ACYANOTIC HEART DISEASES

SUBMITTED TO:

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INTRODUCTION:

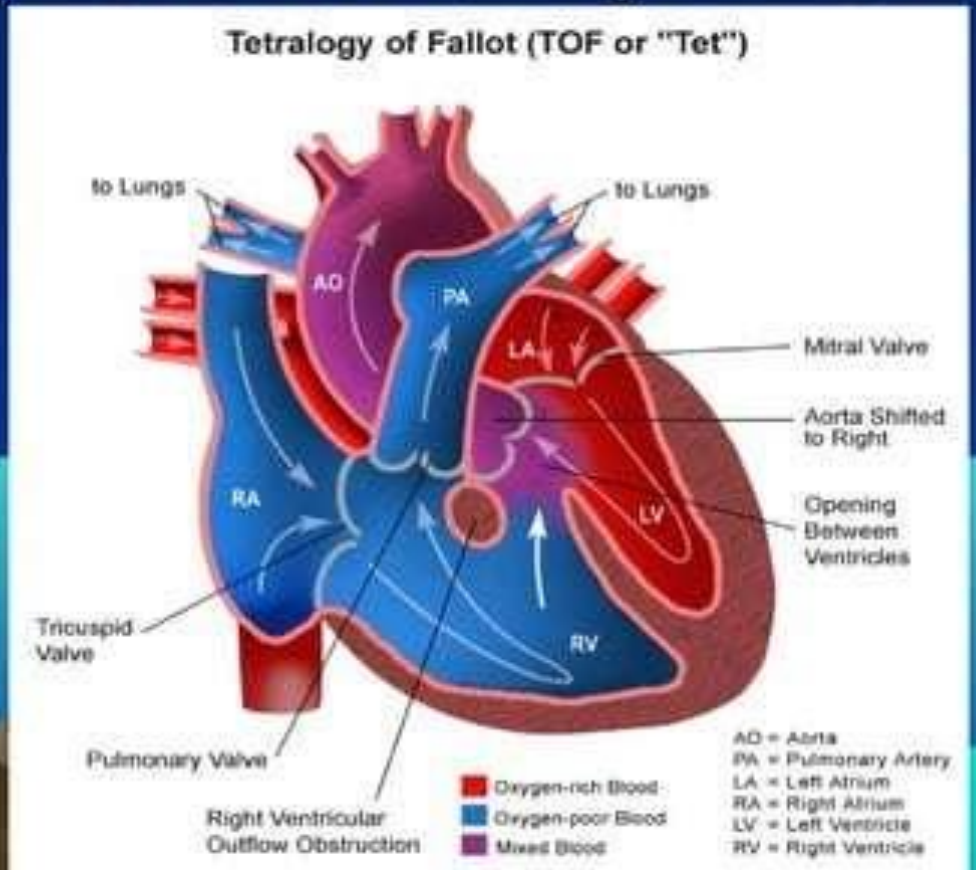
Heart diseases mainly fall into two broad groups. Congenital and acquired. Congenital heart diseases is the structural malformation of the heart while acquired heart diseases are mainly due to inflammatory process.

A cyanotic heart disease is the type of congenital heart diseases and refers to the series of birth defect that affect the heart.

CONGENITAL HEART DISEASES

DEFINITION:

- Congenital heart disease is defined as the structural, functional or positional defect of the heart in isolation or in combination present at birth but may manifest at anytime after birth or may manifest at all.

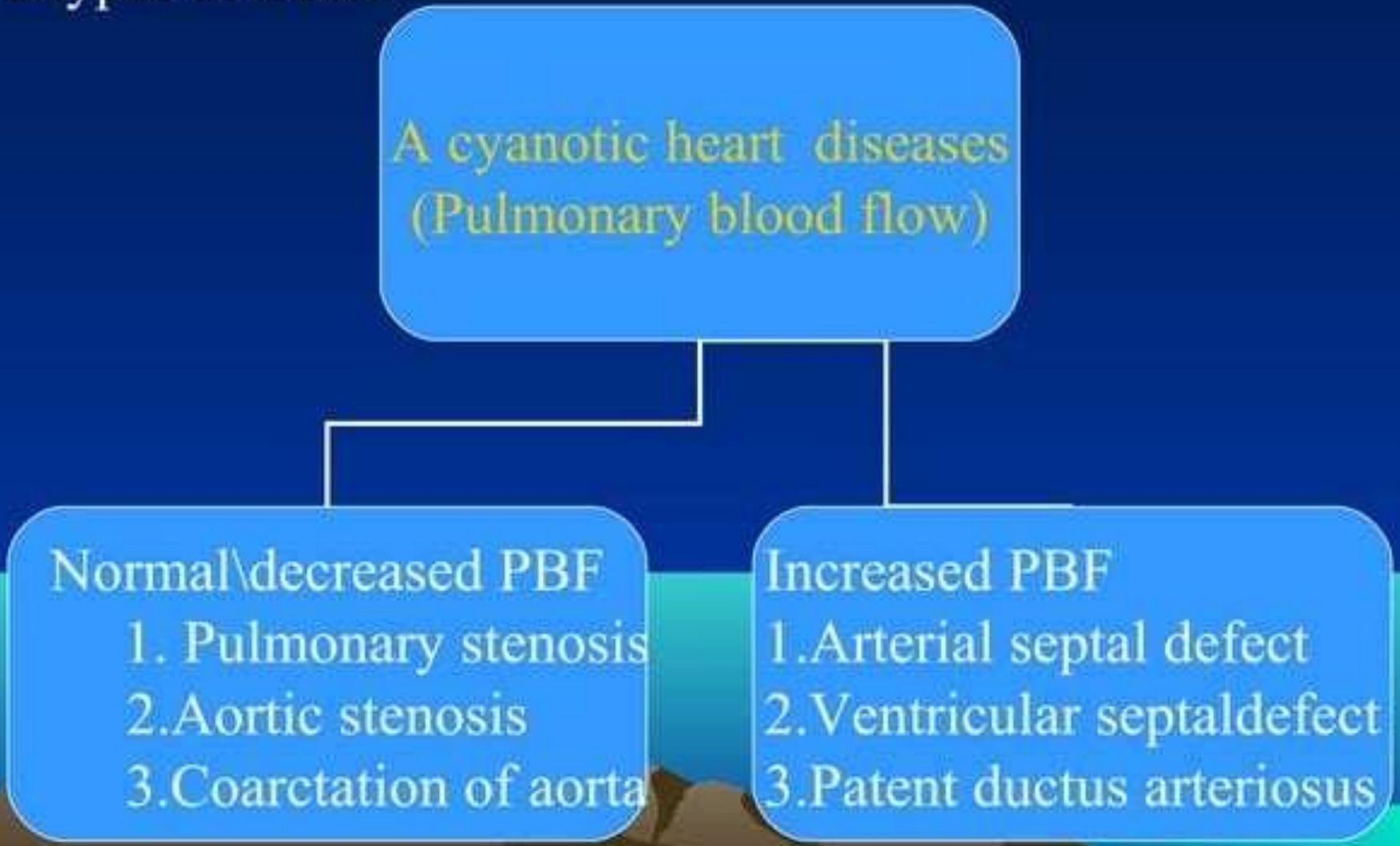


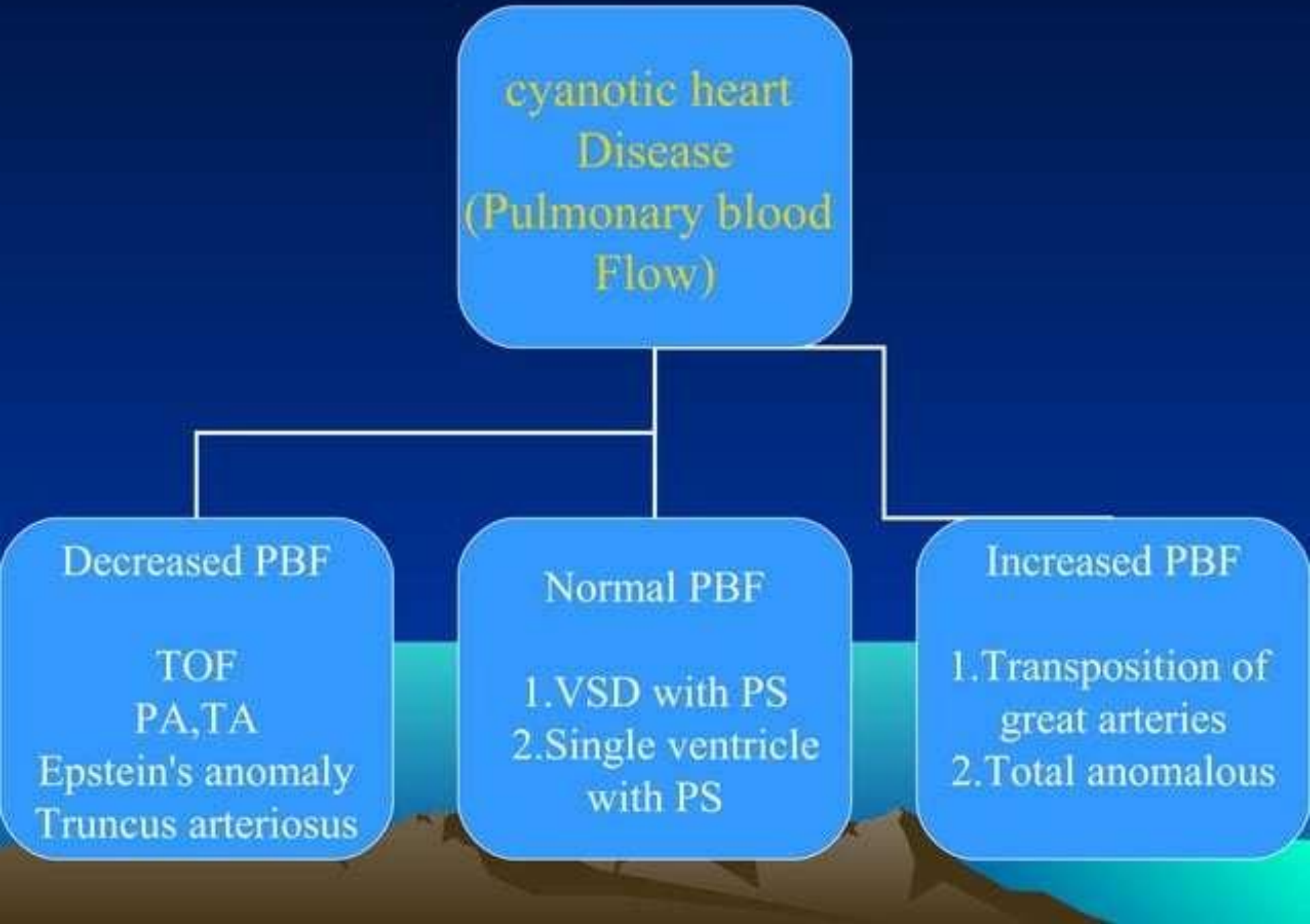
INCIDENCE OF CONGENITAL HEART DISEASE

1. 6 -8 per 1000 live births.
2. 1 per 1000 at 10 yrs of age.
3. 2% of total death is due to CHD
4. A Cyanotic heart disorders are more common than cyanotic ones.
5. For girls –PDA,ASD
6. For boys –PS,AS,transposition and coaretation are more common.

Classification of CHD

- 2 types of CHD.




cyanotic heart Disease (Pulmonary blood Flow)
Decreased PBF
TOF
PA,TA
Epstein's anomaly
Truncus arteriosus
Normal PBF
1.VSD with PS
2.Single ventricle with PS
Increased PBF
1.Transposition of great arteries
2.Total anomalous

ACYANOTIC HEART DISEASE

- A cyanotic heart disease is a congenital heart defect in which the infant has no cyanosis because there is no mixing of unoxygenated blood in systemic circulation.

DEFINITION:

- It is a circulatory problem that is congenital &it is atypical of most congenital heart defects in that it doesn't cause the child to present with blue skin or finger nails.
- Is a congenital heart defect where the blood contains enough oxygen but it is pumped abnormally around the body.

INCIDENCE OF ACYANOTIC HEART DISEASE

1. Females: males ratio is 3:1
2. VSD: 25% of total CHD
3. PDA: 9%
4. PS :10%
5. AS :5%
6. COA : 4%
7. ASD :10%
8. A cyanotic : 60 -65% of total CHD
9. Cyanotic :30-35%

CAUSES OF ACYANOTIC HEART DISEASE

1. Exact cause is unknown
2. Abnormal embryonic development.
3. Possible causes are

a) Fetal and maternal infection

b)Maternal disease like

→ German measles, cytomegalovirus infection

→ Teratogenic effects of drugs &alcohol

e.g)Lithium,thalidamide.

→Maternal dietary deficiencies

e.g)Poor nutritional status

→ Hereditary &consanguineous marriage.

→ Maternal age greater than 40

→Alcohol intake by mother, irradiation.

→ Maternal insulin dependent diabetes

FETAL CAUSES:

1.Fetal hypoxia, birth asphyxia

2.Genetic disorder &Chromosomal aberrations

e.g)

Down syndrome –VSD

Turner syndrome – COA

Trisomy 13,18 _VSD,ASD PDA.

REASON FOR NO CYANOSIS:

1. No abnormal communication between pulmonary &systemic circulation
2. The peripheral blood is therefore oxygenated as in normal infant and cyanosis doesn't result

TYPES OF ACYANOTIC HEART DISEASE:

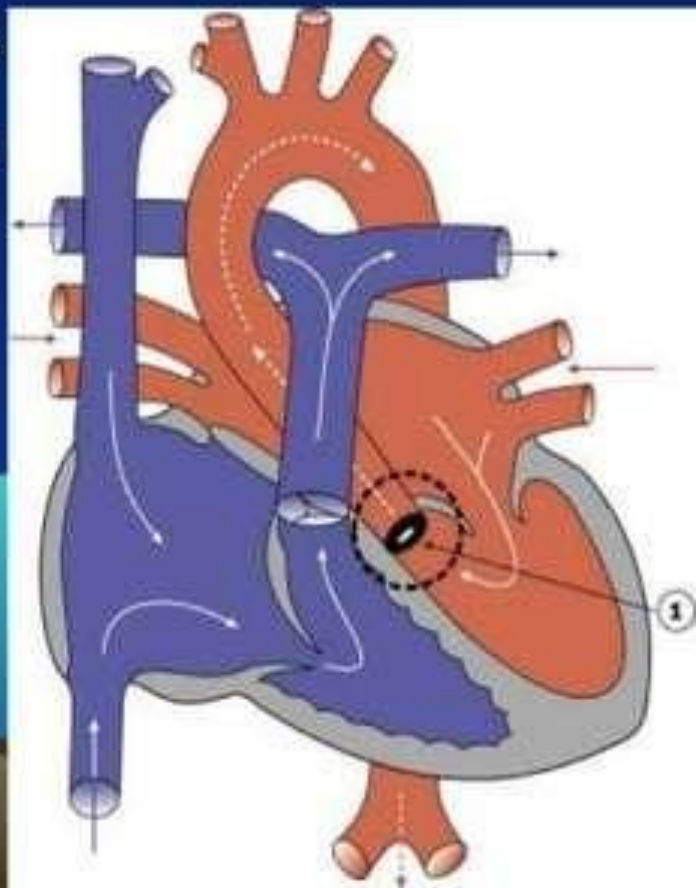
According to pulmonary blood flow

Normal or decreased pulmonary blood flow:

1.PULMONARY STENOSIS:

Definition:

Ps is an obstructive lesion that interferes with blood flow from the right ventricle.



Description:

1.PS is the narrowing at the entrance to the to the pulmonary artery.

2.Resistance to blood flow cause right ventricular hypertrophy & decreased PBF.

3.Pulmonary atresia is the extreme form of PS.

Types of pulmonary stenosis:

On the basis of their anatomical presentation

2 types of pulmonary stenosis:

1.Valvular stenosis

2.Infundibular stenosis

VALVULAR STENOSIS:

1. Occurs more than 90% of cases
2. Cups of the pulmonary valves are fused
3. Cause dome like stenotic valve &Right ventricular hypertrophy.

INFUNDIBULAR STENOSIS:

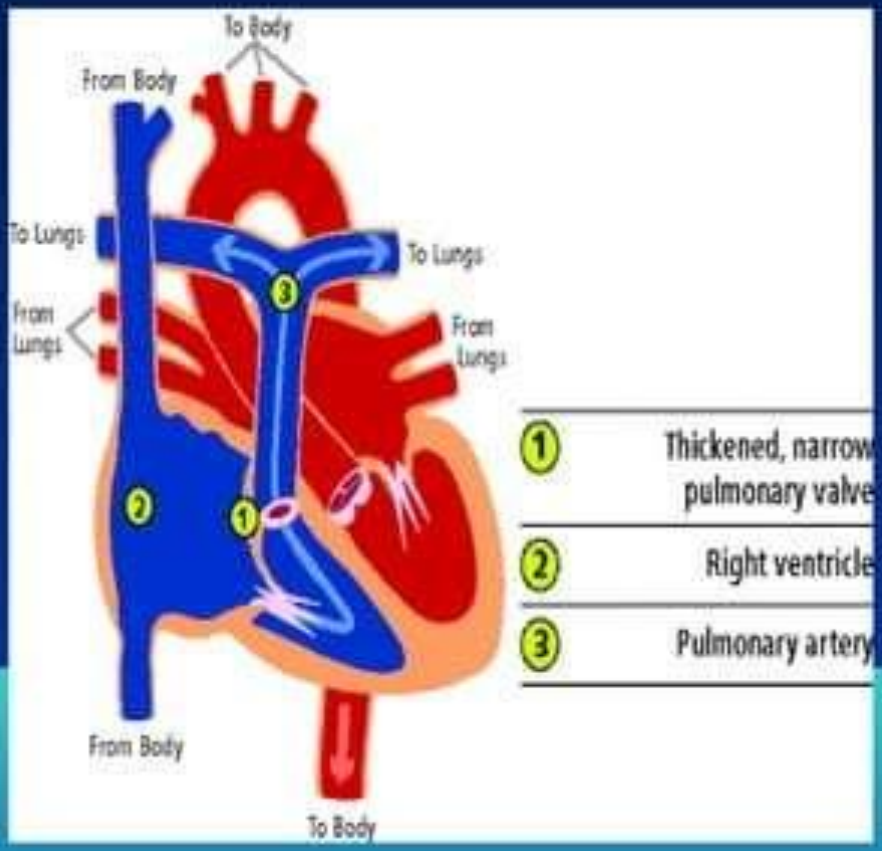
1.Less common

2.Pulmonary valve is normal but outflow of right ventricle is narrow.

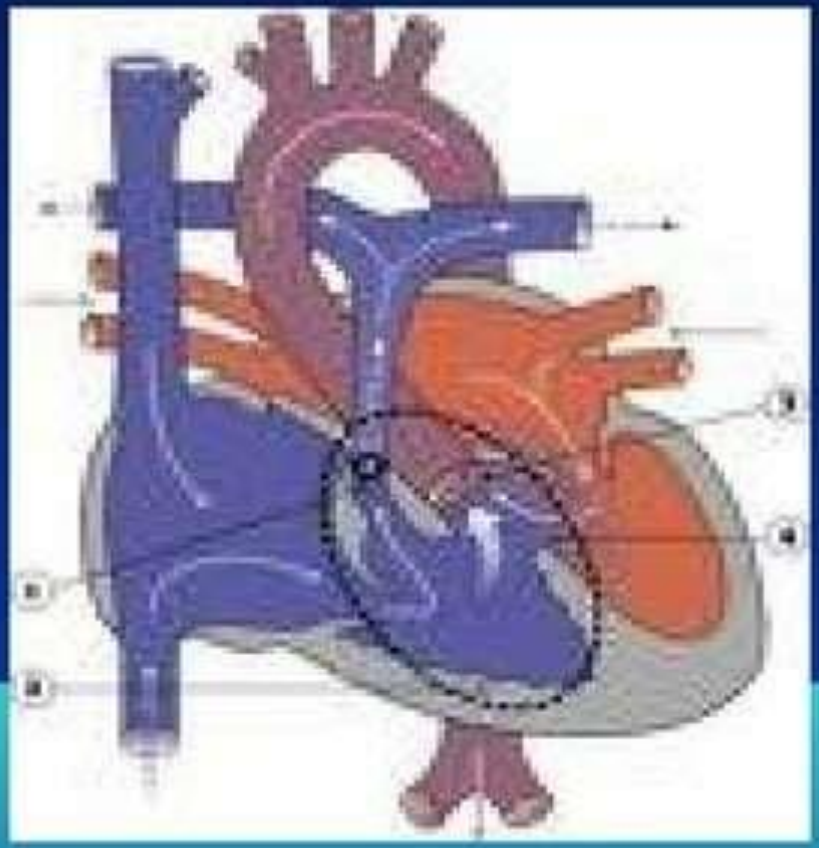
3.Converting the narrowed region into an infundibular channel.

4.It is called as third ventricle.

Valvular stenosis



Infundibular stenosis



Pathophysiology & Haemodynamics:

Etiology

Pulmonary valve is obstructed by fusion of cups

Résistance to blood flow from RV to PA

Increased pressure in the RV

Increased pulmonary stenosis

Blood is backed up into the RA

↓

Reopening of the foraman ovale

↓

Shunting of unoxygenated blood to the LA

↓

Systemic cyanosis occur only PS

↓

If @ with PDA,it compensate the obstruction by

↓

Shunting of blood from aorta to PA &to lungs

↓

No cyanosis

Clinical manifestation:

No cyanosis\asymptomatic.

Mild PS produce:

1. Exertional fatigue
2. Chest pain with exercise
3. Systolic murmur

In severe PS produce:

1. Dyspnoea
2. Cardiac failure
3. Cyanosis
4. Child may squat to relief dyspnoea

Investigations:

1. Electrocardiogram – Right ventricle hypertrophy

2.Radiography –Enlargement of the heart

3.Cardiac catheterization –PS pressure

Management:

Medical management:

Prophylaxis - Bacterial endocarditis

Surgical management:

Indications:

1.CCF

2.Right ventricular pressure

3.Cyanosis

Name of the surgery:

1.Pulmonary valvotomy\ Brock procedure

2.Ballon angioplasty by cardiac catheterization.

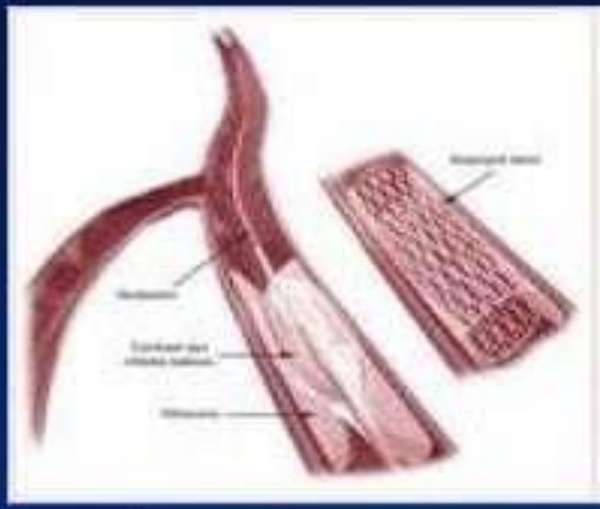
Complication:

1.CCF.

2.Bacterial endocarditis

3.Primary tuberculosis

4.Anoxic spells

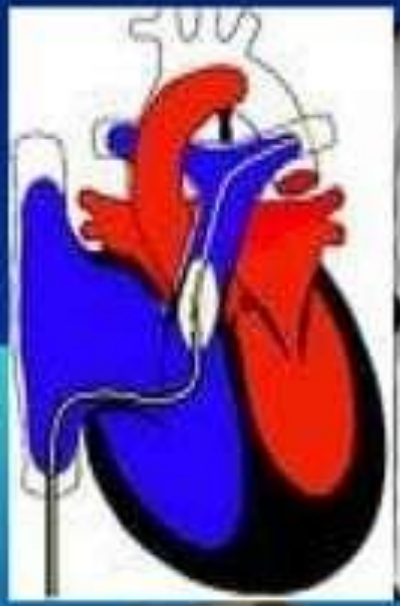


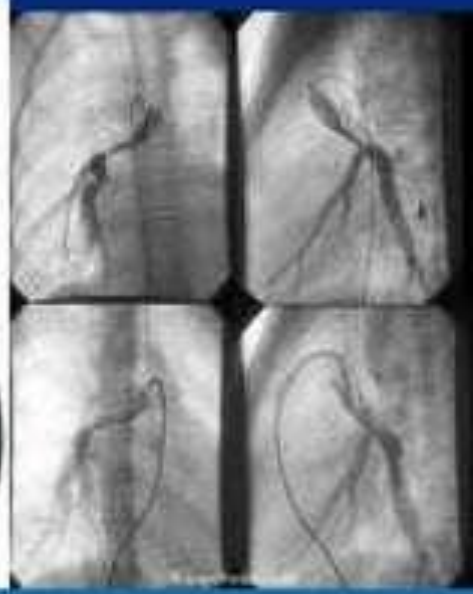
Prognosis:

1. Mortality-2 to3%

2.Good for children with mild PS

3.Severe PS – cyanosis,CHF.





2.AORTIC STENOSIS

Definition:

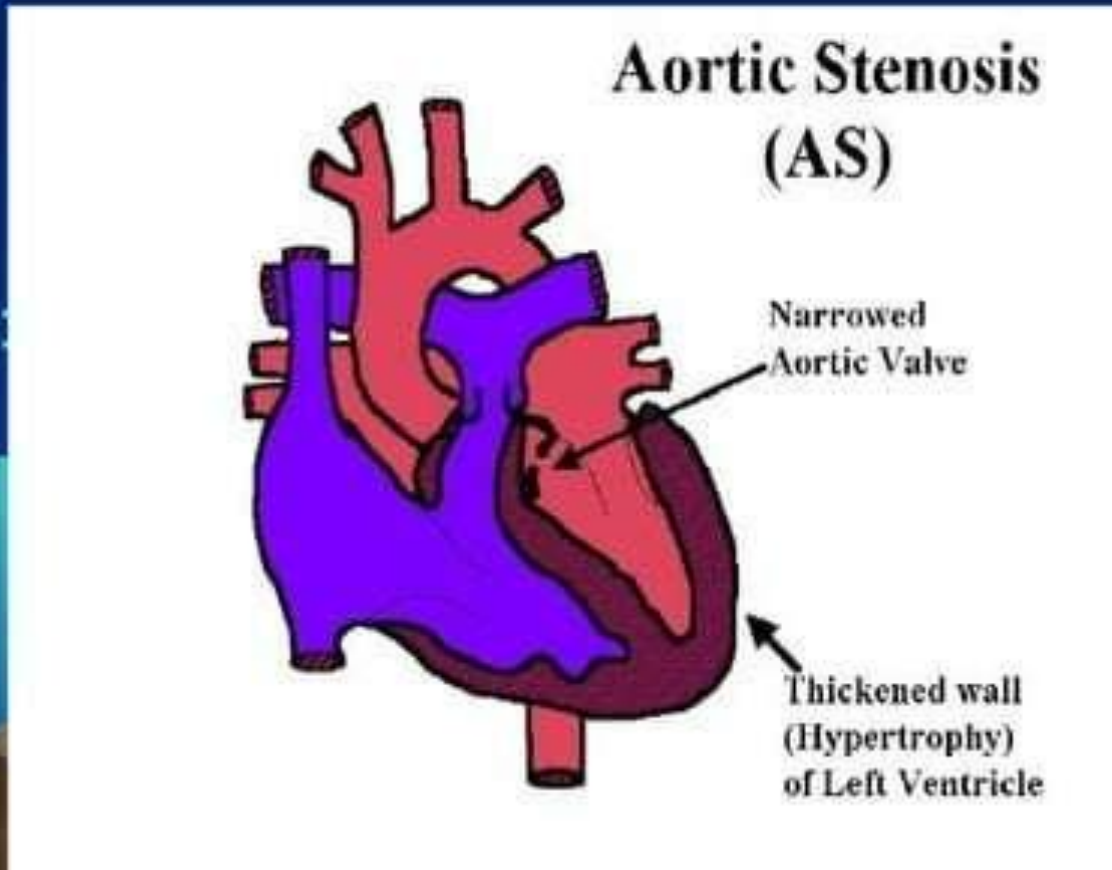
AS is narrowing or stricture of the aortic valve, causing resistance to blood flow in the LV,decreased cardiac output,left ventricular hypertrophy & pulmonary vascular congestion.

Incidence:

1. Males >Females
2. 80% of CHD is AS

Types of aortic stenosis:

1. Valvular stenosis
2. Subvalvular stenosis
3. Supravalvular stenosis



1.VALVULAR STENOSIS:

It is a most common type is usually caused by malformed cups resulting in a bicuspid rather than tricuspid valve or fusion of the cups.

It accounts about 75%.Male>Female(2:1)

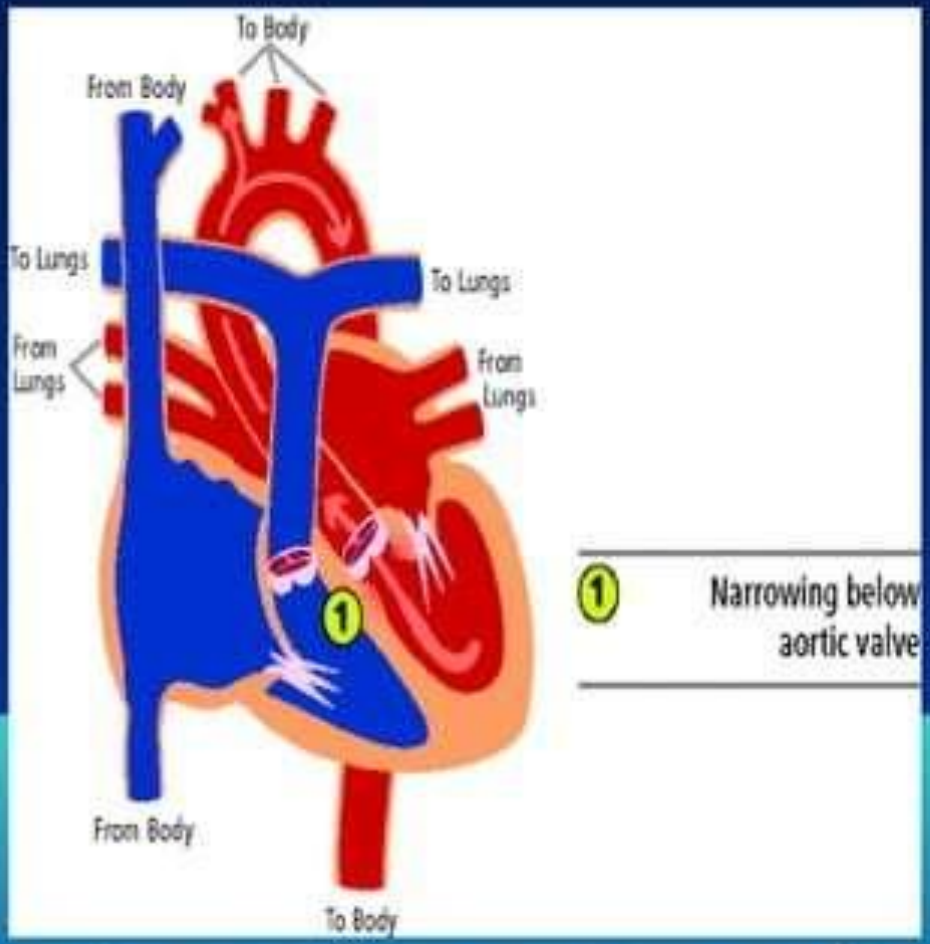
2.SUBVALVULAR STENOSIS:

It is a stricture caused by a fibrous ring below a normal valve. Accounts about 20% of cases. Fibrous muscular obstruction forms ring 5-10 mm the aortic valve.

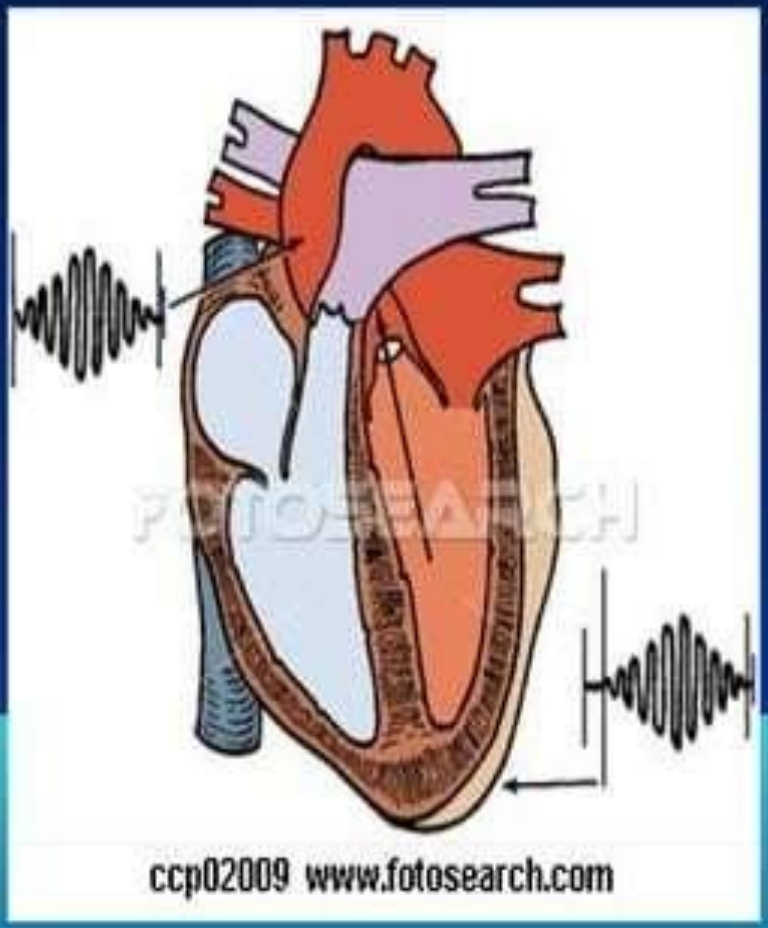
3.SUPRAVALVULAR STENOSIS:

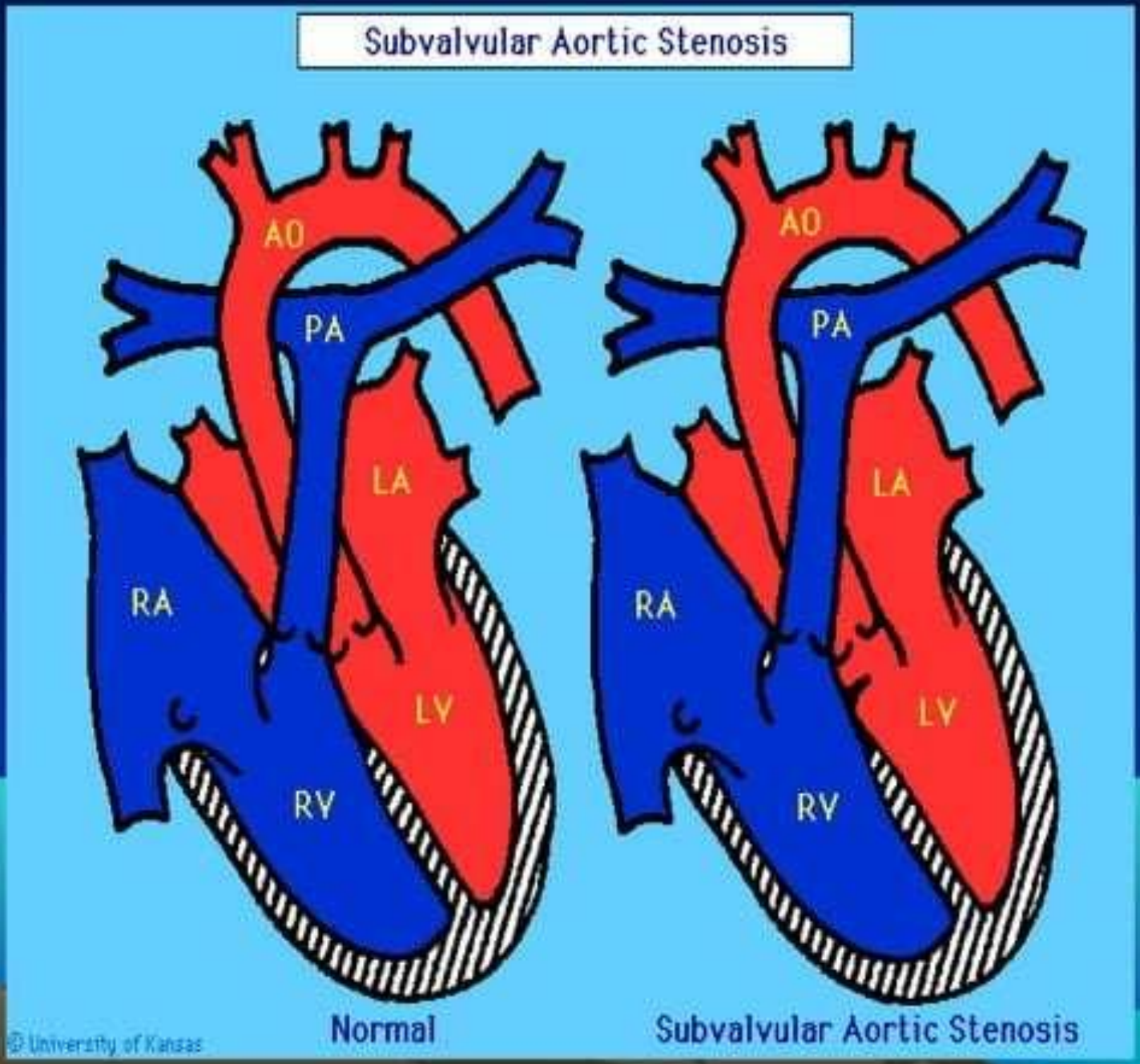
Stenosis occurs just above the coronary arteries. It occurs infrequently.

valvular stenosis



Supra valvular stenosis




Subvalvular Aortic Stenosis
AO
PA
LA
RA
LV
RV
AO
PA
LA
RA
LV
RV
Normal
Subvalvular Aortic Stenosis
© University of Kansas

Pathophysiology &haemodynamics:

Etiology

↓

Narrowing of the aortic valve

↓

Resistance to blood outflow from the left ventricle to the aorta

↓

Extra workload in the LV.

↓

Left ventricle hypertrophy.

↓

Blood backs up in the left atrium

Increased pressure in the left ventricle

↓

Increased pressure in the pulmonary veins

↓

Pulmonary vascular congestion

↓

Pulmonary edema due to AS

Clinical manifestation:

1. Hypotension
2. Decreased cardiac output with faint pulse.
3. Tachycardia
4. Poor feeding
5. Exercise intolerance

6.Chest pain

7.Dizziness when standing for long period

8.Murmur

9.Bacterial endocarditis

10.Coronary insufficiency

11.Ventricular dysfunction

Investigation:

1.Chest x ray: No cardiomagaly,Aortic knuckle is prominent.

2.ECG : Normal ECG.

3.Echocardiography: Find out changes in heart sounds.

4.Angiocardiography : Enlargement of the LV.

5.Cardiac catheterization:

Demonstrate the pressure differential between the left ventricle &Aorta.

The following computation is used for assessing the severity of stenosis

1.Mild : Gradient < 40 mmhg

2.Moderate : Gradient 40-75 mmhg

3.Severe : Gradient > 75 mmhg

Management:

MEDICAL MANAGEMENT:

1.Treatment of CCF

2.Treatment of bacterial endocarditis

SURGICAL TREATMENT:

VALVULAR AS:

1.Aortic valvotomy:

Mortality -10-20%

25% of patient require additional surgery within 10 yrs of recurrent stenosis.

SUBVALVULAR AS:

1.Incision or cutting of fibro muscular ring.

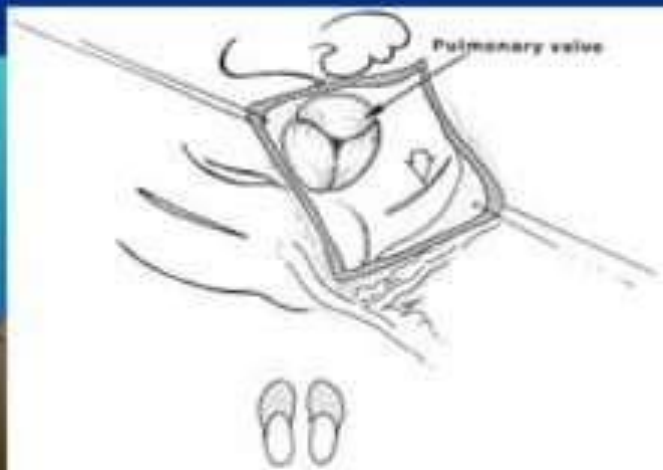
2.A patch to enlarge LV outflow.

3.Konno procedure –Replacement of Aortic valve

4.Ross procedure -Pulmonary valve may be moved to the aortic position &replaced with homograft valve& also known as extended aortic root replacement

NON SURGICAL:

Dilating narrowed valve by balloon angioplasty in cardiac catheterization

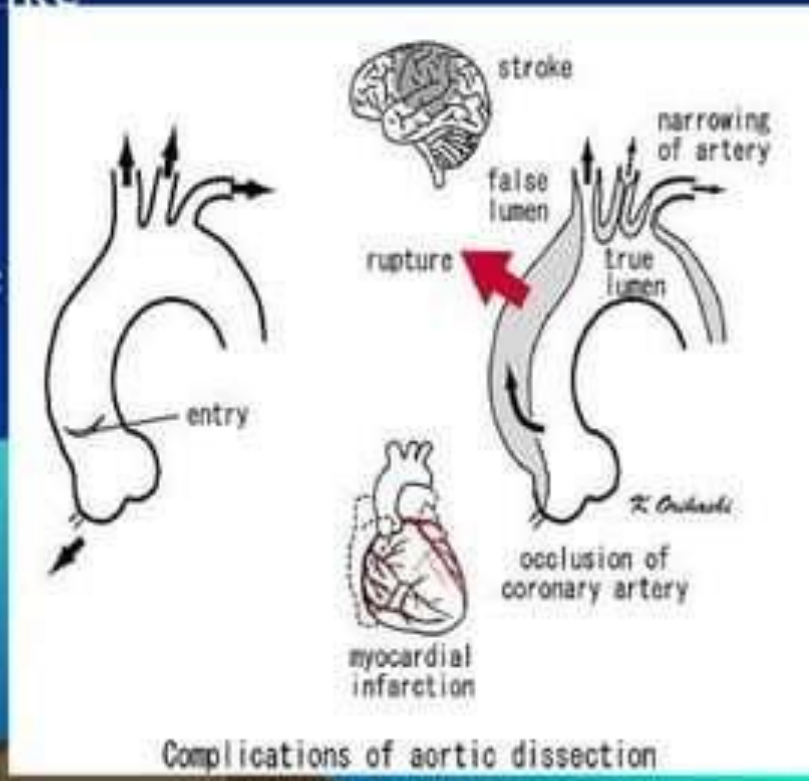


Complication:

1. Sudden death
2. Endocardial fibroelastosis .
3. Associated malformation like ASD,VSD,PS,COA.

Prognosis:

It is fair.Incases of severe stenosis &those with @ anomalies the ultimate outlook is not bright due to sudden death



Complications of aortic dissection

3.COARCTATION OF THE AORTA

Definition:

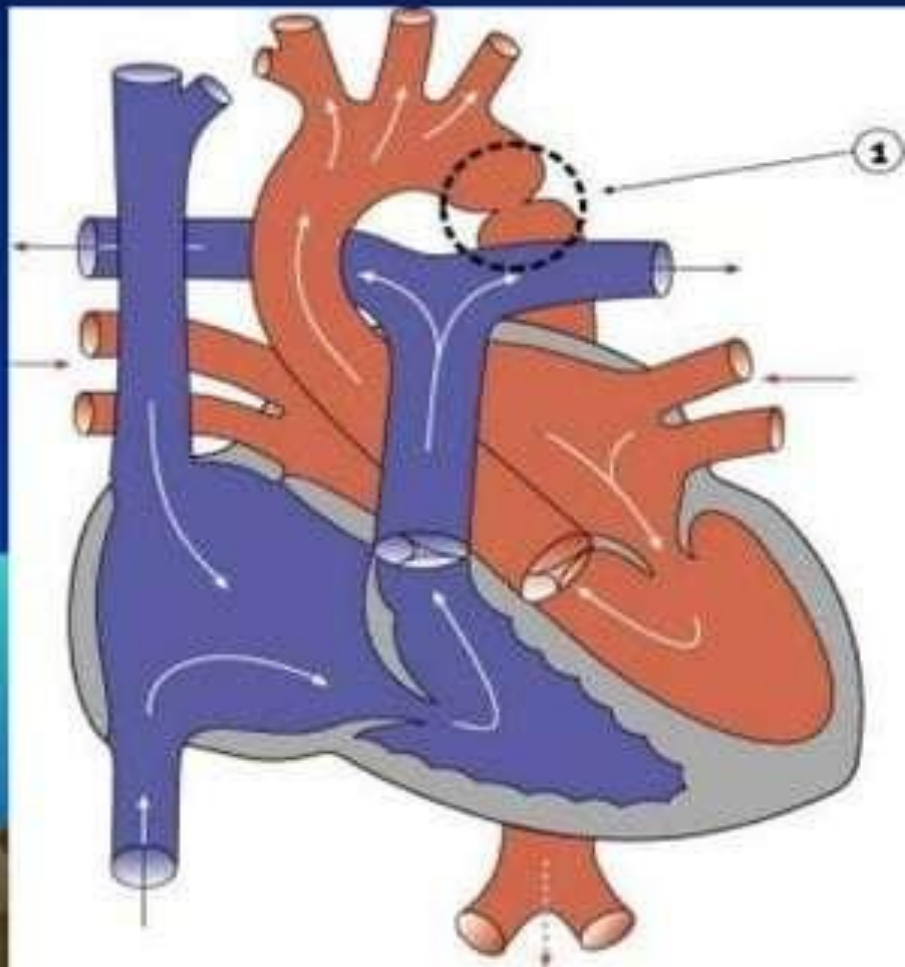
COA is a localized malformation caused by a deformity of the Aorta that results in a narrowing of the lumen of that vessels.

Incidence:

1.Accounts about 5% of CHD

2.1\3 of it present after childhood.

3.Male>Females(2:1)



Anatomy:

On the basis of their anatomical presentation COA is classified into 2 types:

1.INFANTILE\ PREDUCTAL TYPE:

There is a constriction between the subclavian artery &the arteriosus.98% is more common. It located at near the region of the aortic isthmus.

2. POSTUCTAL TYPE:

Constriction at on distal to the ductus arteriosus.

Description:

1.Narrowing near the insertion of the ductus arteriosus.

2.Increased pressure to the proximal to the defect (Head& Upper extremities

3. Decreased pressure to the distal part of the defect (Body& Lower extremities)

Pathophysiology and Haemodynamics:

Congenital causes

↓

Narrowing within Aorta

↓

Decrease pressure to the distal part of the defect

↓

Increase pressure to the proximal part of the defect

↓

Increase left ventricular workload

↓

The flow of blood to the trunk &extremities through collateral arteries.

↓

Collateral arteries bypass the coarctation

It connect arteries the branches of the subclavian artery to the arteries which arise from Aorta below coarcation

Suzman's sign(Dilatation of collateral arteries are often seen over the scapular regions of the back)

HAEMODYNAMICS:

Preductal type:

The lower half of the body supplied by

Right ventricle through the ductus arteriosus

Postductal:

RV cannot maintain blood flow to the decending Aorta

Collateral arteries develops

It maintain flow from ascending to the decending Aorta.

Clinical manifestation:

1. High BP (Upper part of the body)
2. Bounding pulses in arms, weak femoral pulse
3. cool lower extremities with lower BP
4. Signs of CHF
5. Increase pressure it resulting in headache.
6. Dizziness
7. Fainting

8.Epistaxis

9.Cerebrovascular accidents.

10.Muscle cramps in the leg while exercise due to anoxia.

INVESTIGATION:

1.X- ray:

Shows Dock's sign

2.Retrograde aortography:

It passes via brachial artery may demonstrate the presence &extent of coarcted area & state of collateral circulation.

3.Angiography:It shows COA

4.Cineangiography:Shows extent of the COA

5.Cardiaccatheteriztion:Estimate the progression of COA.

6.Echocardiography:Shows @ anomalies.

7. In radiology (Barium swallowing): Shows 'E' signs

MANAGEMENT:

Medical management:

1. Administer prostaglandin E1 (Ductal patency)
2. Treatment of Hypertension
    a) beta blockers
    b) M dopa
    c) Captopril.

Surgical management:

1. End To End Anastomosis
2. Grafting
3. Percutanious balloon angioplasty

Prognosis:

1. Mortality < 5%
2. Preductal is poor. Postductal is better.
3. Increase risk in infants with other complex cardiac defects

Complications:

1. Cardiac failure
2. Subauute bacterial endocarditis
3. Intracranial hemorrhage.
4. Rupture of the aorta.

According to increased pulmonary blood flow the acyanotic heart disease classified into 3

1. Atrial septal defect
2. Ventricular septal defect
3. Patent ductus arteriosus.

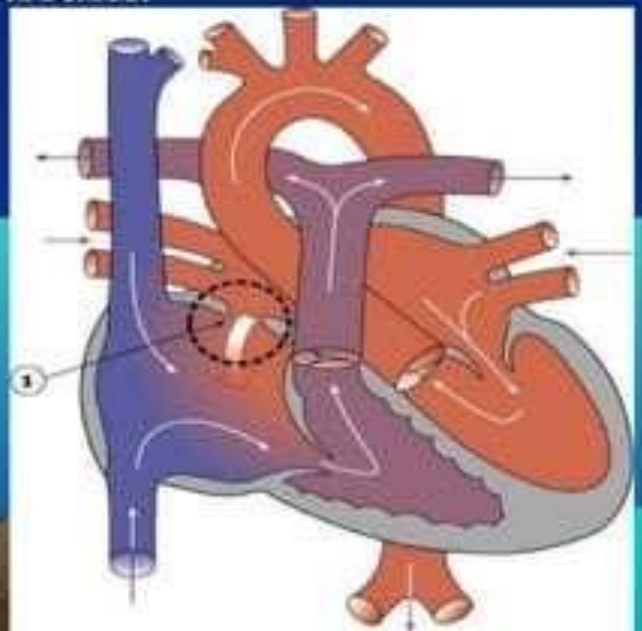
ATRIAL SEPTAL DEFECT:

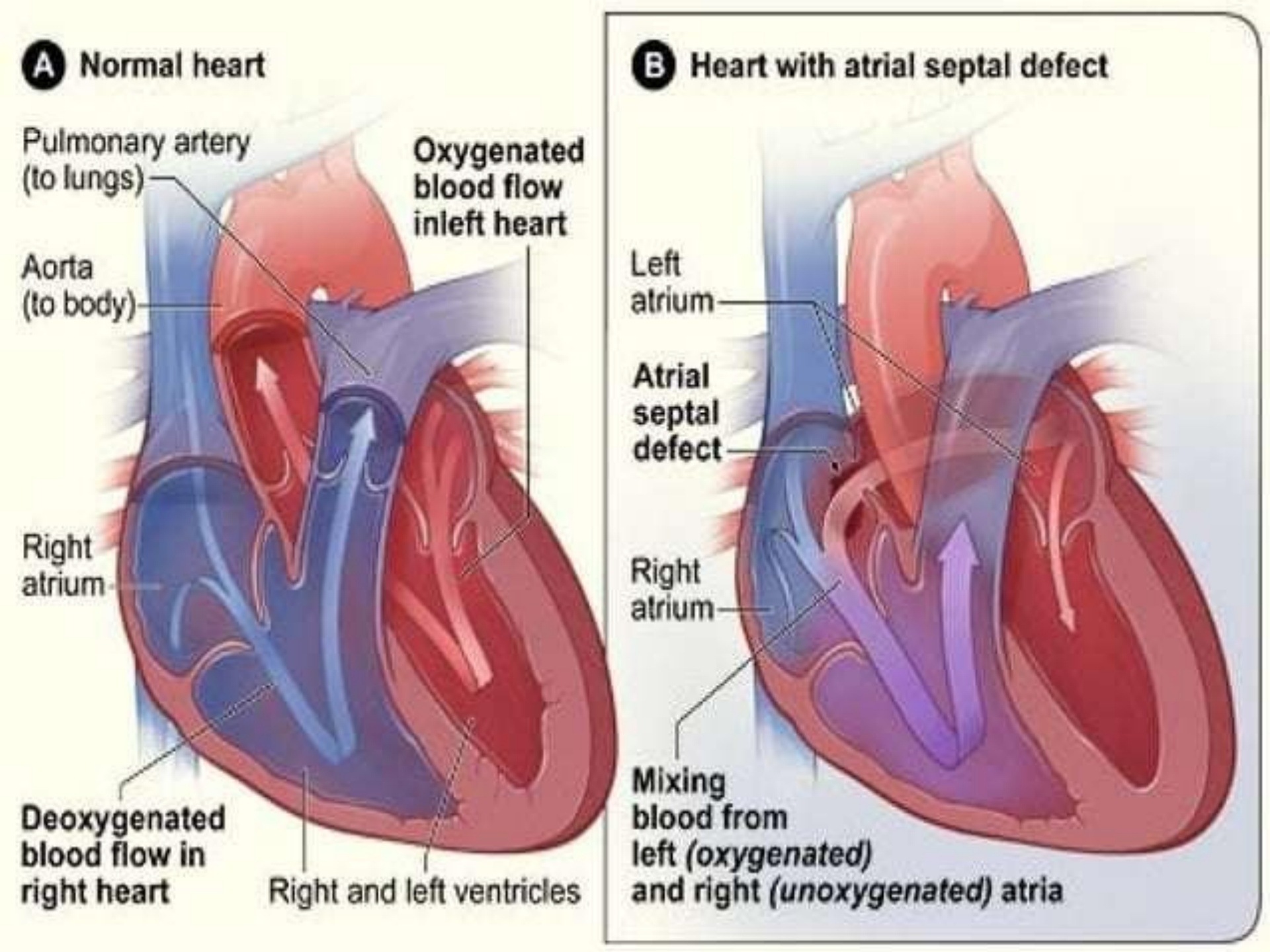
Definition:

ASD is a defect in the septum between the atria that allows shunting of blood from the left to right atrium.

Incidence:

1. Females >Males (3:1)
2. Occurs 10% of total CHD.




A Normal heart
Pulmonary artery (to lungs)
Oxygenated blood flow inleft heart
Aorta (to body)
Right atrium
Deoxygenated blood flow in right heart
Right and left ventricles
B Heart with atrial septal defect
Left atrium
Atrial septal defect
Right atrium
Mixing blood from left (oxygenated) and right (unoxygenated) atria

Types of atrial septal defect:

1.Ostium primum (ASD):

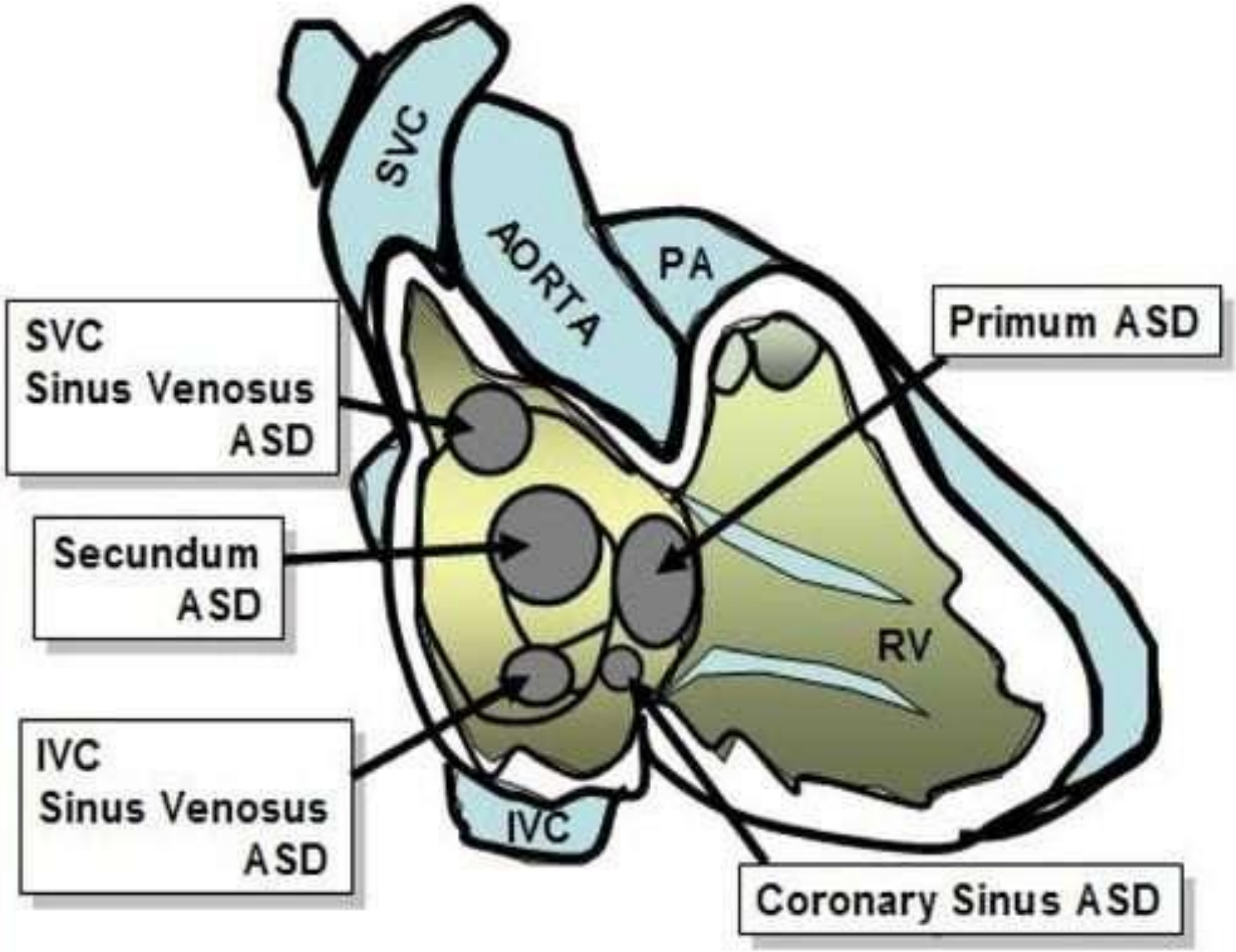
Opening at lower end of septum may be associated with mitral valve abnormalities. It accounts about 20%

2.Ostium Secundum:

Opening near centre of septum. It accounts about 70%.

3.Sinus venous defect:

Opening near junction of superior venacava & RA may be associated with partial anomalous pulmonary venous connection. It accounts about 5-10%


SVC
AORTA
PA
SVC
Sinus Venosus
ASD
Secundum
ASD
IVC
Sinus Venosus
ASD
Primum ASD
RV
IVC
Coronary Sinus ASD

Anatomy:

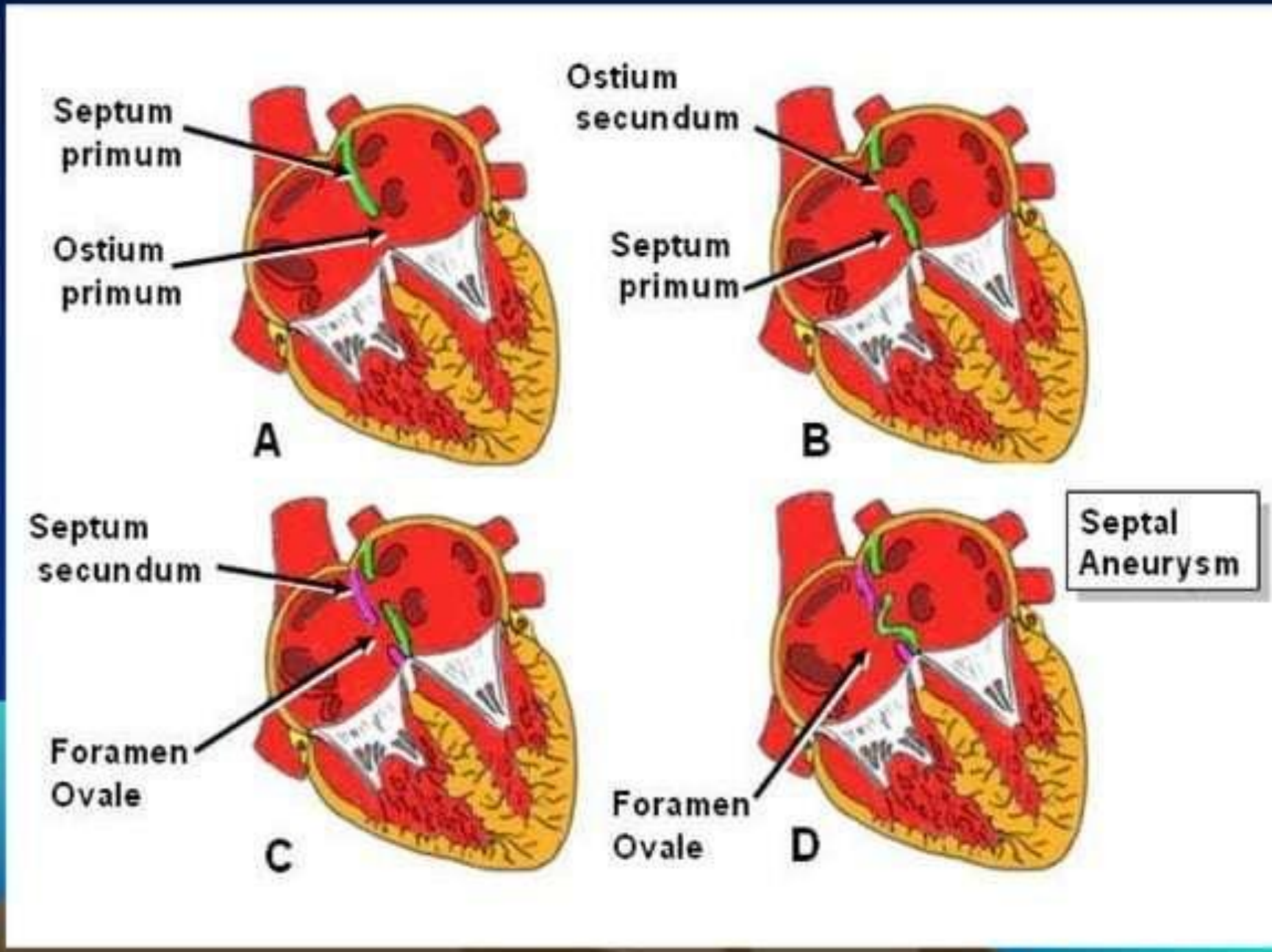
1.A patent foramen ovale is not an ASD But it is the normal which remains patent for months. Afterwards it is occluded by a flab valve. For any reason it can open to allow a shunt from right to left artrium.It known as ASD.

Location of the types of ASD:

Ostium primum –Lower part of the atrial septum.

Ostium secondem-Region of fossa ovalis.

Sinus venous defect-Upper part of the septum& pulmonary veins.


Septum primum
Ostium primum
A
Ostium secundum
Septum primum
B
Septum secundum
Foramen Ovale
C
Foramen Ovale
D
Septal Aneurysm

Pathophysiology & Haemodynamics:

Patent foramen ovale (Fails to close)

↓

Blood shunted from LV to RV

↓

Increase burden on the right side of the Heart

↓

Increase pulmonary blood flow

↓

If it is @ with pulmonarystenosis

↓

Increase pressure in RV

↓

Eisenmenger's complex

Biventricular Hypertrophy

It results cyanosis, cardiac failure, Right atrial \ventricular enlargement.

Clinical manifestation:

1.Congestive heart failure.

2.Murmur

3.Atrial dysrhymias.

4.ASD child will appear

-Thin

-Undernourished

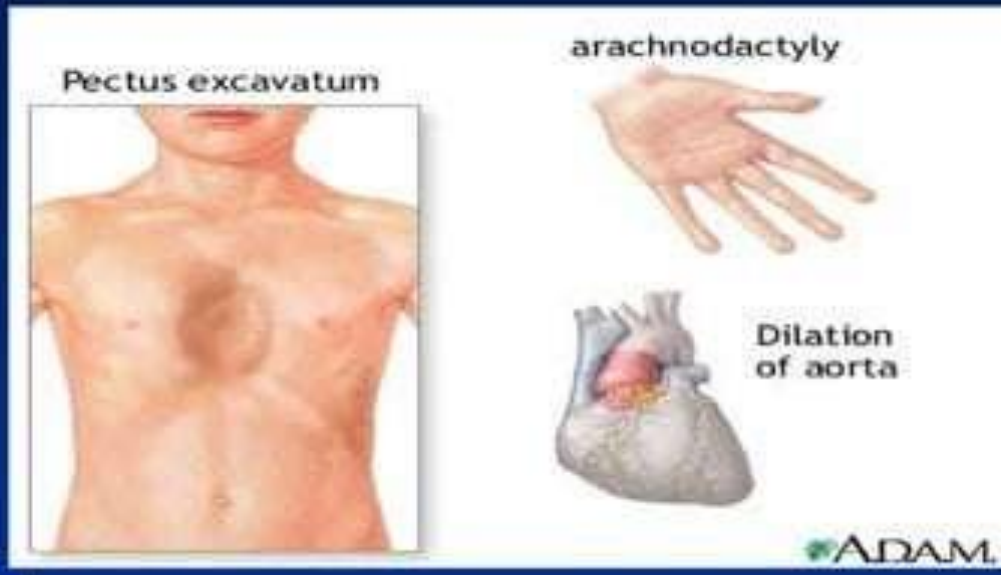
- Arachnodactyl

5.Marfan's syndrome:

- High arched palate

-Laxity of ligaments

-Hypermobility of joints



6.Lutembacher's syndrome:

Diastolic murmur at the apex with or with out mitral stenosis.

7.Protrusion of left chest along with a slender build.

8.Frequent episodes of pulmonary inflammatory disease.

9.Ostium primum:

Systolic murmur will be loud ,harsh &long, high pitch, loudest at the apex.

Investigation:

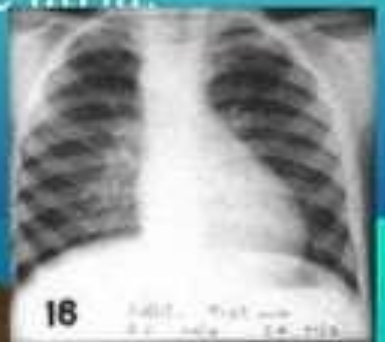
X-ray :Shows heart enlargement, PA enlargement.

Electrocardiogram : Right ventricular hypertrophy.

Cardiac catheterization :Denotes the left to right shunt.

Angiocardiography : Reveals opacification of both the atria.

Echocardiography :Right ventricular over load.



Management:

Surgical treatment:

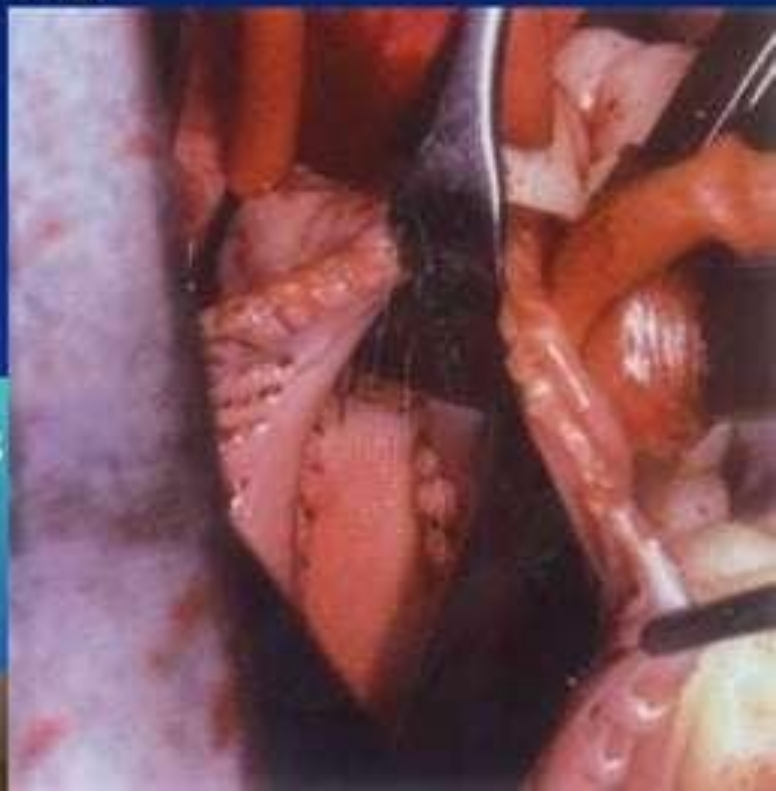
1. Dacron patch closure of moderate to large defects.
2. Open repair with cardiopulmonary bypass is usually performed before school age.
3. ASD I require : Replacement of mitral valve.
4. ASD II require :Closed using prosthetic devices during cardiac catheterization.
5. Sinous venous defect: Patch placement.

Prognosis:

Very low operative mortality less than 1%

Complication:

1. Pulmonary hypertension
2. Congenital mitral valvulitis
3. Death due to pulmonary infections &cardiac decompensation



5.VENTRICULAR SEPTAL DEFECT

It is the most common congenital cardiac anomaly. This frequently occurs with both the cyanotic types of heart disease like Fallot's tetralogy and acyanotic varities like COA,ASD,PS,AS.

Definition:

VSD is the abnormal opening between the right &left ventricle.

Incidence:

1.20 -25% of all cardiac lesions

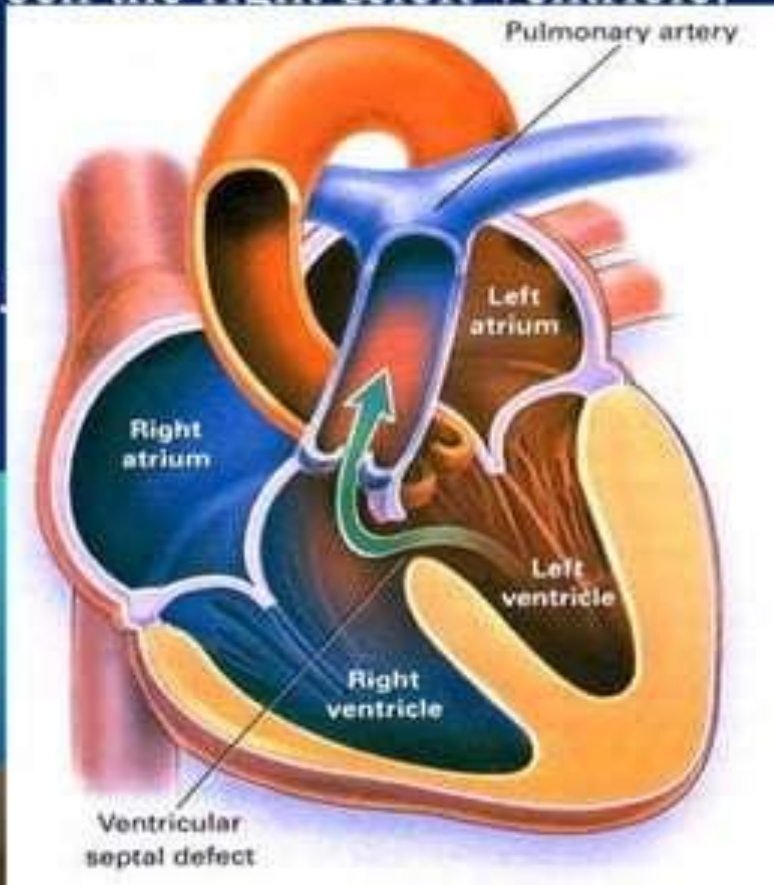
2.More common in premature babies.

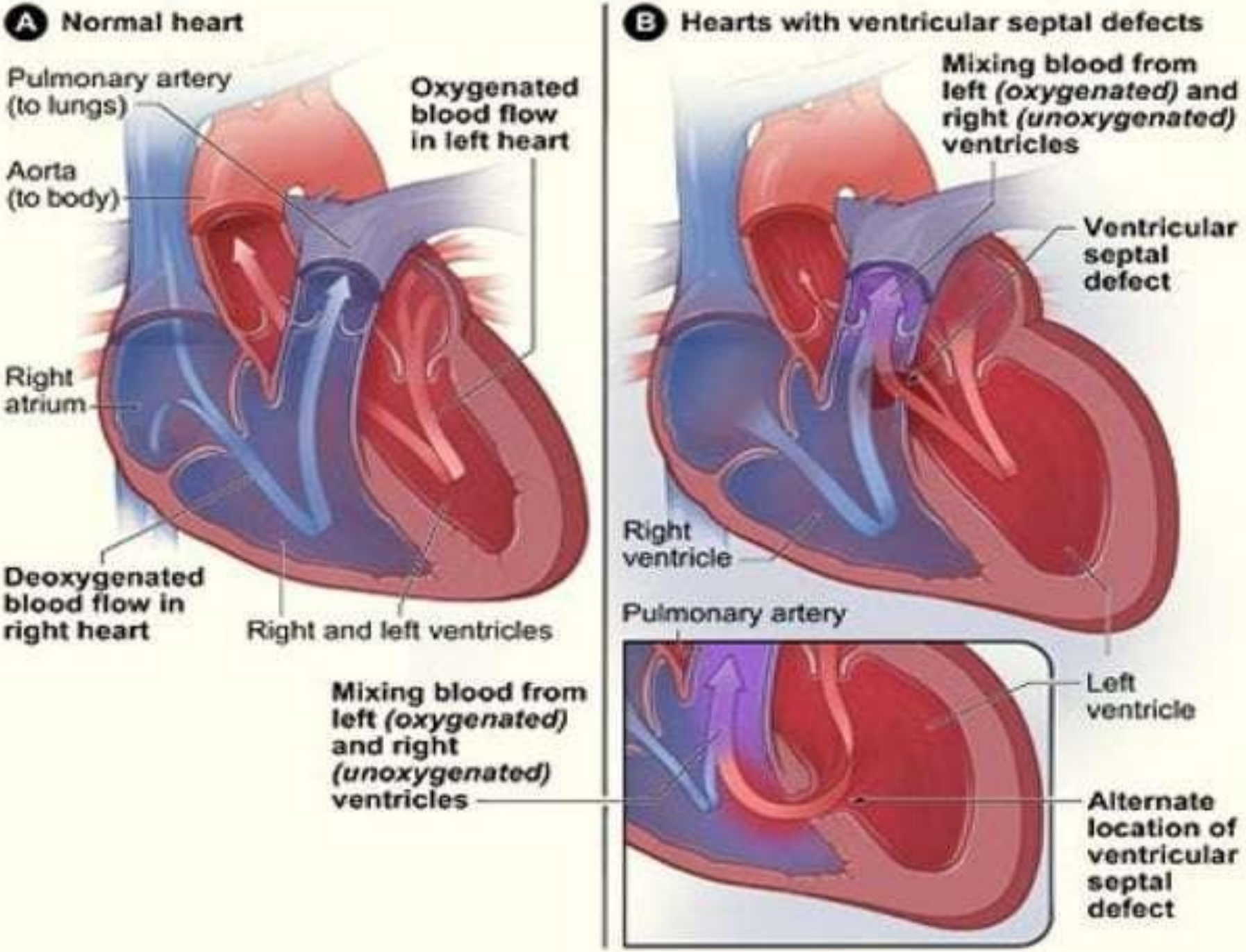
3.Equal Male :Female ratio.

4.VSD is most common CHD in

Edward syndrome

Down syndrome




A Normal heart
Pulmonary artery (to lungs)
Oxygenated blood flow in left heart
Aorta (to body)
Right atrium
Deoxygenated blood flow in right heart
Right and left ventricles
Mixing blood from left (oxygenated) and right (unoxygenated) ventricles
B Hearts with ventricular septal defects
Mixing blood from left (oxygenated) and right (unoxygenated) ventricles
Ventricular septal defect
Right ventricle
Pulmonary artery
Left ventricle
Alternate location of ventricular septal defect

Types of VSD:

1.PERIMEMRANOUS VSD:

a) Defect in the membranous septum is called as high or memraneous VSD

b) It accounts for 70 -80% of all VSD

c) It frequently @ with other defects like COA,PDA

2. MUSCULAR VSD:

a)The defect present at interventricular septum of the muscle portion.

b) It is called as low or muscular VSD.

c) Accounts about 10%

d) It can be single or multiple.

e)Occasionally entire ventricular septum may be absent resulting single ventricle.

3.Inlet(Inflow) VSD:

1.It is called canal VSD.

2.Because it may form a part of AV canal.

3.It is found in 5 to 8% of all VSD.

4.Outlet(Subpulmonic) VSD:

1.It is called subarterial VSD

2.More common in south east Asian population-Japan

3.Accounts about 5 to 8%

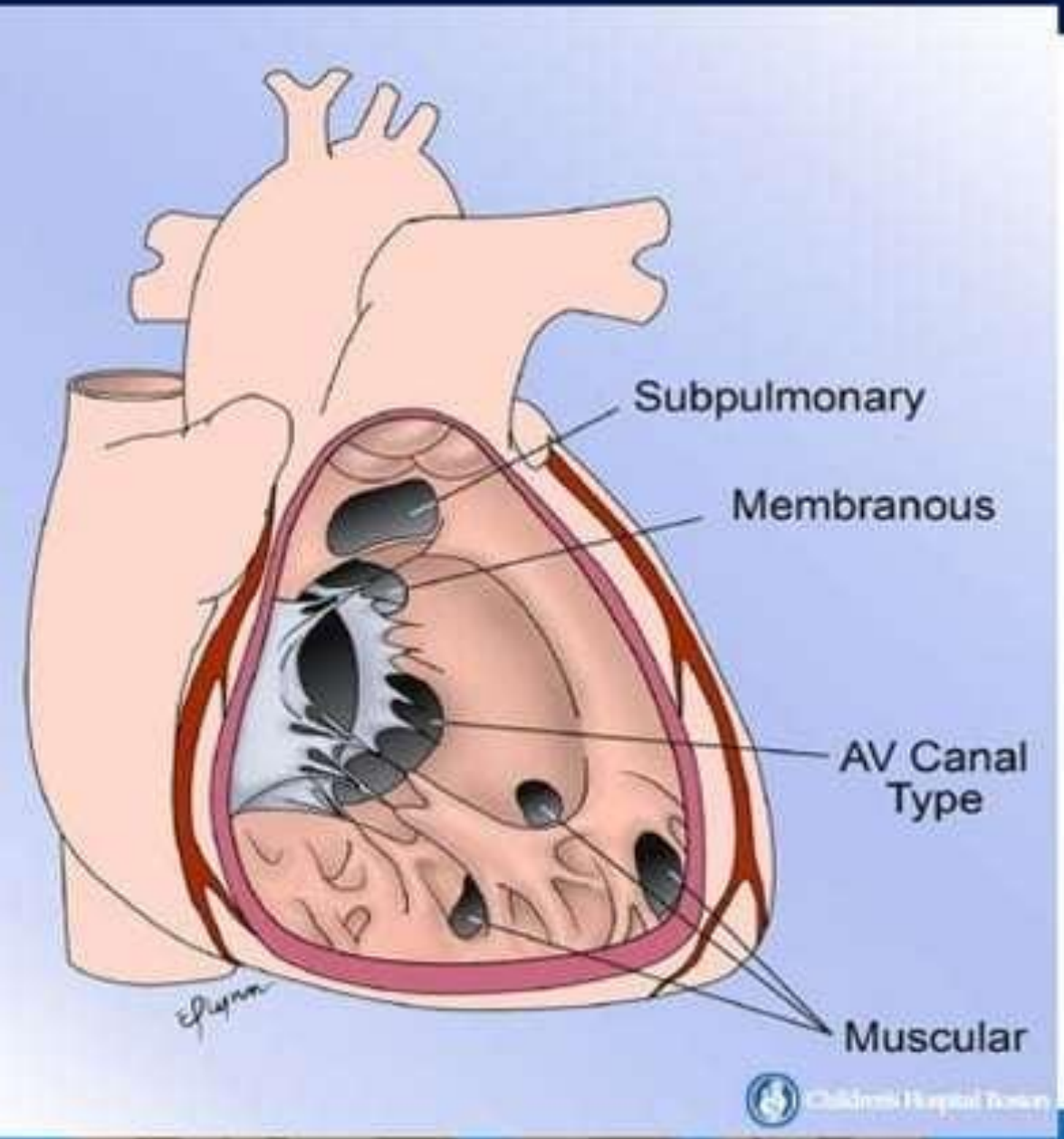
4. Aortic valve can prolapse into this VSD

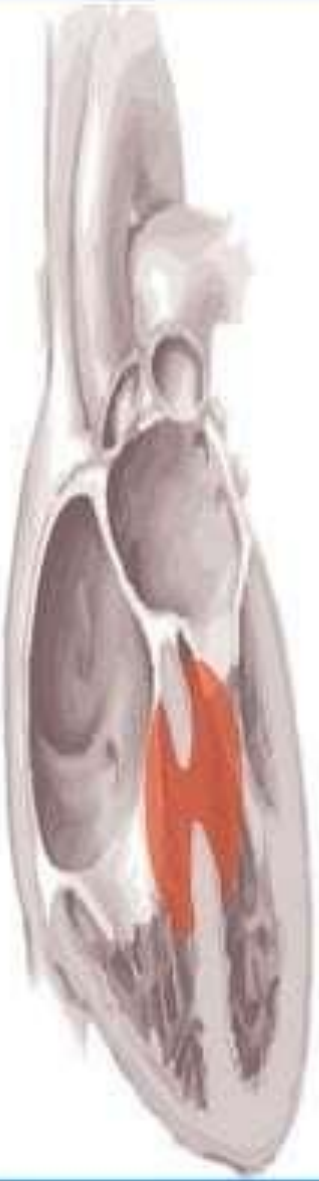
5.It causing aortic regurgitation.

1.Small VSD : Whendefect is about < 5mm

2.Moderate VSD : 5 to 10 mm

3.Large VSD : > 10mm


Subpulmonary
Membranous
AV Canal Type
Muscular



Description about VSD:

1. Many VSD 20-60% are though to close spontaneously.

2.It occur during I st yr of life in children having small or moderate

3.Left to right shunt develops in VSD.

Pathophysiology:

Congenital causes

⬇

Abnormal opening between the RV &LV

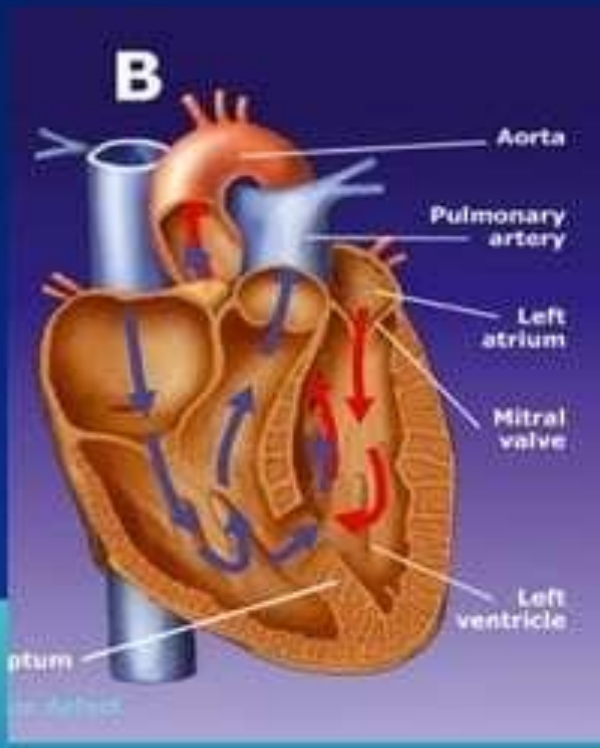
⬇

Pressure in the LV is higher than RV

⬇

Blood is shunted from left to right ventricle

⬇



Increase blood flow to the PA

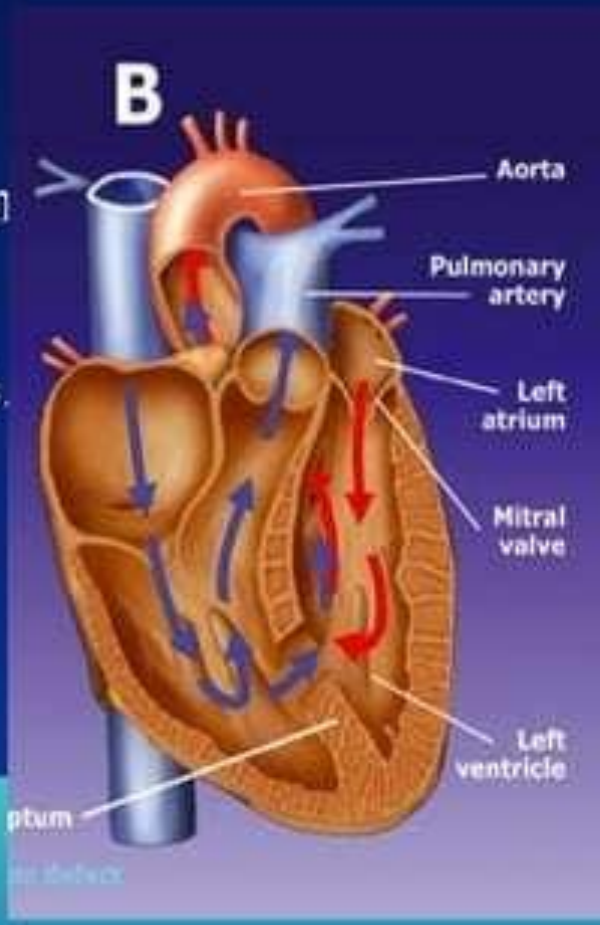
Increase blood flow to the lungs.

Increase pulmonary vascular resistan

Increase pressure in right ventricle

Right ventricular hypertrophy

Eisenmenger syndrome



Haemodynamics of VSD is depend upon the size of the defect:

Small defect:

Found in muscular portion

↓

Effect is slight

↓

Small amount of O2 passess from LV to RV.

Large defect:

Found in membranous portion of the septum

↓

Pulmonary hypertention

↓

Greater amount of oxygenated blood passess from RV to LV

3.Extreme defect:

In extreme defect there may be only one ventricle.

Clinical manifestation: Large defects:

1.Harsh,loud,pansystolic murmur

2.Breathlessness

3.Difficult in feeding

4.Failure to thrive.

5.Congestive cardiac failure

6.Peripheral pulse is small because of poor systemic bloodflow

7.Tachypnea

8.Slowphysical development

9.Frequent pulmonary infection.

10.Cardiac enlargement

Small defect::

1.Mostly asymptomatic

2.Pansystolic murmur.

3.Bacterial endocarditis

4.Pulmonary vascular obstructive disease

Investigation:

Chest X ray- Cardiomagaly & RV,LV enlargement.

Electrocardiogram: It shows right axis deviation & notched R waves are present.

Echocardiography : Shows size & Haemodynamics & associated lesions.

Angiocardiography: Shows level of shunt.

Management of VSD:

1.GENERAL:

a) Treat iron deficiency if present

b) Ineffective endocarditis prophylaxis

c) Treat chest infection promptly

d) Follow up

2.TREATMENT OF CHF

3.SURGICAL TREATMENT:

Indication: Uncontrolled CHF

A) Small Defect : Conservative treatment

Large Defect : Open heart surgery\Cardiopulmonary bypass.

B) Septal defect are patched up by

a)Prosthetic dacron

b)Direct suture

C).Pulmonary artery banding:

Placing band around the main PA to decrease PBF.

D) Complete repair by:

Small defect : Purse string approach.

Large defect : Knitted Dacron patch seen over opening

Prognosis:

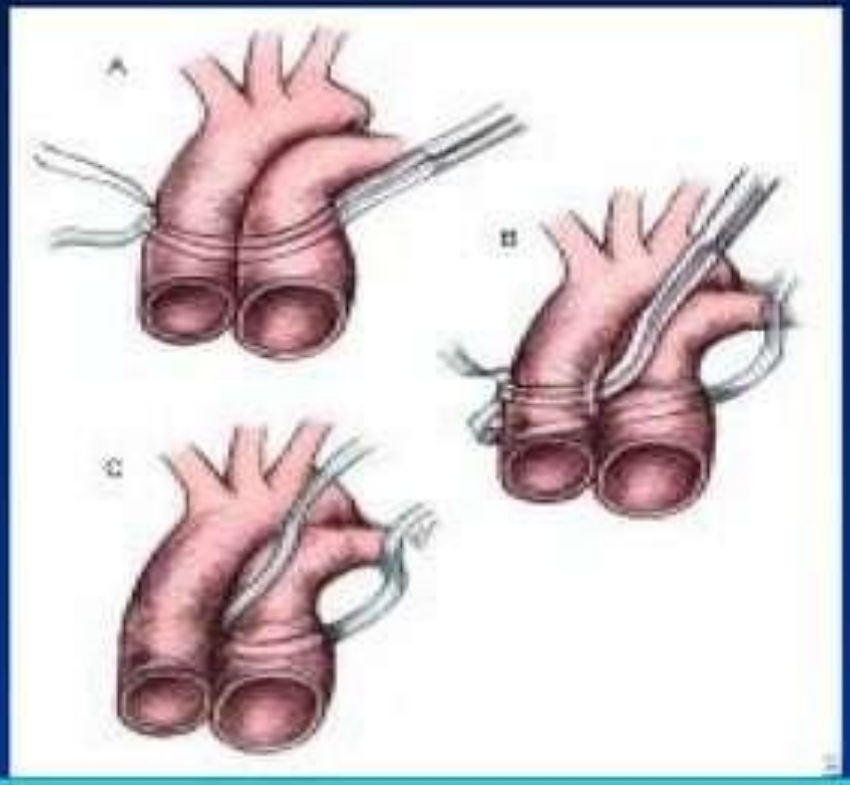
1.Membranous defect : Low mortality < 5%

2.Multiple muscular defects: High mortality >20%

Complication:

"THE GOOD"	"THE BAD"
1.Spontaneous closure	1.CHF.
2.Reduction in size	2.PAH
	3.Eisenmengers syndrome
	4.Ineffective endocarditis
	5.Growth failure

PULMONARY ARTERY BANDING AND PURSE STRING APPROACH:



6.Recurring pneumonia

7.AR 3%

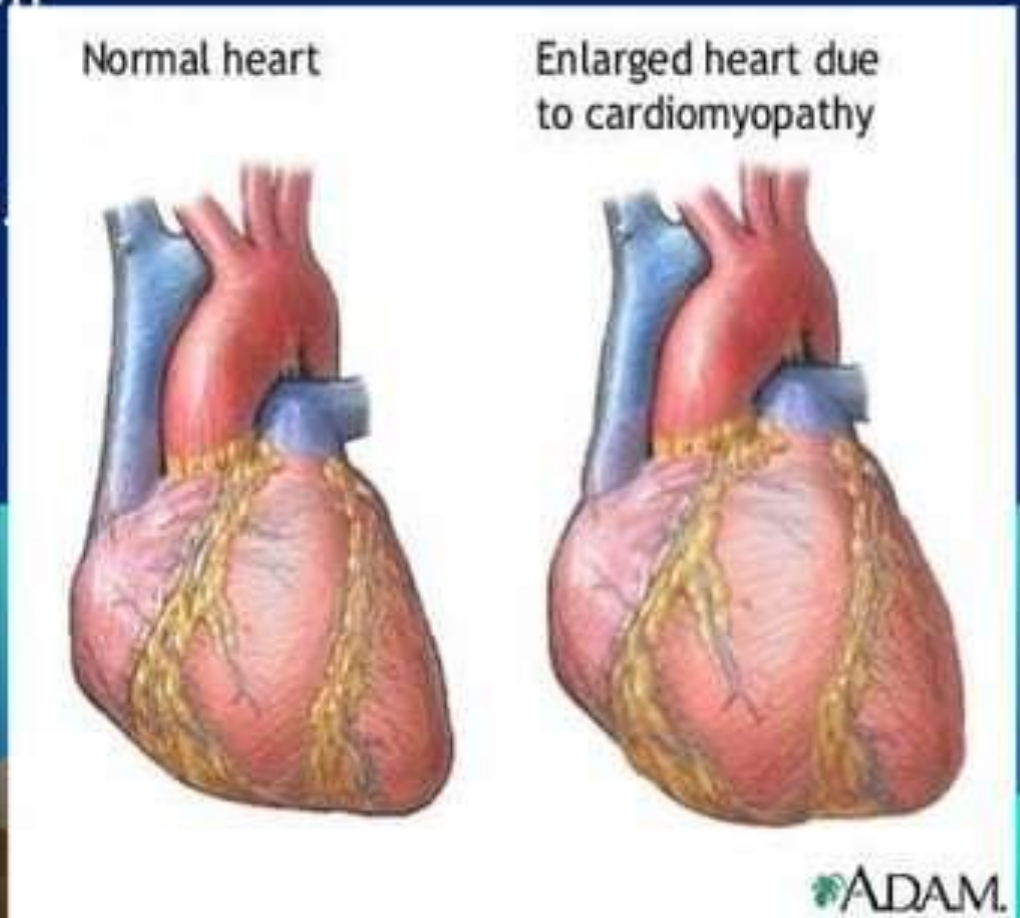
8.Acquired PS 3%

Other complication:

1.Pulmonary hypertension

2.CCF

3.Ineffective endocarditis



6.PATENT DUCTUS ARTERIOSUS.

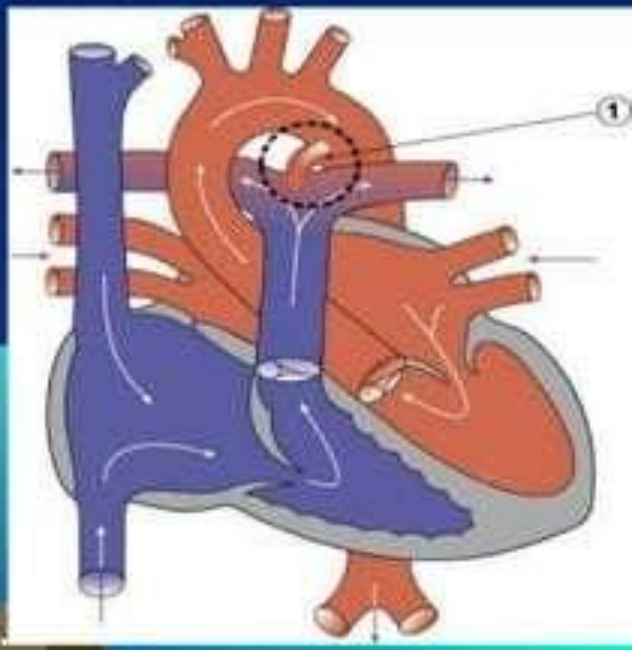
PDA is the third most common CHD in children

Definition:

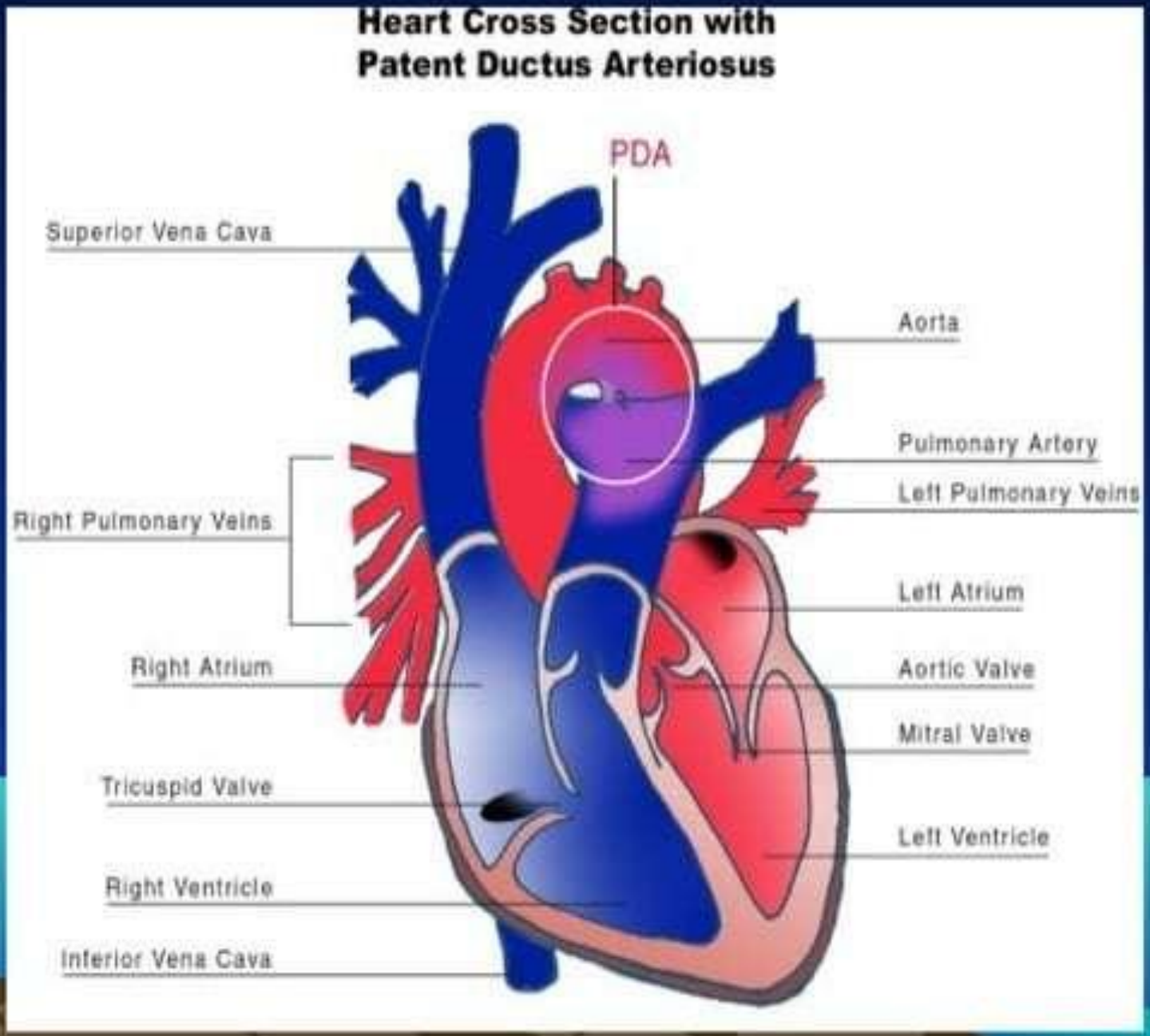
PDA is the continuing patency of the ductus arteriosus,a communication between the PA & ascending aorta.

Incidence:

1. Most common in premature infants
2. Less often in preterm infants
3. Occurs with other cardiac lesions
4. Ocurs about 7 -10 %
5. Female : male (2: 1)
6. Most common in klinefelters syndrome.



Heart Cross Section with Patent Ductus Arteriosus



Description:

1. DA - Artery connecting the aorta & PA
2. PDA – Is the failure of the fetal ductus arteriosus to close within the I st weeks of life.
3. Continued patency of this vessel allows blood flow from the higher pressure aorta to lower pressure PA.

Types of PDA:

1. Small
2. Moderate

1. SMALL PDA:

a) The opening usually less than 4 mm size at aortic end
b)Usually asymptomatic
c)Nogrowth failure
d)CHF
e)No murmur

2. Moderate:

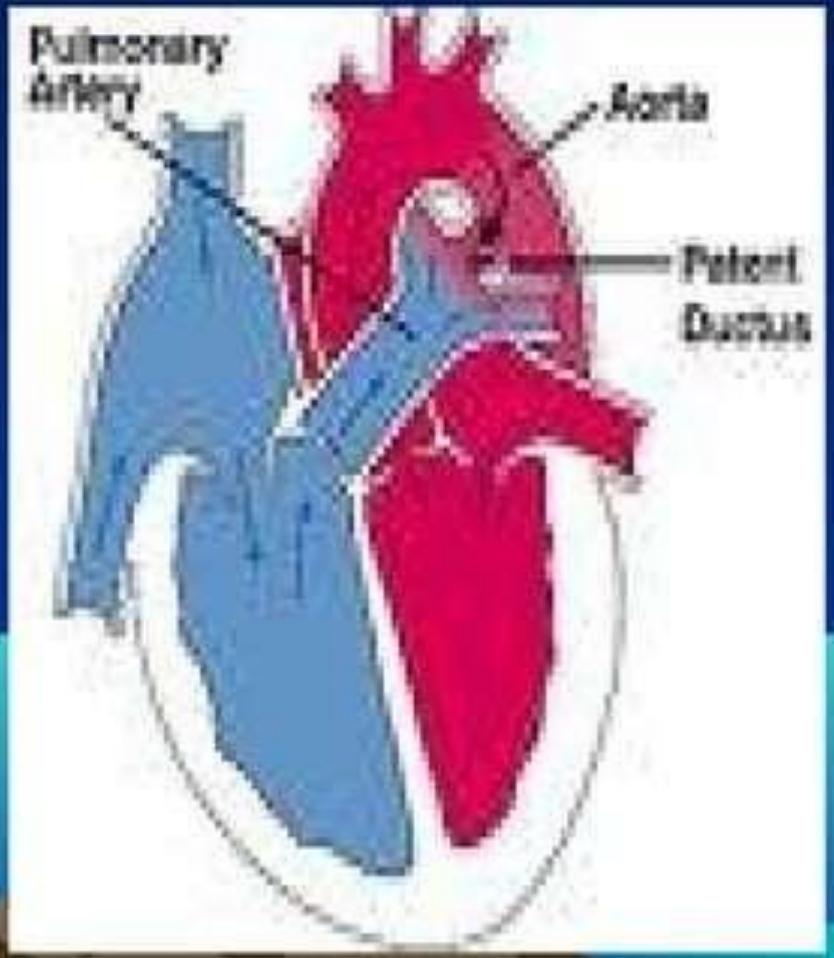
a) It can be symptomatic

b) Mild growth failure

c) The defect size is more than 4mm

d) Cardiomagaly

e) Murmur.



Embryology:

5th & 7th wk of gestation the aortic arch is formed

It form from the apex of the truncus arteriosus

Pulmonary arch gives a branch to develop lung

Right side of the lung

Left side of the lung

It becomes PA It disappears Left side PA DA

It serve as connection between PA & the aorta

As soon as the baby is born the ductus is functionally closed.

Anatomical closure occurs around 6th month of life

If it is remain for some reasons cause

Patent ductus arteriosus

Pathophysiology &Haemodynamics:

During fetal life

↓

DA connects PA to the aorta

↓

Blood reaches the descending aorta from PA to DA

↓

Respiration begins at birth

↓

Decrease pulmonary vascular resistance

↓

Pulmonary arterioles dilate when PBF is increase

↓

O2 level is increase

It cause the ductus to contract during 1 st 24 hrs to 72 hrs

It forms fibrous becoming ligamentum arteriosum

If this obliteration is not occur

Left to Right shunt

Blood flow from aorta to PA through PDA

Recirculation of oxygenated blood

Increase burden on left side of the heart

Increase pulmonary congestion

↓

Left arterial, ventricular enlargement

↓

Left ventricular hypertrophy.

Clinical manifestation:

1. Signs of CHF
2. Machinery like murmur
3. Widen pulse &bounding pulse
4. Bacterial endocarditis
5. Pulmonary vascular obstructive disease.
6. Dyspnea
7. Physical underdevelopment
8. Increased respiratory infections
9. Heart rate 150 b\mt

10.Gallop Rhythm (Due to rapid filling of the ventricle)

11.Cough

12.Heptospleenomagaly

Investigation:

1.X-ray : Left & Right ventricular enlargement

2.Electrocardigraph : Left ventricular hypertrophy.

3.Echocardiography : Size of PDA,

4.Cardiac catheterization : Reveals increase pressure in RV.

Management:

Medical:

1.Treatment for CHF

2.Inefective endocarditis

3.Iron supplementation

4.Indomethacin

Surgical management:

1. Ligation of the patent vessel via left thoracotomy
2. Visual assisted thoracoscopic surgery
3. Smaller ductus –Triple ligation
4. Larger PDA -Division &suture
5. Coil occlusion
6. Device closure-Amplatzer
7. Other modalities:
   a) Video assisted thoracoscopic ligation
   b) Video assisted thoracoscopic clipping

Preterm with PDA >10 days:

Indomethacin.-0.1 mg \kg 12 hr *2 doses

Ibuprofen –syrup -10mg\kg

Prognosis:

1.Average life expectancy 23-40 yrs.

2.Prognosis following surgery is excellent

Complication:

1. Sub acute bacterial endocarditis.

2.PH

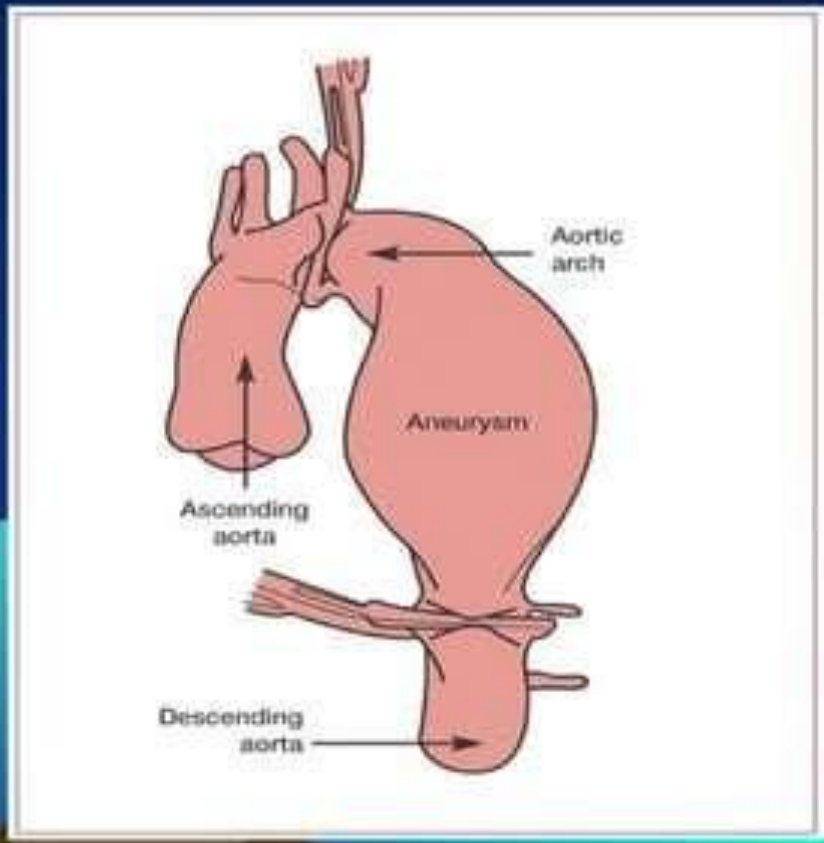
3.CCF

4.Bronchitis

5.Aneurysm

6.Rarely rupture of the greatly dilated ductus & PA

7.Reversal of shunt



NURSING MANAGEMENT

Nursing management of infant with acyanotic heart disease includes helping family members to adjust to the child's care &both preoperative &post operative care.

1. General nursing care:

a) Helping family members to adjust

b) During episodes \ Dyspnoea

c) Need for comfort & rest

e) Nutrional needs

f) Psychosocial needs

g) Continuing care

h) Family relationship

i) Financial support

2.Preoperative care:

It includes

a) Pre operative assessment

b) Pre operative teaching

A)PRE OPERATIVE ASSESSMENT:

1.Admission history &physical examination

2.Pre operative studies

3.Baselines vital status.

4.Anthropometric measurement

5.Additional nursing observation.

B)PRE OPERATIVE TEACHING:

1. Introduction to environment.

2. Introduction to equipment

3.Introduction to postoperative procedures

3.Post operative care:

1. Transfer to ICU
2. Monitor vital signs.
3. Assist in restoring the optimal functioning of the
   - -Cardiopulmonary
   - -Gastro intestinal
   - -Renal
   - -CNS.

NURSING DIAGNOSIS

1.Inefective breathing pattern related to decreased PBF

Nursing intervention:

1. Assess the general condition.
2. Check breathing pattern
3. Poisoning & Head elevation
4. Avoid any constructing clothing
5. Administer humidified O2
6. Assess the respiratory rate
7. Assess O2 saturation
8. Provide calm & warm place
9. Provide comfort bed.

2.Decreased cardiac output related to structural defect.

Nursing intervention:

1. Assess cardiac function
2. Provide quite environment
3. Provide tender loving care
4. Provide appropriate play to reduce anxiety
5. Administer Digoxin as order
6. Observe signs of hypokalemia
7. Observe for signs of hypotension
8. Monitor electrolyte level
9. Observe cardiac monitoring carefully.

3.Imbalance nutritional status less than body requirement related to less food intake.

Nursing intervention:

1. Assess the child's nutrional status
2. Assess the child's Nausea,vomiting,inability to eat
3. Check the weight daily
4. Provide small amount of formula &food frequently
5. Feed slowly &Buddle to prevent distention of stomach
6. Provide low fat diet
7. Provide fruits &fiber rich diet

4.Activity intolerance related to imbalance between O2 supply & demand

Nursing intervention:

1. Assess the child's response to activity
2. Maintain neutral thermal environment
3. Respond promptly ti crying
4. Provide calm &comfortable environment
5. Feed small volume at frequent intervals
6. Provide divertional activity
7. Change the position of the child every 2 hours
8. Teach the parents ,about child's activity

5.Risk for infection related to reduced body defences

Nursing intervention:

1. Assess the child for any changes
2. Maintain aseptic environment
3. Use sterile equipment
4. Maintain good hand washing
5. Isolate child if nosocomal infection
6. Provide nutritional diet according to child's preference
7. Teach family about manifestation of illness
8. Maintain disposal method
9. Administer antibiotics

6. Risk for complication related to improper care or no early treatment

Nursing intervention:

1. Assess the condition of the child
2. Monitor vital signs
3. Response immediately for cry
4. Maintain aseptic technique
5. Administer O2 to prevent brain damage
6. Take early intervention
7. Frequent observation
8. Explain complication
9. Explain about early treatment

7.Altered family process related to illness or hospitalization

Nursing intervention:

1. Assess the current knowledge.
2. Assess the current scoping skills
3. Recognize parental concern
4. Explore family feelings &problems surrounding
5. Clarify the doubts
6. Provide support as needed
7. Provide information on resources available

8.Altered growth & development related to impaired blood supply to the brain

Nursing intervention:

1. Check development of the child
2. Check anthropometric maseaurement
3. Encourage learning of self care skills
4. Provide nutritional diet
5. Provide play therapy

9.Fear & Anxiety related to difficult breathing ,unfamiliar procedures

Nursing intervention:

1. Establish rapport with child & parents
2. Explain about the disease condition
3. Provide calm &quite environment
4. Explain unfamiliar procedure
5. Provide frequent attendance
6. Provide comfort
7. Instill confidence
8. Provide divertional activities

10.Knoledge deficit related to treatment and follow up care

Nursing intervention:

1. Assess the knowledge of mother
2. Allow the mother to ask doubts
3. Explain the procedures
4. Explain about medication
5. Explain about nutrition
6. Explain the importance of surgery & follow up care.

Summary :

Conclusion :

Assignment :

Bibliography :