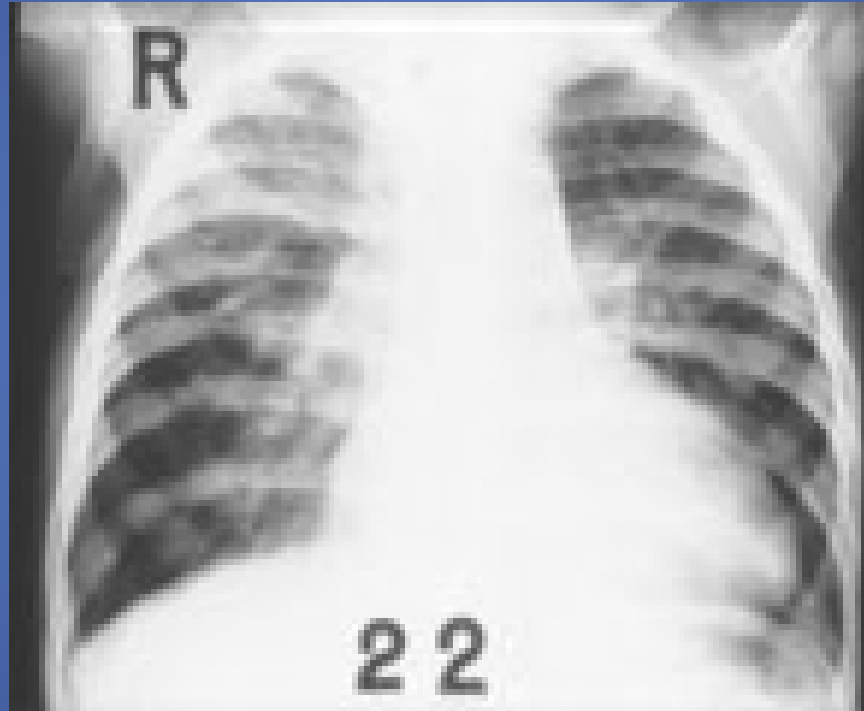
Truncus Arteriosus

- Less than 1% of all CHD
- Single great artery supplies systemic, pulmonary and coronary circulation
- Degree of cyanosis depends on amount of pulmonary blood flow
- **Classically grouped with cyanotic lesions, also pulm overcirculation with some degree of CHF
- Can present with tachypnea, cardiomegaly, + murmur, bounding pulses 2nd to pulmonary run off

Truncus

- OR cyanotic from R -> L shunt 2nd to respiratory disease/infection (parallel circulation)
- Early surgical intervention with RV to PA conduit

Truncus



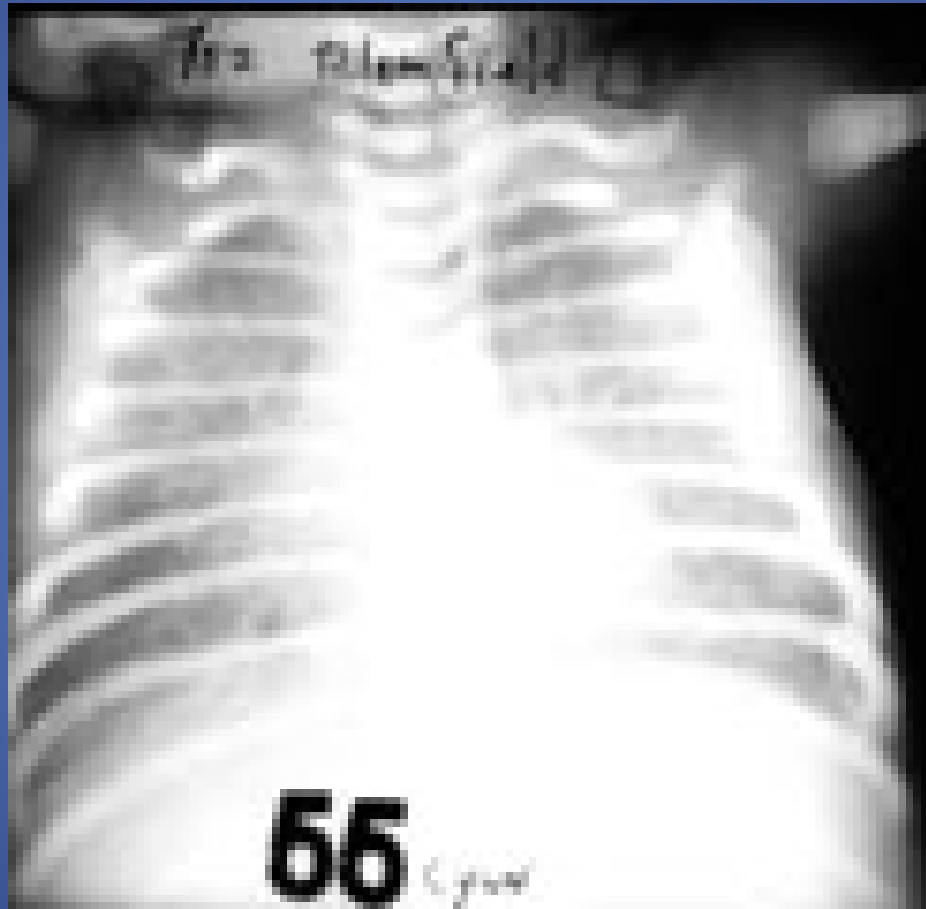
Total Anomalous Pulmonary Venous Return

- Pulmonary veins drain into systemic venous circulation
- Supracardiac, cardiac, infracardiac (SVC, RA, or portal circulation)
- Systemic circulation maintained by R → L shunt at atrial level (ASD)
- Obstruction most often with infradiaphragmatic connection

TAPVR

- Significant obstruction presents with cyanosis and pulm edema
- Partial or unobstructed will present later, generally within the first 2 months with hx of poor feeding, tachypnea, wheezing and mild cyanosis

TAPVR - infracardiac



Cardiovascular Collapse

- First 2 weeks of life
- Medical emergency
- Infants are discharged prior to the ductus closing
- Present in shock
- PGE₁ life saving

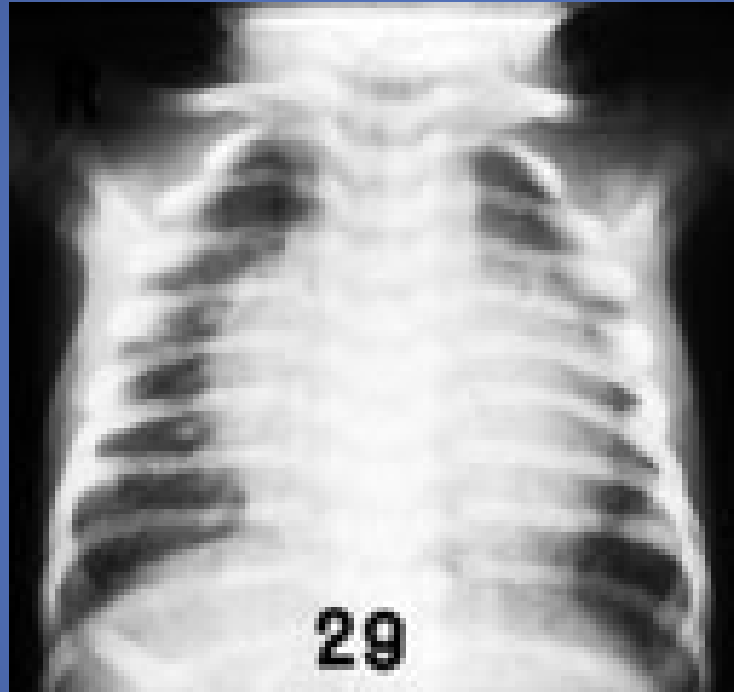
CV Collapse - Lesions

- Hypoplastic left heart syndrome
- Critical aortic stenosis
- Coarctation of the Ao and interrupted aortic arch

HLHS

- Most common cause of death during first week of life
- Diminutive LV, critical AS, mitral stenosis, hypoplastic proximal aorta
- Ductus closes, PVR reduced
- Shock ensues rapidly to decrease systemic flow
- PGE₁ maneuvers to inc PVR (parallel circ)
- Three definitive options: transplant, Norwood, nothing

HLHS



Critical AS

- Shock 2nd to compromised systemic flow
- Ductus closes
- PGE₁ again is life saving
- Airway support, inotropes, treat acidosis
- If LV too small may need Norwood
- If LV adequate, valvotomy or balloon valvuloplasty in cath lab

Coarc/IAA

- Coarc 5-8% of CHD, IAA < 1%
- Ductus closes, severe shock
- Discrepancy of upper and lower pulses and BP
- PGE₁ , airway support, treat acidosis, inotropes for severe early presentation
- Milder forms may present later but still with significant shock

Congestive Heart Failure

- Presents more gradually, beyond neonatal period
- Symptoms vague – poor feeding, wt. gain etc.
- + murmurs usually appreciated
- Two groups – pulmonary **overcirculation**, most common
- **Ventricular failure**, less common

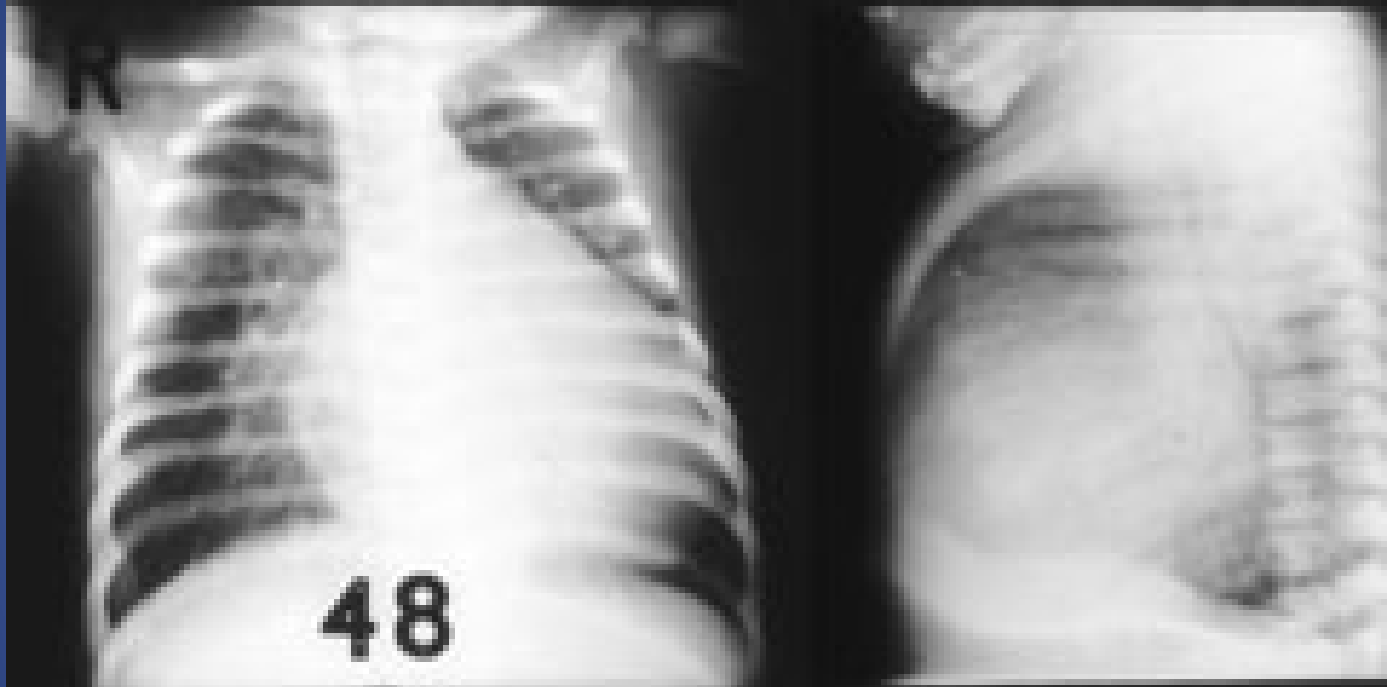
Pulm Overcirculation

- Mod to large VSD
- AVC or AV septal defect
- Large PDA

Overcirculation

- Signs of CHF
- PVR falls, pulmonary blood flow increases
- + murmur
- CXR – large heart, pulm edema
- Airway support, diuretics, afterload reduction
surgical repair

VSD



PDA



Ventricular Failure

- Anomalous left coronary artery
- Myocarditis
- Cardiomyopathies

Anomalous Left

- Coronary Artery originates from the pulmonary artery
- PVR falls
- Steal as blood actually flows retrograde from coronary circulation to pulmonary circ
- Severe shock, ischemic myocardium
- Increase PVR, volume to perfuse heart

Myocarditis/Cardiomyopathy

- Poor feeding, pallor, tachypnea, sweating
- Shock
- Hepatomegaly
- Cardiomegaly, increased pulmonary markings
- Milder inotrope, afterload reduction, diuresis
- Severe need intubation

Cardiomyopathy



END