MARROW

2024 NEET-SS

UPDATED PEDIATRICS NOTES

CARDIOLOGY

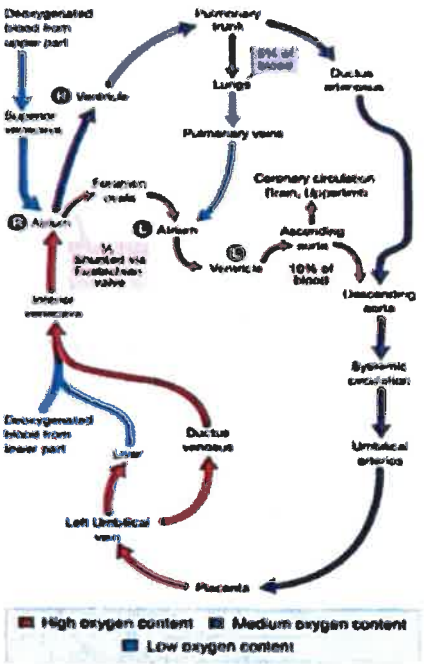
FETAL CIRCULATION AND NEONATAL TRANSITION

Introduction

00:00:55

Features :

- The heart and circulatory system is the first organ system to become fully functional which is necessary for embryogenesis and further fetal development.
- most of the work has been done in fetal lambs whose circulation closely resembles to that of human fetal circulation.
- The first detailed account of fetal circulation as a whole was described by William Harvey in 1632.



Fetal circulation

Fetal circulation vs Adult circulation :

- Parallel arrangement of two main arterial systems and their respective ventricles (Adult circulation is a series circuit).
- Placenta is the site of gas exchange.
- Presence of shunts.
- Preferential streaming of blood.
- Pulmonary circulation : High PVR and low flow.
- Systemic circulation : Low SVR and high flow.

Features of Fetal circulation

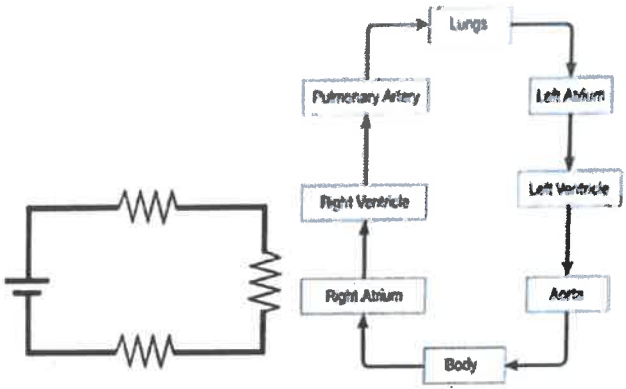
00:02:55

Circuit :

Series circuit :

Blood has to pass through each and every component.

Active space

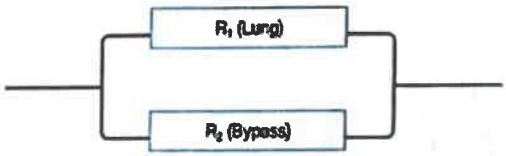


Series circuit. Adult circulation.

Parallel circuit :

Blood can pass through any one channel.

Fetal and neonatal circulations



$1/R_{eq} = 1/R_1 + 1/R_2$

Before birth R1 is high. Thus most of blood bypasses the lung.

After birth R1 decreases and blood is directed through the lungs.

Consequence of parallel nature of fetal circulation :

- Fetus tolerates the obstructive lesions reasonably well.
- Fetal circulation is jeopardized by regurgitant lesions and myocardial disease.

Causes of fetal heart failure :

1. Severe valvular regurgitation : AVCD, Ebstein.
2. Uncontrolled fetal arrhythmias : Tachycardias,CHB.
3. Fetal myocarditis, fetal cardiomyopathies.
4. Recipient twin in TTTS.
5. AVF with high output cardiac failure : Vein of Galen malformation.
6. Some congenital porto systemic shunts.
7. In utero restriction of foramen ovale and ductus.

Feedback

Shunts :

1. Placenta.
2. Ductus venosus.
3. Foramen ovale.
4. Ductus arteriosus.

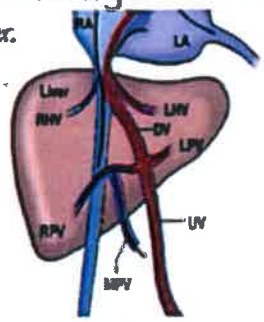
Parts of fetal circulation 00:08:31

It can be divided into :

1. Prior to RA (Placenta, UV, IVC).
2. Inside RA
3. After RA (ventricles and great arteries).
4. Return of blood to placenta.

Prior to RA (Placenta, UV, IVC) :

- Oxygenated blood (80% saturation) leaves the placenta through the umbilical vein and reaches up to the porta hepatis of the liver.
- 50 % of blood bypasses the liver through the Ductus venosus and enters the IVC.
- 50% of blood in the umbilical vein enters the left branch of the portal vein, perfuses the hepatic sinusoids and eventually enters the IVC through the left hepatic vein
- 2 streams in the IVC (Preferential streaming). They do not mix d/t different velocities.



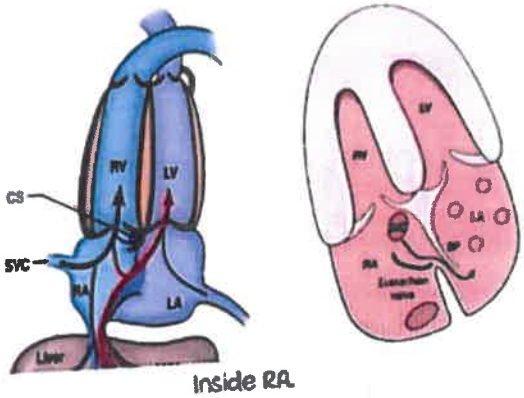
Prior to RA

Inside RA :

- The SVC blood streams along the lateral atrial wall into the RV, due to the superior and left ward course of the eustachian valve.
- The coronary sinus is located caudal to fossa ovalis and hence flows down to the RV across the TV .
- 4 main streams of inflow into right atrium :
  - i. From SVC carrying poorly saturated blood (40%) from the upper body and brain.
  - ii. Coronary sinus is highly desaturated which is 25% and two streams in the IVC are relatively well oxygenated .
  - iii. Blood from the umbilical vein via the ductus venosus and left hepatic vein ie 1/3 rd IVC blood.

iv. Poorly oxygenated blood from the rest of the lower body and right lobe of liver which is a/3rd IVC blood.

- Red stream is directed directly to LV via foramen ovale. (D/t eustachian valve and crista dividens).
- Blue stream is directed to RV.



Inside RA

After RA (ventricles and great arteries):

From RV:

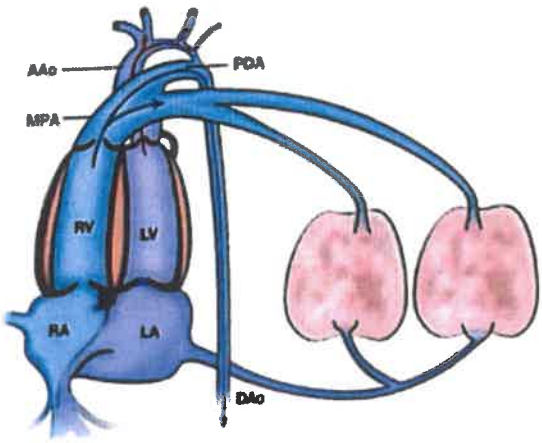
- To lungs: 15%.
- To PDA (Supplies lower limbs): 85%.

From LV:

- Aorta: Supplies head and neck.
- Lower limbs (Small proportion).

Note:

- RV output is 1.3 time LV CO: RV is thicker at birth.
- RV 55% of CVO.
- LV 45% of CVO.
- RV pressure = LV pressure.



Feedback

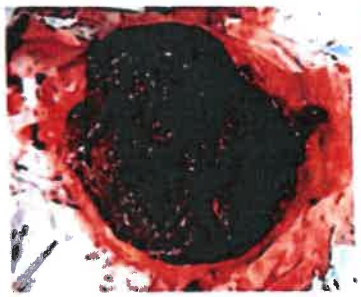
Note :

- most of the desaturated blood reaches the right ventricle and this blood is channeled via the ductus arteriosus and descending aorta to the placenta for oxygenation.
- most of the saturated blood reaches the left ventricle which is driven into the ascending aorta to the heart and brain.

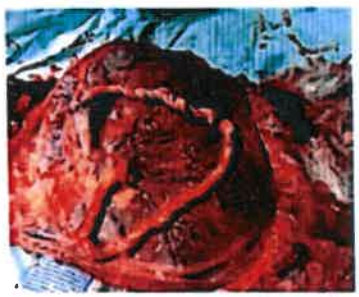
Placenta & umbilical vessels :

Placenta :

- Organ which receives max CVO.
- Site of least vascular resistance.
- Efficiency of oxygenation is less than lungs.



maternal side.

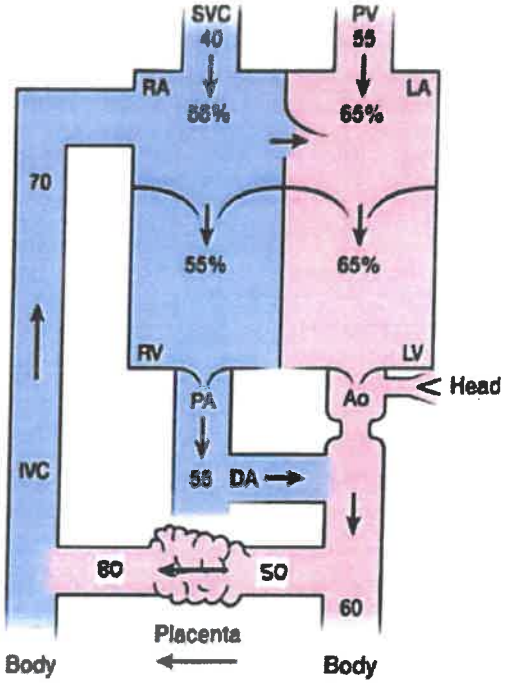


Fetal side.

Umbilical vessels :

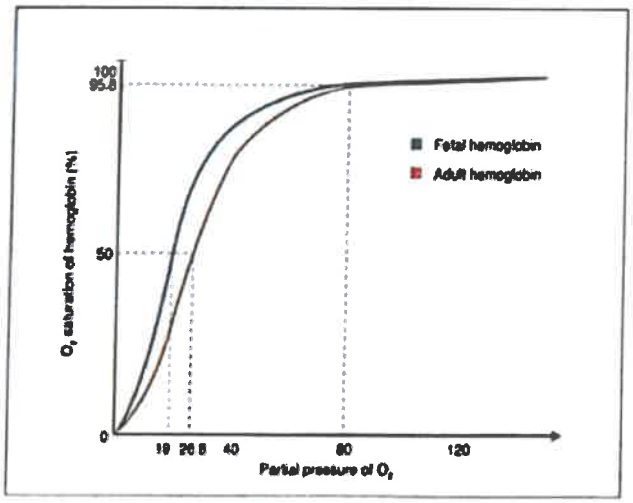
Single umbilical vein	2 umbilical arteries
• SpO2 : 80% • PO2 : 30-35 mm Hg	• Branch of Anterior division of maternal internal iliac artery.

Oxygen saturation at various levels :



Why fetus tolerates low oa levels :

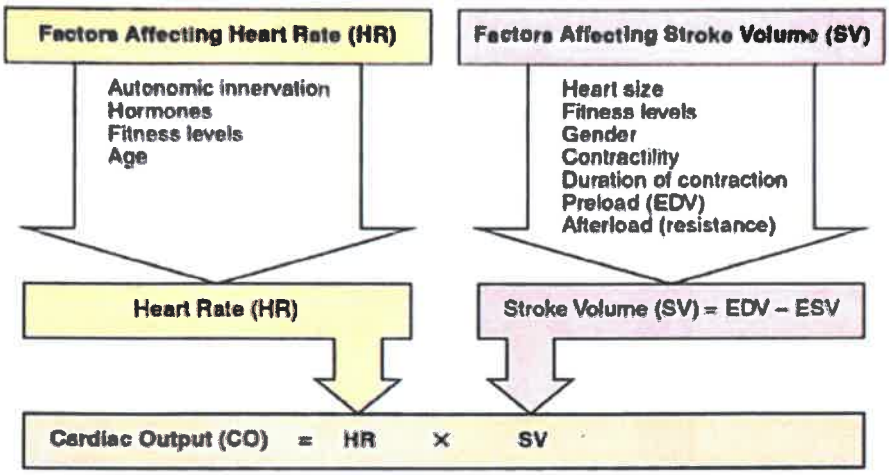
- High CVO : 400 to 450 mL/kg/min.
- High HbF levels.



Oxygen hemoglobin dissociation curve.

Feedback

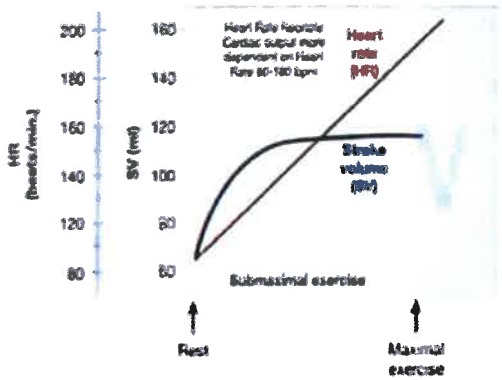
How fetus /neonate increases its CO :



Differences between infant myocardium and adult myocardium :

- Neonatal myocardium is characterized by higher water to collagen and connective tissue to contractile tissue ratios which is less compliant.
- The immature myocyte has fewer myofibrils, and these myofibrils are less organized.
- Neonatal myocardium has fewer mitochondria, which are smaller, and morphologically immature.
- Neonatal myocyte contractility is more dependent on plasma calcium due to an immature sarcoplasmic reticulum and poorly developed T-tubules.
- In the neonate, parasympathetic innervation is fully developed, whereas sympathetic innervation is incomplete. Responses to stimuli are often vagal in nature resulting in significant bradycardia.

HR, Stroke volume relation with exercise



Feedback

Note :

- Fetal and neonatal myocardium is immature .
- Increase in cardiac output is mainly affected by heart rate hence if there is fetal bradycardia fetus goes into shock.

Transition of fetal circulation 00:25:55

Events at birth :

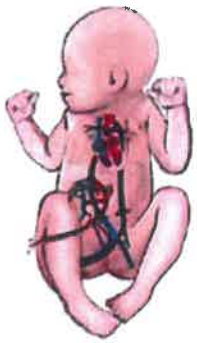
- The change from fetal to postnatal circulation happens very quickly.
- Changes are initiated by baby's first breath.

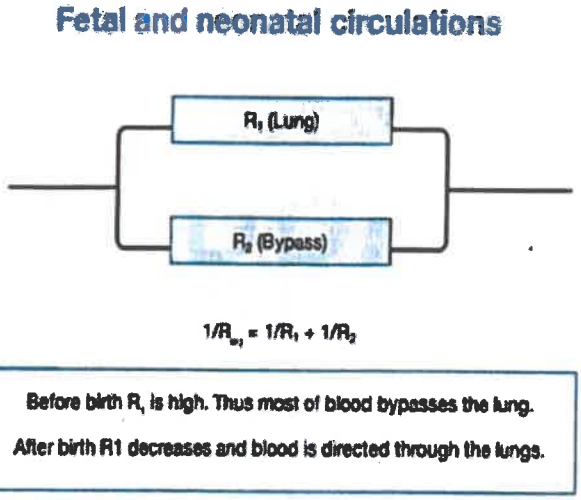
Changes at birth :

- SVR increases.
- Large fall in PVR.
- Closure of shunts (mainly ductus arteriosus).

Reason for rise in SVR :

- Placenta is removed from circulation .
- Large rise in SVR as the lowest resistance portion is eliminated .





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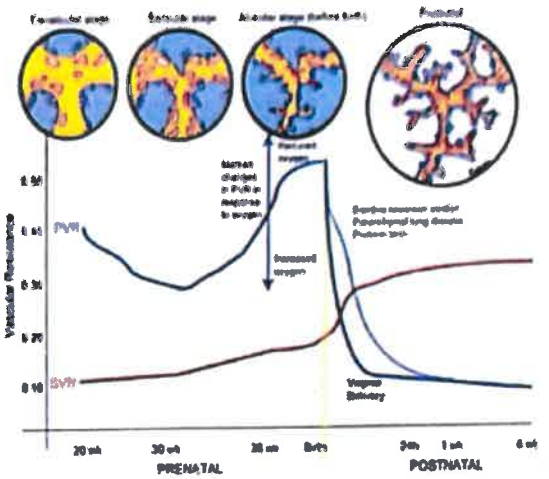
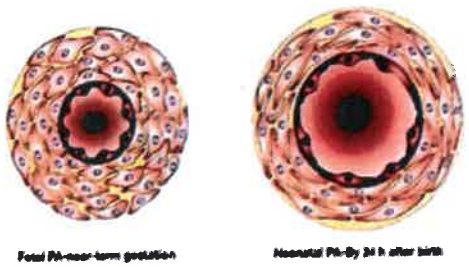


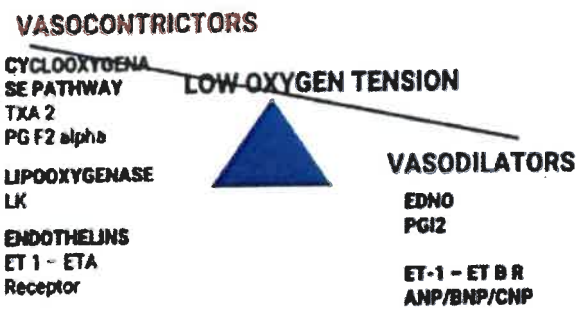
PVR and SVR.

Changes in pulmonary circulation :

Reason for high PVR in fetus :

- The human fetus has evolved to maintain a high PVR in utero to allow the majority of the fetal circulation to bypass the lungs, which do not participate in gas exchange, towards the low resistance placenta, hence high PVR seen in fetus.
- mechanical factors :
  - Fetal lungs are collapsed with minimum blood flow.
  - Thick walled muscular pulmonary arteries.
- Chemo reflexes :
  - more vasoconstrictors vs vasodilators due to relative hypoxia.

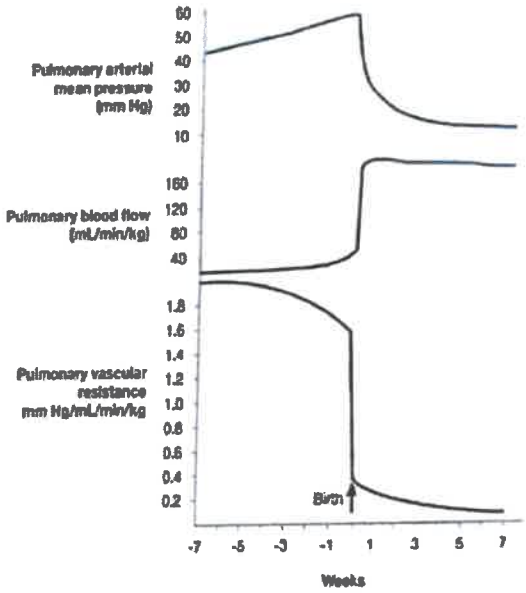




PVR in preterm VS term infants :

- PVR rises at term d/t increase in vasocontrictors.
- Hence, preterm babies have lower PVR.

Pulmonary parameters :



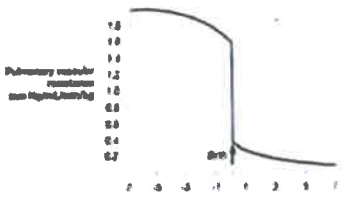
Phases of changes in PVR :

Slight fall of PVR just before birth :

- Removal of fetal lung fluid begins before birth by active Na transport.
- Triggered by hormones : Catecholamines, vasopressin, prolactin, glucocorticoids.

Phase I :

- Early rapid fall : 48-96 hours.
- Immediate recruitment of lung vasculature.
- Increase PO2 : From 25 mm to 50 mm Hg.



Phase I.

Factors causing delayed fall in PVR :

- Hypoxia :
  - High altitude.
  - Pre term infants with delayed lung maturity.
  - Lung diseases : Pneumonia /MAS.
- Caesarian section :

Feedback

- TTN
- Preterm birth

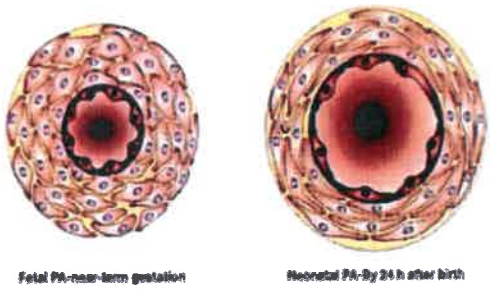
Note :

Pre term with hypoxia : No fall in PVR

Pre term with normal oxygenation : Fall in PVR seen

Phase a :

Late slow decline in PVR from 4 to 6 weeks due to loss of muscular coating



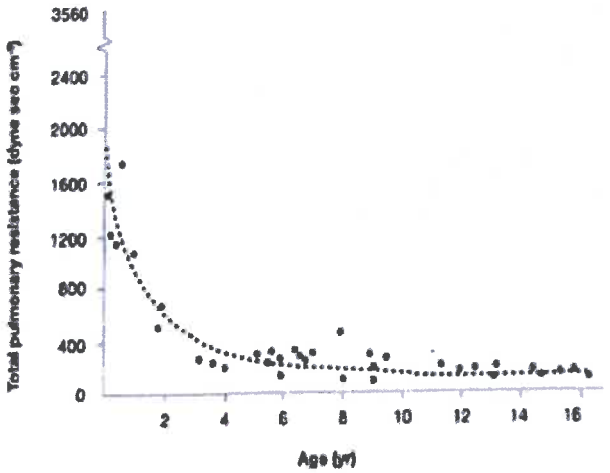
Fetal PA-near-term gestation Neonatal PA-By 24 h after birth

PA involution :

- In the first 4 to 6 weeks after birth, there is progressive involution of the circumferential medial smooth muscle with overall reduction in medial muscular thickness of the walls of the small pulmonary arteries.

Phase 3 :

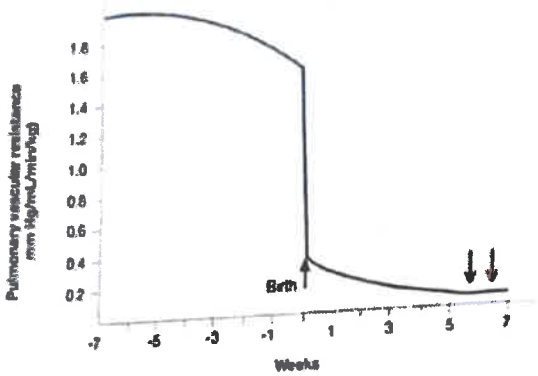
3rd decline occurs after 2 years due to increase in number of alveoli and pulmonary vessels.



Feedback

Clinical importance of fall in PVR :

- Persistent pulmonary hypertension.
- Large post tricuspid shunts present after 6 weeks with HF when when PA media becomes thinner.



Large post tricuspid shunts presentation

Cases where post tricuspid shunts present earlier than 6 weeks :

1. VSD :
   - Pre term VSD (Normal Oxygenation).
   - CoA, Aortic interruption.
2. PDA
   - Pre term PDA : (Normal Oxygenation).
3. AP window.
4. AVCD : Associated severe MR.

Note :

- High O2 causes vasodilation of PA.
- PG causes vasodilation of PA.

Closure of shunt vessels

00:40:45

Ductus venosus closure :

Closes immediately after birth : From first few hours to within 7 days due to cessation of umbilical flow.

Foramen ovale closure :

It is due to rise in LA pressure and fal in RA pressure.

25% adult population are estimated to have probe patent foramen ovale.

Feedback

Ductus arteriosus closure :

CCHD and Ductus closure :

- Functional complete closure usually occurs within 10 to 15 hours of birth in term neonates : Due to vasoconstriction.
- Anatomical closure in 2-3 weeks after birth causes SMC proliferation leading to ligamentum arteriosum formation.
- Functionally closed ductus arteriosus may reopen with reduced Po2.

Factors keeping ductus arteriosus open :

- Low PO2.
- High PGE2.
- High altitude.

Factors causing premature closure :

- Maternal Aspirin intake can cause ductus constriction in utero and can lead to PPHN.

Interventions :

To keep ductus open	To close the ductus
• Alprostadil : PGE1.	• O2. • NSAIDS. • Increase Ach. • Increase bradykinin.

Note :

- O2 and acidosis constricts the ductus and dilates the pulmonary artery.
- PG dilate both PA and ductus.

Problems faced by premature newborns :

- Higher incidence of PDA :
  Ductal smooth muscle less responsive to o2.
  Immature lungs : Decreased breakdown of PGE2.
- PVR :
  Pre term with hypoxia (Eg HMD).
  Pre term with normal O2 .

Fate of different structures :

Fetal structure	Develops into
• Foramen ovale	Fossa ovalis
• Ductus arteriosus	Ligamentum arteriosum
• Extra-hepatic portion of the fetal left umbilical vein	Ligamentum teres hepatis (Round ligament of the liver)
• Intra-hepatic portion of the fetal left umbilical vein (ductus venosus)	Ligamentum venosum
• Proximal portions of the fetal left and right umbilical arteries	Umbilical branches of the internal iliac arteries
• Distal portions of the fetal left and right umbilical arteries	Umbilical ligaments

NEONATAL CARDIOLOGY

Introduction

00:00:21

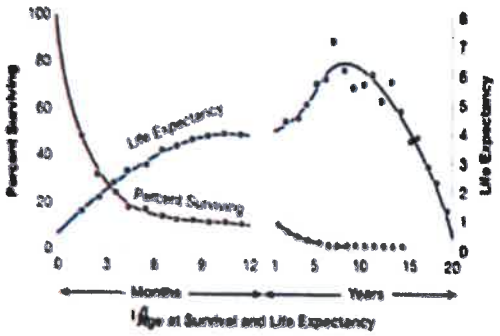
Critical CHD :

Definition :

Structural malformations of the heart that are present at birth and require surgery or intervention in the first year of life to survive.

- 1 in 4 babies born with a CHD have CCHD.
- most CCHD require intervention in neonatal period itself.

Life expectancy graph



80% Mortality at 3 months
>95% by age 5 years
100% by age 22 years

Case 1 :

- A 2 day old neonate presents to the ED unwell with poor pulses and cold peripheries.
- Parents give history of early discharge 24 hrs after normal vaginal delivery.
- O/E : Baby is in shock with no palpable femorals with well felt brachials.
- LL SpO2 and BP are un recordable.
- Soft systolic murmur over left axilla.

Typical history in CCHD :

- Usually benign birth history with nearly normal Apgars.
- Stable during first few hours of life, becomes symptomatic when transition of circulation occurs.
- Closure of the ductus.
- Fall in PVR.

Feedback

Faces of CCHD presentation 00:03:00

3 faces of CCHD presentation :

1. Neonate with cyanosis : Blue baby syndrome.
2. Neonate with systemic hypoperfusion : Grey baby syndrome.
3. Neonate with respiratory distress without cyanosis or systemic hypoperfusion : Pink baby syndrome.

The neonate with cyanosis (Blue baby) :

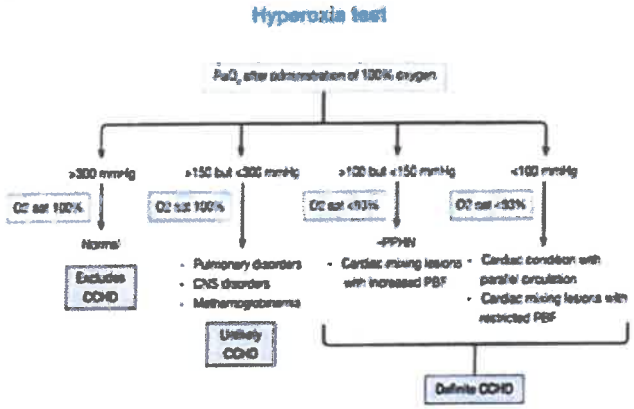
Identify respiratory cause or cardiac cause cyanosis.

Respiratory cause	Cardiac cause
Marked respiratory distress	No/mild respiratory distress
Mild cyanosis	Profound cyanosis
Increase in SpO2 after oxygen Increased PCO2 in ABG	No increase in SpO2 after oxygen Normal PCO2 in ABG
Normal blood pressure in all limbs No murmur, no rate abnormalities	Abnormal cardiac findings : Pansystolic murmur, tachycardia/bradycardia, abnormal heart sounds

Note :

Cyanosis in the absence of significant respiratory distress is almost always caused by a structural HD : Peaceful cyanosis.

Hyperoxia test :



- Insufficient validation of hyperoxia test.
- Mainly developed in an era when ECHO was not available.
- Advised not to do this test.

Feedback

Causes of Cyanotic CHD in newborns :

Decreased PBF :

- DDPC.
- TOF (MC cause).

Variable PBF :

- Admixture Physiology.

Parallel circulation with poor mixing :

- D-TGA.

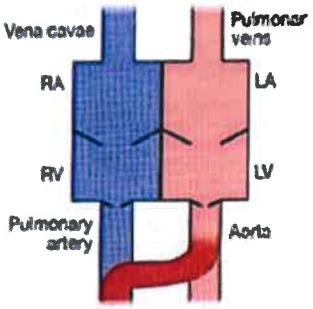
Obstruction to Pulmonary Venous return :

- Obstructed TAPVC, mitral atresia, HLHS.

Duct dependent pulmonary circulation (DDPC) :

- Pulmonary atresia with intact IVS.
- Critical PS.
- Tricuspid atresia with PS (HRHS).
- Severe TOF.
- Severe Ebstein.

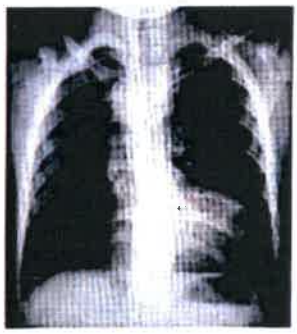
Closure of PDA results in severe cyanosis.



PDA

Tetralogy of Fallot :

- Right side obstructive lesion with large non obstructive VSD.
- Decreased PBF d/t pulmonary stenosis.
- CXR shows boot shaped heart.


Boot shaped heart in TOF.

Admixture physiology :

Definition :

- Congenital Cyanotic heart disease (CCHD) with a cardiac defect which facilitates complete mixing of arterial and venous blood in a common receiving chamber.
- Hallmark : Aortic and pulmonary saturations are equal.

PDA to maintain mixing of blood :

TGA with intact IAS & IVS.

On PDA closure, presents as :

- Cyanosis (Decreased PBF).
- Shock (Decreased systemic flow).

Obstruction to Pulmonary venous return :

- Increased PV pressure → Increased PA pressure → Decreased PBF.

The neonate with systemic hypoperfusion (Grey baby) :

- Usually of a non cardiac etiology : Sepsis.
- Listlessness,lethargy,failure to accept feeds,cold.
- extremities,mottled skin,prolonged CRT.
- Consider ECHO especially when neonatal sepsis fails to improve.

Cardiac causes of shock :

- Duct dependent systemic circulation (DDSC).
- Systemic ventricle dysfunction without obstruction :
  - Myocarditis.
  - Metabolic causes.
  - Tachycardiomyopathy.

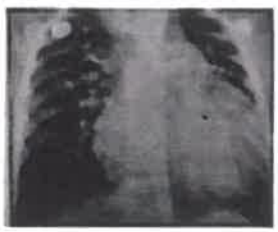
PDA to maintain SBF (DDSC) :

- Hypoplastic left heart syndrome (HLHS).
- Severe AS.
- Severe CoA.
- Interruption of the aortic arch.

PDA closure leads systemic hypoperfusion (Shock).

The neonate with respiratory distress without cyanosis/shock :

Common feature : Excessive PBF.



Increased pulmonary vasculature.

Causes in the neonate

AV septum :

- AVCD with severe MR.

Ventricular level :

- Pre term VSD.
- Associated CoA or AI.

Great arterial level :

- Pre term PDA.
- AP window.

Feedback

Assessment of neonate with suspected CCHD 00:17:55

Clinical examination :

- Pulse/BP.
  - Preductal : Right upper limb.
  - Postductal : Left lower limb.
- Pulse oximeter.
- ECG.
- CXR.
- ECHO (Gold standard).

Presentation :

- Usually benign birth history with nearly normal APGARs.
- Stable during first few hours of life,becomes symptomatic when transition of circulation occurs.
- Group presentation into one of the three faces :
  - Cyanotic neonate.
  - Shock neonate.
  - HF neonate.

Physical examination :

- Look for dysmorphic features :
  Trisomy 13/18/21.
  22q11.
  Noonan,Turners.
  Williams syndrome.
- Temperature/BP.
- Pulse and SPO2 : Pre ductal vs post ductal O2 saturation of >3-5% is significant.