

# VSD

## (Ventricular Septal Defect)

Kwang Ho Choi, M.D.

Department of Thoracic and Cardiovascular Surgery

Pusan National University Yangsan Hospital

# Definition and Prevalence

- Def. : a hole between the ventricles
  - isolated or with a variety of anomalies
- Prevalence
  - Isolated VSD at a rate of about 2 per 1000 live births
  - => Increased using TTE showed remarkable increase in Dx of muscular VSD

# Historical Perspectives

- 1879 : 1st description of clinical manifestations, Roger
- 1897 : noted presence of a large VSD with overriding aorta, Eisenmenger
- 1952 : Muller and Dammann : pulmonary artery banding
- 1954 : 1<sup>st</sup> operative repair, Lillehei -> cross circulation with an adult acting as pump oxygenator
- 1955 : 1<sup>st</sup> operative repair with CPB, Kirklin

# Anatomy



Richard & Stella Van Praagh



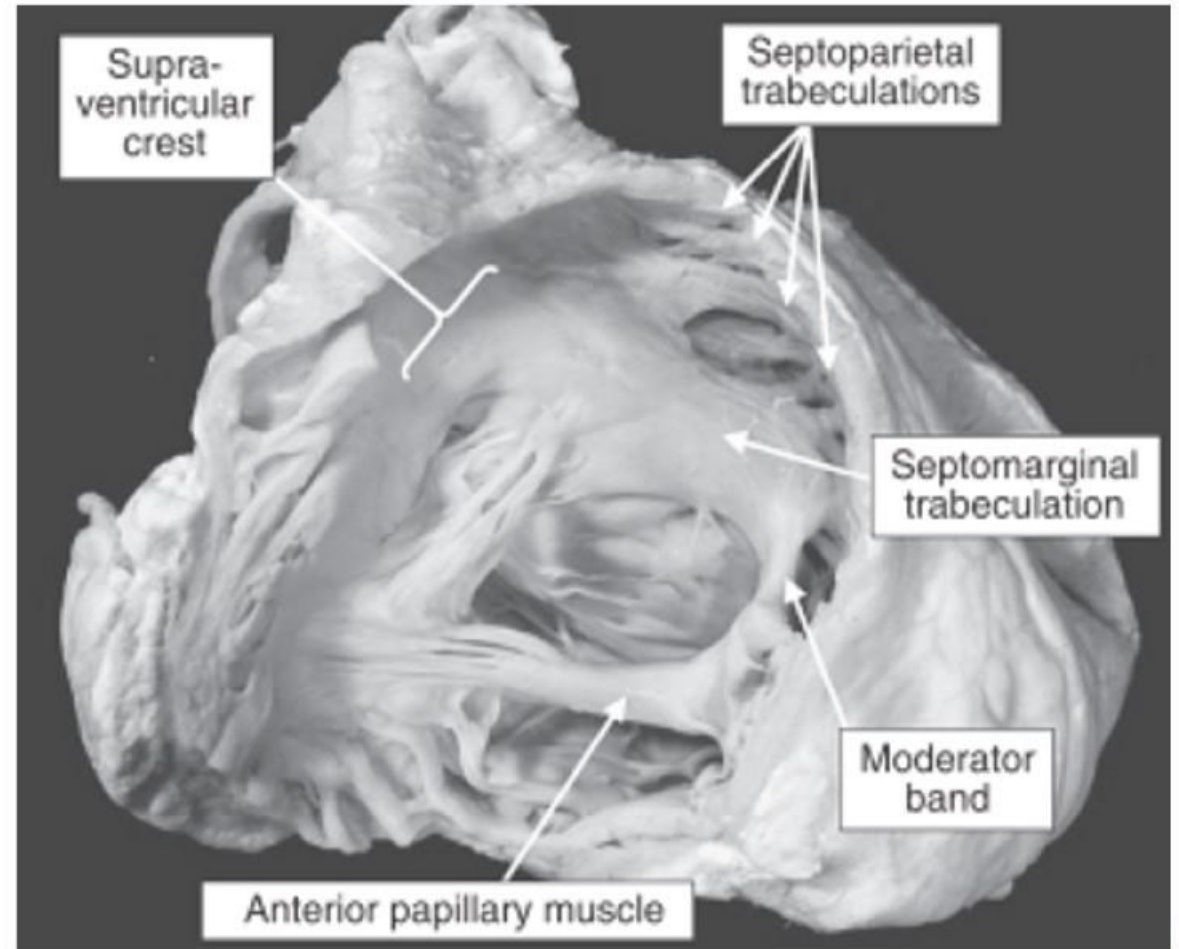
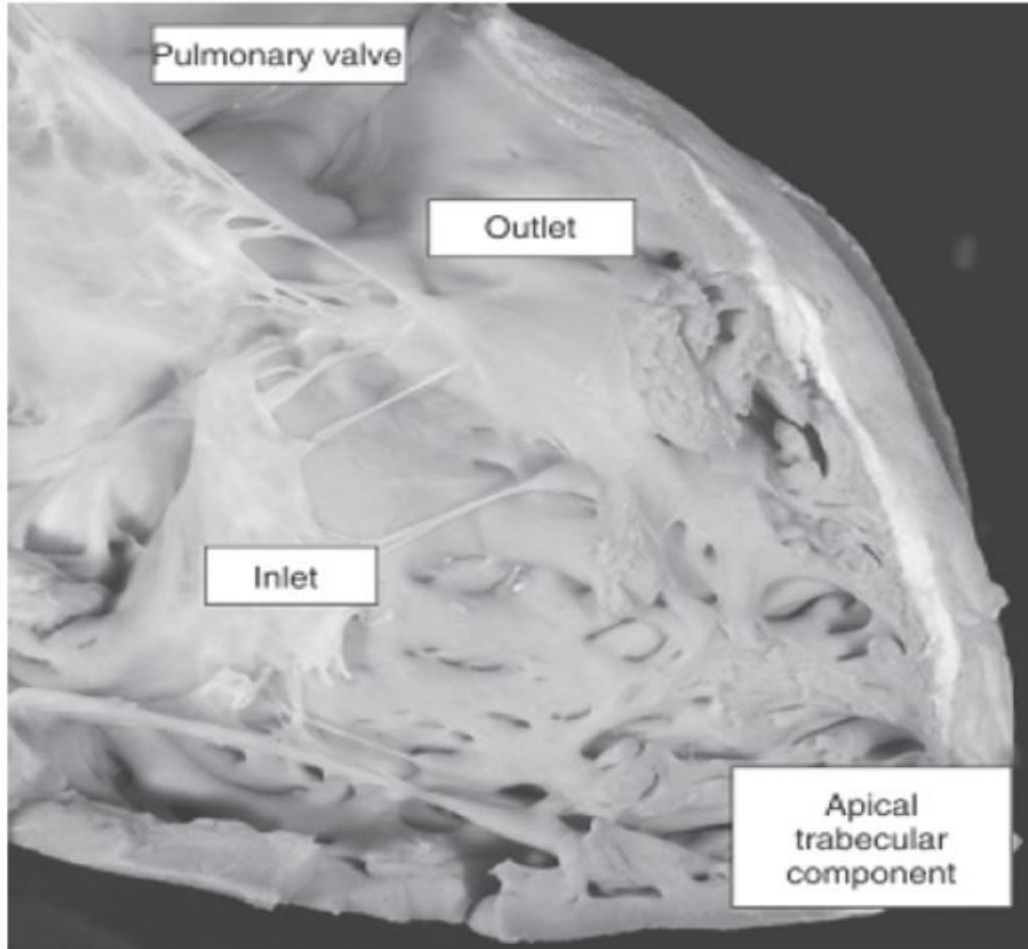
Robert & Christine Anderson



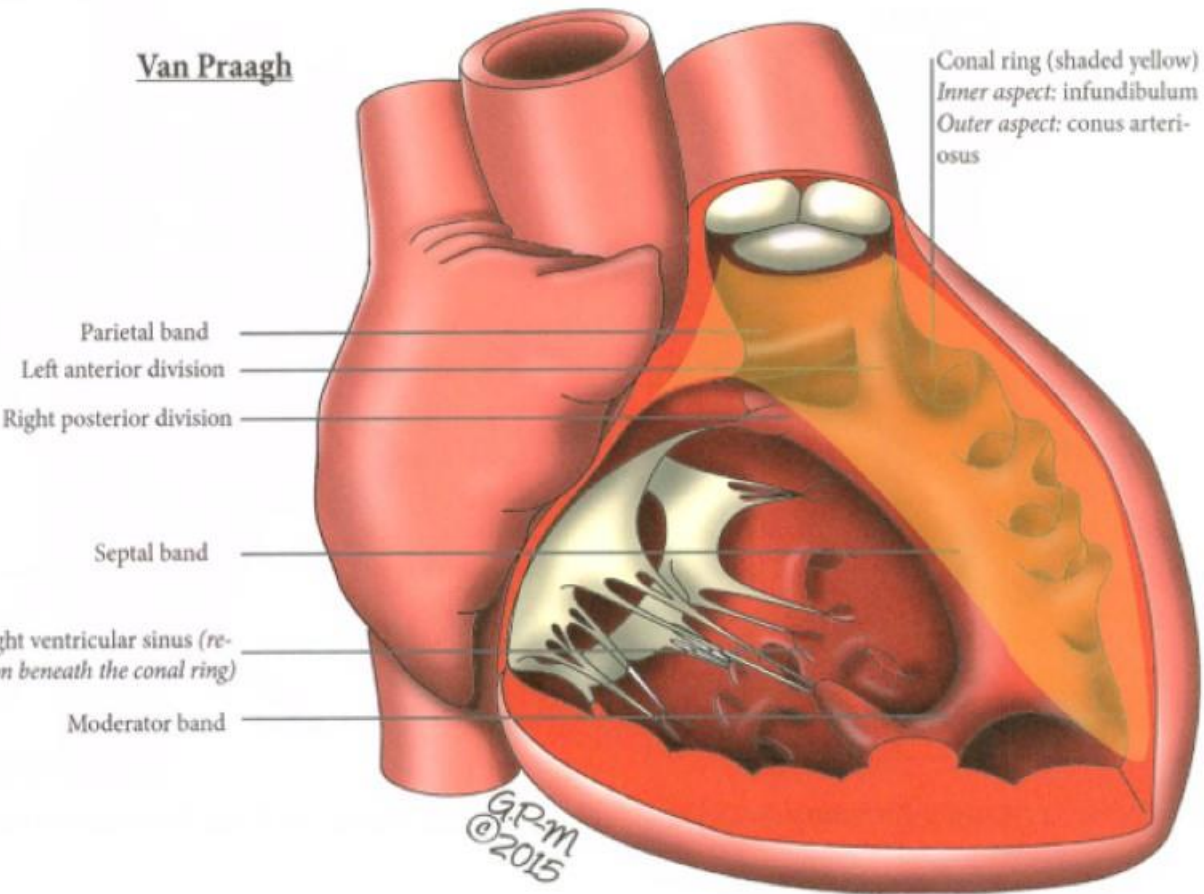
# Anatomy

- Right ventricle
- Tricuspid valve anatomy
- Conduction system

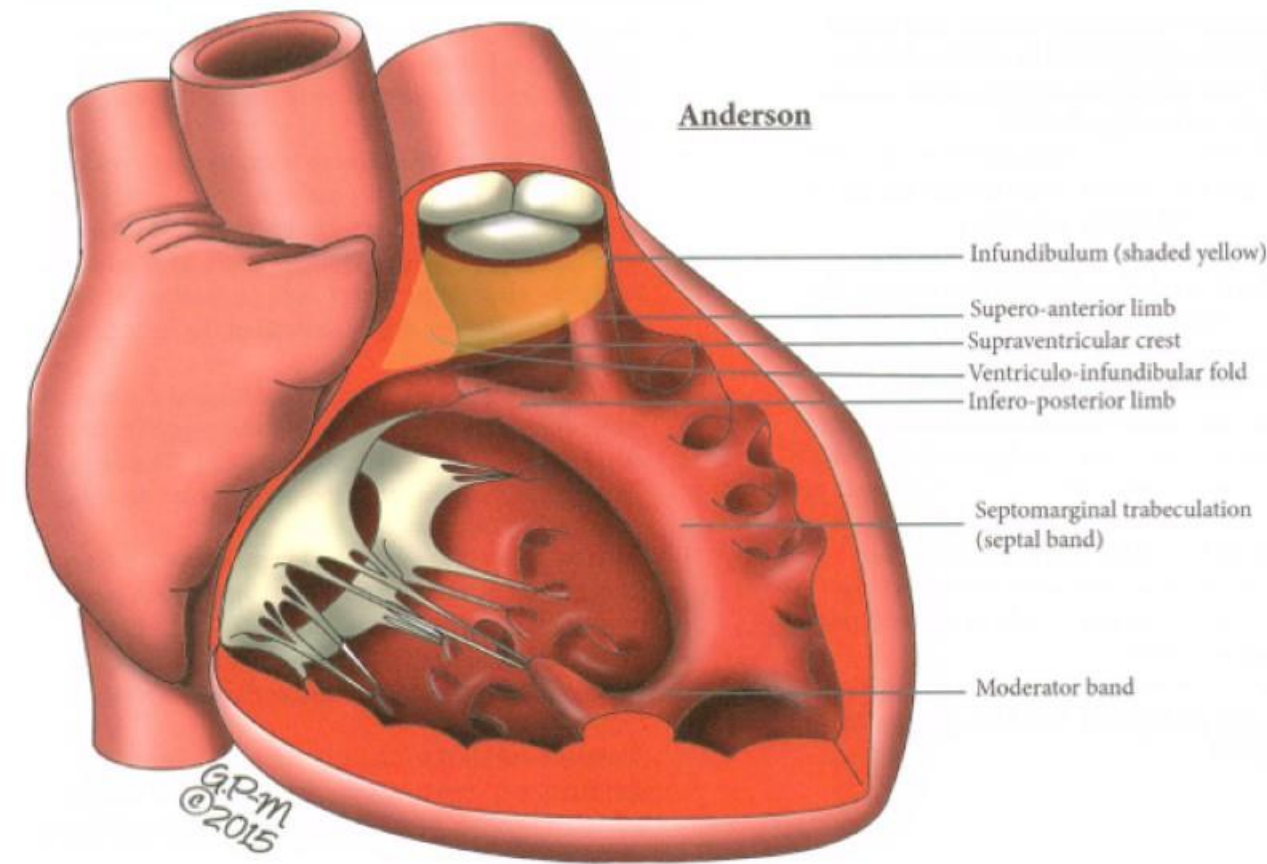
# Right ventricle



**Van Praagh**

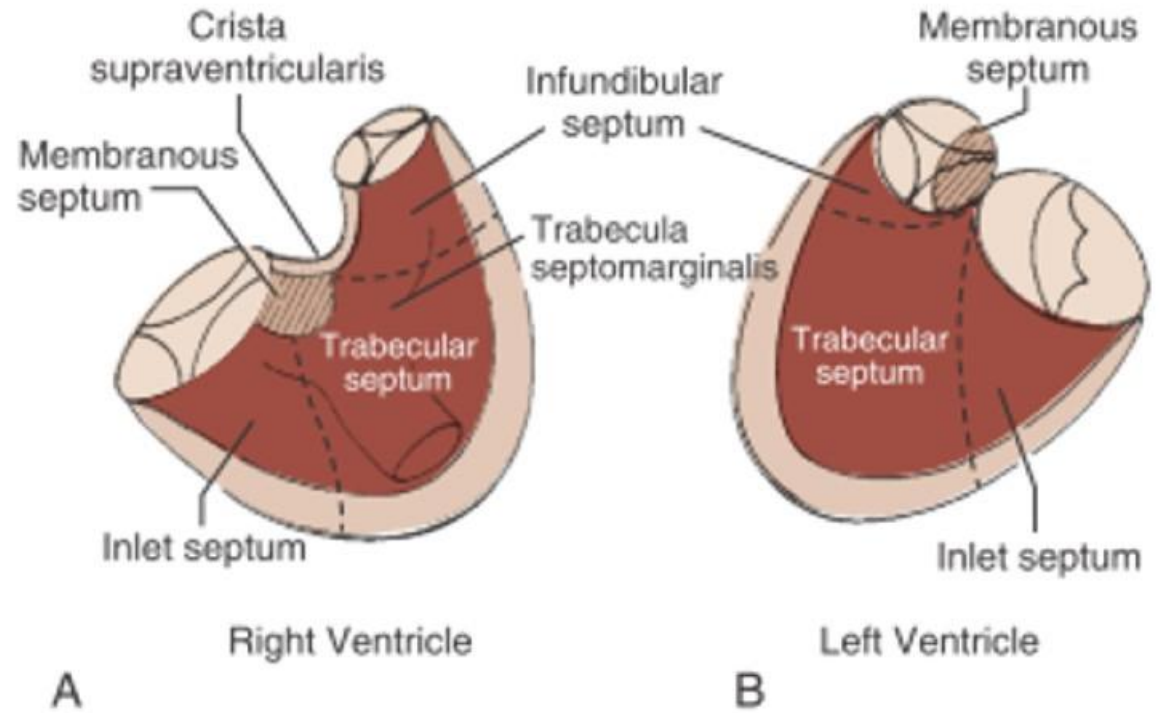


**Anderson**



# Right ventricular septal anatomy

- Membranous septum
- Inlet septum or atrioventricular canal
- Muscular septum
- Trabecula septomarginalis
- Conal septum or infundibular septum

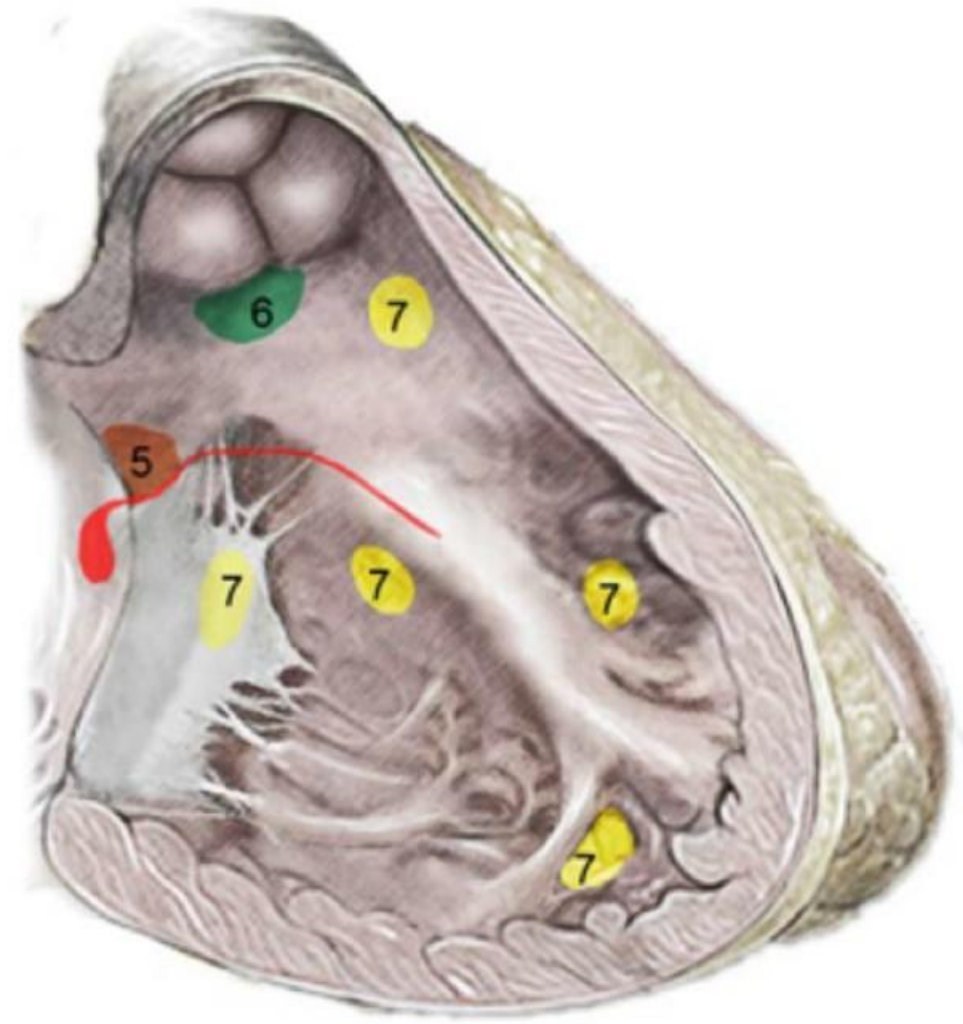






**A**

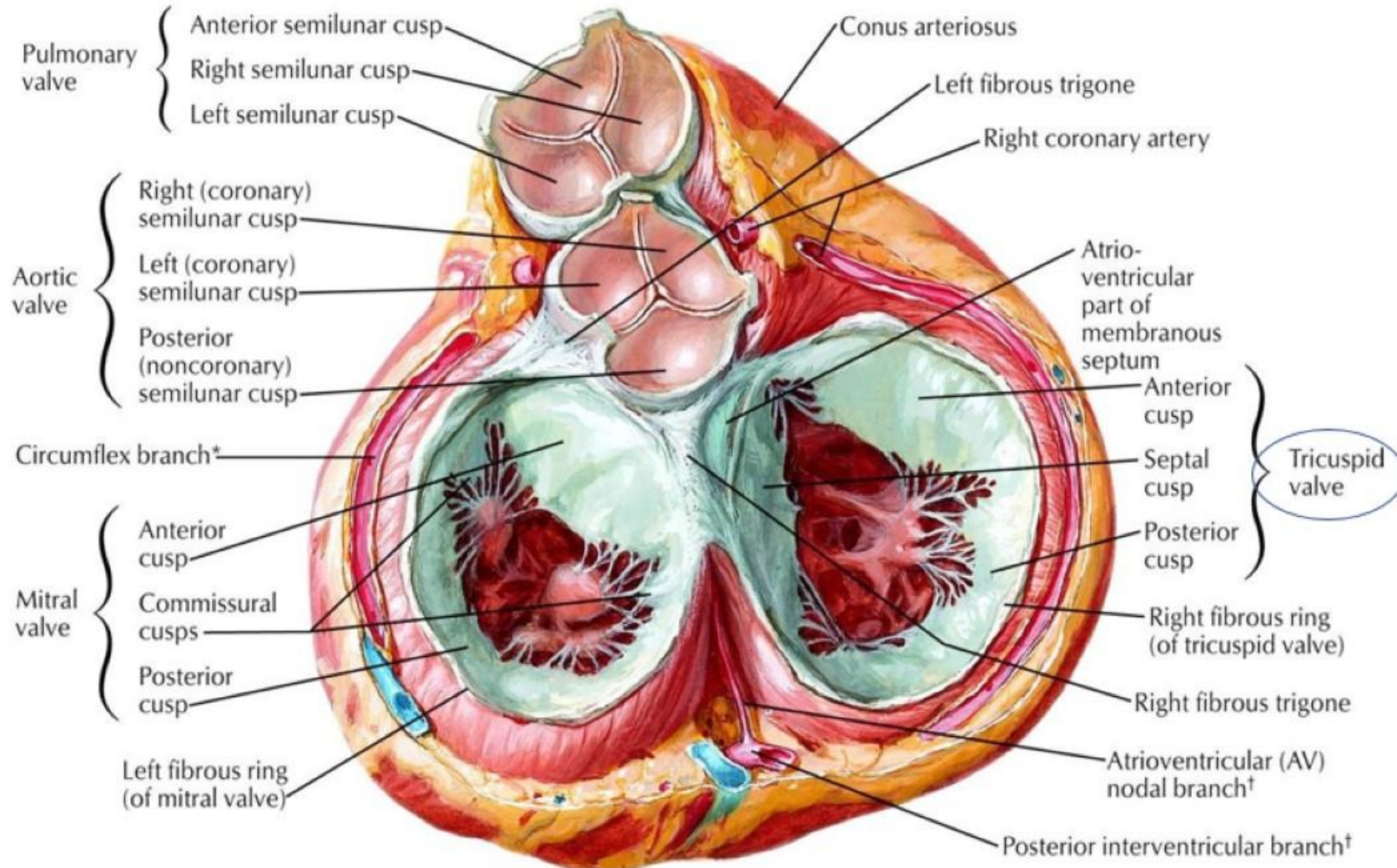
- 1. Membranous septum
- 2. Outlet
- 3. Trabecular
- 4. Inlet



**B**

- 5. Perimembranous defect
- 6. Doubly committed/juxtaarterial defect
- 7. Muscular defects

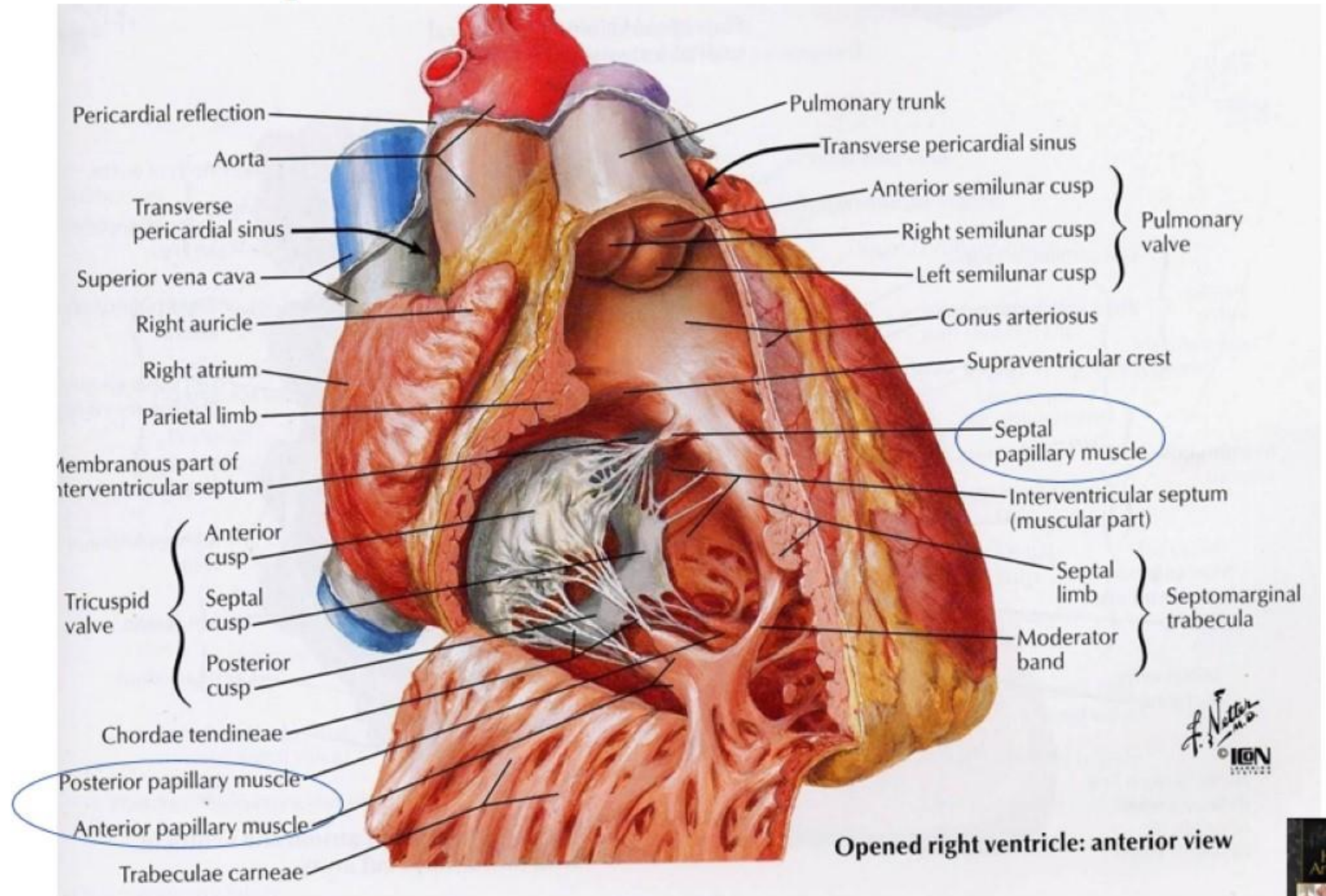
# Tricuspid valve



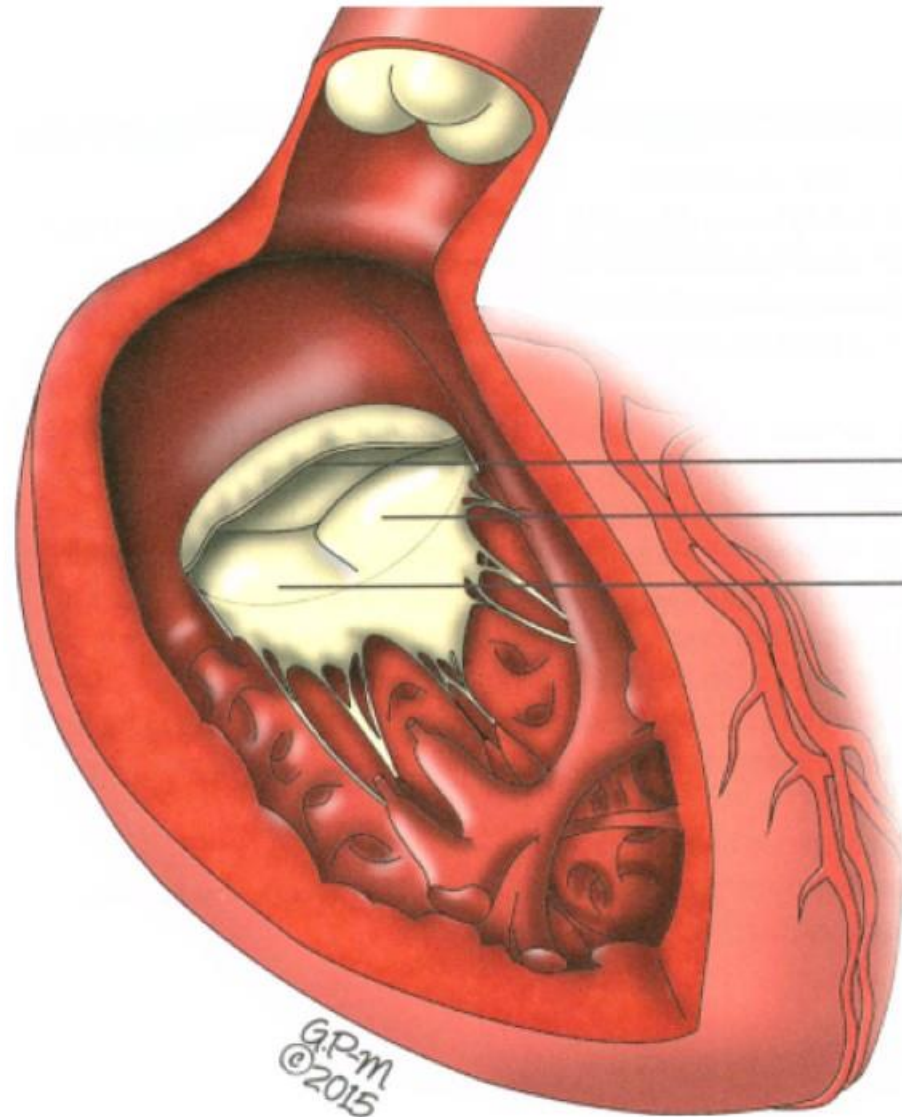
Heart in diastole:  
viewed from base with atria removed

# Tricuspid valve

- Papillary muscles



# Tricuspid valve anatomy



## Tricuspid Valve

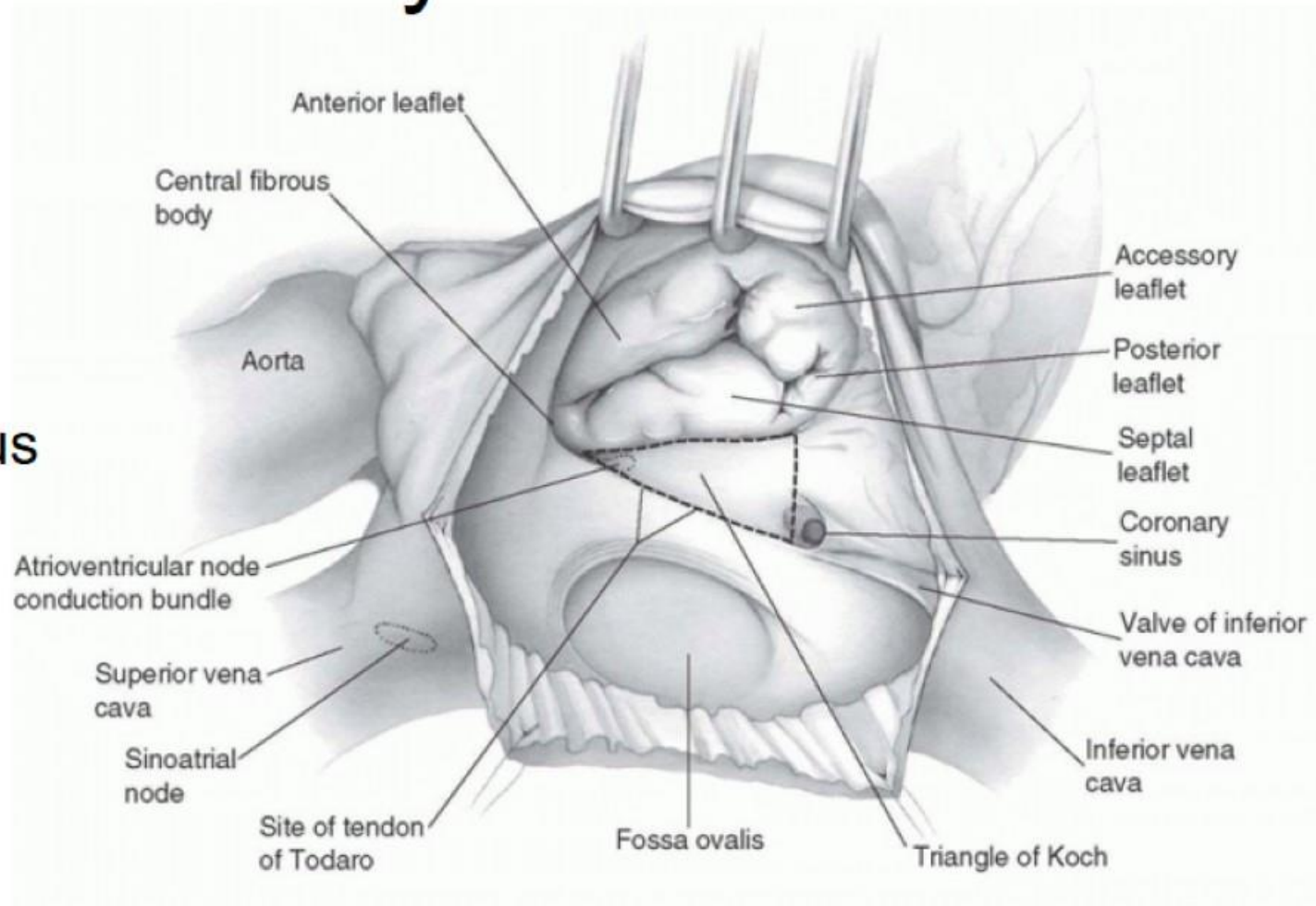
- VP: Anterior leaflet
- AN: Anterior-superior leaflet
- VP: Septal leaflet
- AN: Septal leaflet
- VP: Posterior leaflet
- AN: Inferior leaflet

G.P.M.  
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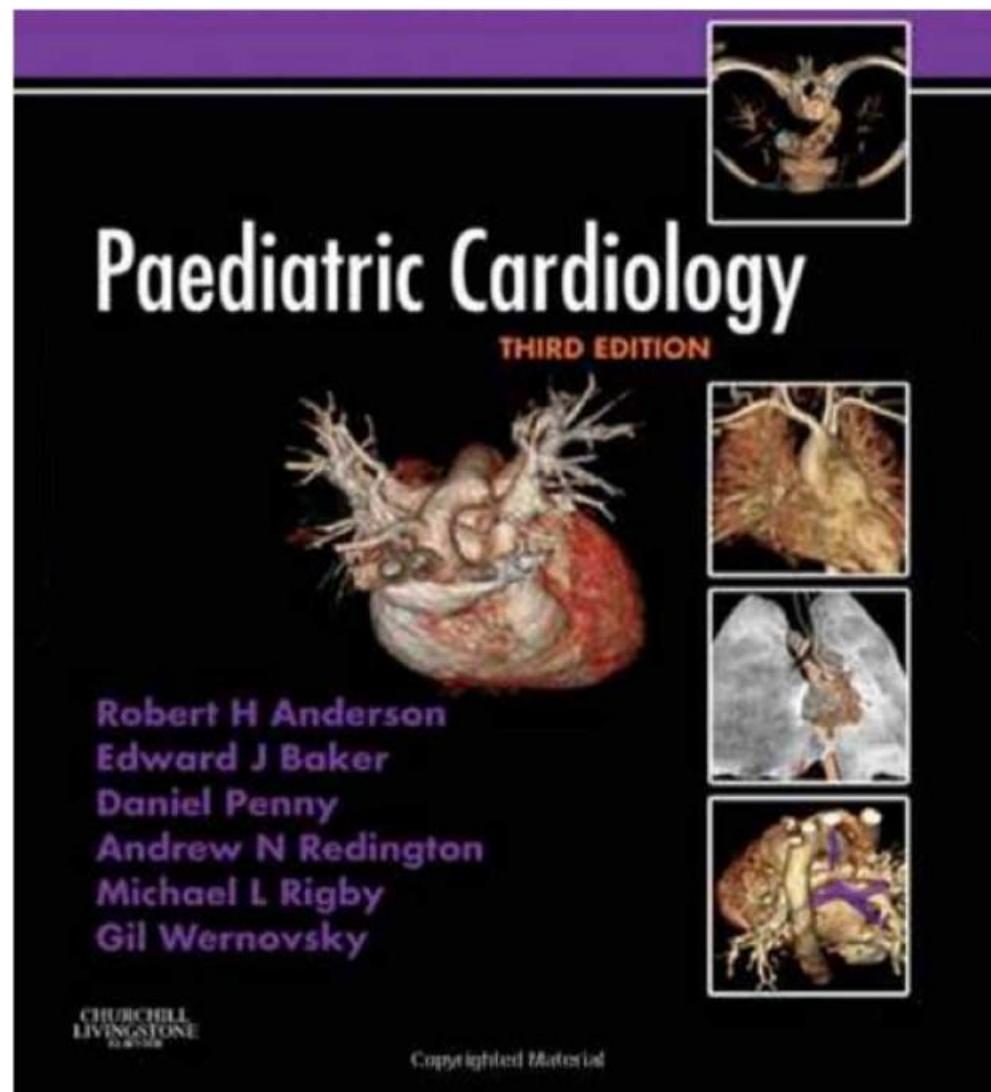


# Conduction system

- Triangle of Koch
  - Tendon of Todaro
  - Orifice of the coronary sinus
  - TV septal annulus

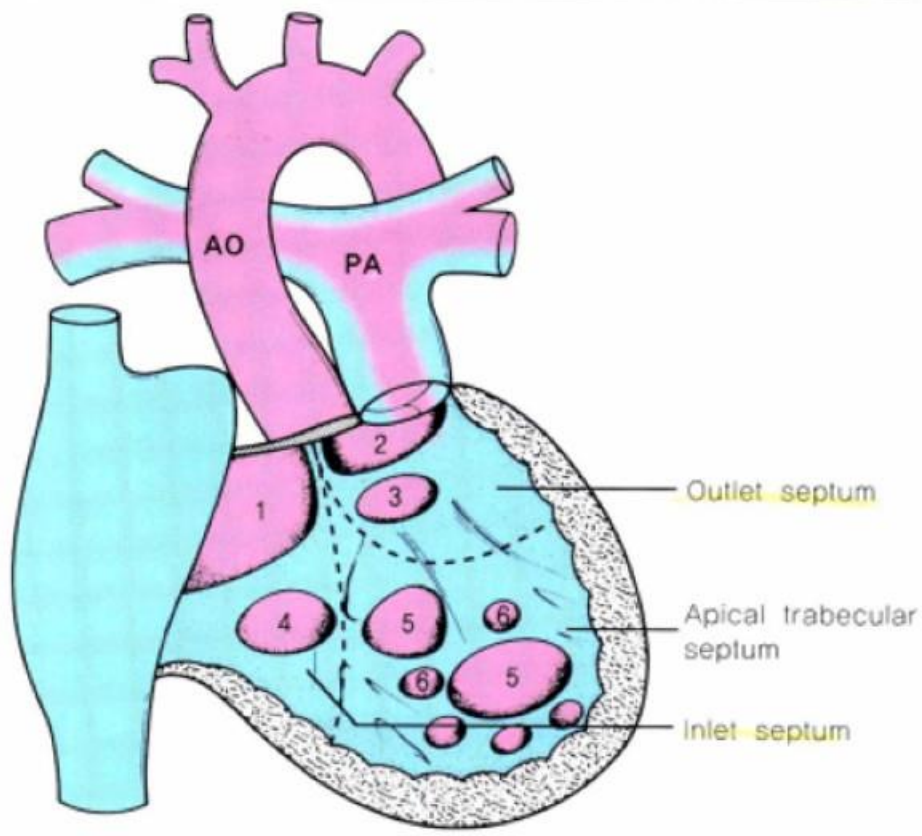


# Classification of VSD



## Categorisation of Defects

It is, perhaps, surprising that, as we start the 21st century, there is still no consensus concerning the best way to categorise and describe holes between the ventricles.



- ① 막양부 결손(PM VSD) : 결손의 천장부분이 삼첨판과 대동맥 판막으로 이루어져 있다.
- ② 판막 하 결손(SA VSD) : 결손의 천장부분이 폐동맥 판막과 대동맥 판막으로 이루어져 있으며 근육이 없으므로 대동맥 판막이 결손을 통해서 빠져 나오는 합병증(대동맥 판막 탈출)이 가장 잘 생길 수 있는 결손이다.
- ③ ④ ⑤ ⑥ 근성 결손(Muscular VSD) : 결손의 둘레가 모두 근육으로 둘러싸여 있다.
- ③ Muscular outlet VSD(MO VSD) : 근성 결손이 우심실 유출로에 가깝다(intracristal VSD).
- ④ Muscular inlet VSD : 근성 결손이 삼첨판 근처 우심실 유입로에 가깝다.
- ⑤ Muscular trabecular VSD : 근성 결손이 심실 중격의 아래쪽에 위치한다.
- ⑥ Swiss-cheese VSD(multiple muscular VSD) : 심실 중격 아래쪽에 크고 작은 근성 결손이 여러 개 있어서 구멍이 숭숭 뚫린 모양이다.

### VSD

- VSD Ventricular Septal Defect (VSD), Type 1 (Subarterial) (Supracristal) (Conal septal defect) (Infundibular)
- VSD Ventricular Septal Defect (VSD), Type 2 (Perimembranous) (Paramembranous) (Conoventricular)
- VSD Ventricular Septal Defect (VSD), Type 3 (Inlet) (AV canal type)
- VSD Ventricular Septal Defect (VSD), Type 4 (Muscular)
- VSD Ventricular Septal Defect (VSD), Type: Gerbode type (LV-RA communication)
- VSD Ventricular Septal Defect (VSD), Multiple

한국 소아 및 선천성 심장 수술 데이터베이스  
 Korean Pediatric and Congenital Heart Surgery  
 Database (KPCHSD)

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## CRITERIONS FOR CLASSIFICATION OF A VENTRICULAR SEPTAL DEFECT

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Define the plane of the defect

Define the boundaries of the defect

Account for the anatomical features:

The relation of the defect to the atrioventricular conduction axis

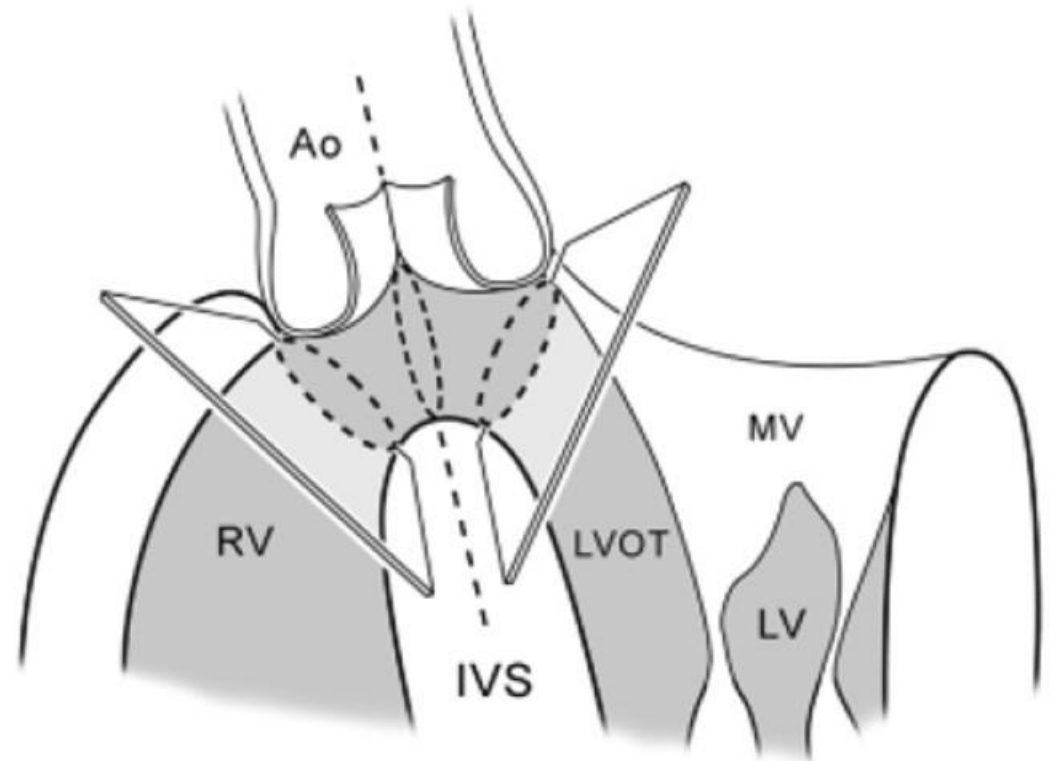
The relation of the defect to the atrioventricular valves

The relation of the defect to the arterial valves

The position of the defect within the ventricular septum: opening to the inlet, apical trabecular, or outlet parts of the right ventricle

The size of the defect

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# Classification of VSD

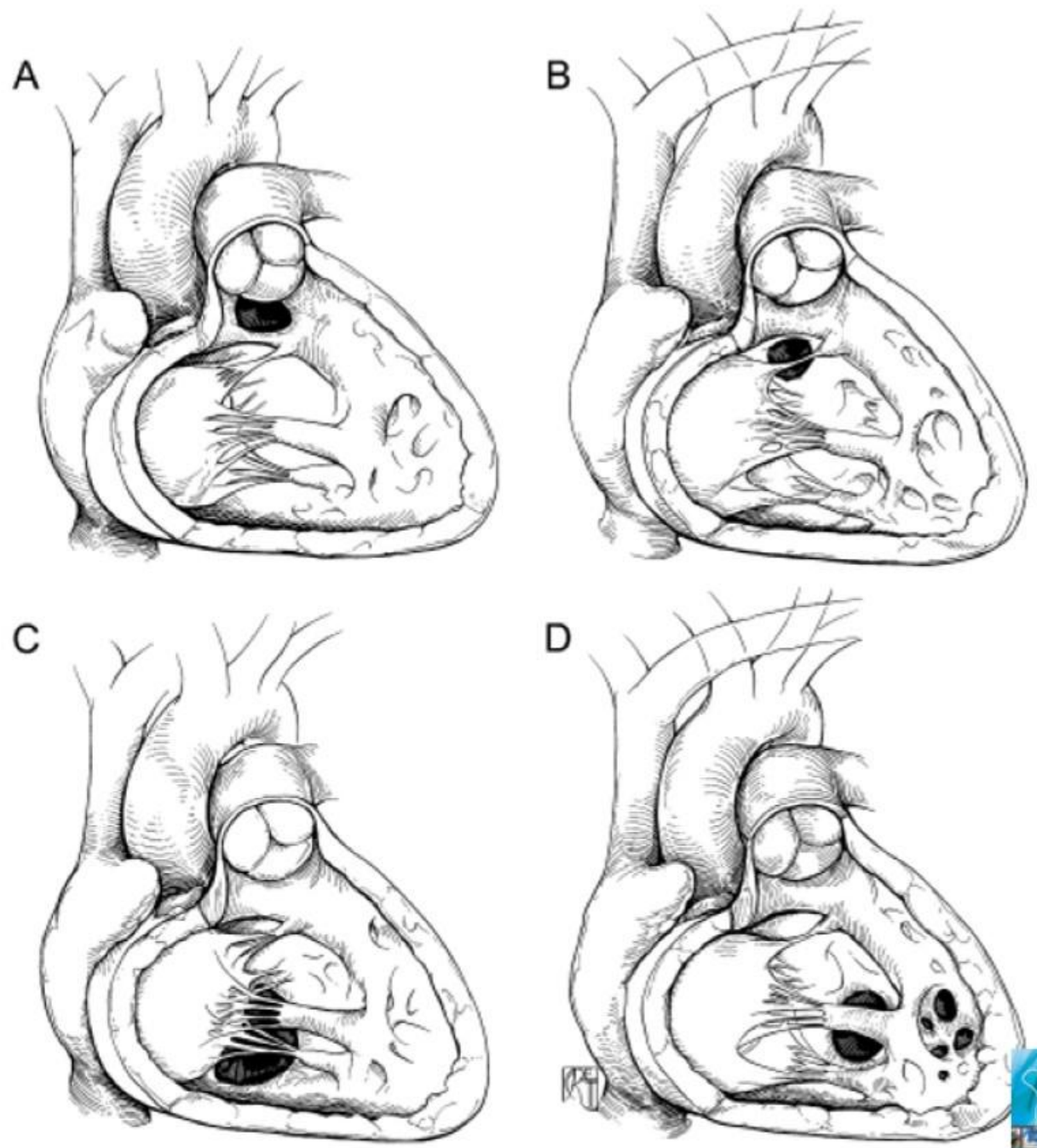
- Classic anatomic type of VSD
- New concepts of nomenclature
  - VSD nomenclature system advocated by van Praagh
  - VSD nomenclature system advocated by Anderson

# Classic anatomic type of VSD

- Type I : doubly committed and juxta-arterial, conal, supracristal, infundibular, and subarterial  
Failure of formation of the muscular subpulmonary infundibulum
- Type II : perimembranous,  
Membranous flap is found in the inferior and caudad margin of defect
- Type III : atrioventricular canal type, inlet  
Common atrioventricular junction
- Type IV : muscular VSDs (exclusively muscular borders)  
Some of the type I and type III defects, having exclusively muscular rims, should properly be defined as type IV

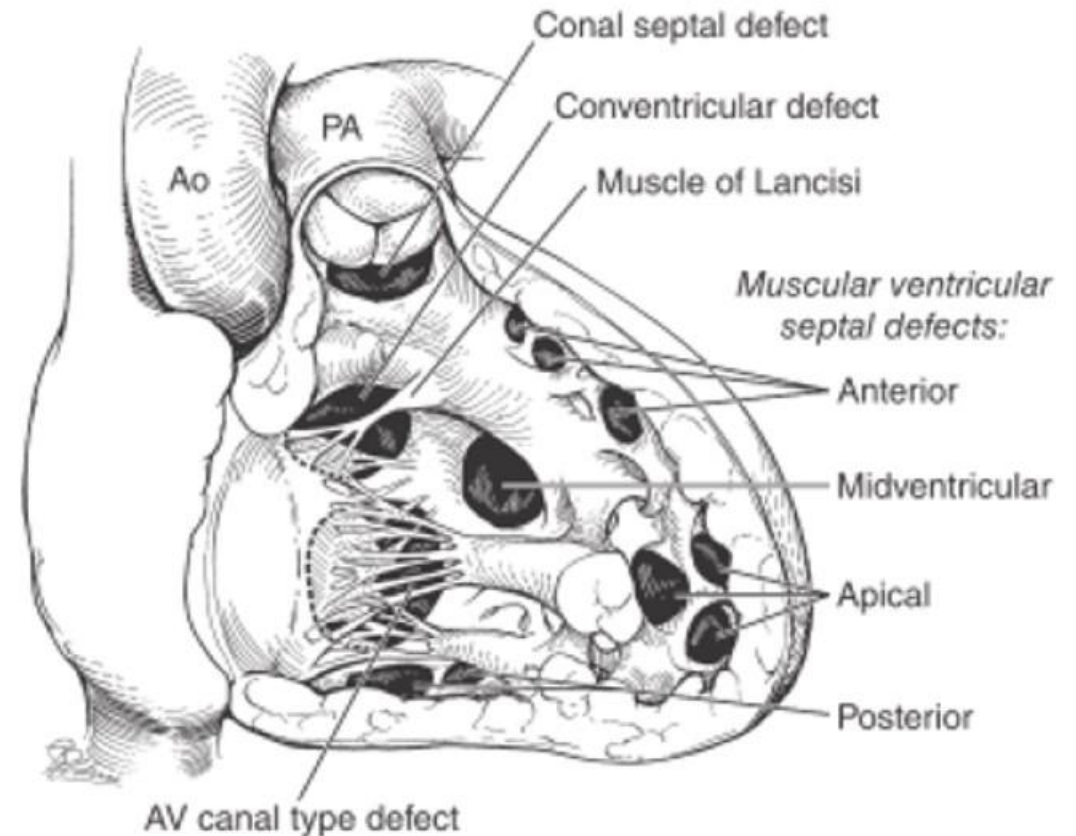


- Type I : doubly committed
- Type II : perimembranous
- Type III : atrioventricular canal type, inlet
- Type IV : muscular VSDs (exclusively muscular borders)



# VSD nomenclature by Van Praagh

- Atrioventricular canal type
  - Beneath the TV and limited by the TV annulus
  - Do not have a common atrioventricular junction but have overriding of the orifice of the TV
- Muscular type
  - Rim totally made up of muscle
  - Anterior, midventricular, posterior, apical type



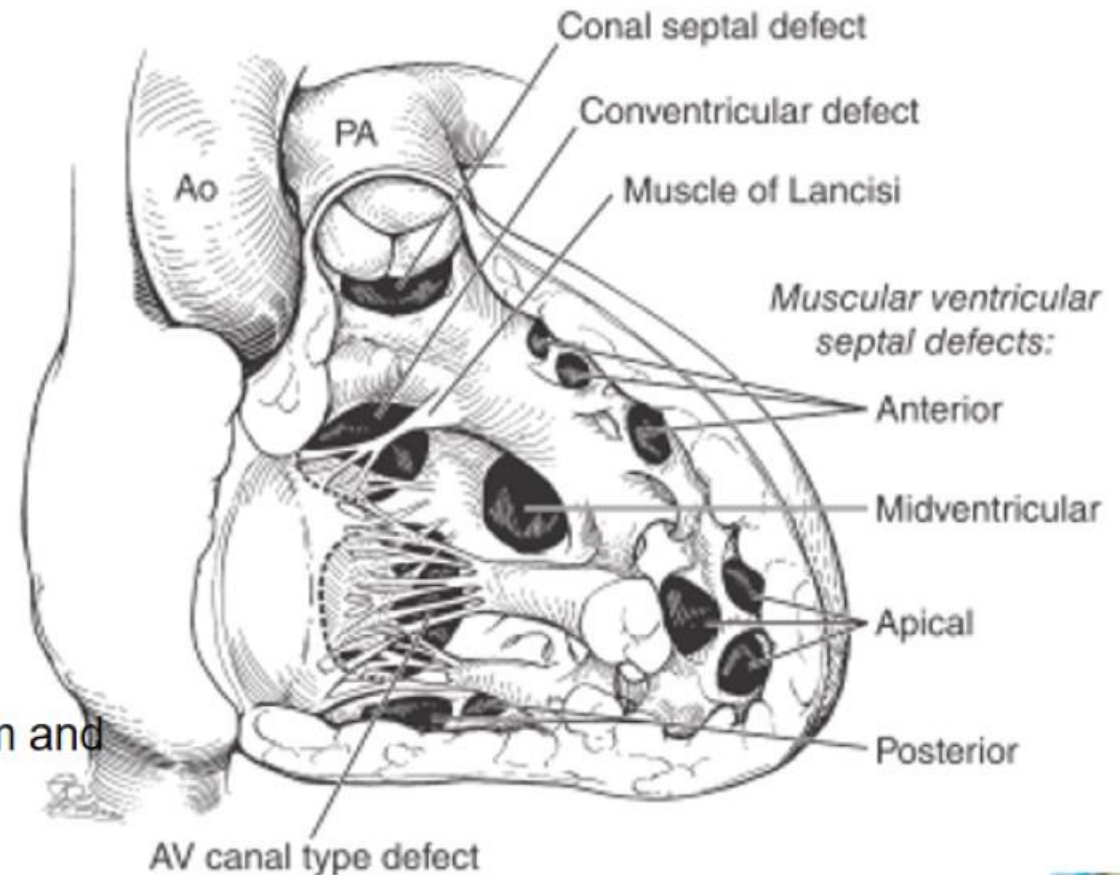
# VSD nomenclature by Van Praagh

- Conoventricular type

- Include defect of membranous septum alone
- Most involve more than just the membranous septum -> paramembranous
- Include malalignment of conal septum

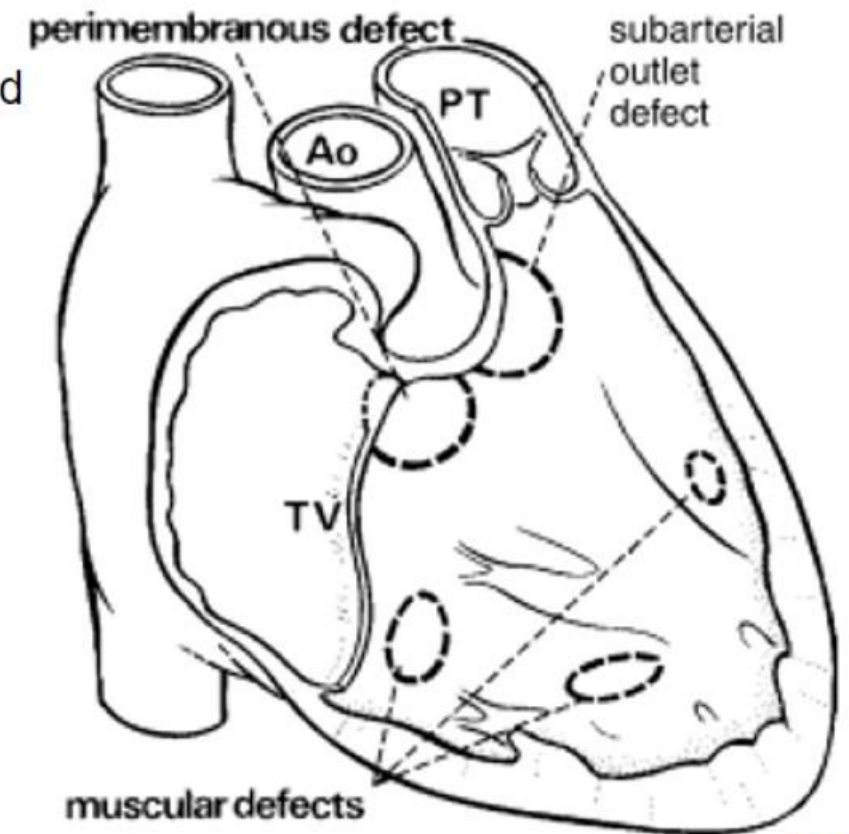
- Conal type

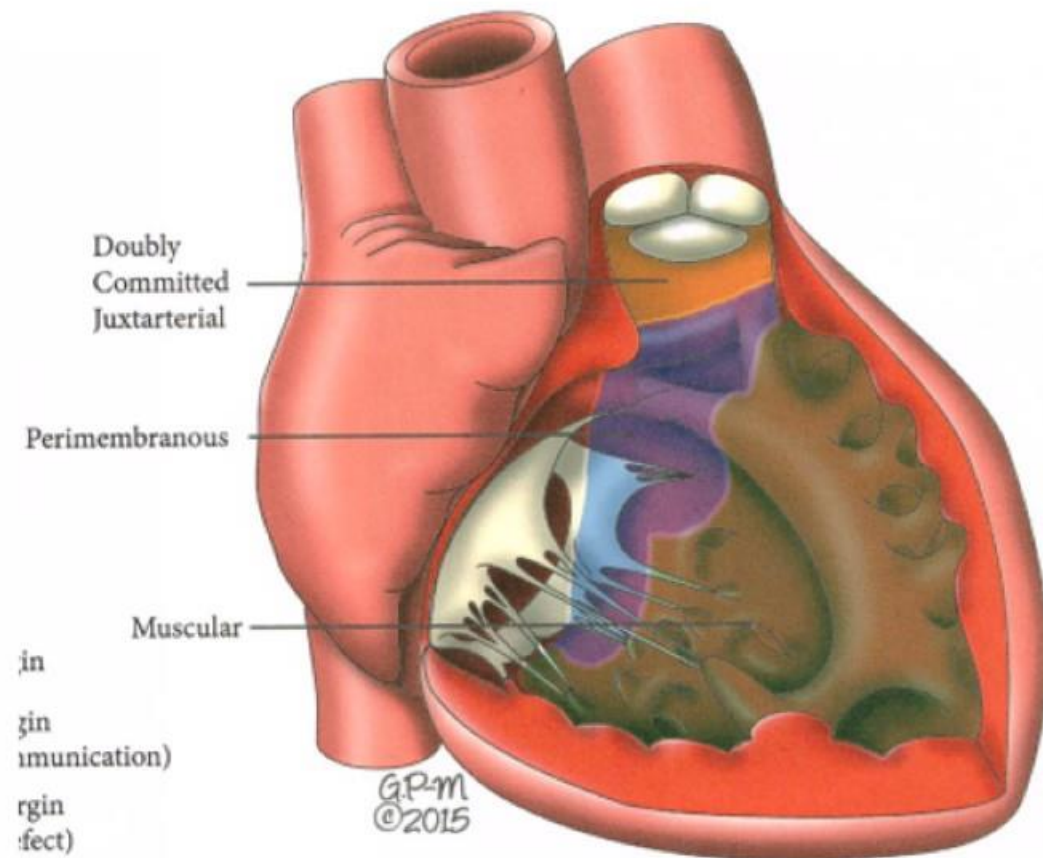
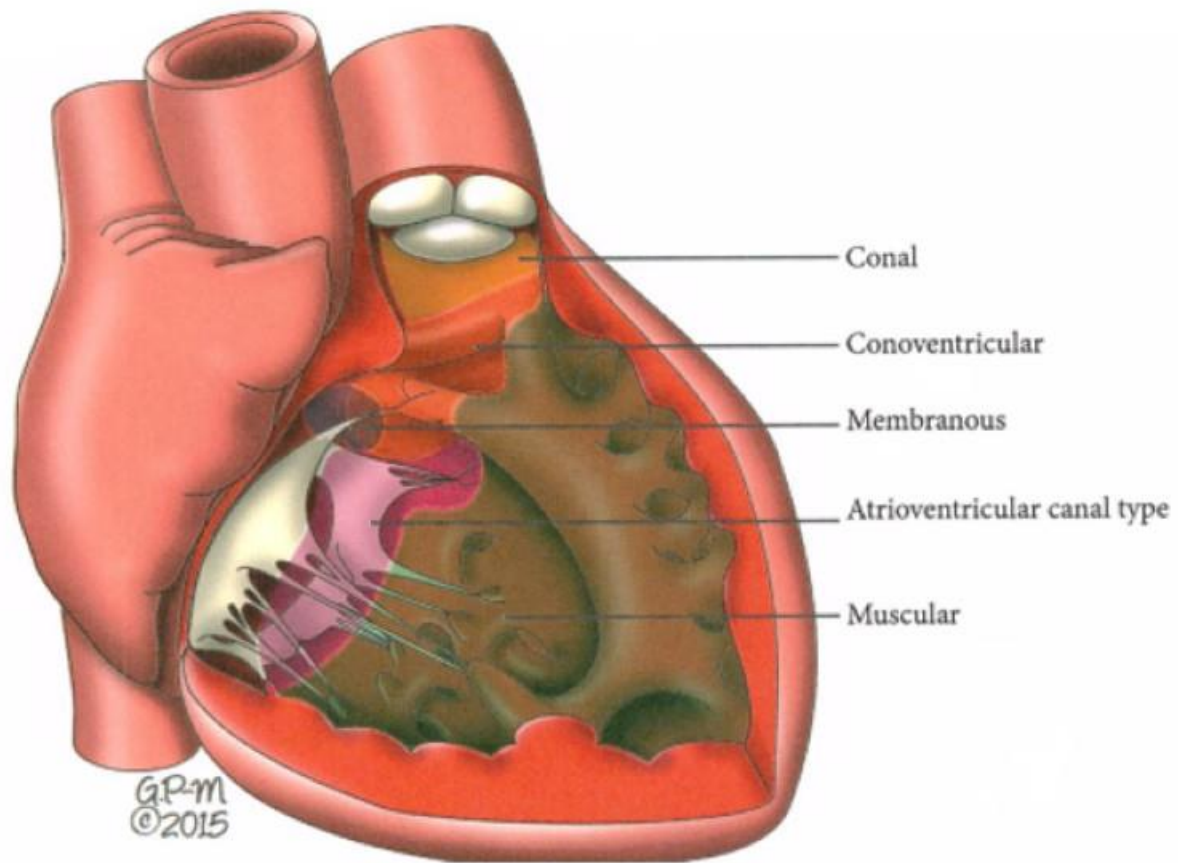
- Defect within the conal septum
- Limited upstream by muscular subpulmonary infundibulum and surrounded by conal musculature

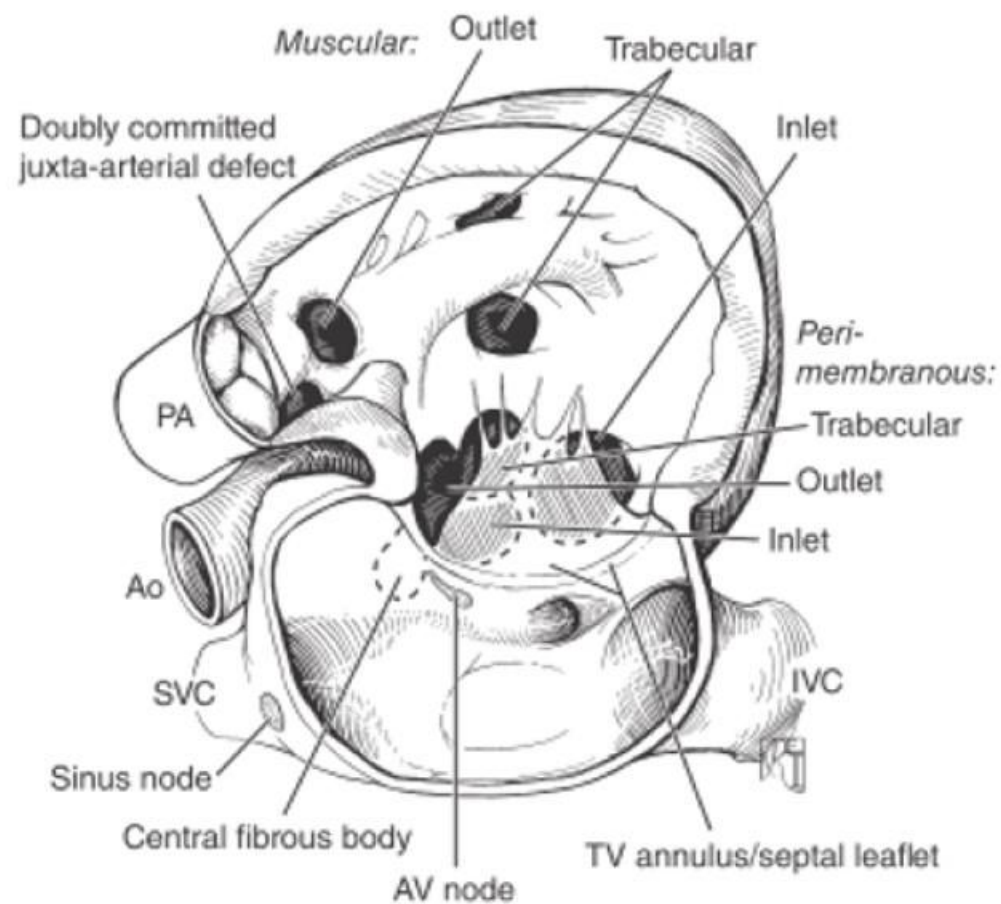
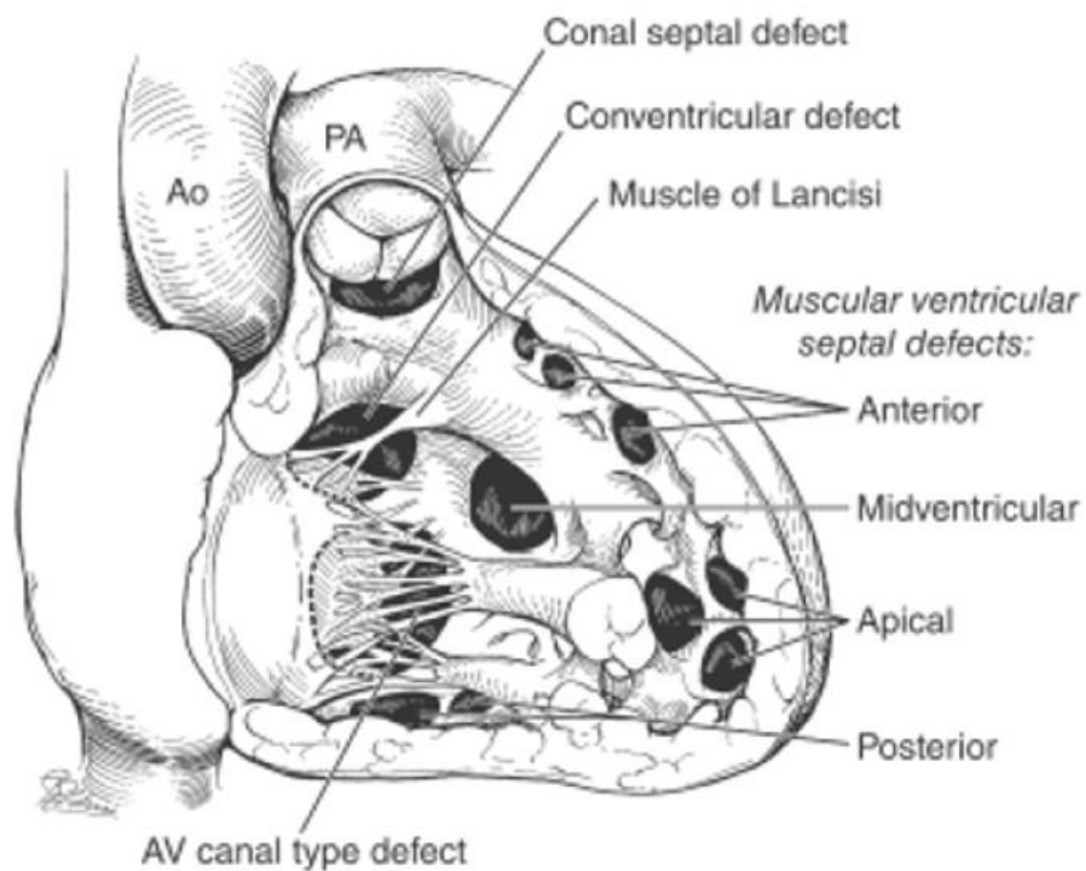


# VSD nomenclature by Anderson

- Perimembranous defects
  - Bordered by area of continuity between one or both AV valves and the arterial valve.
  - Inlet, outlet, central, overriding TV
- Muscular
  - Completely surrounded by muscular tissue
  - Inlet, outlet, apical trabecular
- Doubly committed juxtaarterial
  - Bordered by both arterial valves, with fibrous continuity of the leaflets of each of the arterial valve









# Classification of VSD

- Conoventricular (or membranous) defect : 80%

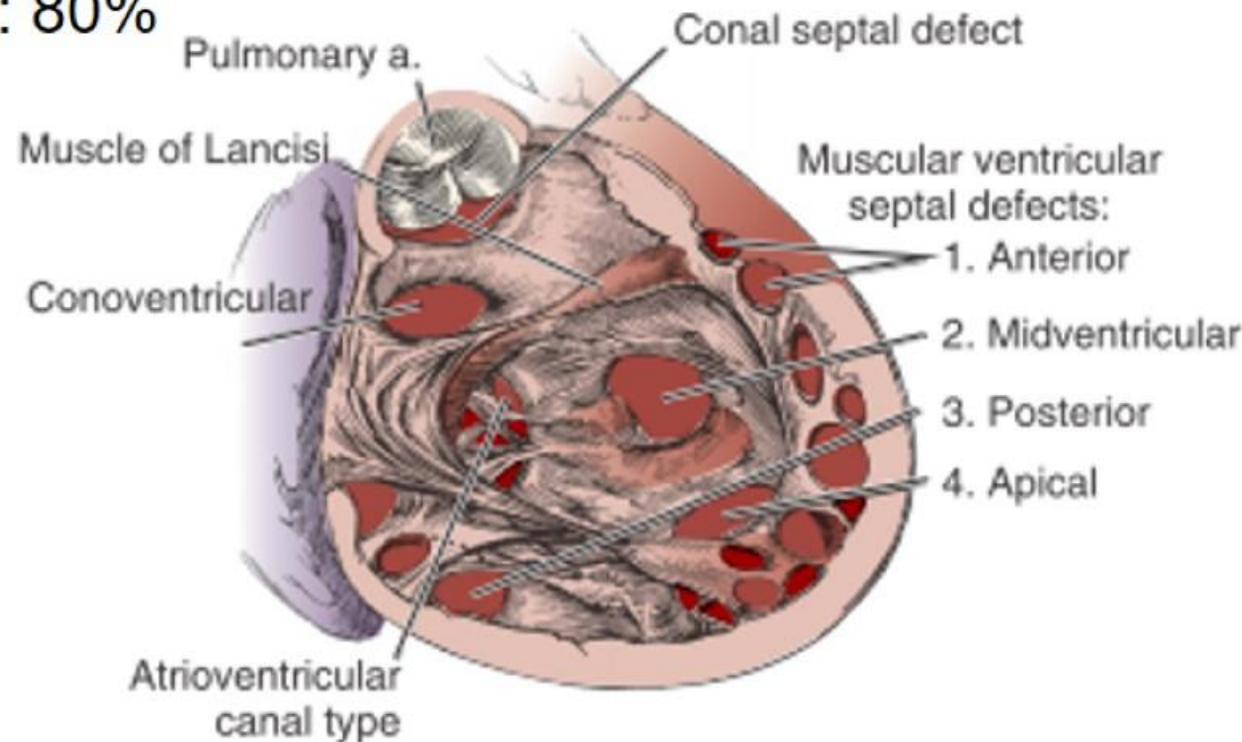
- Membranous septum

- Perimembranosus or paramembranosus

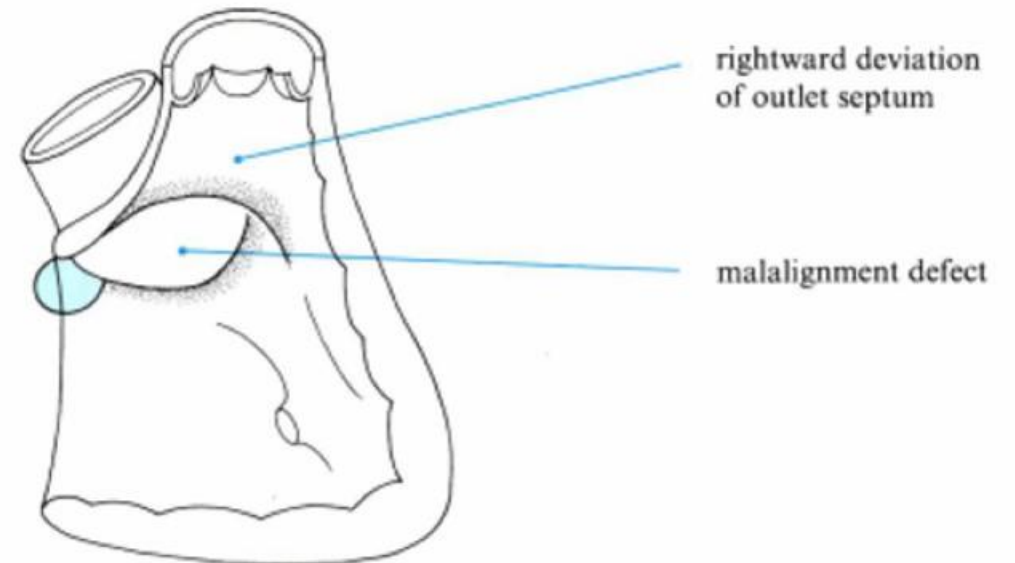
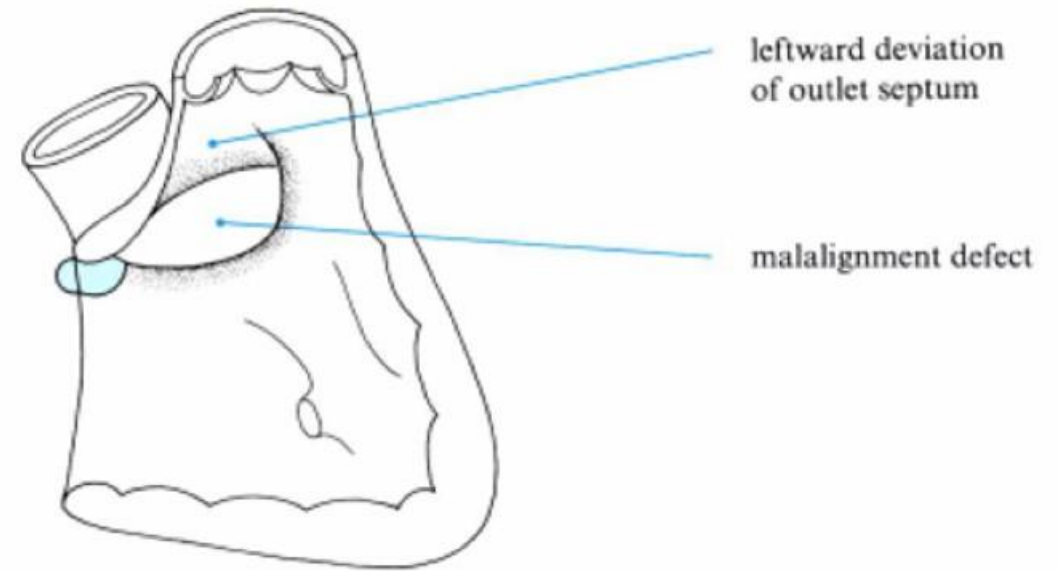
- Landmarks

- Anteroseptal commissure of TV – inf.
- NCC of AV
- Medial papillary muscle (muscle of Lancisi) – inferoposterior

- Malalignment

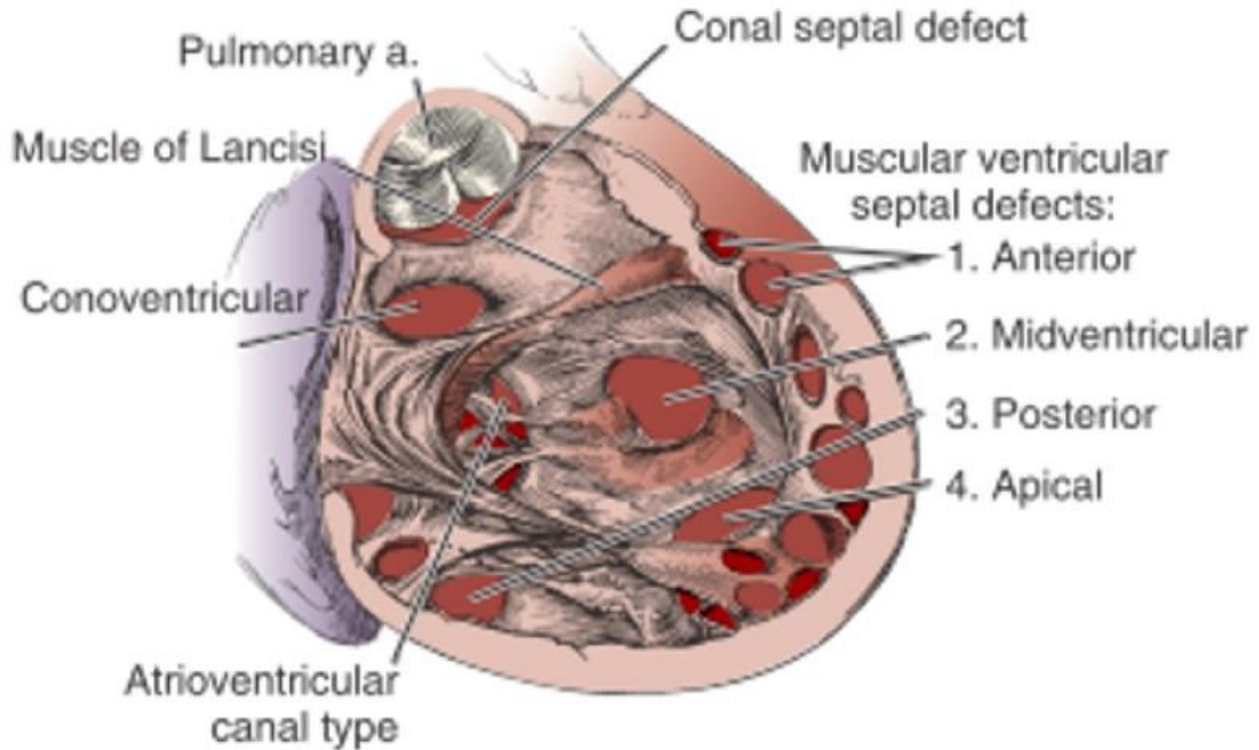


- Malalignment : conal septum
  - Conal septum palne to ventricular septal plane
  - Anterior : TOF
  - Posterior : IAA



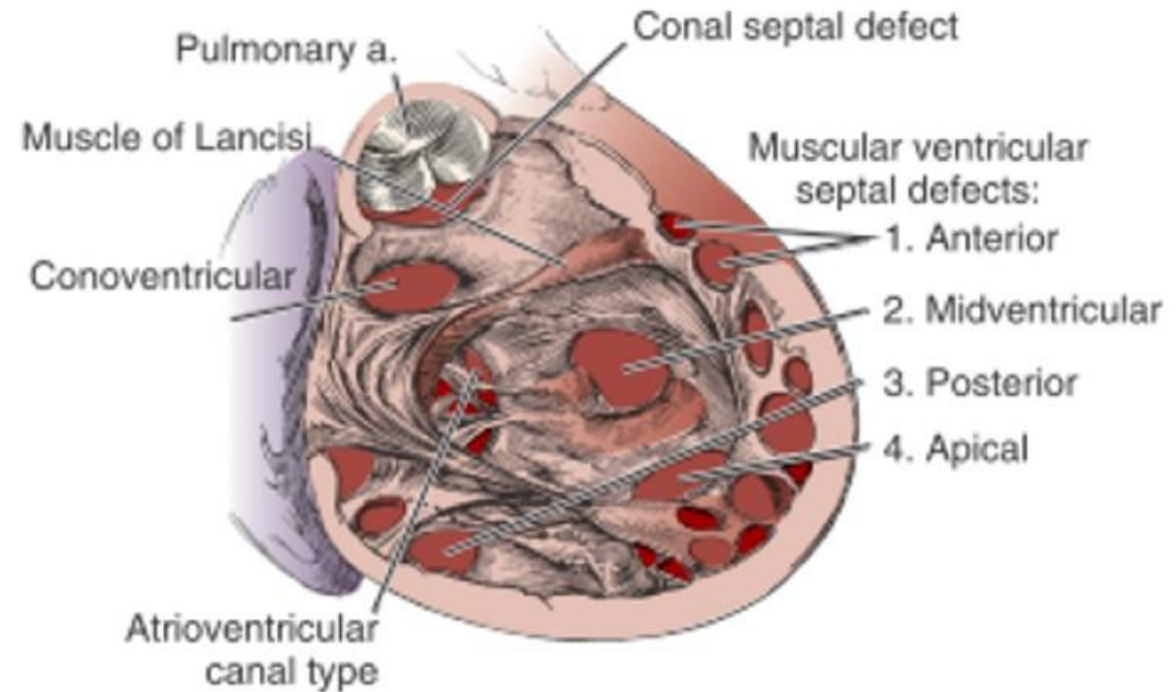
# Classification of VSD

- Conal (or outlet) VSDs : 8%
  - Entirely surrounded by muscle
    - Muscular conal VSD
  - Limited upstream by aortic or pulmonary annuli
    - Subarterial VSD



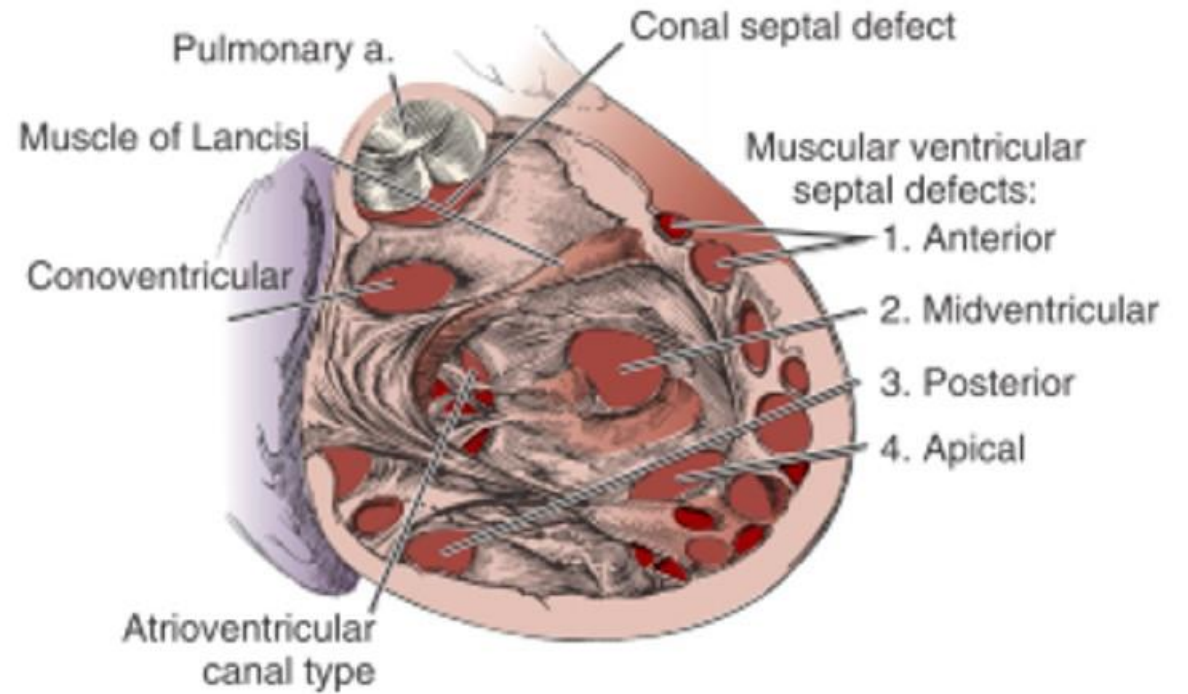
# Classification of VSD

- Inlet (or AV canal type) VSDs : 6%
  - Part or all of AV canal (inlet) septum
  - Immediately underneath septal leaflet
  - No tissue



# Classification of VSD

- Muscular VSDs (single or multiple) : 10%
  - Described by location
    - anterior / midventricular / posterior apical
  - through LV side
    - converge into either a single or two opening



# VSD size

Big! Big.. Big?

Ex) 5mm size of VSD

Infant -> maybe large. Adult -> large??

Small : less than 1/3 of aortic valve diameter

Moderate : 33% < aortic valve annulus < 50%

Large : 50 % > aortic valve annulus

- by Nada

Small : less than 1/3 of aortic valve diameter

Moderate : 33% < aortic valve annulus < 75%

Large : 75 % > aortic valve annulus

- by Hornberger LK



# Pathophysiology

- Shunt direction and magnitude
  - Depends on
    - Size of the defect
    - Pressure gradient through the defect
    - Relative compliance of both ventricles
    - Pressure relationship during the cardiac cycle
  - Non-restrictive
    - RV pressure = LV pressure
    - $Q_p/Q_s$  depends on the ratio of PVR to SVR
  - Restrictive
    - VSD offers resistance to flow

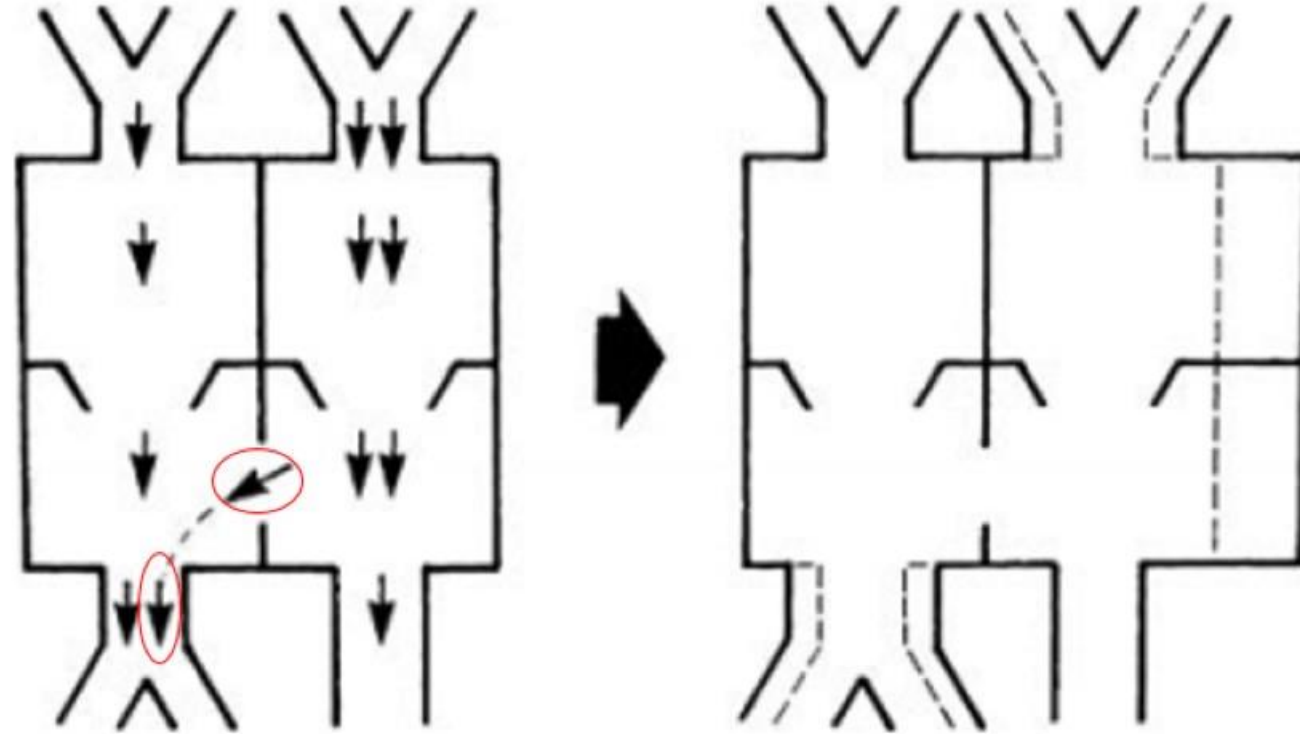
# Pathophysiology

- Sequelae of Left-to-right shunting
  - Increased pulmonary blood flow
    - LA and LV enlargement
    - LAP **↑** -> pulmonary edema -> pulmonary infection
    - Lung compliance **↓** -> the work of breathing **↑**
    - Failure to thrive



# Pathophysiology

- Sequelae of L-R shunting
  - Development of pulmonary vascular disease
    - Pulmonary blood flow ↓ -> Symptom improvement
    - Eisenmenger complex
      - Fixed pulmonary hypertension
      - RV hypertrophy, Normal size LV
      - Often inoperable



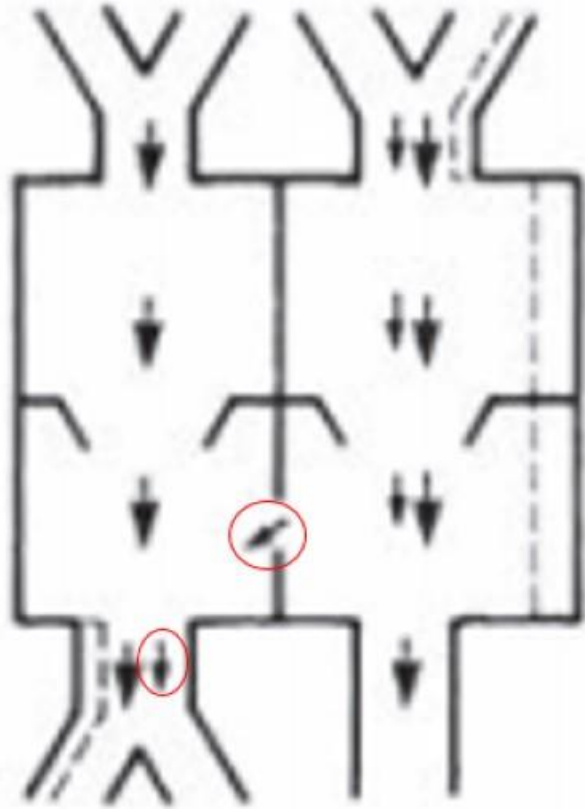
Volume overload ventricle? LV (not RV)

-> results in LV enlargement

Why? Shunt occurs mainly during systole, the shunted blood goes directly to the PA rather than remaining in the RV cavity -> RV remains relatively normal in moderate VSD

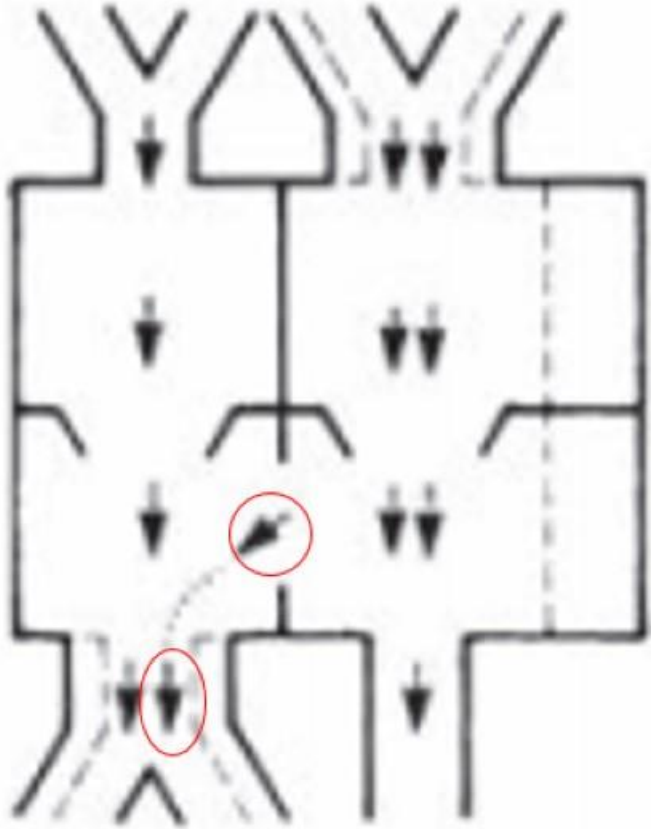
VSD, PDA produce an enlargement of LA, LV

Small



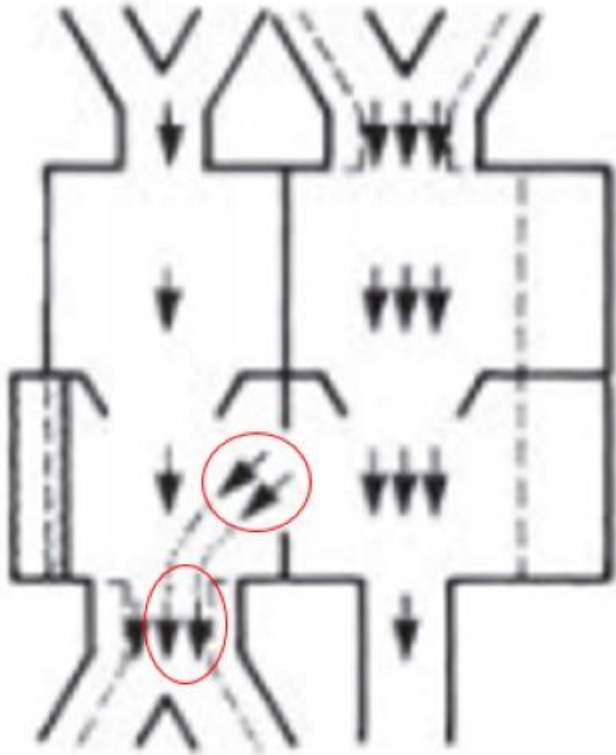
- Pulmonary vascular congestion and chamber enlargement is minimal
- No significant change in chest x-ray

## Moderate (PVR↑)



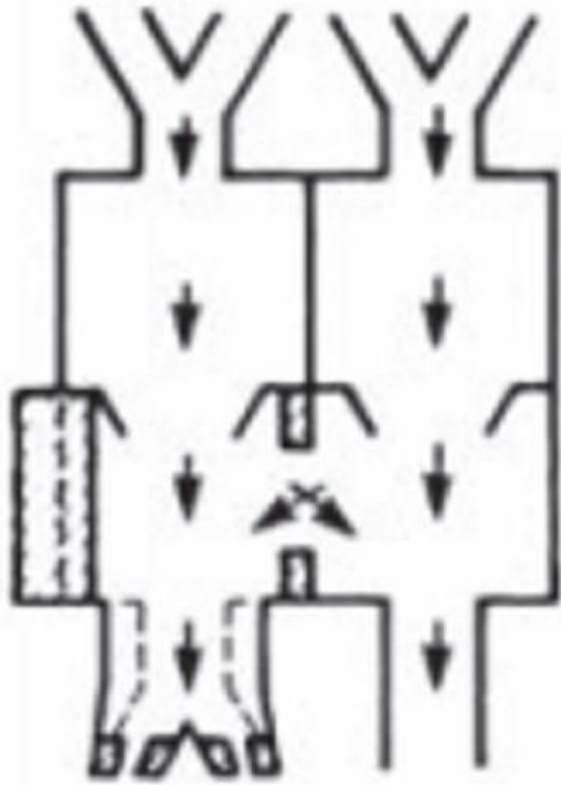
- Left chamber enlarged
- Volume overwork of LV is significant -> ECG shows LVH
- Shunt is large but RV is not significantly dilated -> RV is not under significant volume or pressure overload.

Large (PVR ↑)



- Overall heart size is large than with a moderate VSD
- Direct transmission of the LV pressure through VSD to RV -> RV becomes enlarged and hypertrophied
- Chest x-ray : biventricular enlargement, LA enlargement, increased pulmonary vascularity
- CHF in early infancy

Large (PVR ↑)



- Untreated large VSD
- Irreversible changes in pulmonary arterioles, producing pulmonary vascular obstructive disease (Eisenmenger's syndrome)
- PVR elevated -> RV pressure same as LV -> decreased shunt -> left heart volume loading decrease
- Heart size become small but PA segment remains enlarged

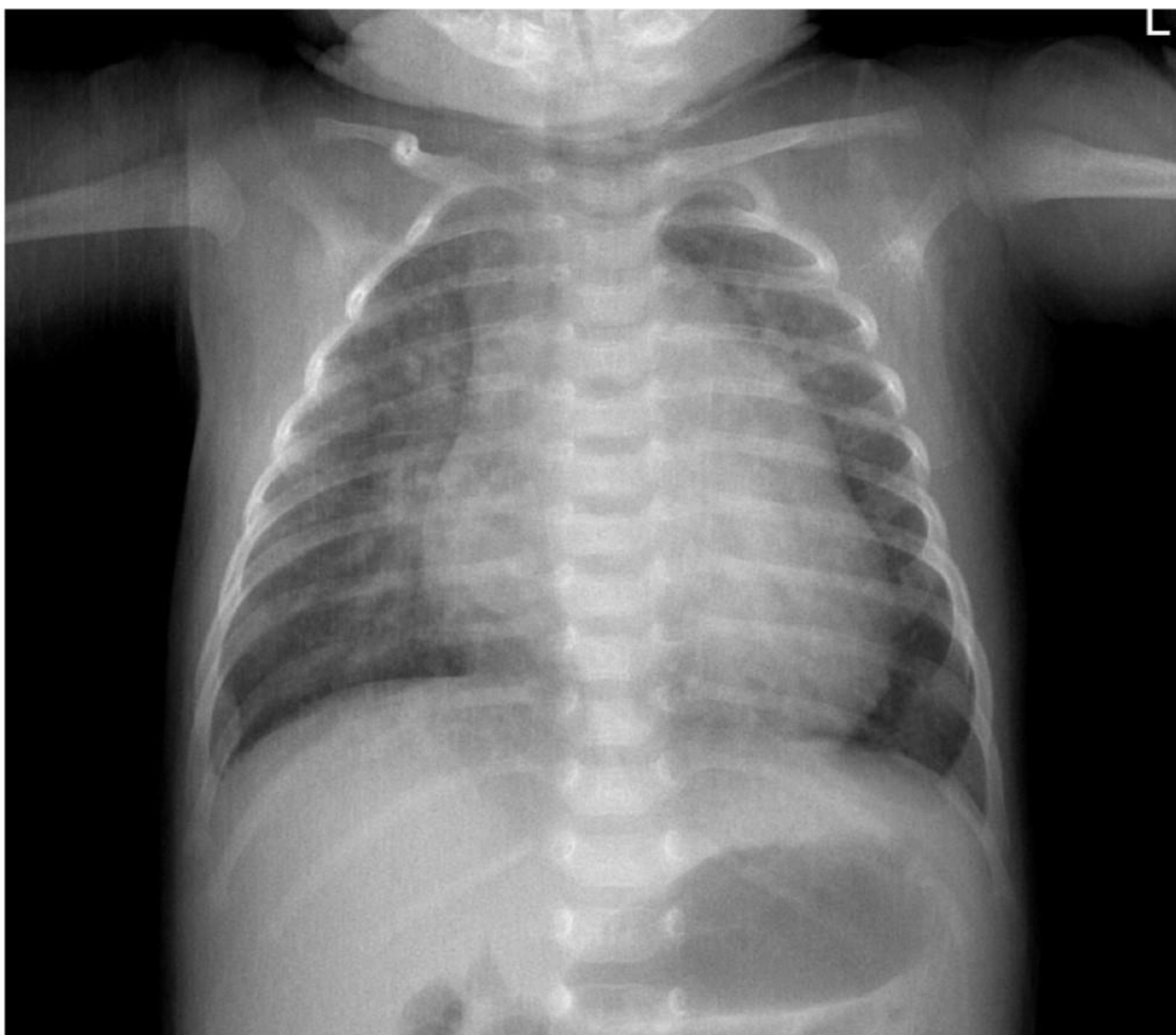
# Diagnosis

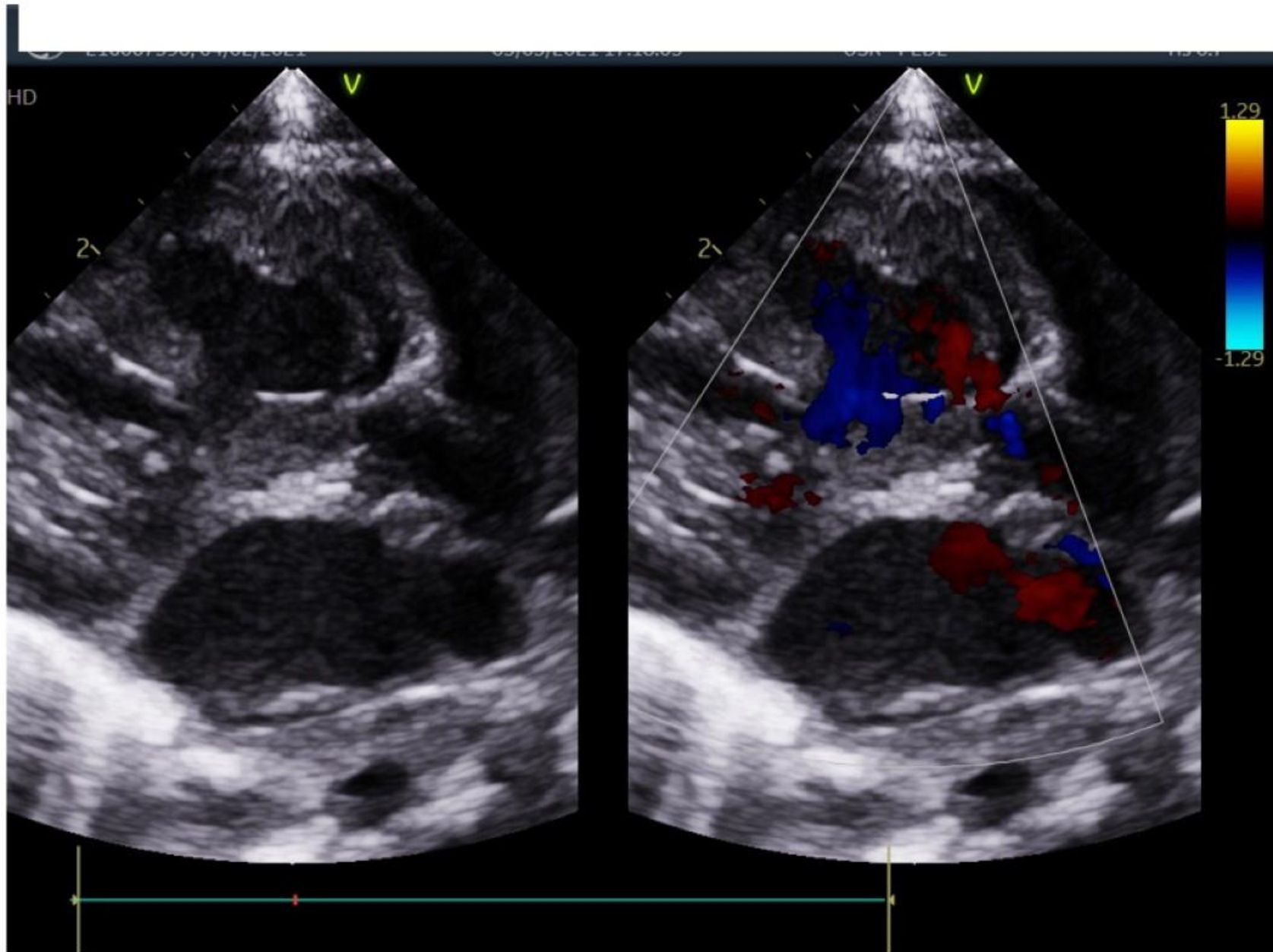
- Symptom
  - Tachypnea, profuse sweating during feeding
  - Growth failure
- Physical examination
  - bulging precordium, pansystolic murmur
  - Enlarged liver, Thready pulse

# Diagnosis

- Chest X-ray
  - Large central and peripheral PA
  - Enlarged LA and LV
- ECG
  - Ventricular hypertrophy
- Echocardiography
- Cardiac catheterization







# Indication for Surgery

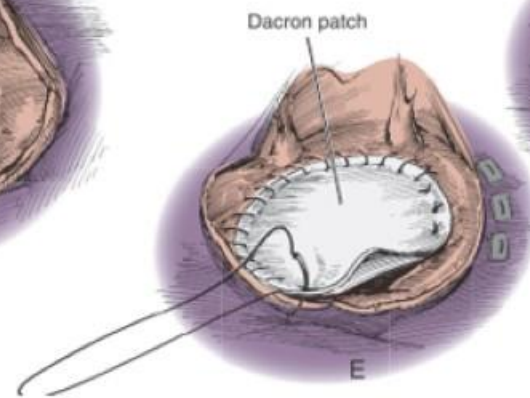
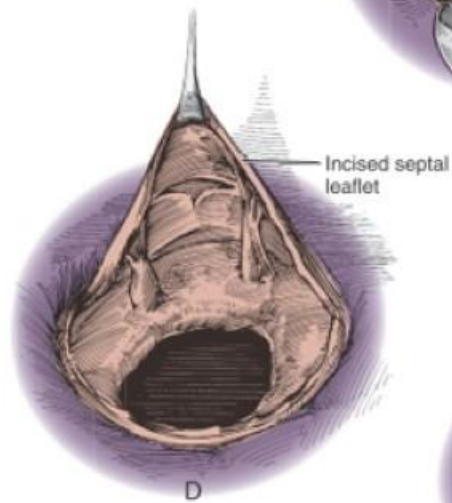
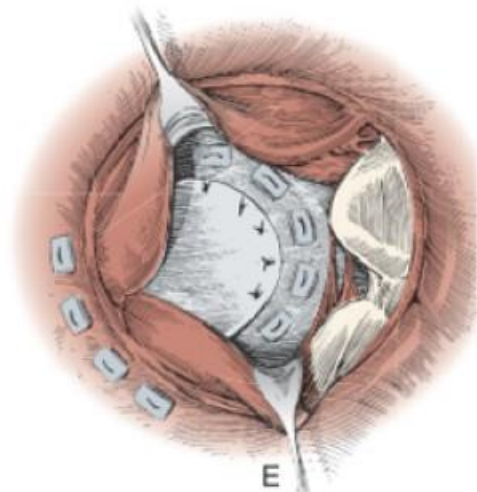
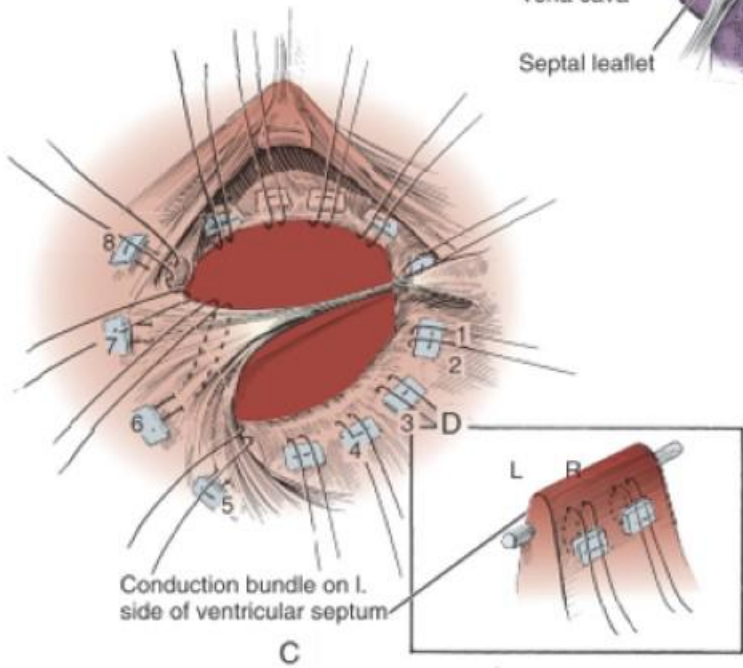
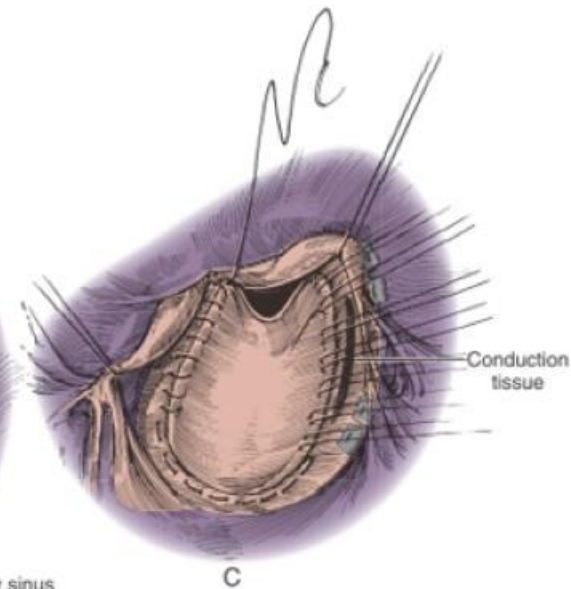
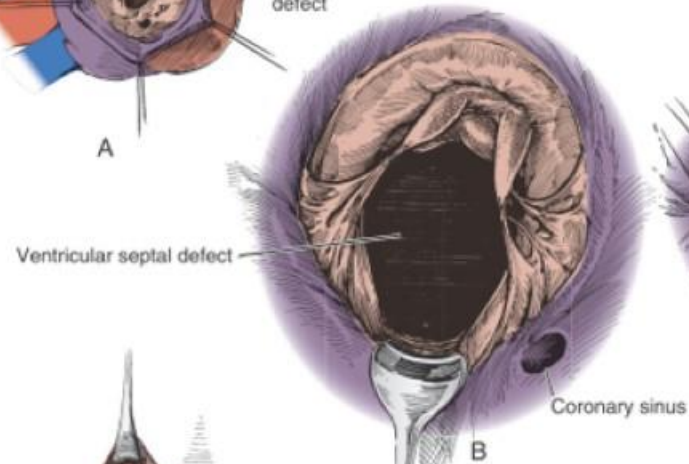
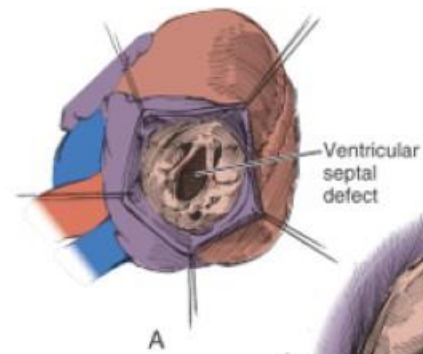
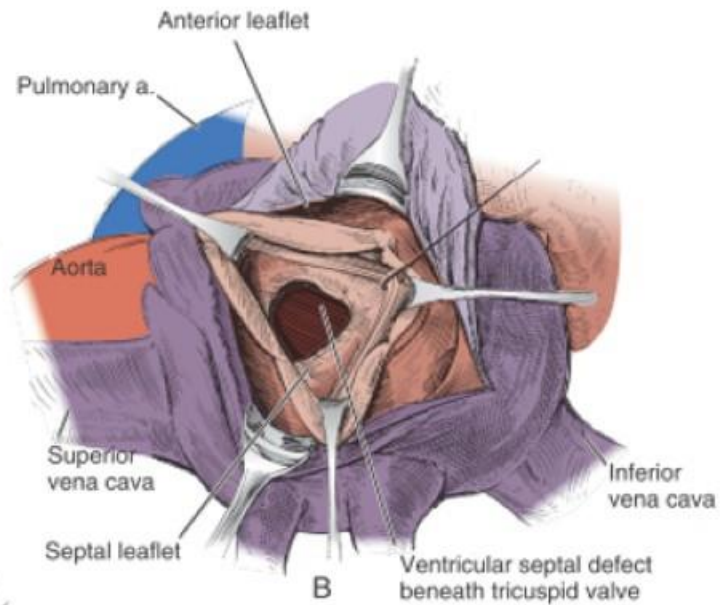
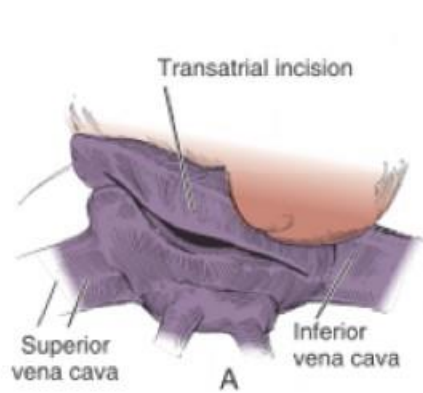
- 30% of infants with severe symptoms requires within first year of life.
- Intractable congestive heart failure, failure to thrive
- Aggressive medical management is indicated
  - Most membranous and muscular VSDs tends to close spontaneously
  - Malalignment conoventricular or inlet VSDs are unlikely to close spontaneously
- Asymptomatic children with isolated VSD can be followed safely

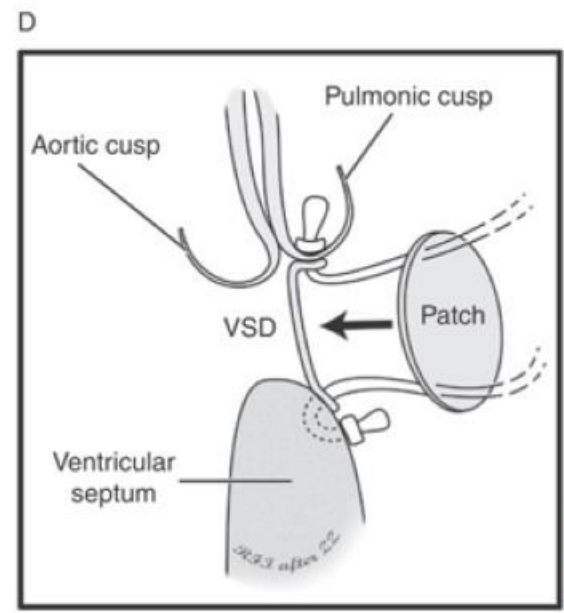
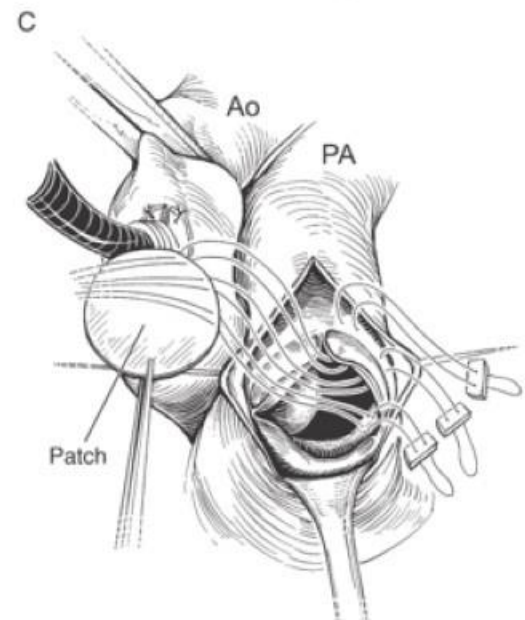
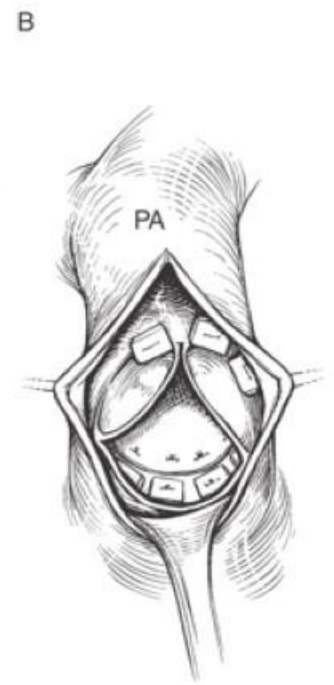
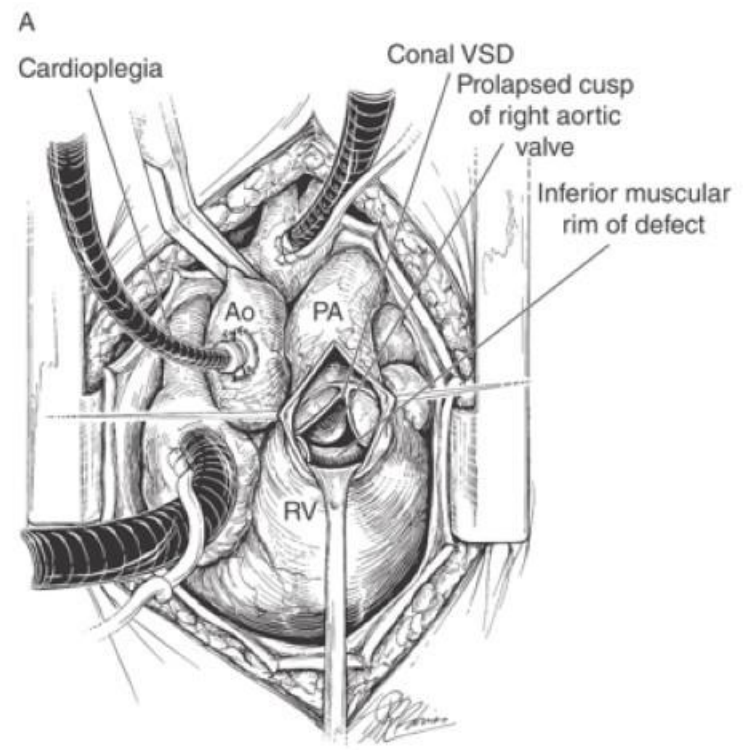
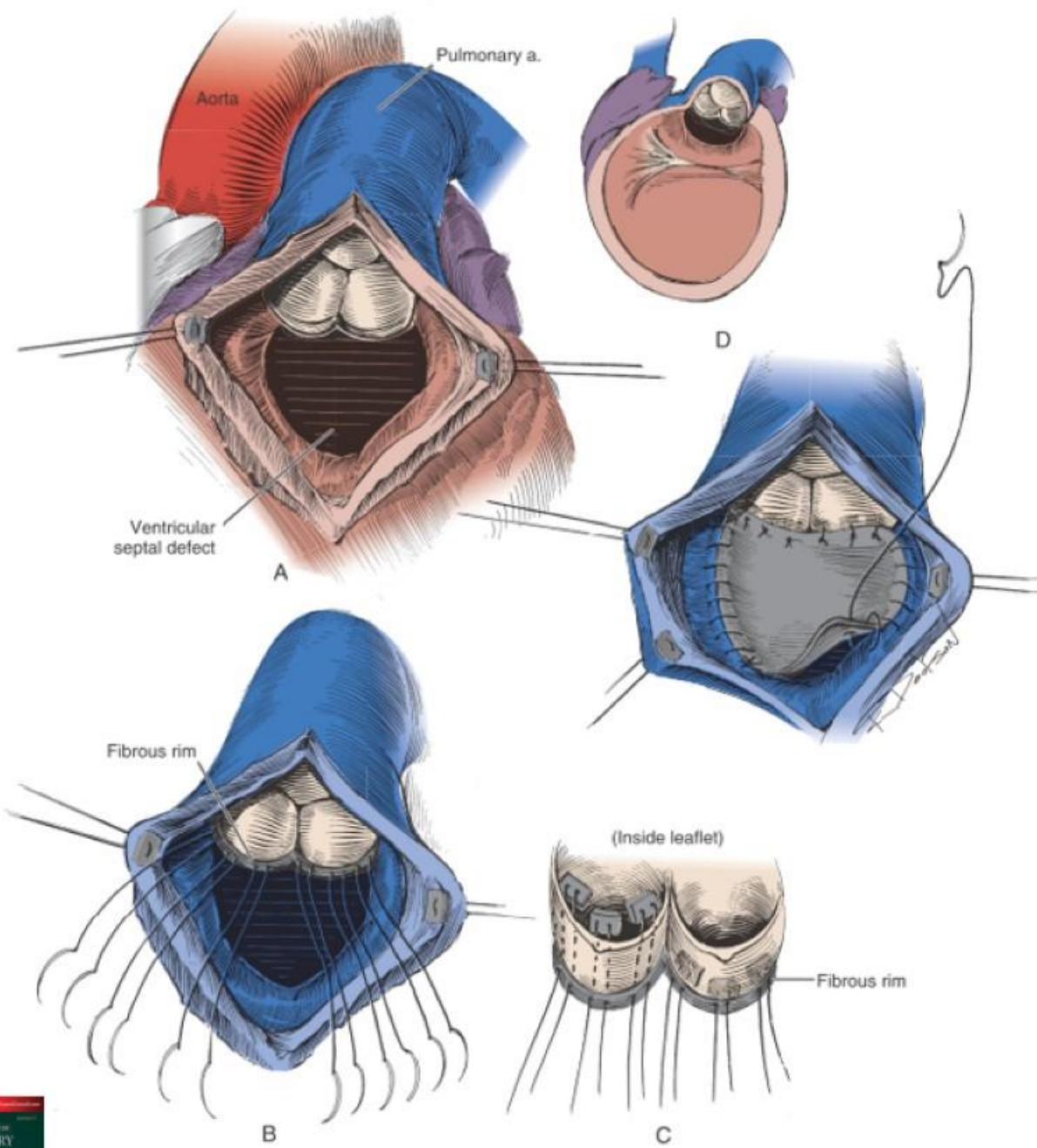
# Indication for Surgery

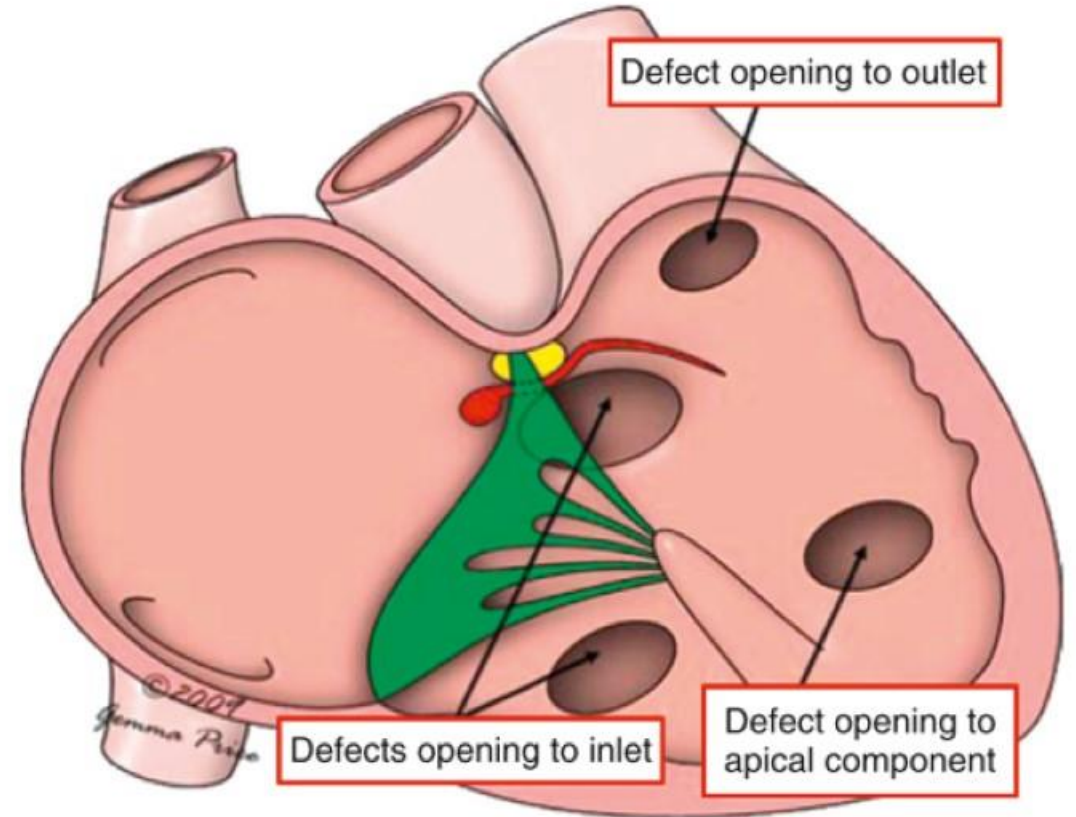
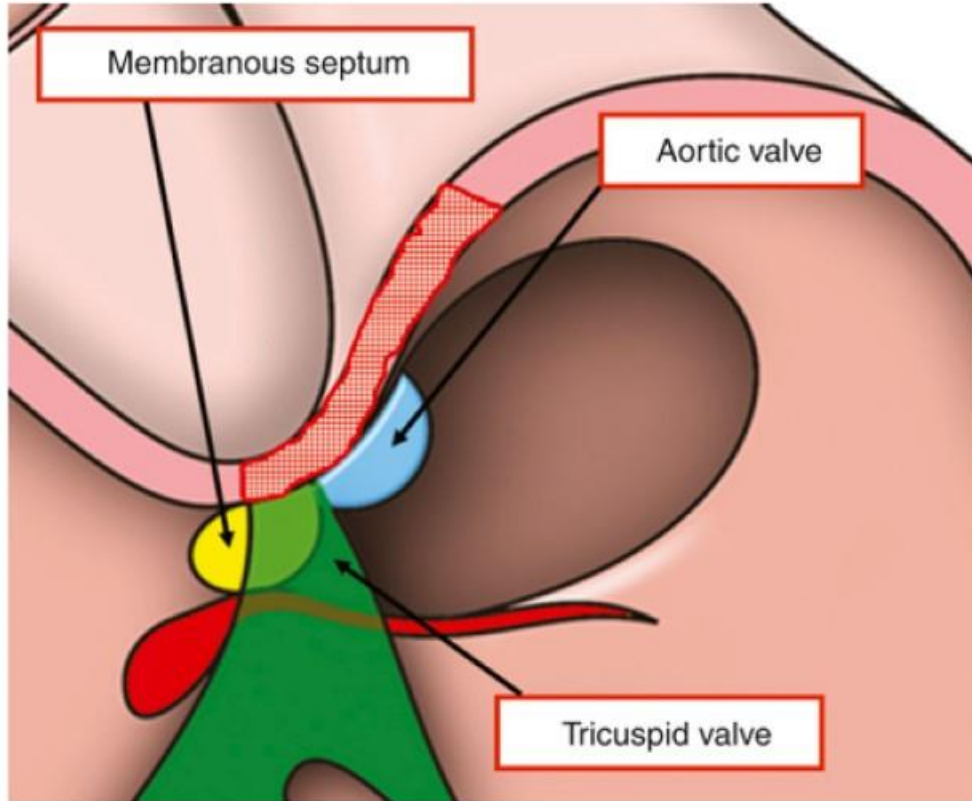
- Development of pulmonary vascular disease
  - PA pressure  $>$  50% of systemic pressure in a child older than 1 year
- Prolapse of aortic cusp in to VSD
  - 5% of membranous or outlet VSD in 1<sup>st</sup> decade of life
- Congestive heart failure
- Increasing aortic valve prolapse and regurgitation
- History of infective endocarditis

# Surgical technique

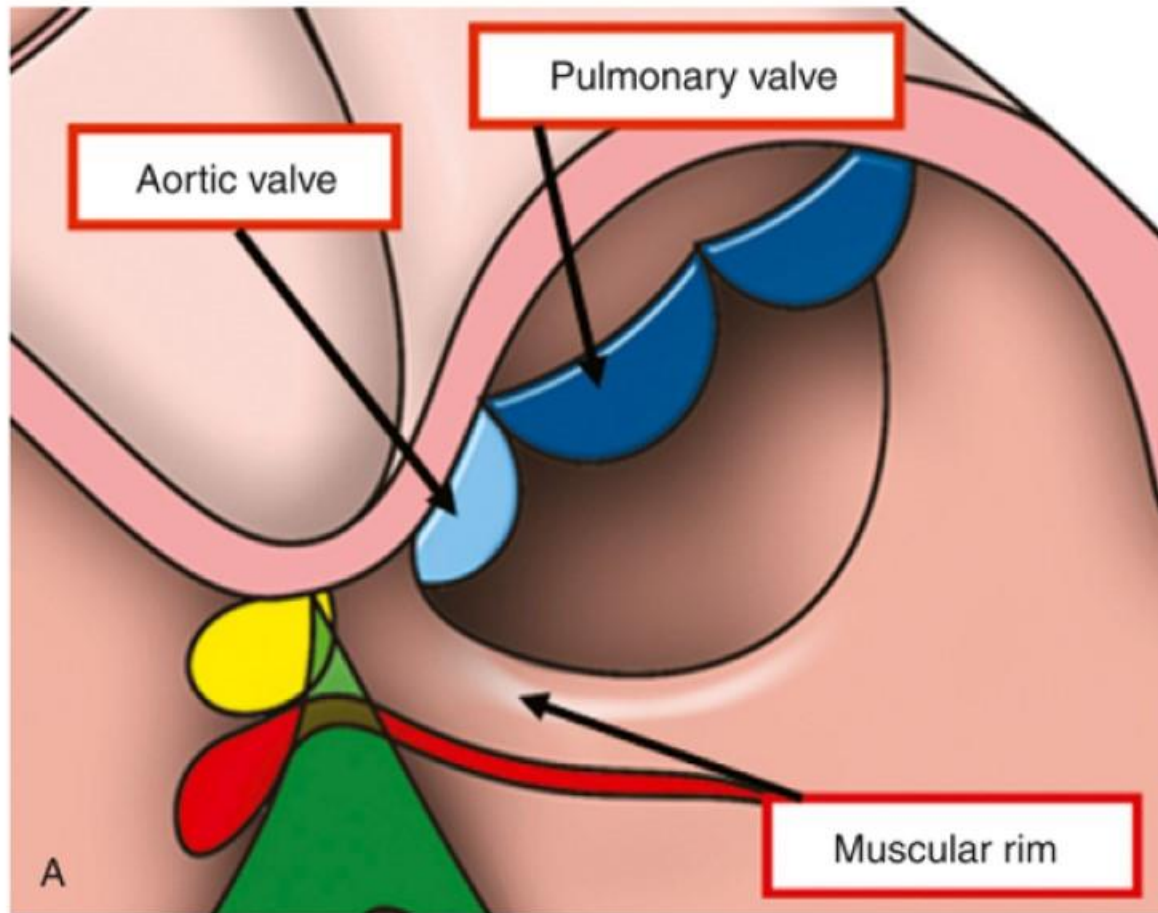
- Normal CPB
- Moderate hypothermia (28°C to 32°C)
- Approach
  - RA / MPA / RV / LV
- TV : retraction or detachment
- AV : infusion of CP solution
- Interrupted or continuous suture

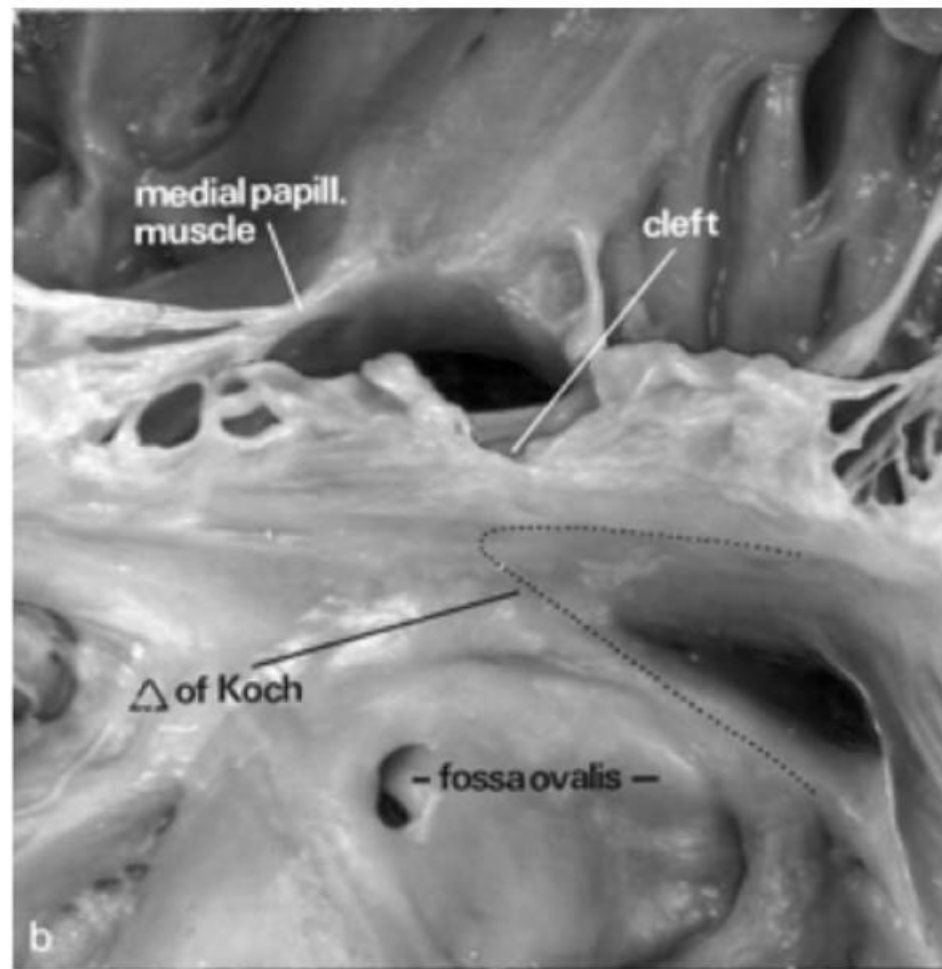
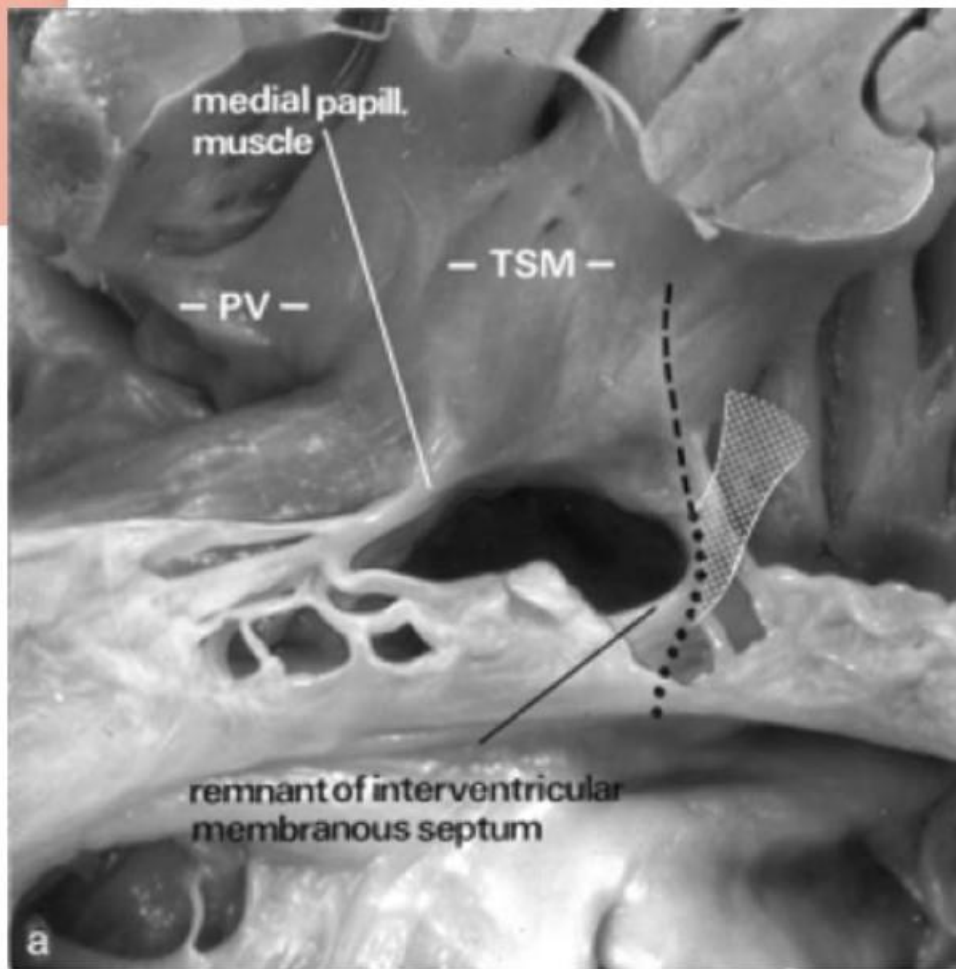
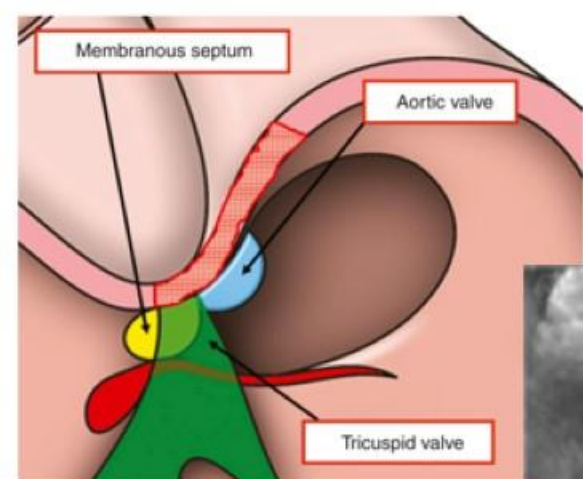


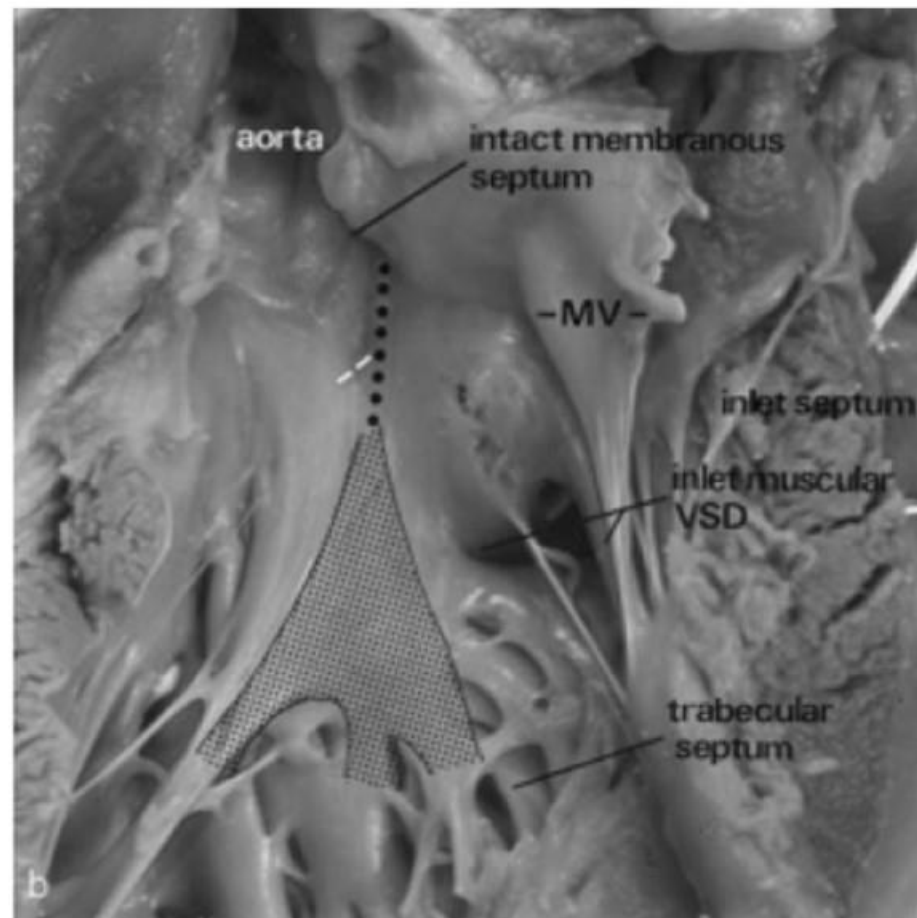
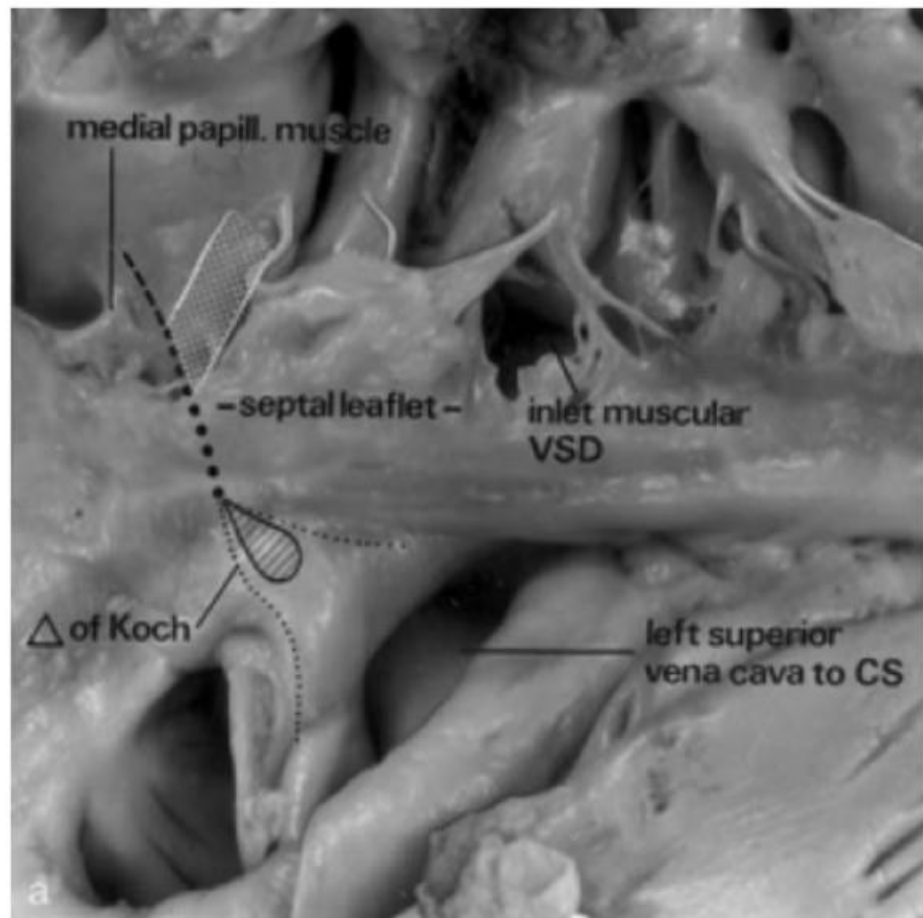
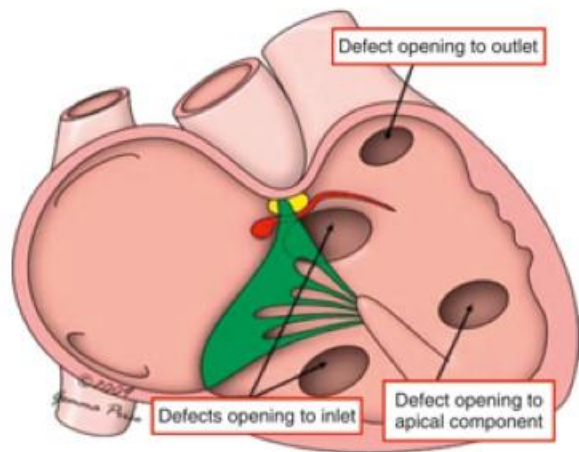








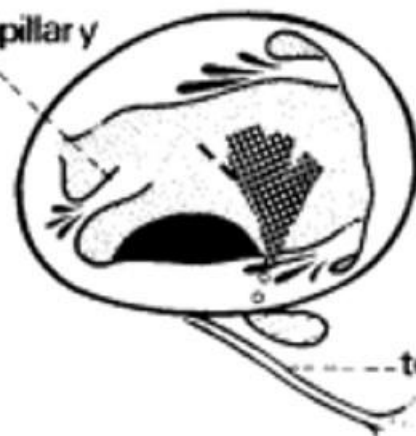






a perimembranous outlet

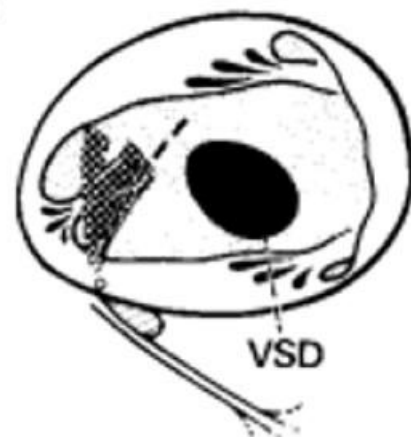
medial papillary muscle



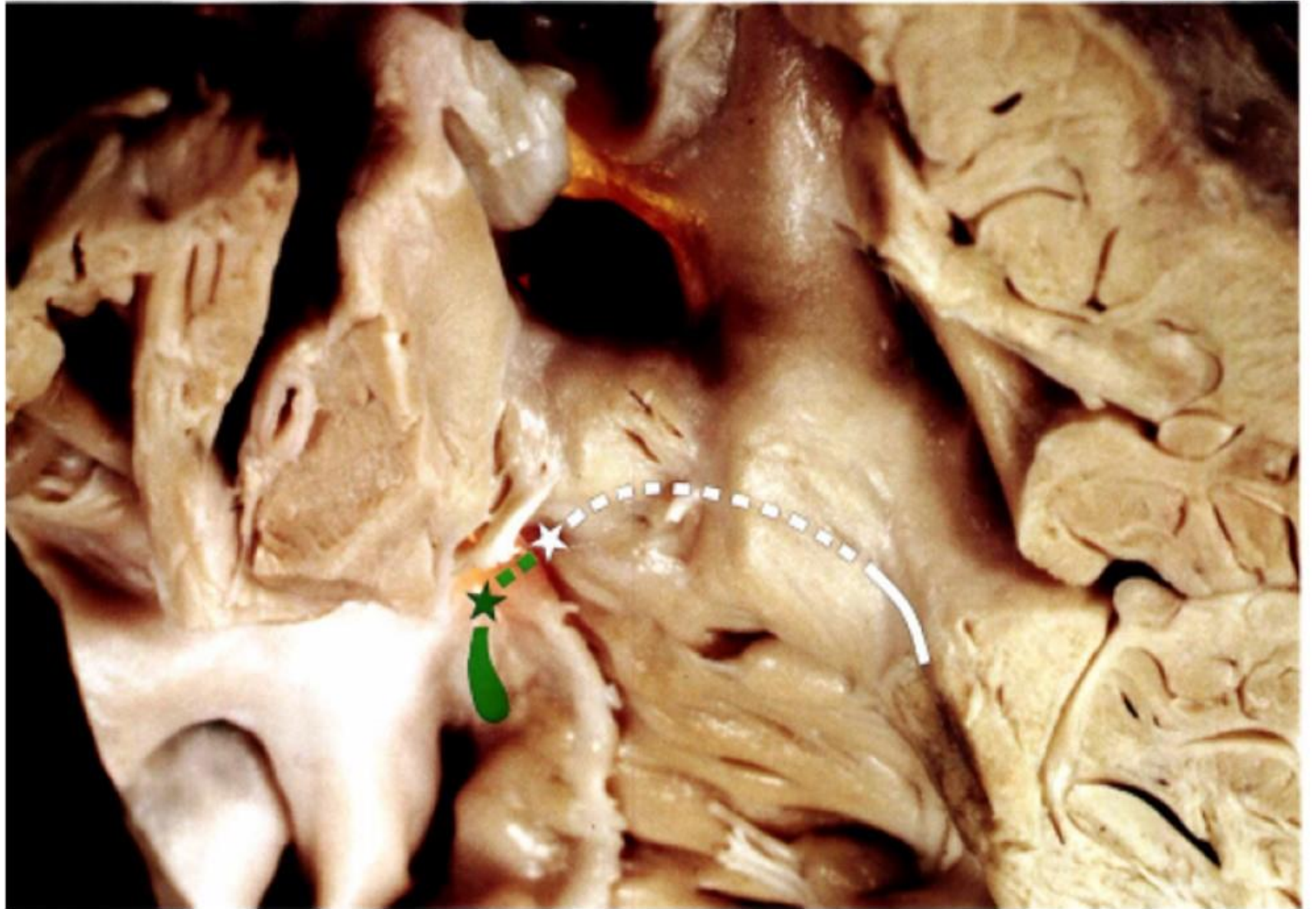
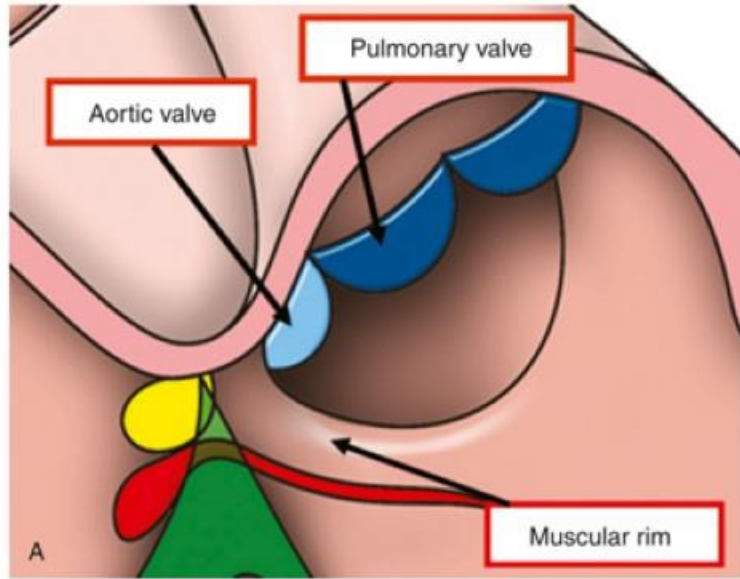
c perimembranous inlet



b perimembranous trabecular

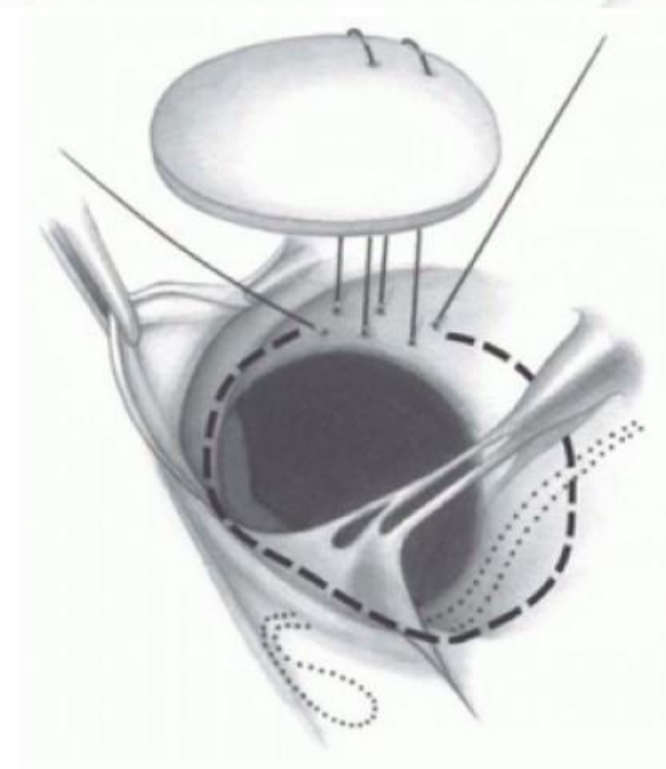
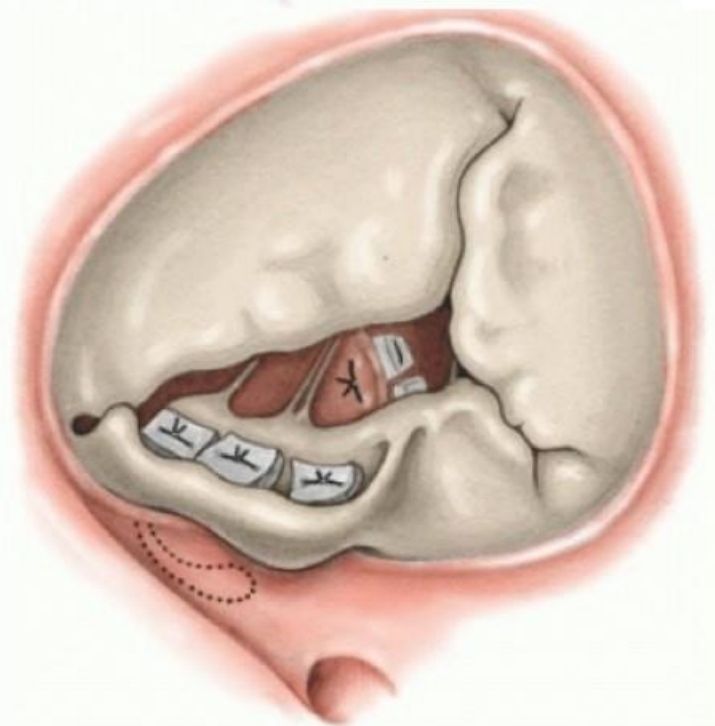
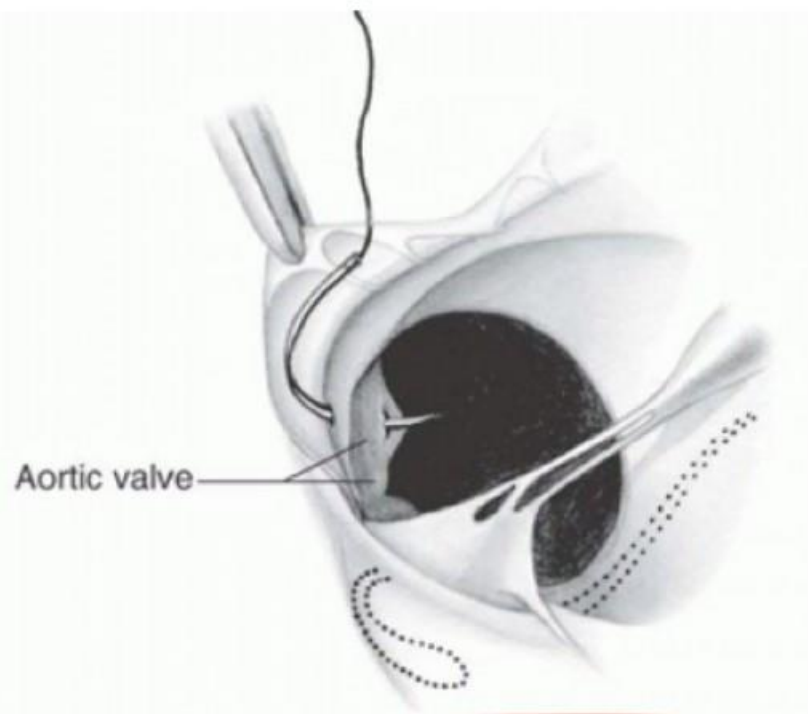


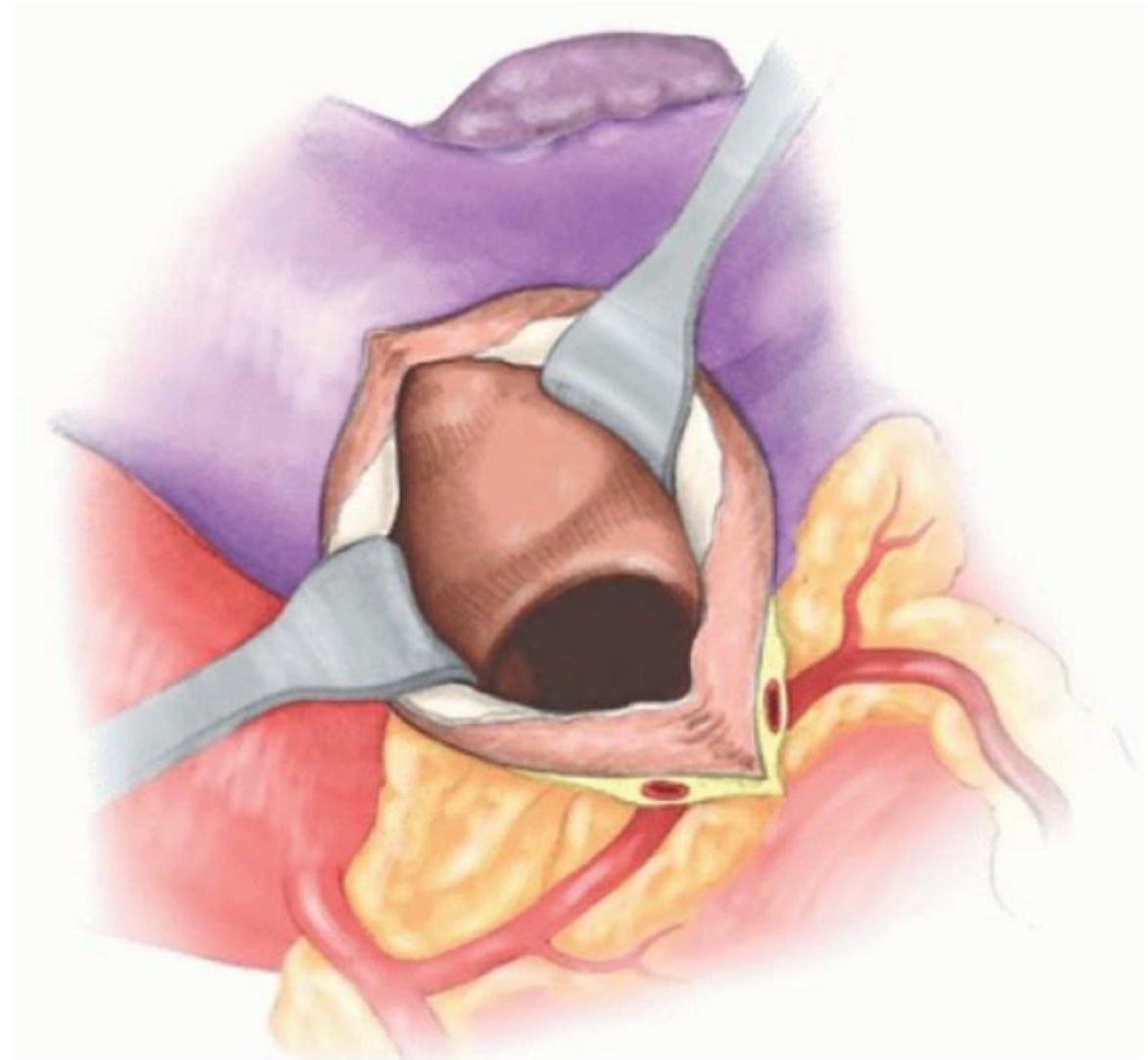
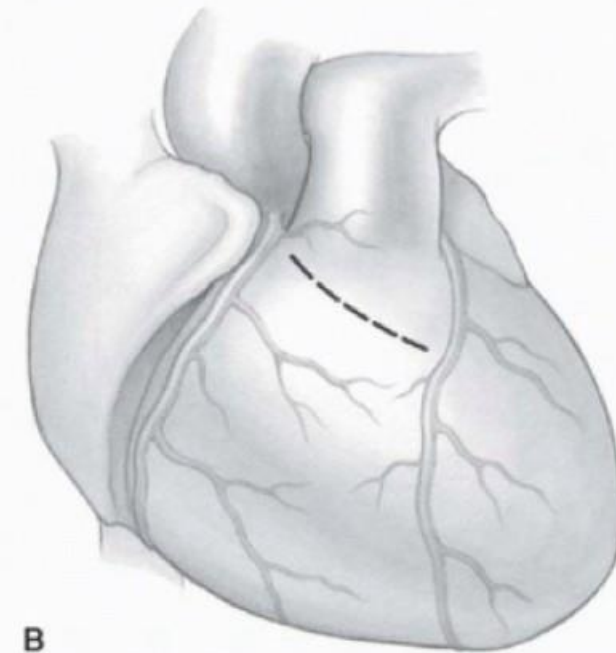
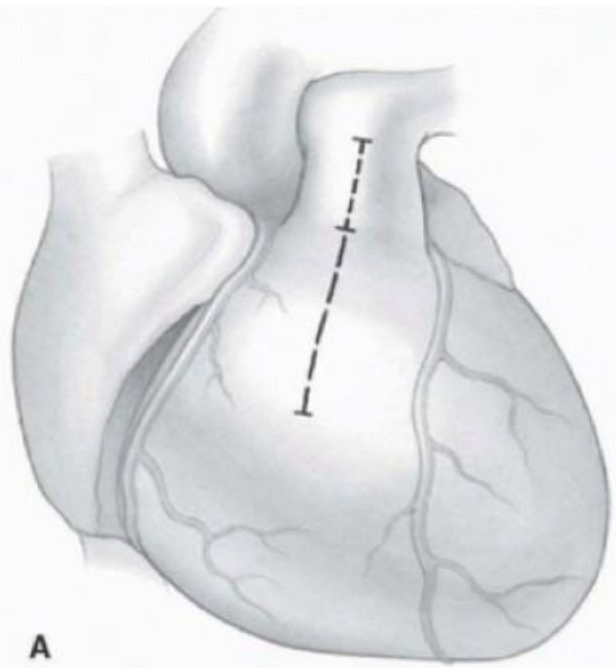
d muscular inlet



# Complications

- Injury to anatomic structure
  - Conduction system
    - Complete AV block – pacemaker 1~2%
  - Aortic valve
    - Inappropriate incision of TV, placement of sutures
    - Aortic insufficiency -> hemolysis
  - TV insufficiency
    - Chordae shortened
    - TV septal leaflet injury
- Incomplete closure
  - Inadequate exposure, disruption of sutures,
  - Less than 5%
  - Reoperation :  $Q_p/Q_s > 1.5$
  - TEE
- Conduct cardiopulmonary bypass
  - Related to deep hypothermia, circulatory arrest







# Postoperative Care

- Complete AV dissociation after CPB
  - Temporary pacing
  - Usually permanent beyond 10 to 14 days
- Pulmonary hypertensive crisis
  - In older patient with large L-R shunt or PV obstruction

# Pulmonary hypertensive crisis

- A serious syndrome characterized by an acute rise in pulmonary artery pressure followed by a reduction in cardiac output and, in patients with residual intracardiac shunts, a fall in systemic oxygen saturation.

Precipitating condition	Management
Noxious stimuli	Sedation
(tracheal suctioning)	Neuromuscular blocker
Hypoxemia	High O2 ventilation
Hypothermia	Hyperventilation
Acidosis	inhaled NO

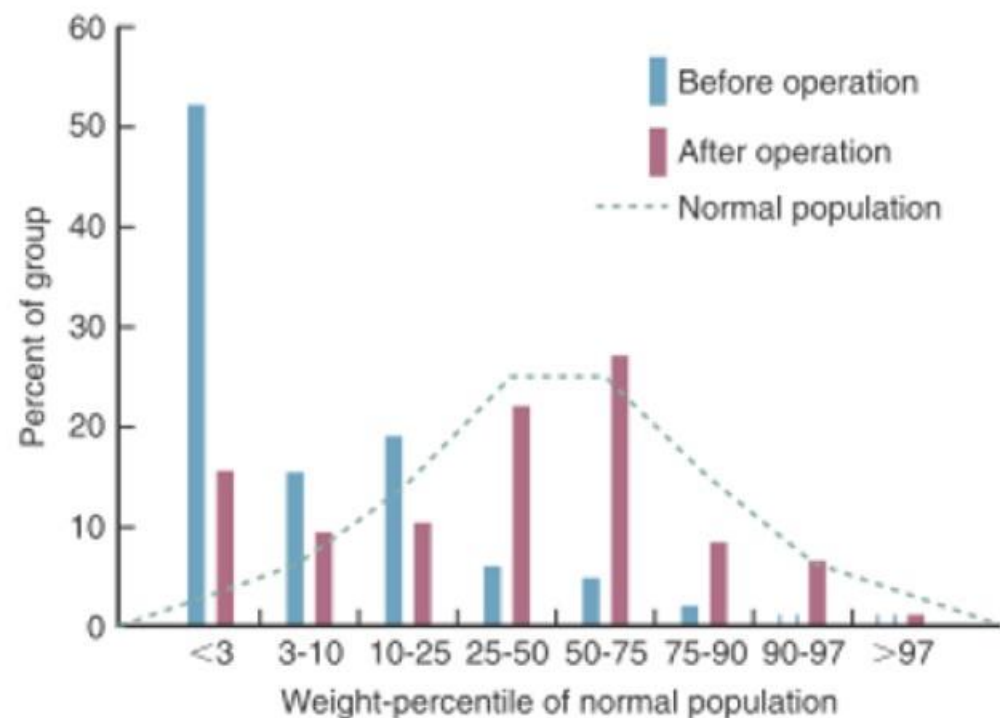


# Early Results of Surgery

- Hospital mortality rate : 1% or less
  - Very low-weight or very young
  - Elevated PVR
  - multiple
- Residual VSD
  - Suture dehiscence in small infants with friable myocardium
  - Small (<3mm) ones close spontaneously over a period of months

# Late Results

- Normal or almost normal
  - Life expectancy, Growth and cardiac function
- Limitation in exercise tolerance
  - Persistent pulmonary HTN and increased PVR
- Mortality
  - Correction after the age of 5 years,  $PVR > 7$
  - Complete heart block.



**Figure 35-27** Changes in weight after repair of ventricular septal defect in 96 patients aged 10 years or less, with ratio of pulmonary and systemic pressures greater than 0.45 and ratio of pulmonary and systemic resistances less than 0.75 preoperatively. (From Cartmill and colleagues.<sup>64</sup>)



*Thank for your attention*

**ppippo77@gmail.com**

