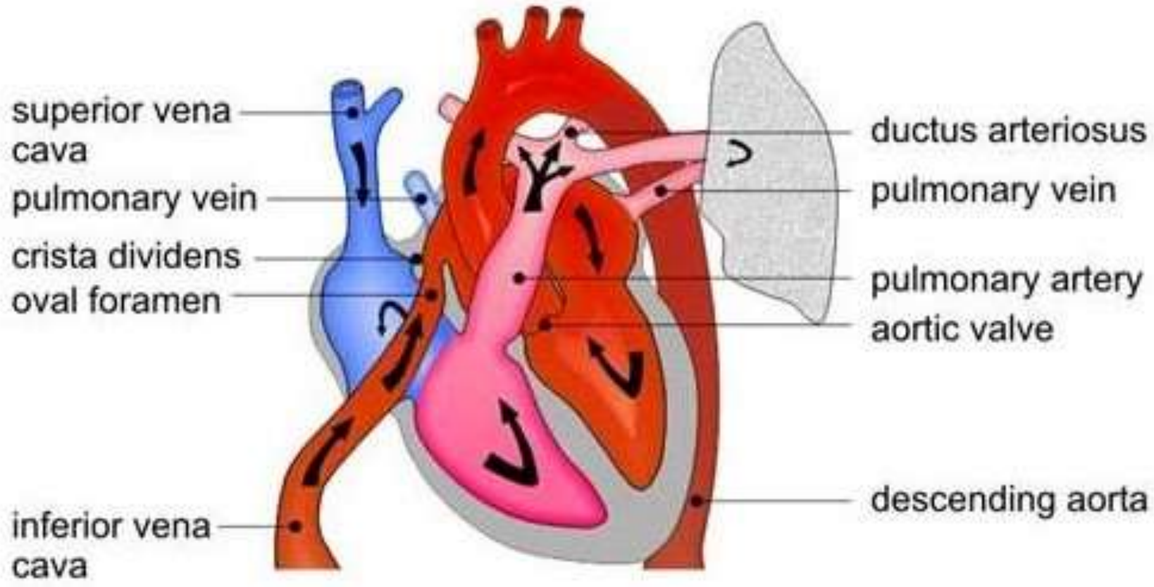
VSD & ASD
&
PDA

Left – to – Right Shunt

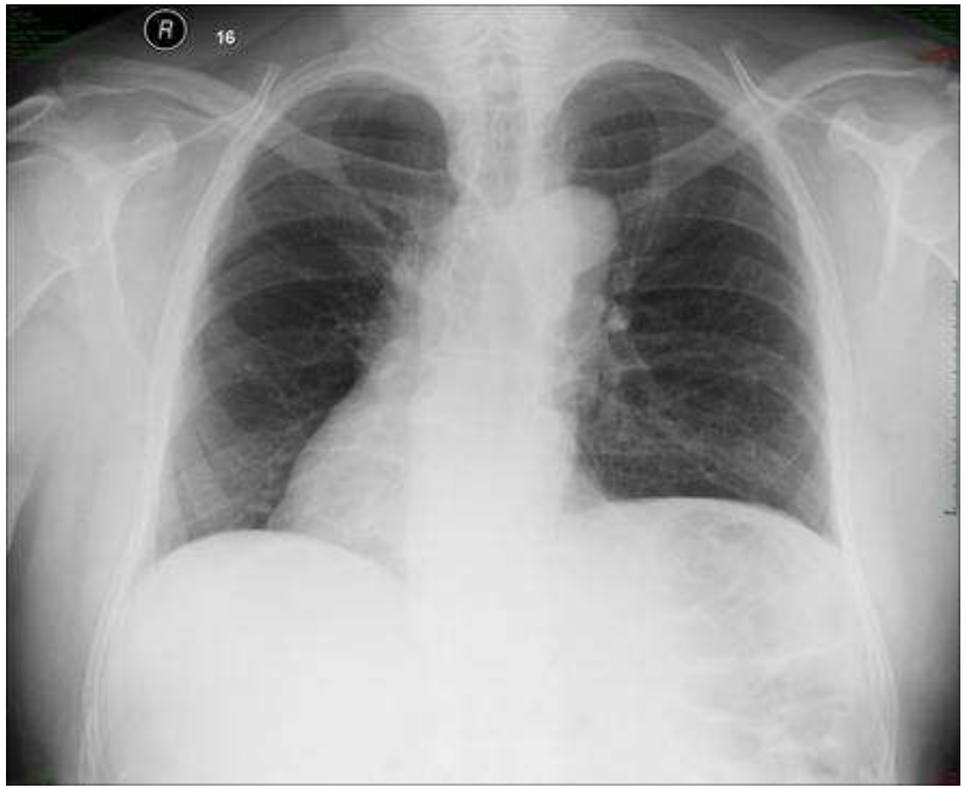
Congenital heart diseases

- Congenital heart disease is the **abnormality of the heart present from birth.**
- The incidence is higher in premature infants.
- It is attributed to multifactorial inheritance involving genetic and environmental influences.



Classification

Congenital anomalies of the heart may be either, ***shunts*** **(left-to-right or right-to-left),** or defects causing ***obstructions*** to flow, ***malposition*** of the heart.



Left – to – Right Shunts

(A cyanotic or Late Cyanotic Group)

Left to Right Shunts (Acyanotic or Late Cyanotic Group)

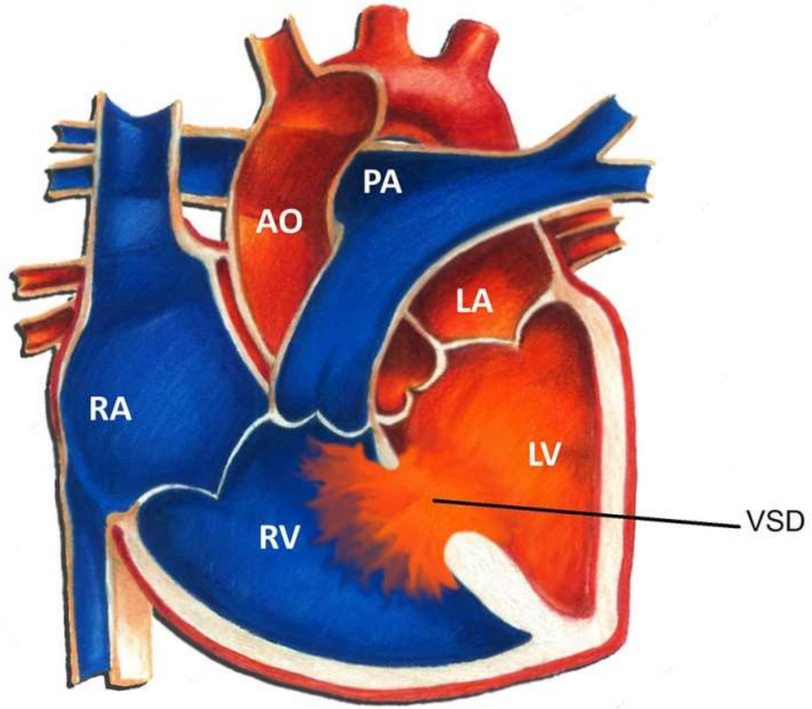
It causes volume overload on the right heart

Pulmonary hypertension and right ventricular hypertrophy

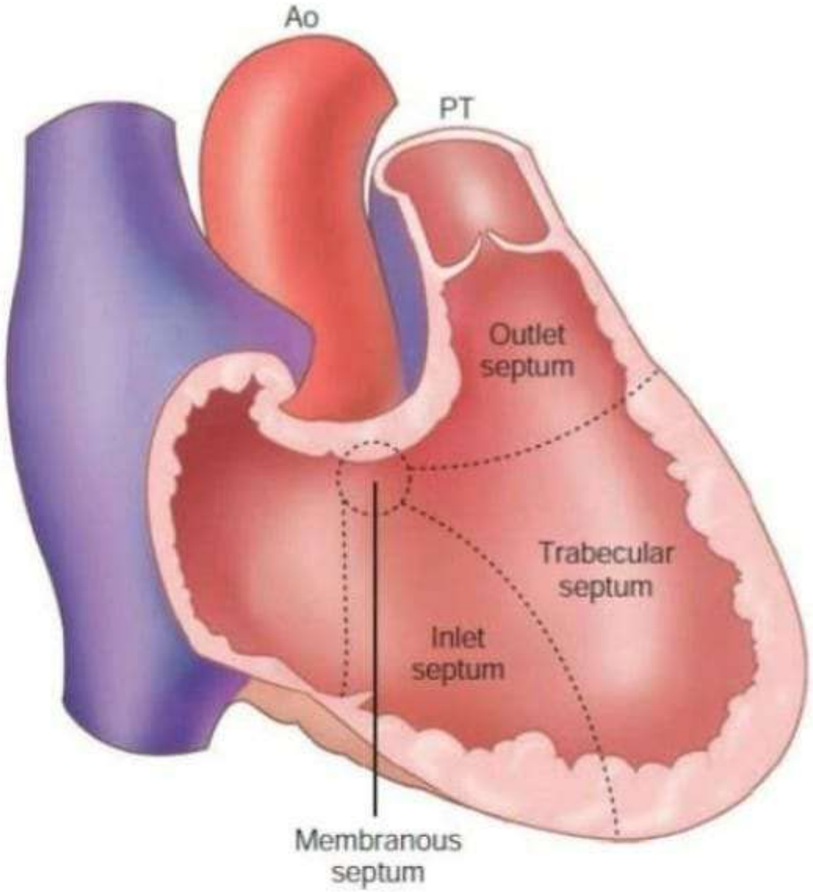
Ventricular Septal Defect (VSD)

- VSD is the most common congenital anomaly of the heart and comprises about 30% of all congenital heart diseases.

- The smaller defects often close spontaneously, while larger defects remain patent and produce significant effects.



Classification: Based on anatomical location



a) A small membranous portion and

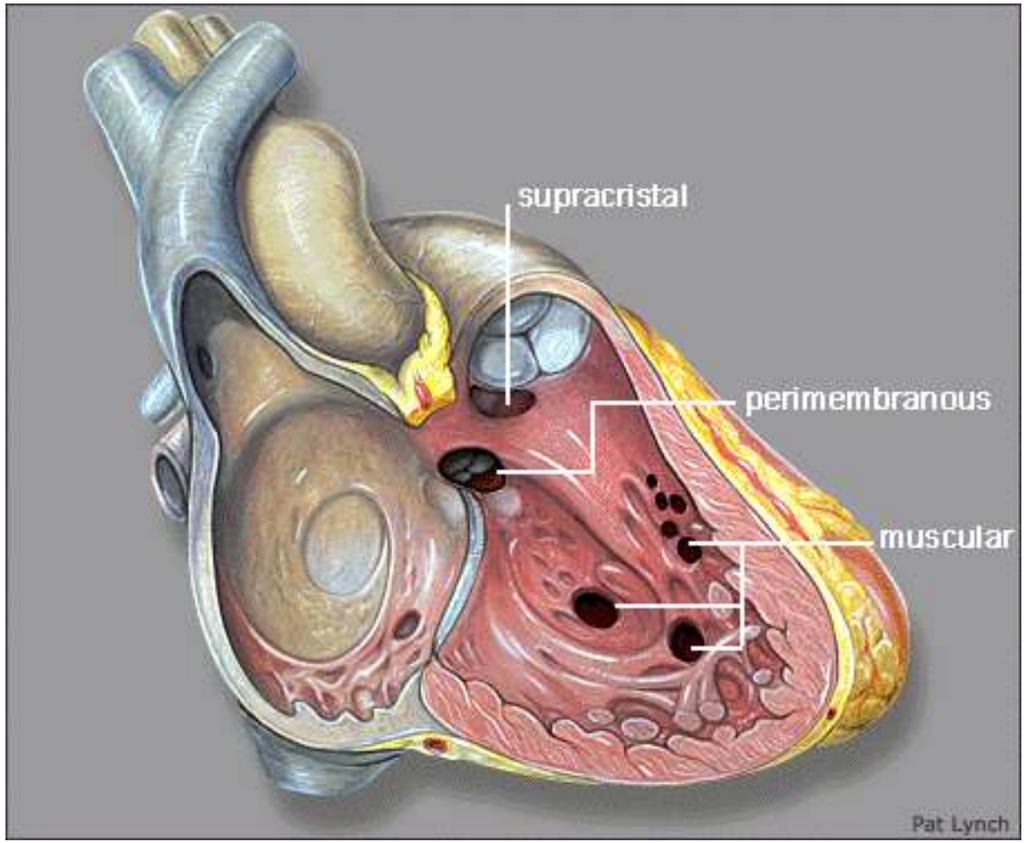
b) A large muscular portion:
   a) The inlet septum,
   b) The outlet septum
   c) The trabecular septum:
      I. Anterior
      II. Posterior
      III. Mid
      IV. Apical

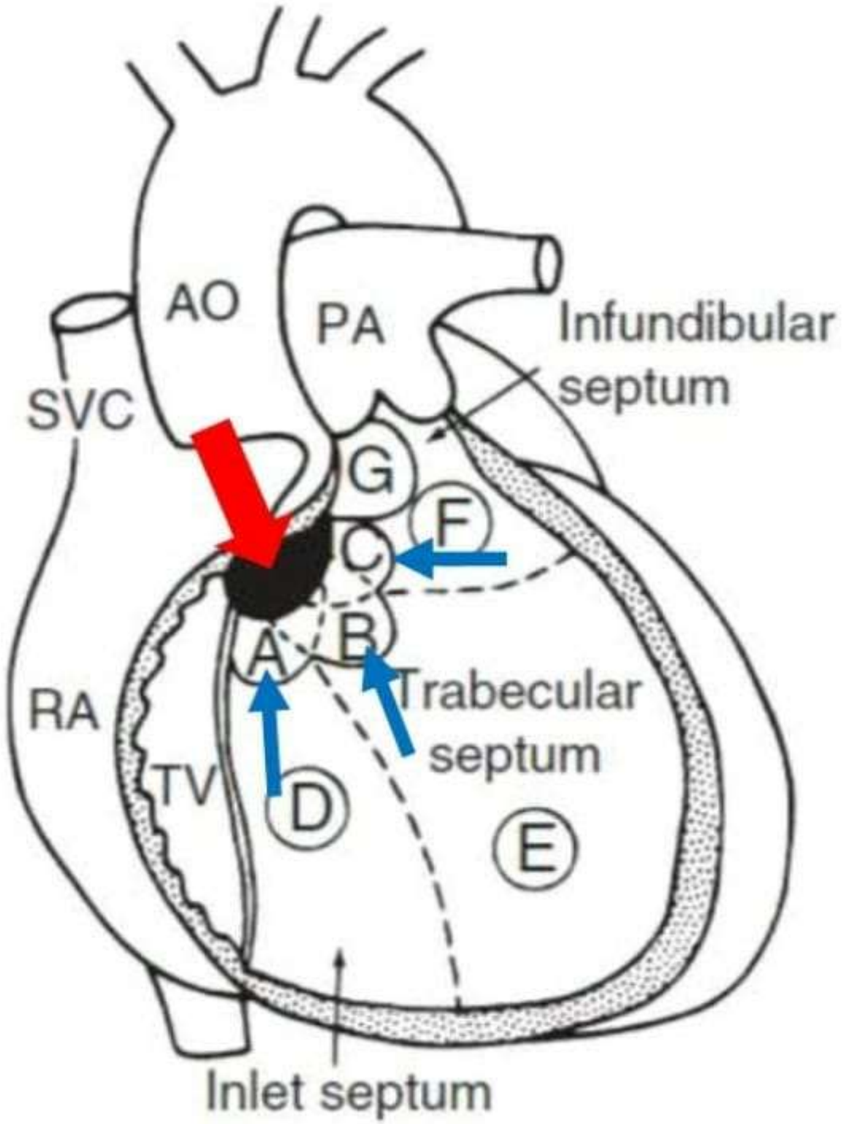
Classification: Based on size

- Small VSD: defect size is less than one-third of the size of the aortic root,
- Moderate VSD: defect size is less than one-half of the size of the aortic root, and
- Large VSD: defect size is equal to or larger than the size of the aortic root.

Types of VSD

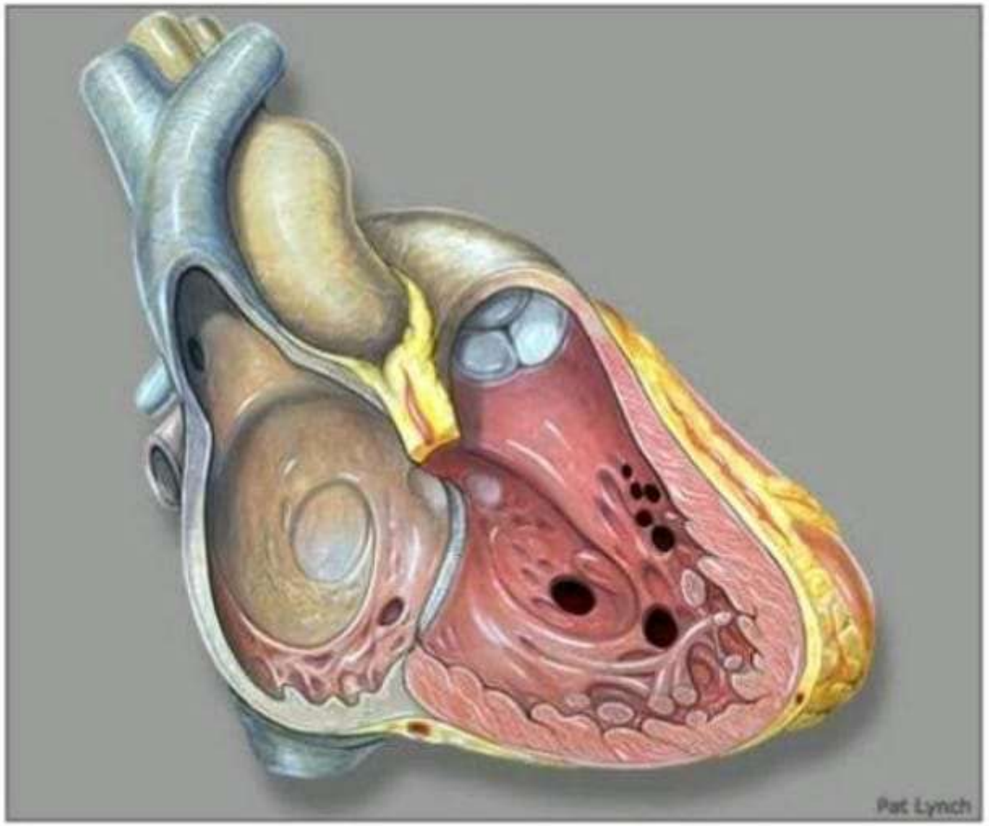
- In 90% of cases, the defect involves ***membranous septum*** and is very close to the bundle of His
- The remaining 10% cases have VSD ***Infundibular VSD (10%)*** immediately below the pulmonary valve **(*sub-pulmonic*),** below the aortic valve **(*sub-aortic*),** or exist in the form of multiple defects in the muscular septum.





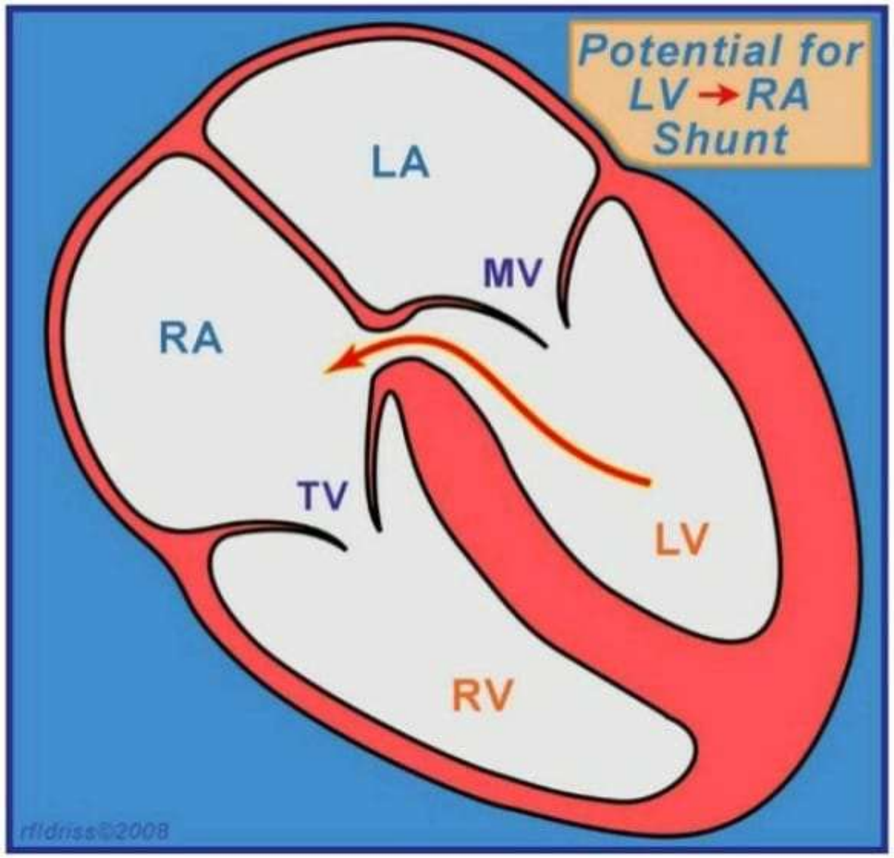
A. Perimembranous inlet ("AV canal-type") VSD
B. Perimembranous trabecular VSD
C. Perimembranous infundibular VSD
D. Inlet muscular VSD
E. Trabecular muscular VSD
F. Infundibular or outlet muscular VSD
G. Subarterial infundibular (supracristal) VSD

Swiss-cheese VSD



- The “Swiss cheese” type of multiple muscular defect (involving all components of the ventricular septum) is extremely difficult to close surgically.

Gerbode defect



- Located in the membranous portion of the atrioventricular septum.
- A Left Ventricular to Right Atrial defect.
- Uncommon, small.

Pathophysiology

LV systolic pressure (120 mm Hg) is higher than the RV pressure (15 mm Hg)

↓

Presence of VSD Leads to development of Left to Right Shunt

↓

Overload to pulmonary circuit

↓

And the volume overload occurs in LA and LV

↓

Pulmonary Artery dilation occurs ----------> Pulmonary pressure rises ----------> PVR rises

↓

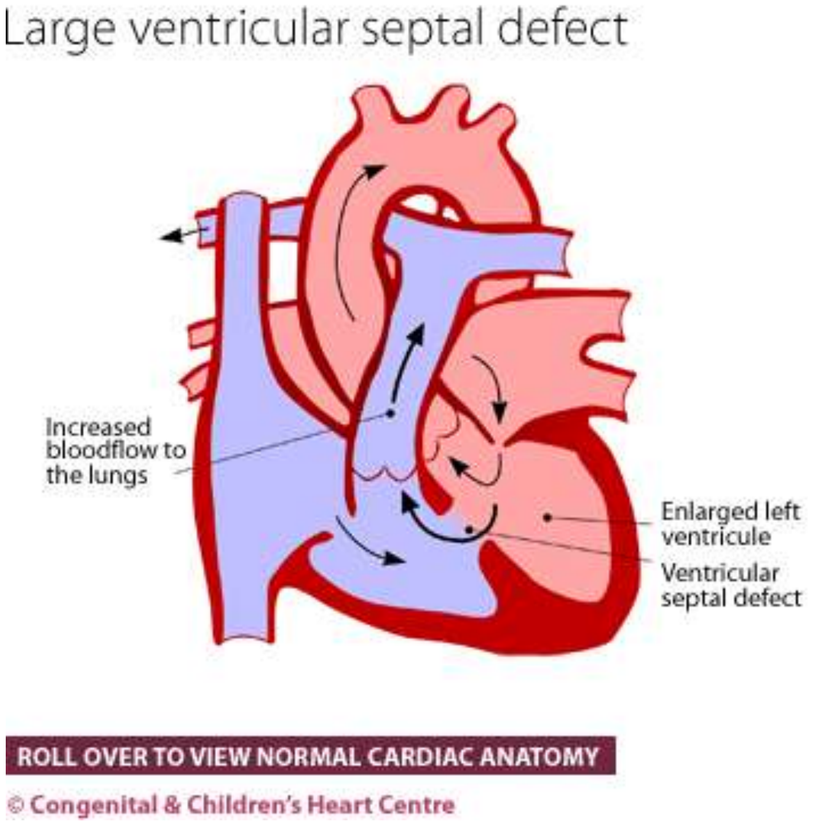
RV and LV pressures become equal and shunt becomes reverse R to L - Eisenmenger Syndrome

Effects of VSD

- VSD ⟶ increased pulmonary flow and increased volume in the left side of the heart

EFFECTS ON HEART

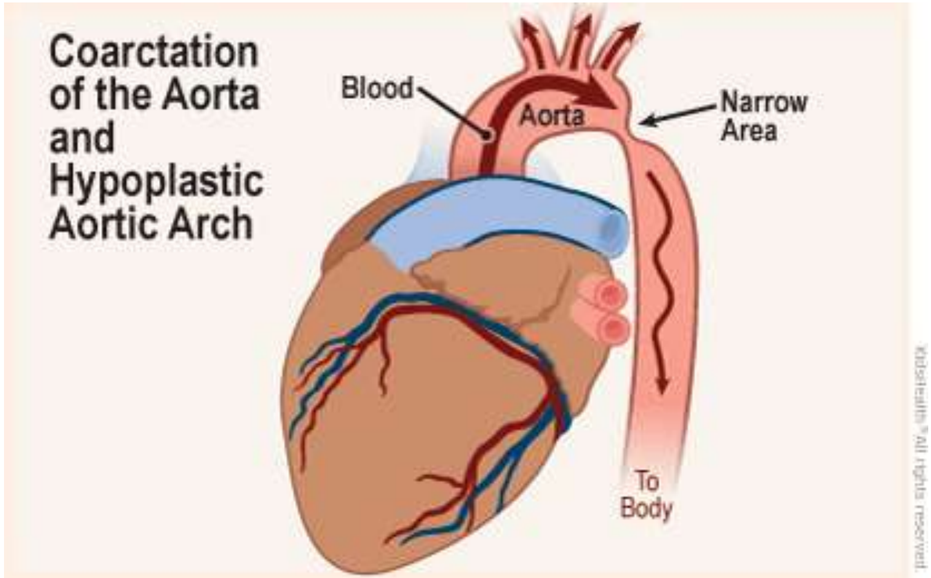
- Volume hypertrophy of RV
- Enlargement of TV & PV
- Endocardial hypertrophy of the RV
- Pressure hypertrophy of the RA



Associated Anomalies

Multiple Shunts ----- due to increased L to R Shunt

Coarctation of Aorta ------- due to increased resistance on the systemic circuit L to R shunt increases



Management

Conservative Treatment

No surgery is advised for a VSD with a diameter <7mm /m2 BSA, without PH, AR or infective endocarditis

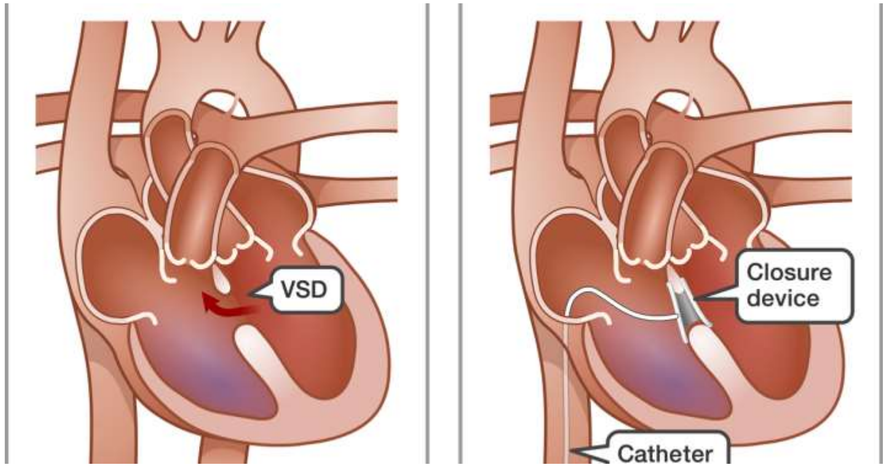
Medical Treatment

It is offered to babies who are awaiting for surgery.

Afterload decreasing agents like ACE Inhibitors, Diuretics

Decreased afterload improve LV output and reduce SVR

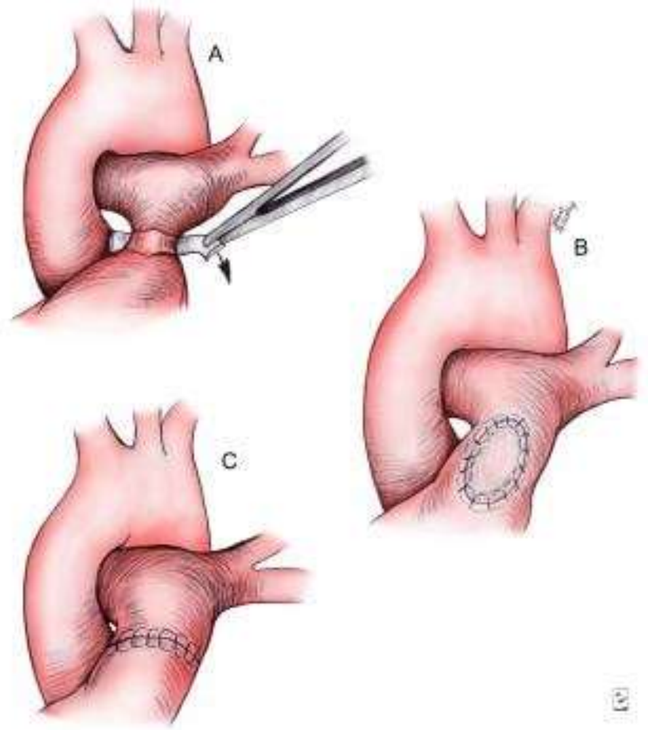
Interventional Treatment ---- Device closure of VSD



Management

Surgical Treatment - MPA Banding

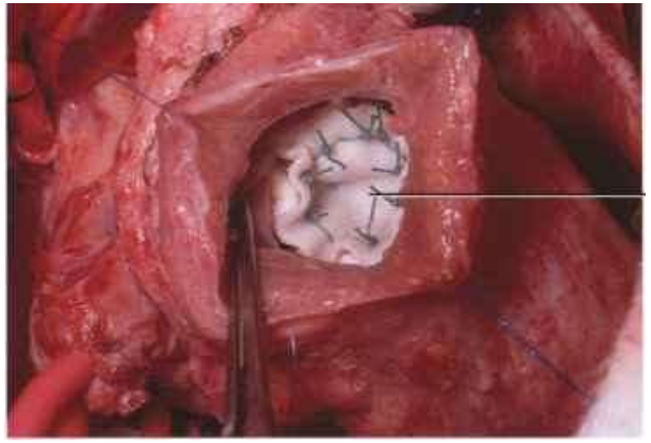
- Pulmonary artery banding (PAB) creates a narrowing, or stenosing, of the main pulmonary artery (MPA) that decreases blood flow to the branch pulmonary arteries and reduces PBF and pulmonary artery pressure
- It is done for too small babies by weight and age
- It is also done for patients having multiple (Swiss Cheese Type) VSD.
- Closure of RVOT, creates an increased resistance to flow of blood from LV to PA, thus reduces the L to R shunt, and increase the forward flow through aorta.

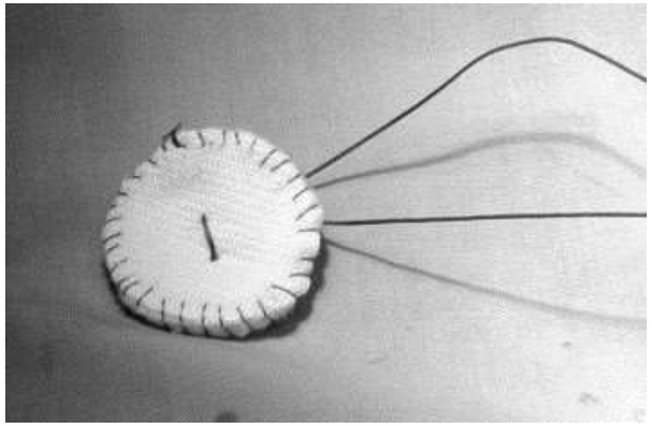


Management

VSD Closure

- Performed through median sternotomy
- VSD is approached through RA, RV, PA or aorta depending upon the situation of VSD
- VSD is closed directly if small or with a patch.
- If VSD is large, patch could be of autologous pericardium, Dacron or Poly Tetra Fluoro Ethylene



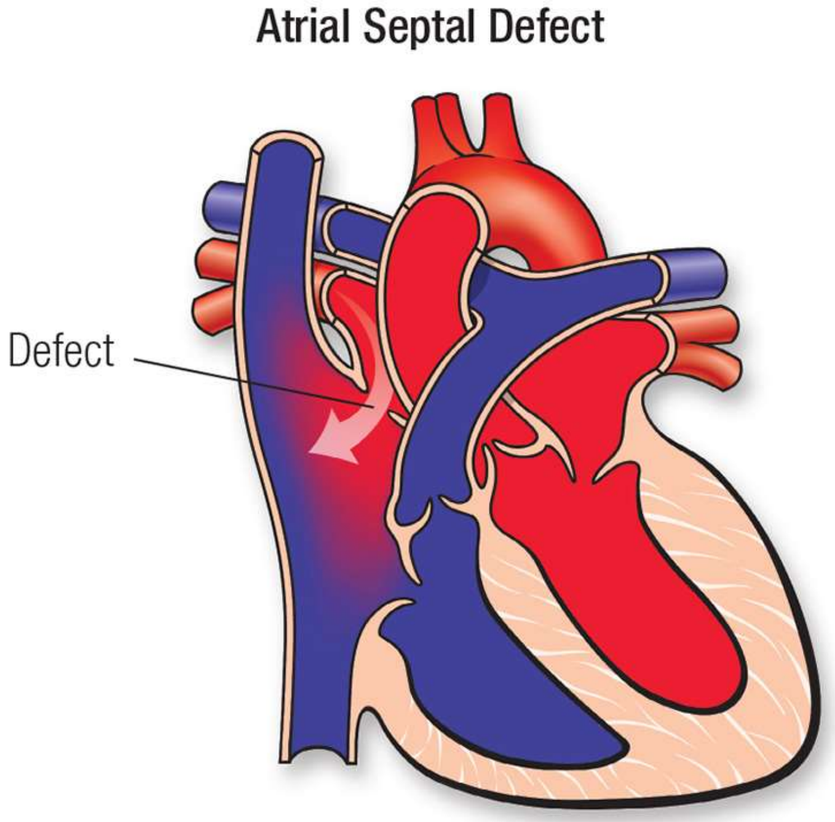


Atrial Septal Defect

- It comprises of 10% of congenital heart diseases

- The condition remains unnoticed in infancy and childhood till pulmonary hypertension

is induced causing late cyanotic heart disease and right-sided heart failure.



Types of ASD

Depending on location of defect, they are

- **Fossa ovalis type or ostium secundum type** is the most common form comprising about 90% cases of ASD **(located in fossa ovalis)**

- **Ostium primum type** comprises about 5% cases of ASD **(low in the interatrial septum)**

↓

adjacent to atrioventricular valves

- **Sinus venosus type** accounts for about 5% cases of ASD **(high in the interatrial septum)**

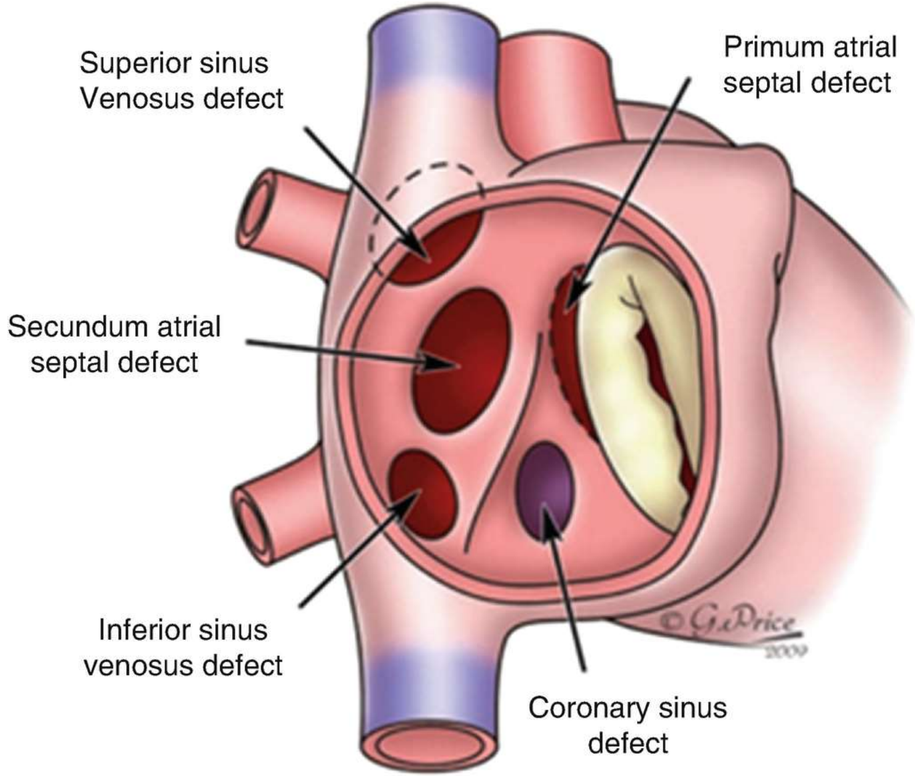
↓

near the entry of the superior vena cava.

Types of ASD

The term single atrium is applied when Inter Atrial Septum (IAS) is absent

Fenestrated ASD: Some time there are multiple small (2-3 mm in diameter) ASD . Collectively they produce a significant hemodynamic load



Pathophysiology

Shunts through ASD are of two types:

Facultative Shunt --------- > It is seen in isolated ASDs. The shunt is not essential for sustaining life of the patient. The shunt is basically L to R shunts

Obligatory Shunt ---------- > ASD is a part of a larger pathological complex. This shunt is essential for sustaining life of the patient.

Pathophysiology

- Defect ----- >IAS blood flows from Left Atrium to Right Atrium

- Blood flows from Left to Right,
- LA Pressure (4 – 12 mm Hg) is higher than RA Pressure (0 – 7 mm Hg)
- RV Compliance is much more than the LV Compliance, so blood will flow easily via LA-RA- RV

- The quantity of shunt is described as **Amount of blood (Q) passing through pulmonary circuit (Qp) to the amount of blood passing through systemic circuit (Qs).**
- The ratio in normal person is 1.0 : 1.0
- An ASD requiring surgery or intervention has Qp : Qs of more than 1.5 : 1.0

Pathophysiology

RA receives blood from SVC, IVC and CS
RV receives shunted blood from LV

↓

RAVO and RVVO and volume load in PA occurs

↓

RV dilation occurs, this increases pressure in PA

↓

Pulmonary Hypertension occurs

↓

Due to PH, RV has to generate more pressure to pump blood into pulmonary circuit.

↓

Prolonged PH leads to RV Failure

RVEDP Rises → Decrease in L to R Shunt → R to L Shunt occurs → Eisenmenger Complex

Associated Anomalies

Partial Anomalous Pulmonary Venous Connection (PAPVC)

- Associated with superior sinus venosus (SV ASD)
- One or two right pulmonary vein drain anomalously into RA or SVC

Mitral Valve Disease (called Leutembacher's syndrome) : Secundum ASD is associated with mitral stenosis or regurgitation

Multiple Shunts : ASD associated with additional VSD or PDA

Valvular Pulmonary Stenosis: In PS, the shunt through ASD is usually left to right, but when RV fails, shunt decreases and later becomes right to left

Left SVC : An additional L SVC producing bilateral SVC is found in many cardiac anomalies with bridging left innominate vein

Management

Medical:

- Drug therapy is mainly for symptomatic purposes
- Antibiotics: to control frequent lower respiratory infections (LRTI) which patients with ASD have due to excessive pulmonary flow

Interventional:

- Less than 2cm in diameter
- Has well defined margins around
- Has anomalies which can be treated by intervention (Absence of PAPVC & MR)

Surgical Management:

- Patch will be placed based on the size of the ASD - Autologous Pericardium, Dacron cloth or PTFE

PDA

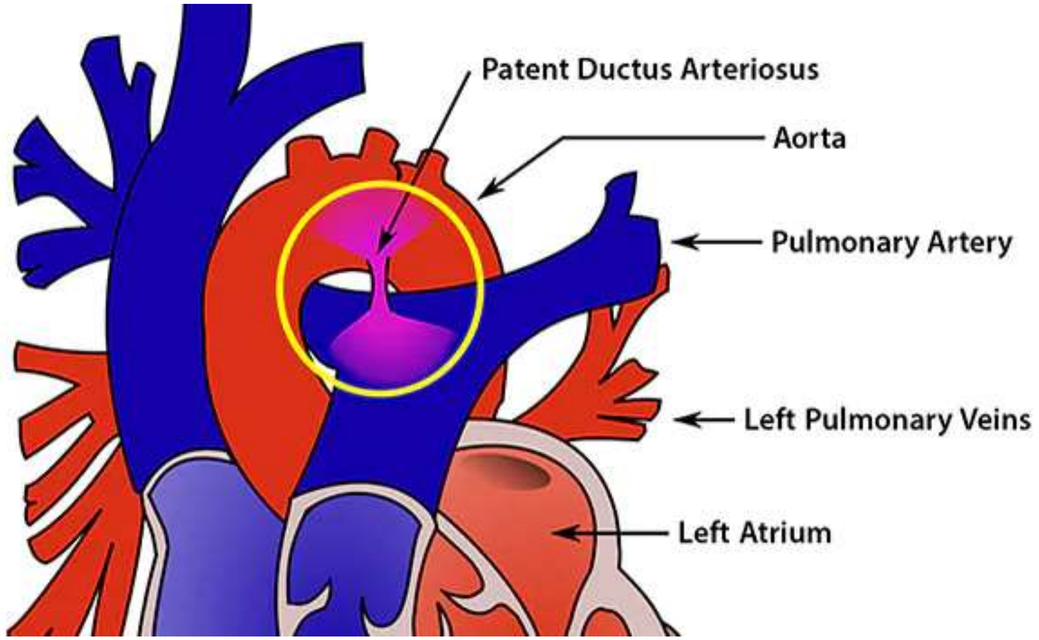
- The **ductus arteriosus** is a normal vascular connection between the aorta and the bifurcation of the pulmonary artery.

- Normally, ductus closes functionally within the first or second day of life

- More than 3 months of age ---- its abnormal

Etiology:

- The cause for patency of ductus arteriosus is not known, but possibly it is due to continued synthesis of PGE2 after birth

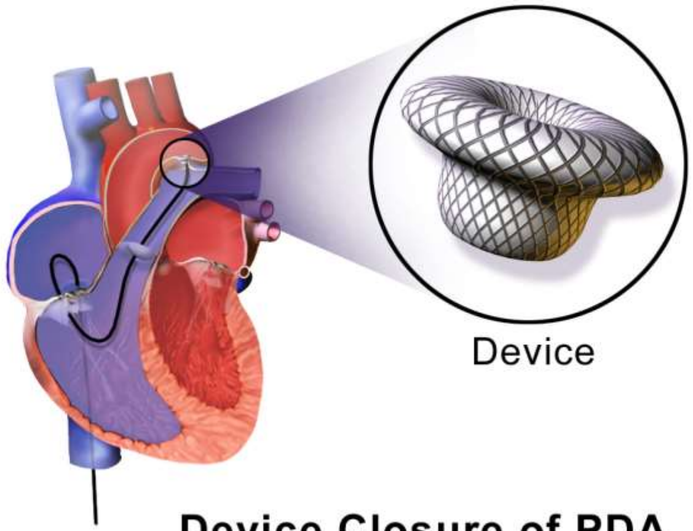


Morphological Changes in PDA

- Volume hypertrophy of the LA & LV
- Enlargement of the ascending aorta
- Enlargement of MV and PV

PDA

- Patient experience respiratory distress syndrome
- Pharmacologic closure of PDA with administration of indomethacin to suppress PGE2 synthesis



Device Closure of PDA