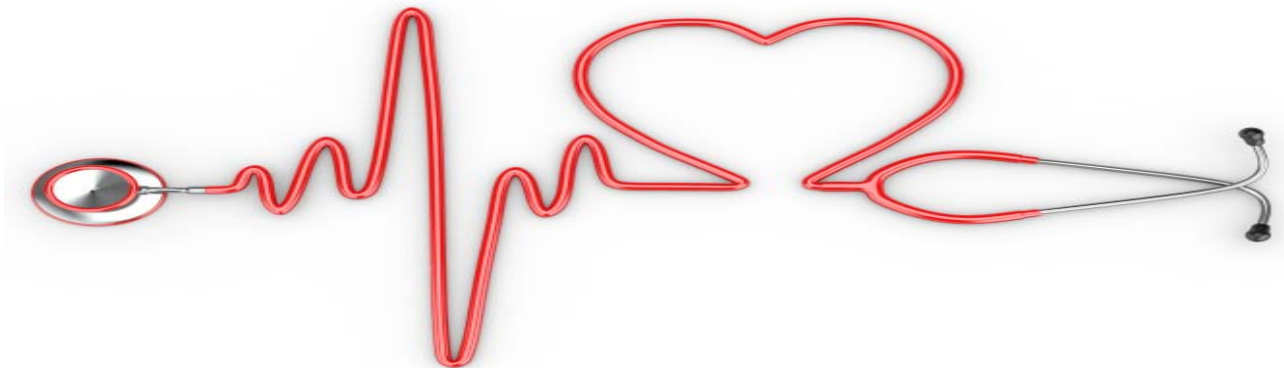


# Cardiovascular System Examination

Part 2

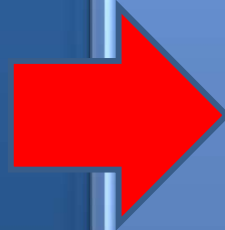
Farah Abuazzam





**Precordium**

**Before  
Examination**

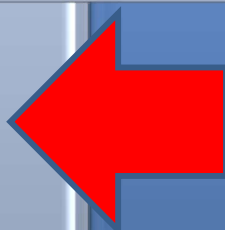


**Inspection**

**CVS  
examination**



**Auscultation**



**Palpation**

# Before Examination

**Introduce  
your self**

**Take  
permission**

**Explain**

**Privacy and  
ask for  
chaperon**

**Good light**


**Ideal  
Position**

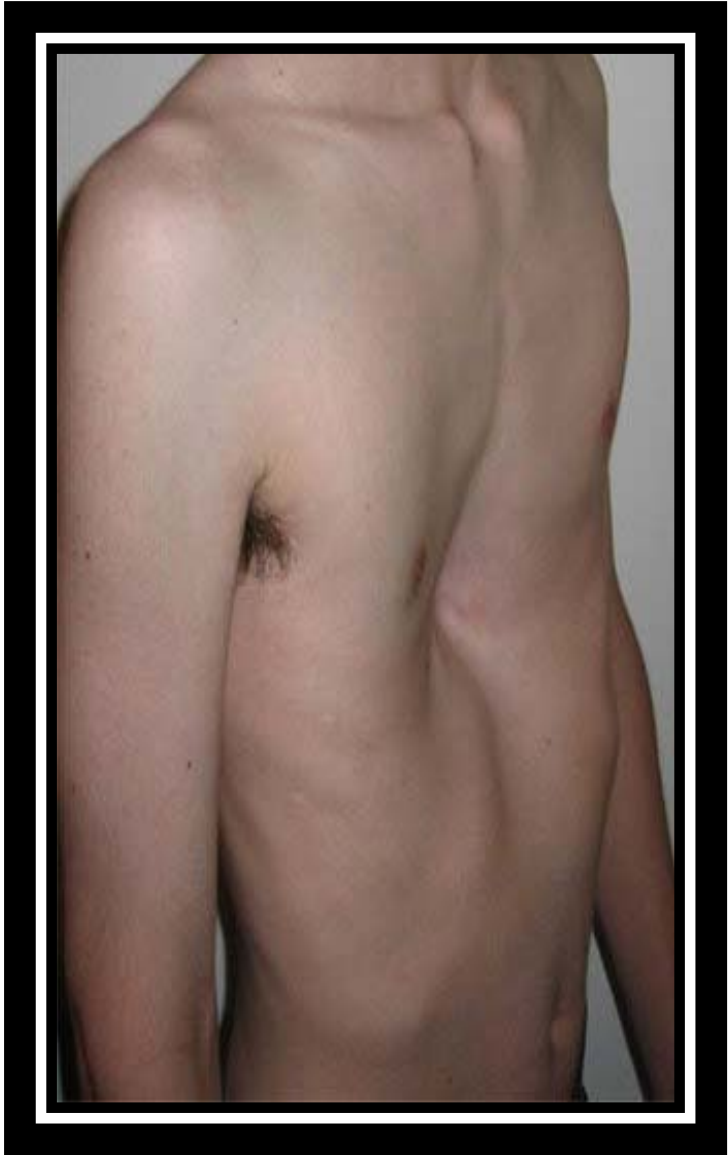
**Exposure**



# Inspection

**From the foot of the pt:**

- 
- **Symmetry**
  - **Deformity**
  - **Moves with respiration**



# From the right side:



- **Hair distribution**



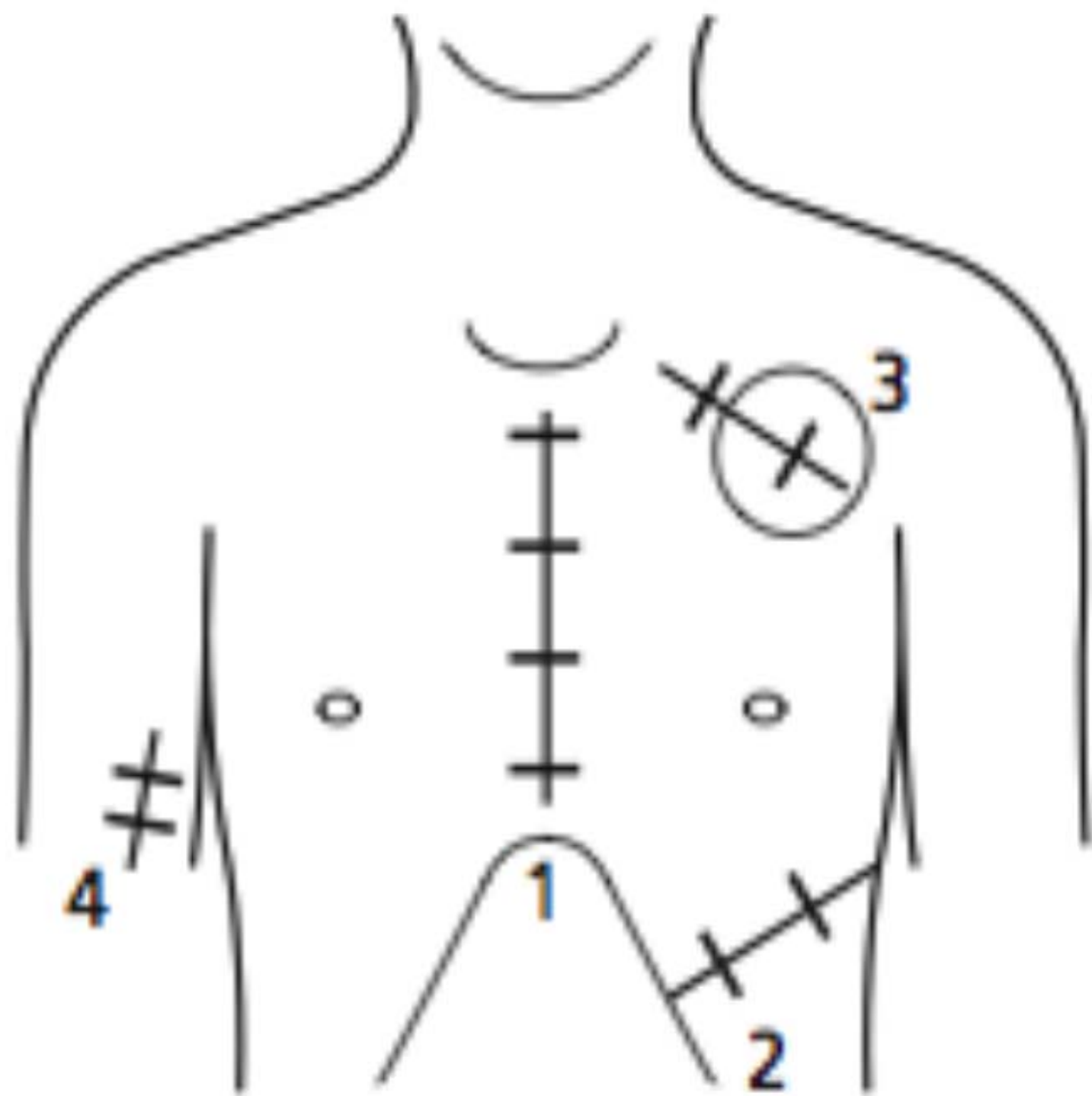
- **Scars and skin lesions**



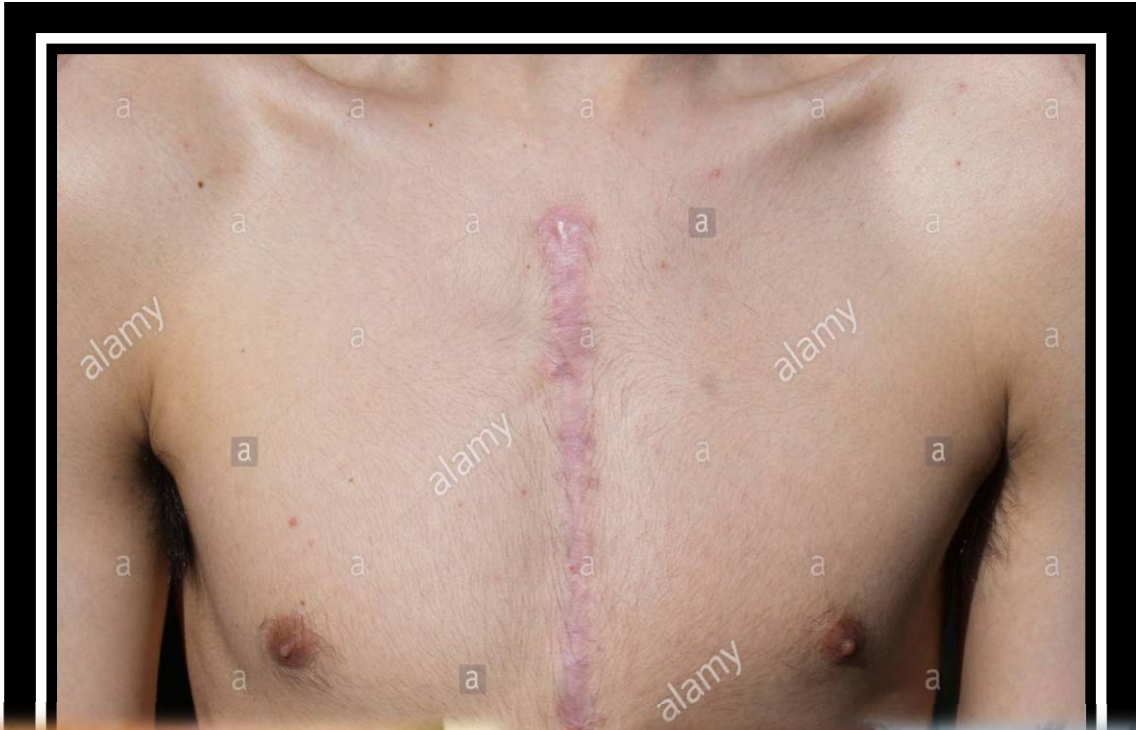
- **Dilated veins**

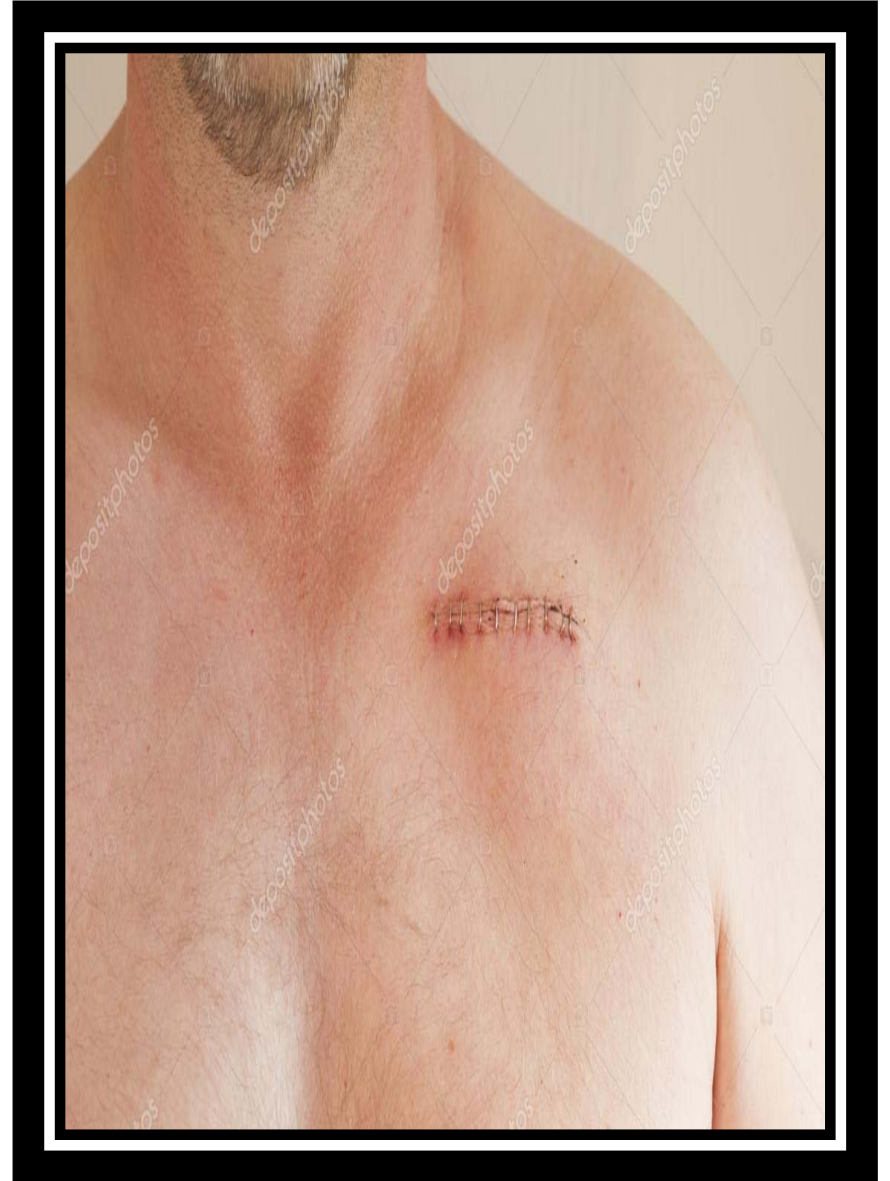
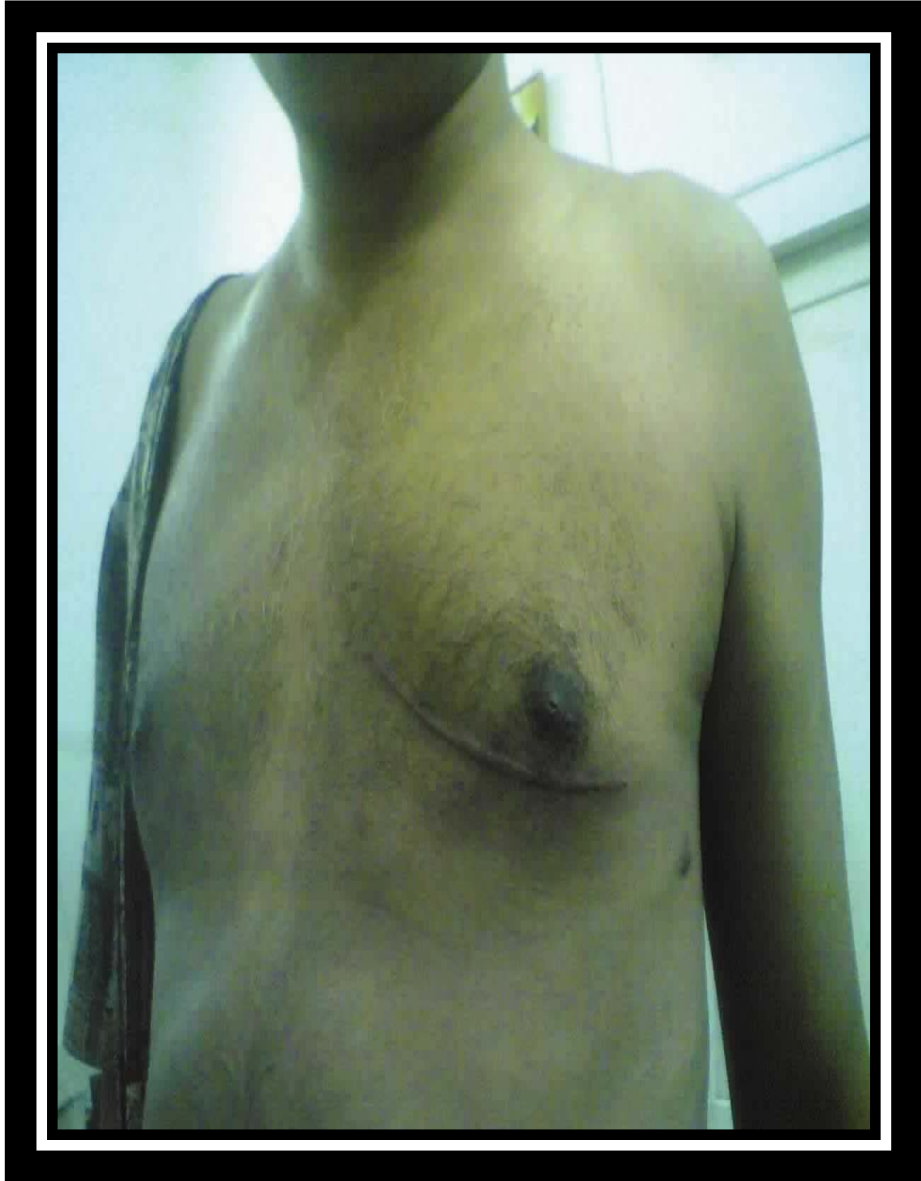


- **Visible pulsation and apex beat**



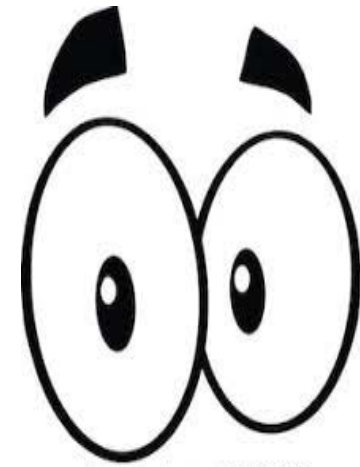






# Palpation

**Eye contact**



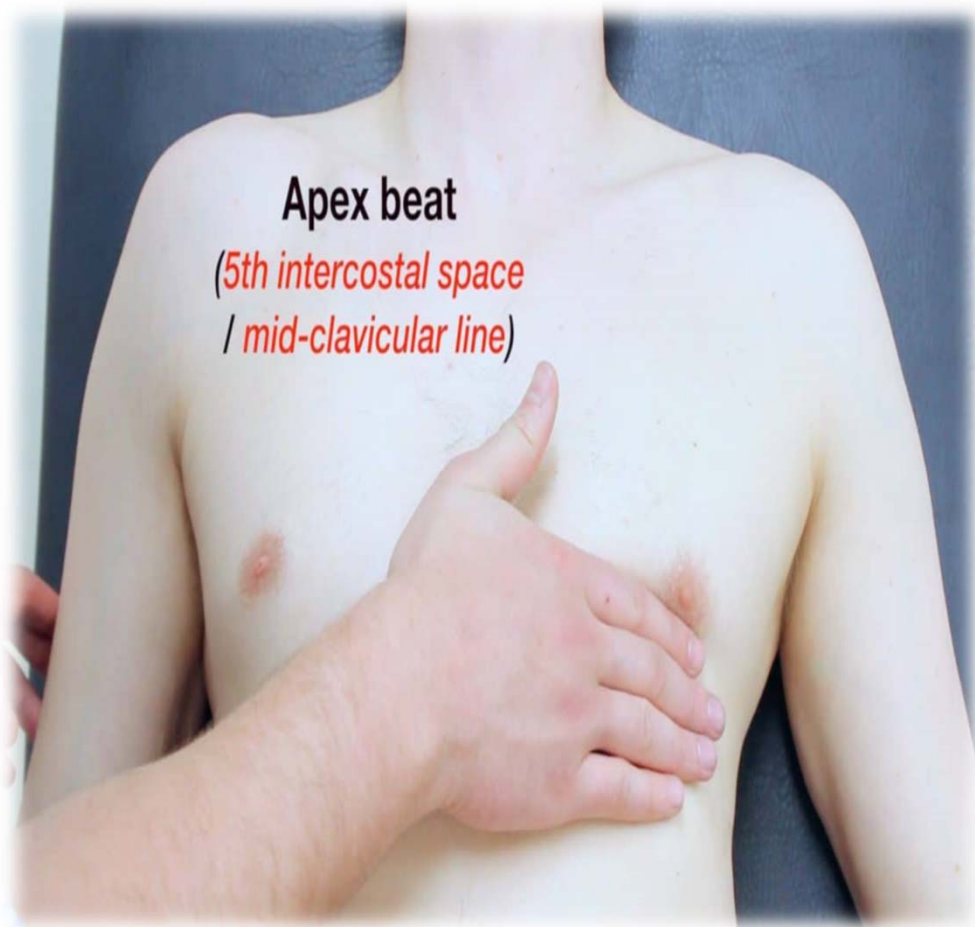
shutterstock.com - 177695390

**Ask about tender areas**



# 1. Apex beat

## position and character



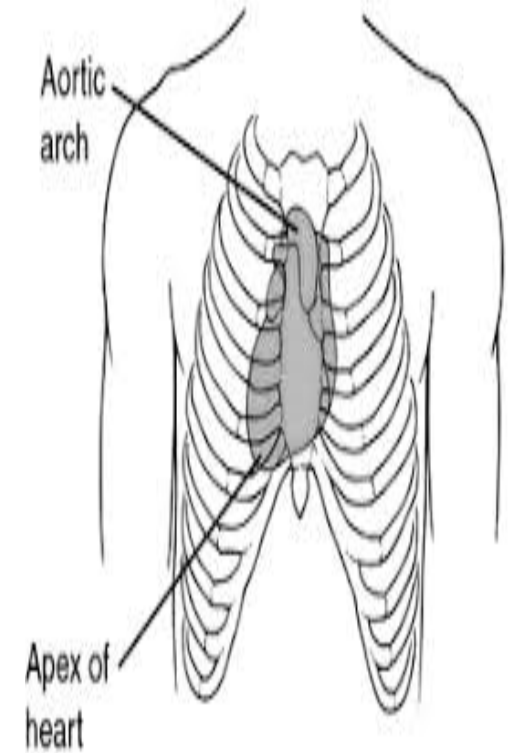
- General palpation using flat of your right hand over the precordium for general impression, then locate it by your fingers lying parallel to ICS then locate with 2 fingers.
- If not palpable, roll the patient to the left side

\*\* **Position:** Lt 5<sup>th</sup> ICS, mid-clavicular line

\*\* **Character:** gentle tapping

## Abnormal location of apex beat:

- Impalpable apex beat
- Displaced inferiorly and laterally
- Palpable on right side



## Abnormal Character of apex beat:

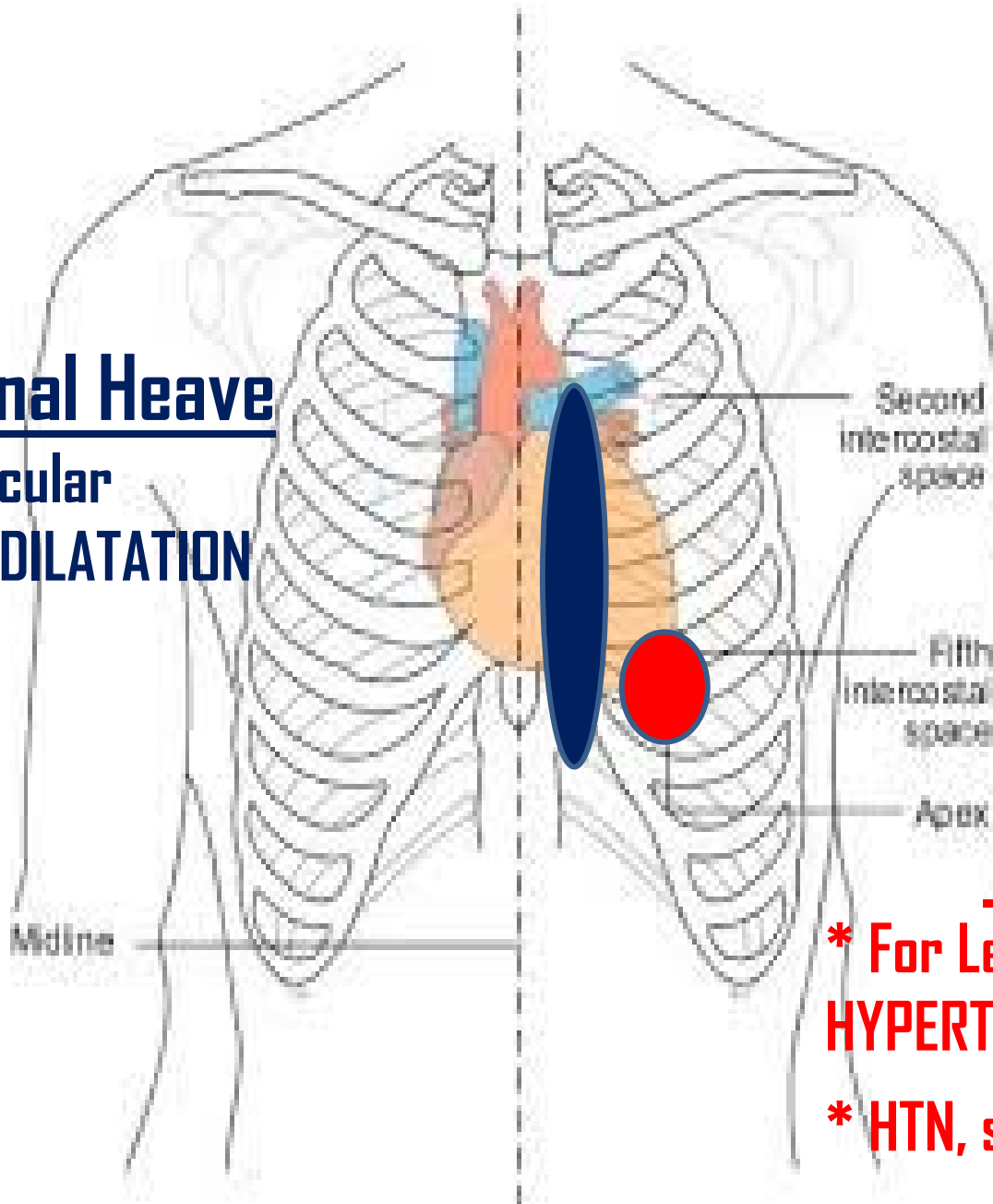
- Forceful pulsation (APICAL HEAVE)
- Tapping apex beat
- Double apical impulse

## **2. Heave**

- **Abnormal palpable impulse that noticeably lifts your hand**
- **Palpate with the heel of your right hand firmly over 2 areas:**
  - 1) Lt lower parasternal area (hold breath in expiration)**
  - 2) Apex area**

## Left parasternal Heave

- \* For Right Ventricular HYPERTROPHY or DILATATION
- \* Pulmonary HTN



## Apical Heave

- \* For Left Ventricular HYPERTROPHY
- \* HTN, severe aortic stenosis



# 3. Thrill

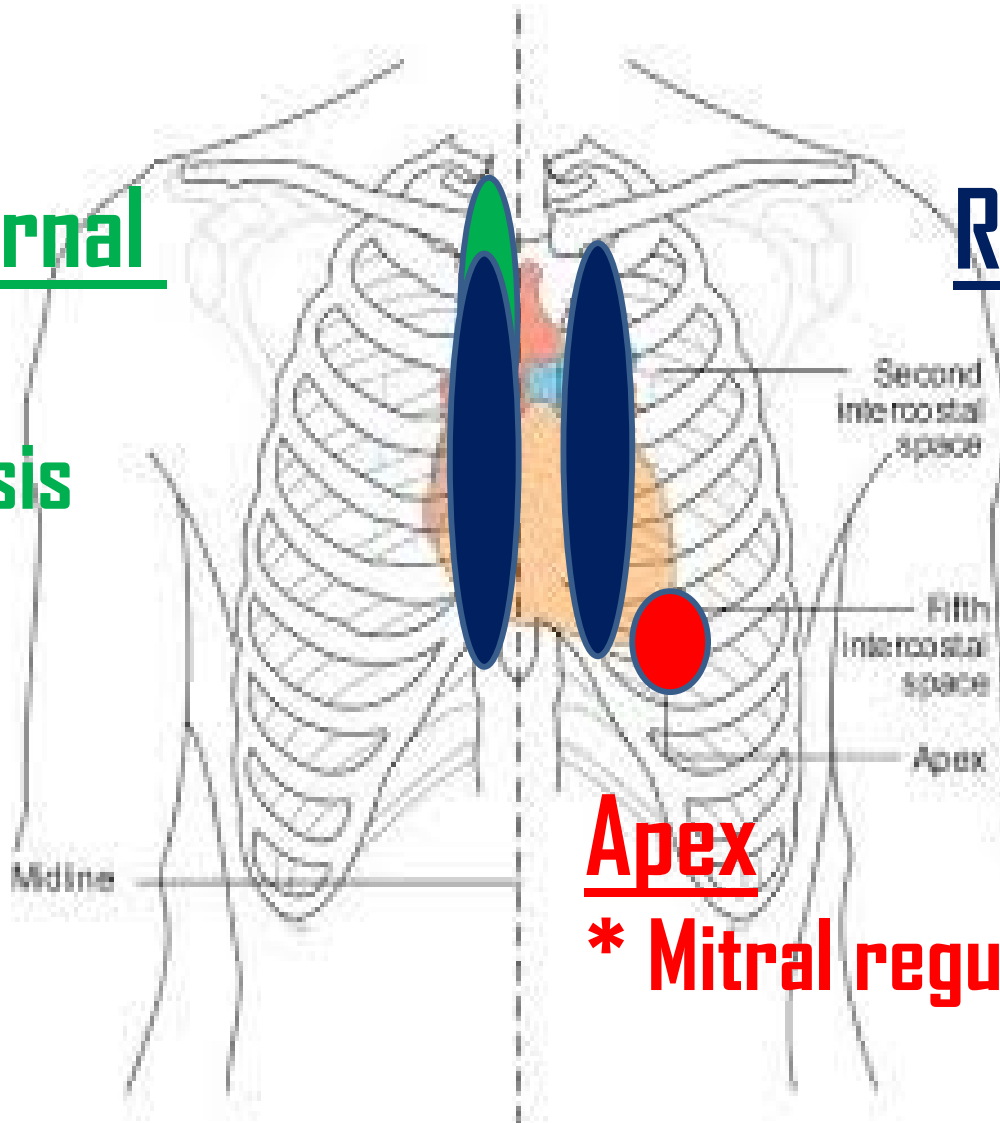
- The tactile equivalent of a murmur, palpable vibration  
( **PALPABLE MURMUR**)

Palpate with the palmar aspect of fingers ( **PLACED VERTICALLY**) over 3 areas:

- 1) Apex
- 2) Left parasternal area
- 3) Right parasternal area

Rt upper sternal border

\* Aortic stenosis



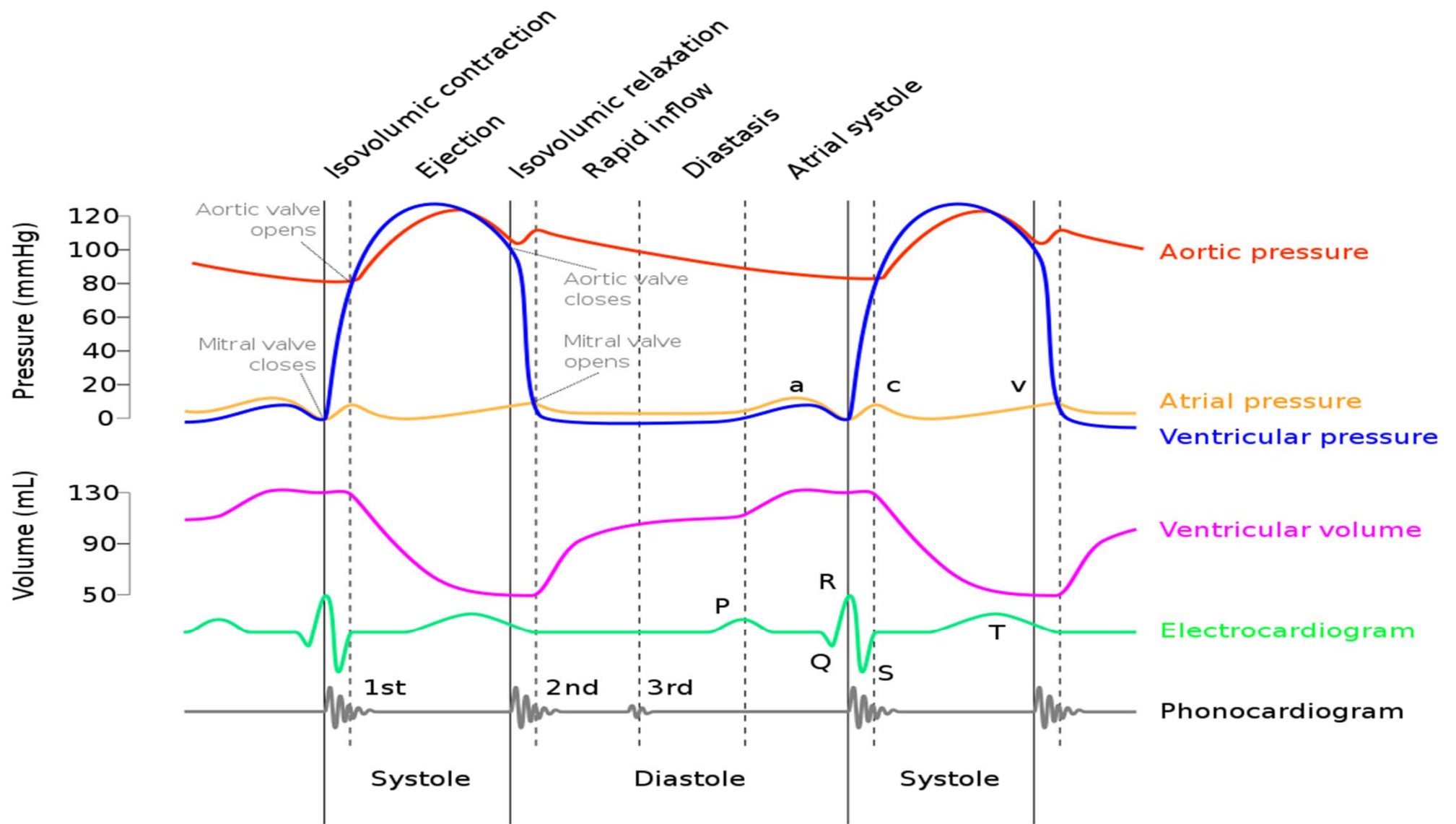
Rt and Lt sternal borders

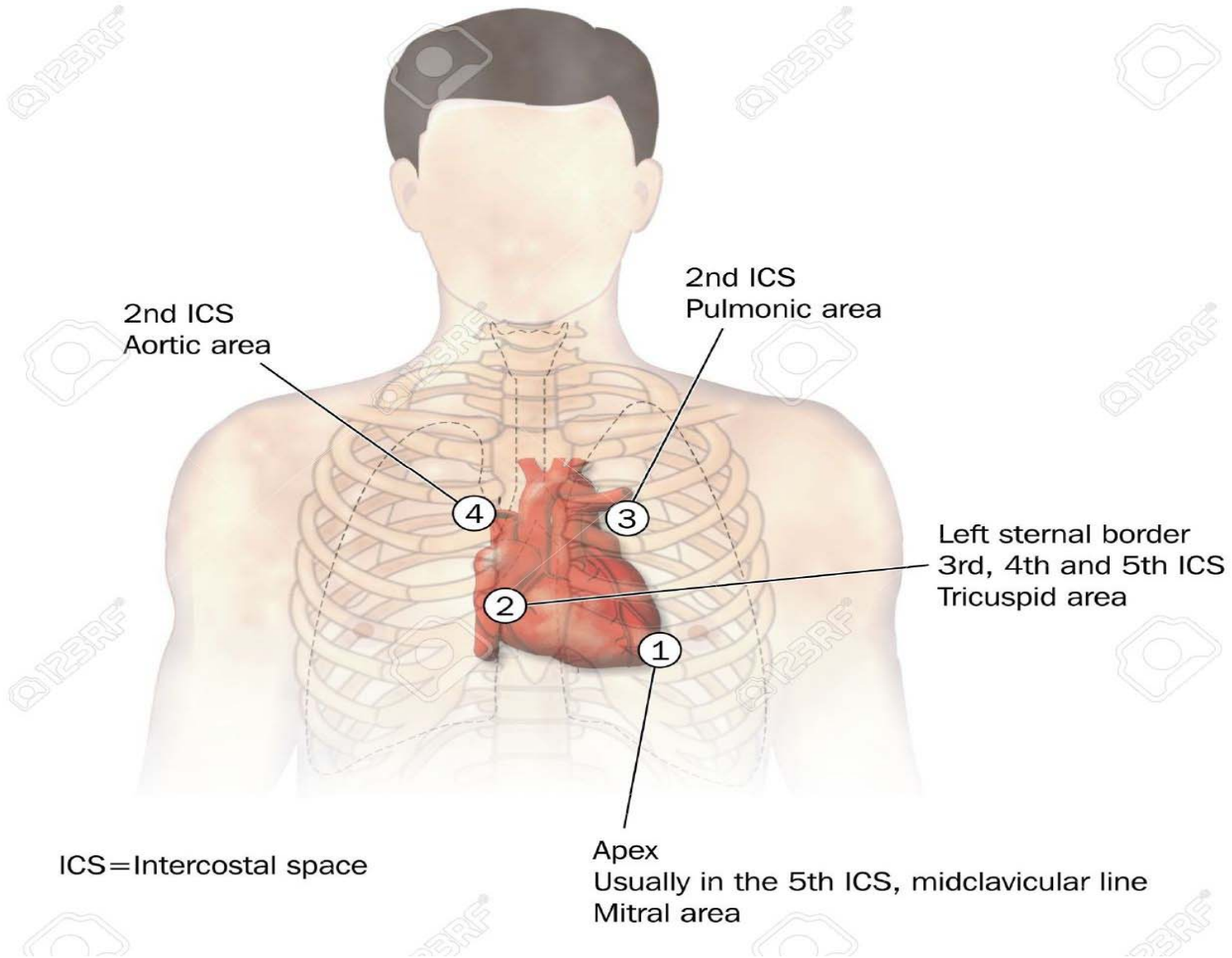
\* VSD

Apex

\* Mitral regurgitation

# Auscultation





# Heart sounds



**PLEASE,  
PUT ON HEADPHONES**

## First heart sound, S1

- **Closure of mitral and tricuspid valve**
- **At onset of ventricular systole**
- **Heard at the apex**

# Abnormal intensity of S1

## Quiet

- Low cardiac output
- Poor Lt ventricular function
- Rheumatic mitral regurgitation
- Long PR interval

## Loud

- Increased cardiac output
- Large stroke volume
- Mitral stenosis
- Short PR interval
- Atrial myxoma

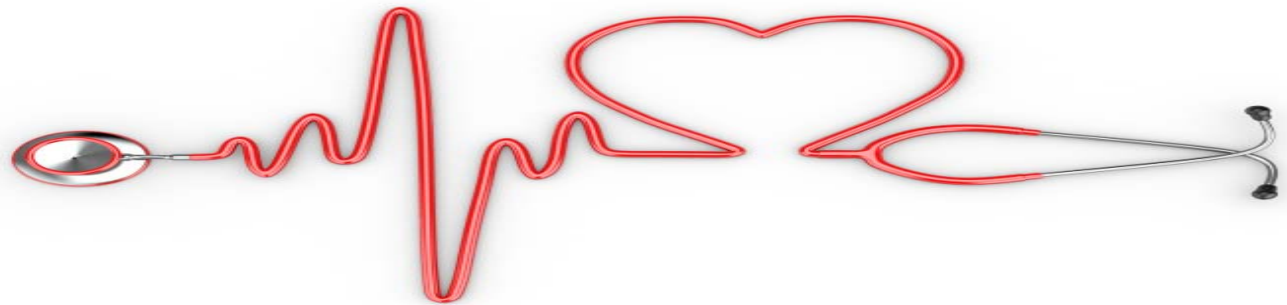
## Variable

- Atrial fibrillation
- Complete heart block
- Extrasystole



# Second heart sound, S2

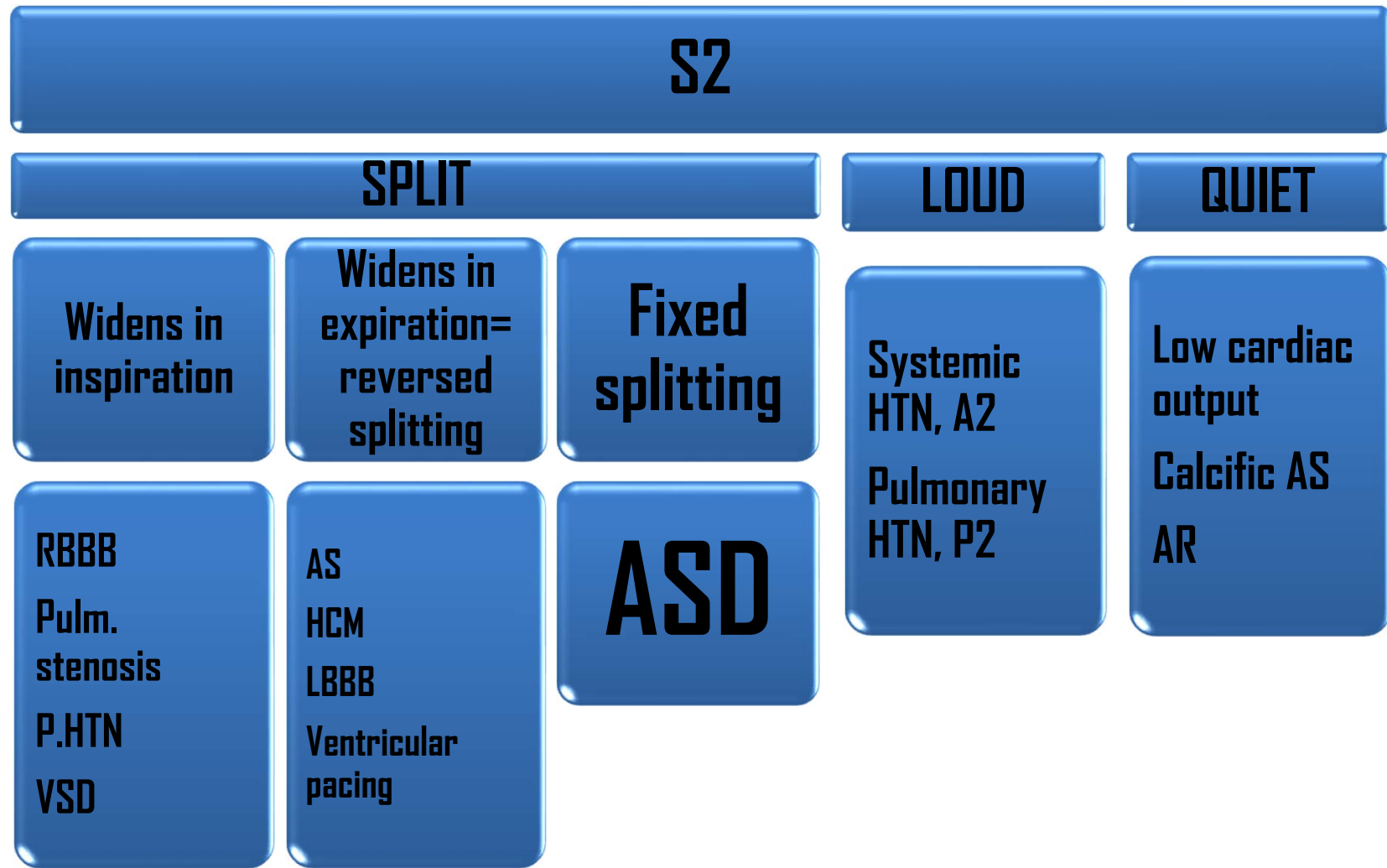
- Closure of Aortic and pulmonic valves.
- At end of ventricular systole.
- Heard on **left sternal edge**.
- Has 2 components;
  - 1) aortic component A2
  - 2) Pulmonic component P2

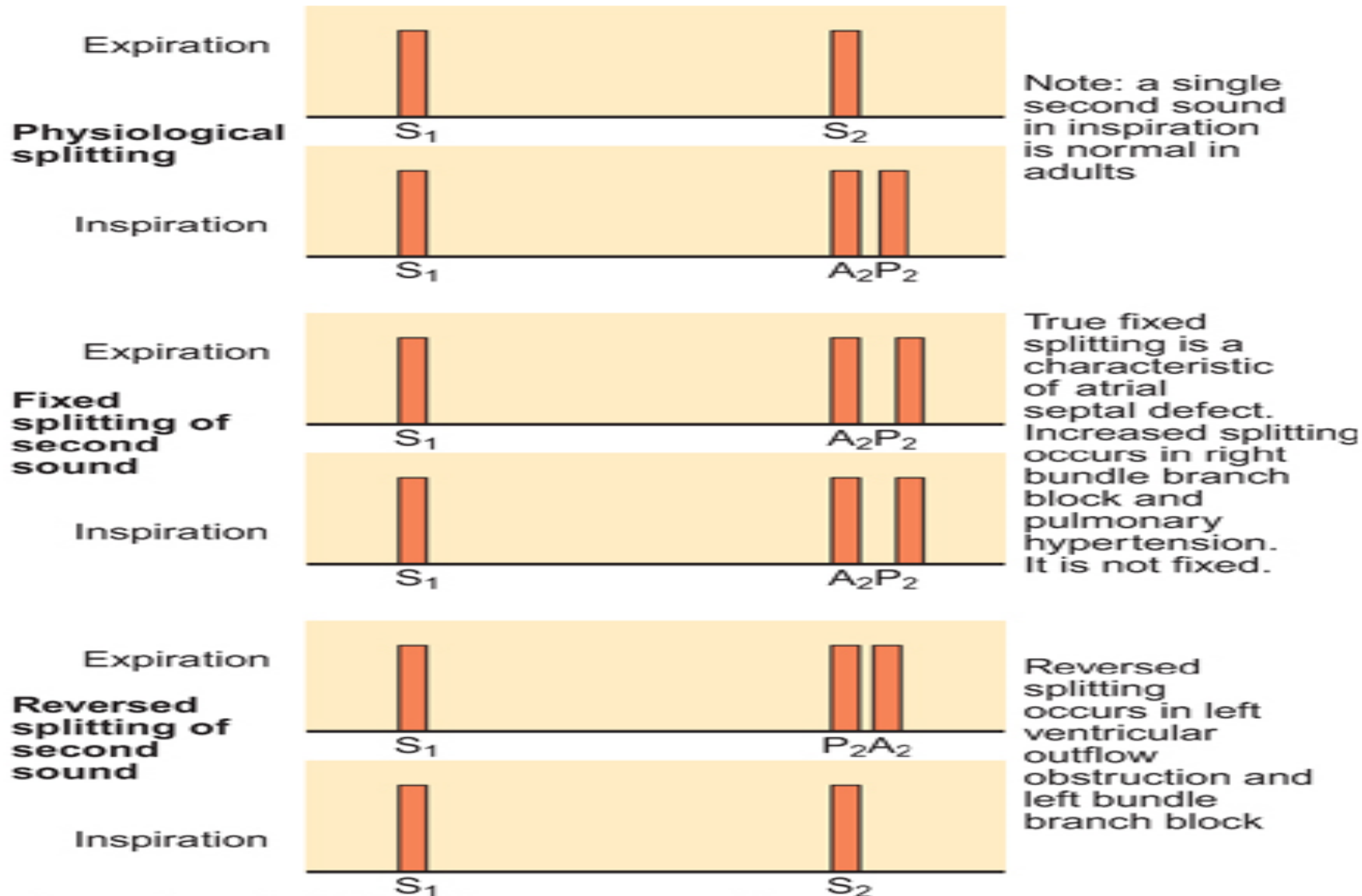


# S2 splitting

- Normally A2 is louder than P2.
- Physiological splitting occurs because LV contraction slightly precedes RV contraction.
- This splitting physiologically increases at end-inspiration (RV VR-related), and disappears on expiration.

# Abnormal intensity and splitting of S2

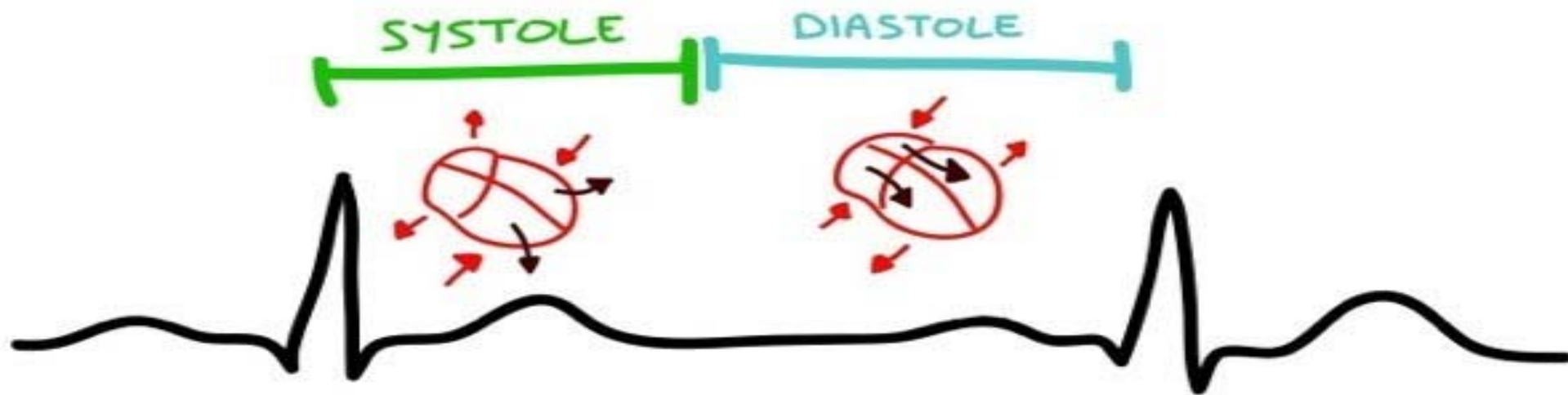




Note: a single second sound in inspiration is normal in adults

True fixed splitting is a characteristic of atrial septal defect. Increased splitting occurs in right bundle branch block and pulmonary hypertension. It is not fixed.

Reversed splitting occurs in left ventricular outflow obstruction and left bundle branch block



S<sub>3</sub>

S<sub>4</sub> S<sub>1</sub>

S<sub>2</sub> S<sub>3</sub>

S<sub>4</sub> S<sub>1</sub>

S<sub>2</sub>



Dilated Ventricle  
"TO — RON — TO"



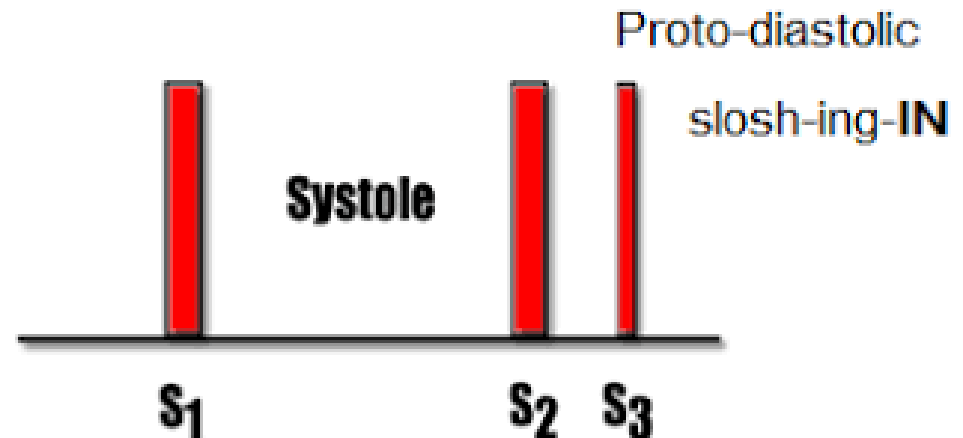
"Stiff Ventricle"  
"KEN — TUCK — KY"

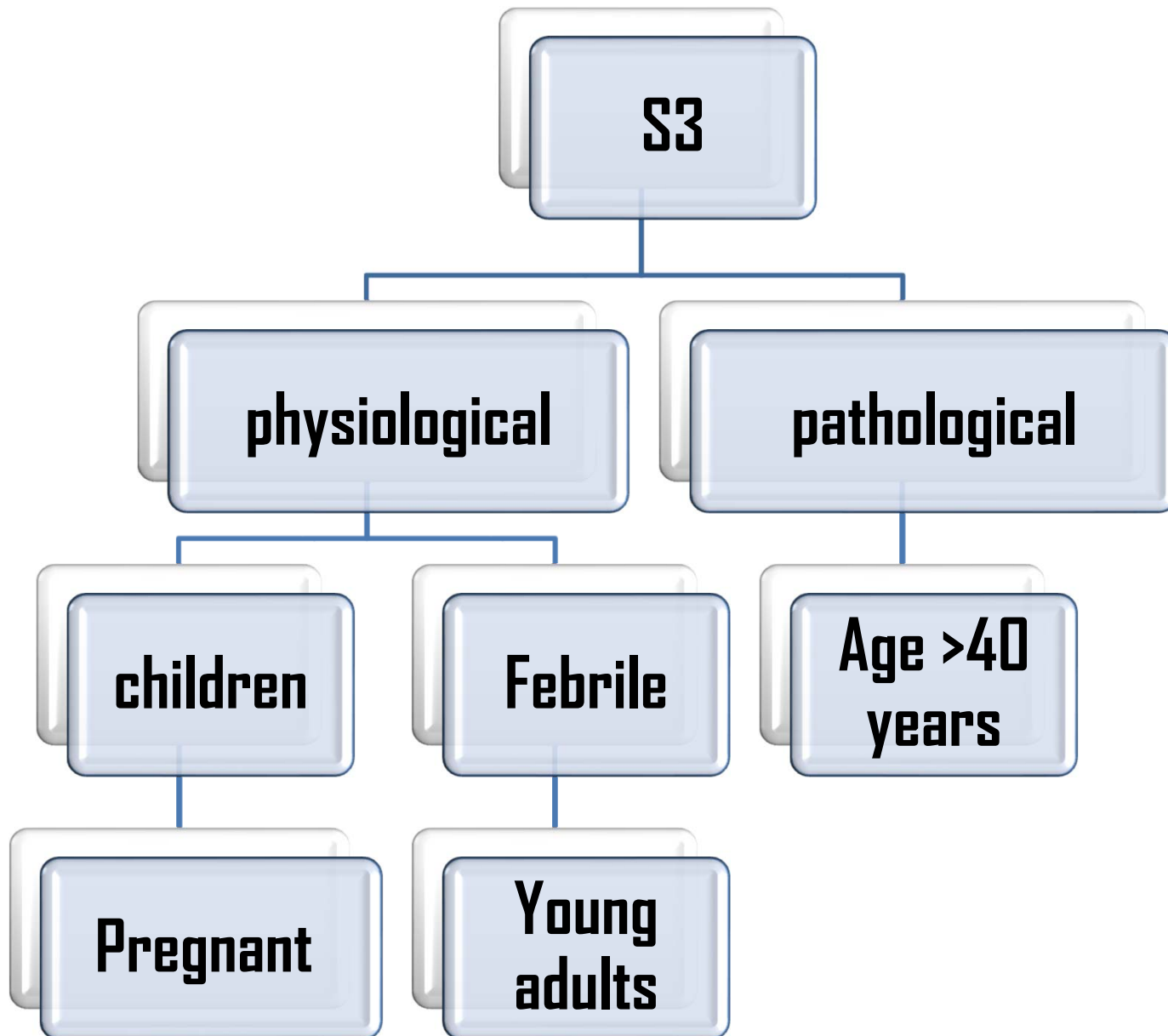
sketchymedicine.com

## Third heart sound, S3

- Low-pitched early diastolic sound.
- Best heard with the **bell** at the **apex**.
- **Due to rapid ventricular filling immediately after opening the atrioventricular valve**

Please put on headphones





## Pathological S3 causes:

1) LV failure

2) MR

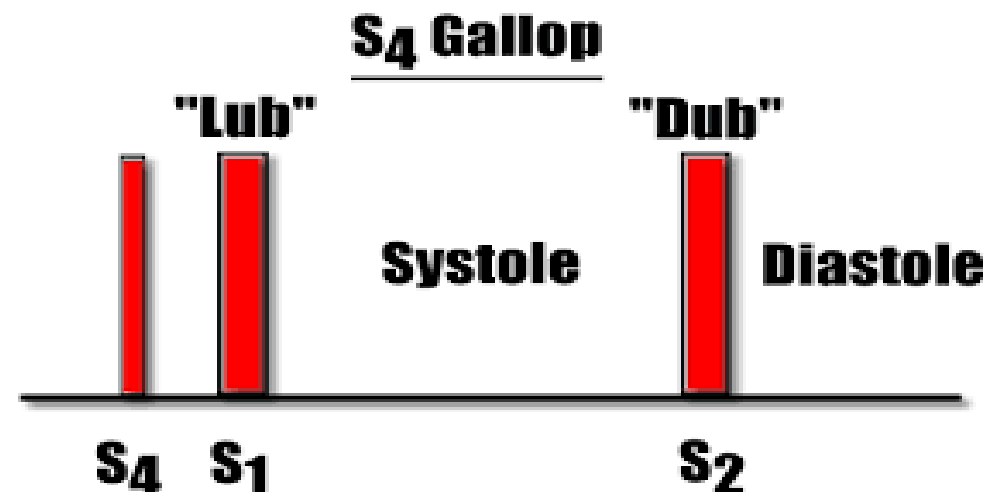
- **Ventricular gallop = S3 gallop = S3+ tachycardia**

**In HF, with quiet S1 and S2**



## Fourth heart sounds, S4

- **ALWAYS PATHOLOGICAL**
- **Soft low-pitched sound at late diastole.**
- **Best heard at the **apex** with the **bell**.**
- **It occurs before S1**
- **Due to forceful atrial contraction against stiff ventricle secondary to LVH.**



- Causes of S4:

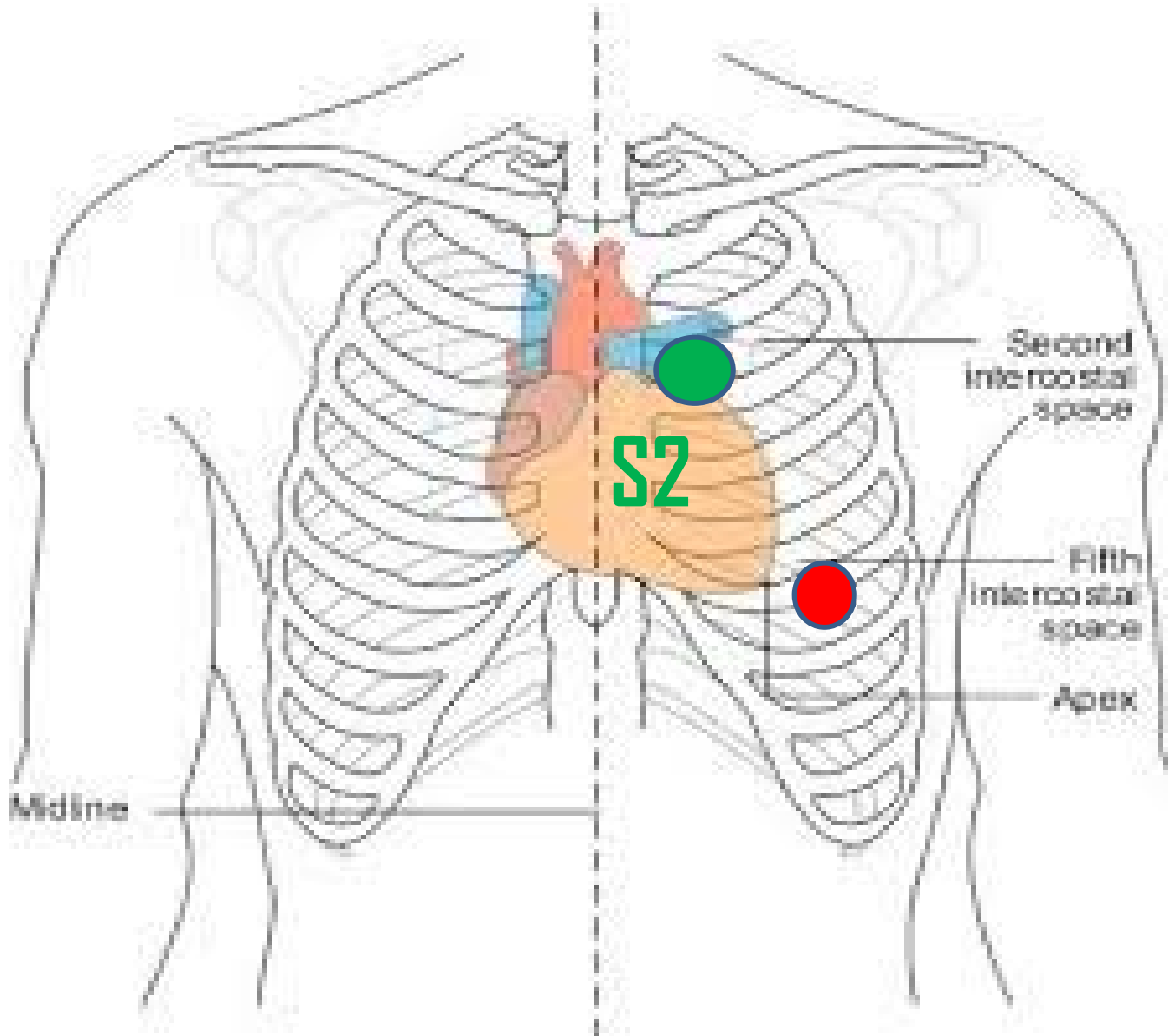
1) HTN

2) AS

3) HCM

**\*\* CANNOT OCCUR IN CASE OF ATRIAL FIBRILLATION.**

- **Atrial gallop= S4 gallop= S4+ tachycardia**

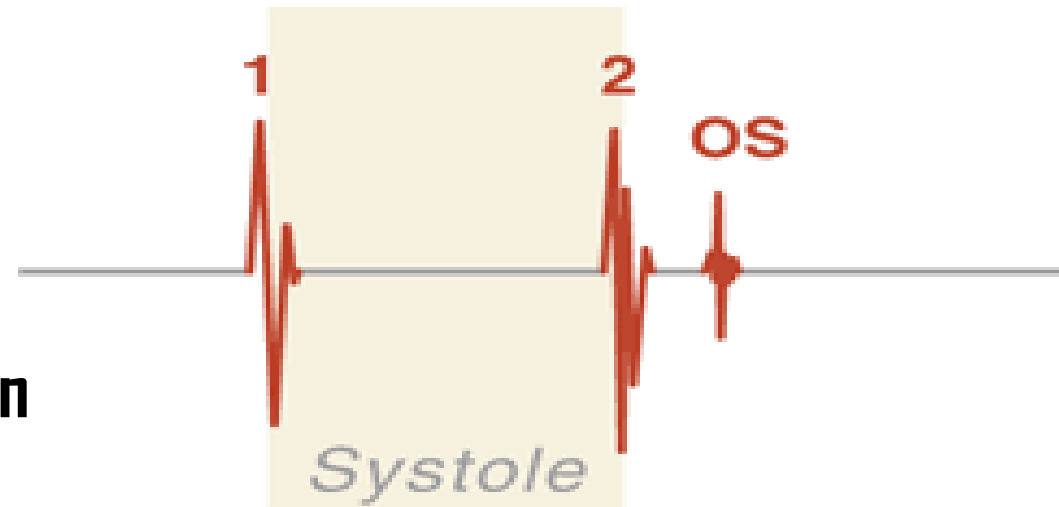


S2

S1  
S3  
S4

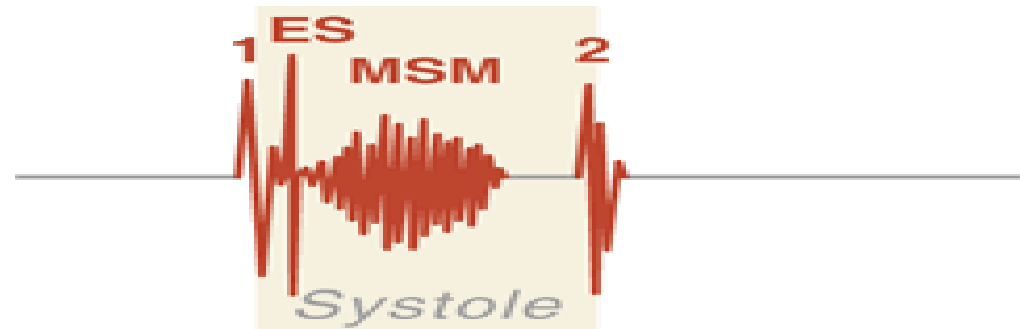
# Added Sounds

## Opening snap



- Sudden opening of stenosed valve in **DIASTOLE**.
- **MS**
- High-pitched, medial to **apex** via the diaphragm.
- Just after **S2**, in early **diastole**.

# Ejection click



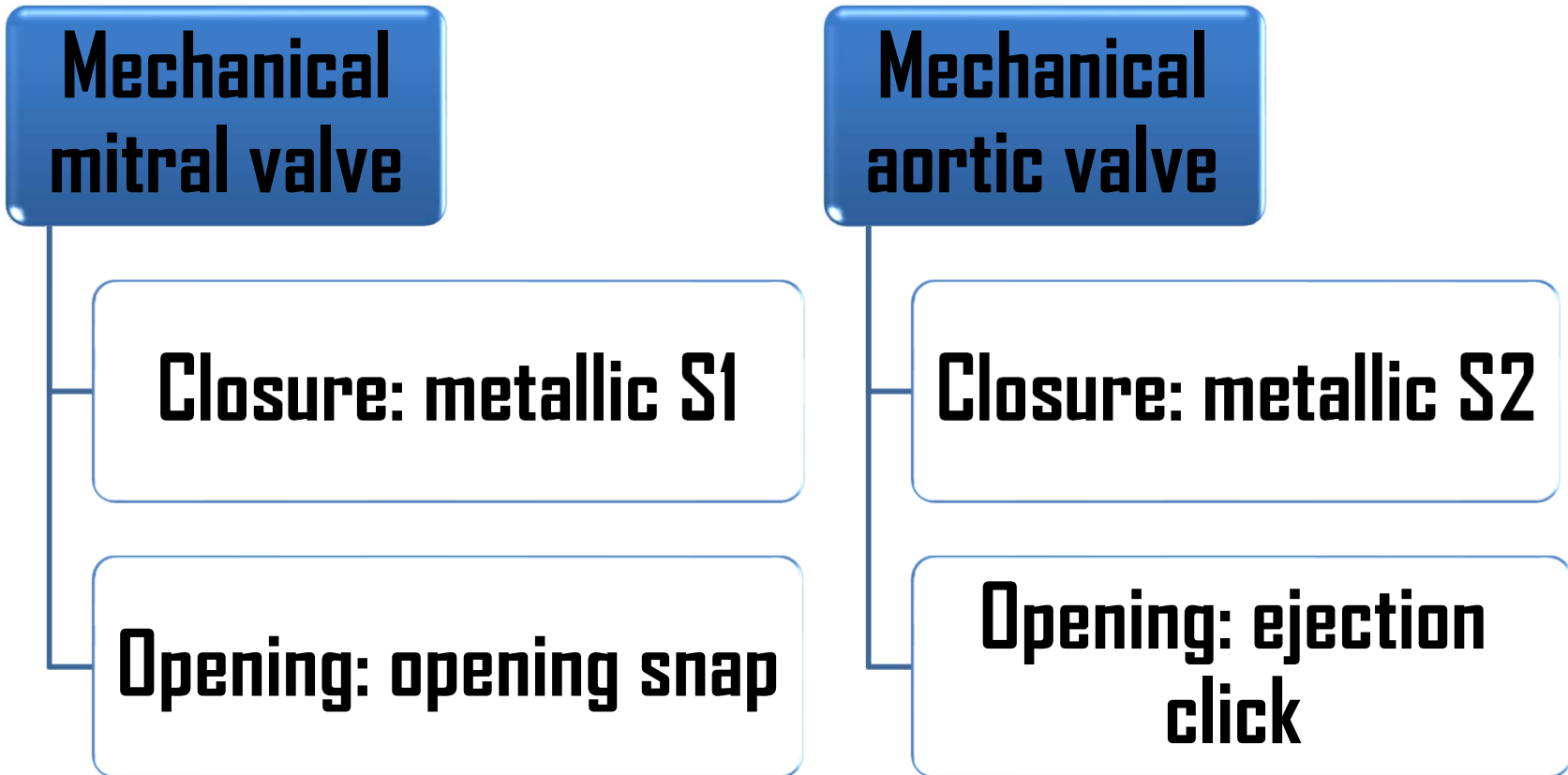
- Opening of stenosed valve in **SYSTOLE**.
- **Congenital pulm./aortic stenosis.**
- High-pitched, at the **Rt and Lt upper sternal borders** via diaphragm
- Just after **S1**, in early **systole**.

**\*\* if calcific valve (rigid cusps)>> absent sound**

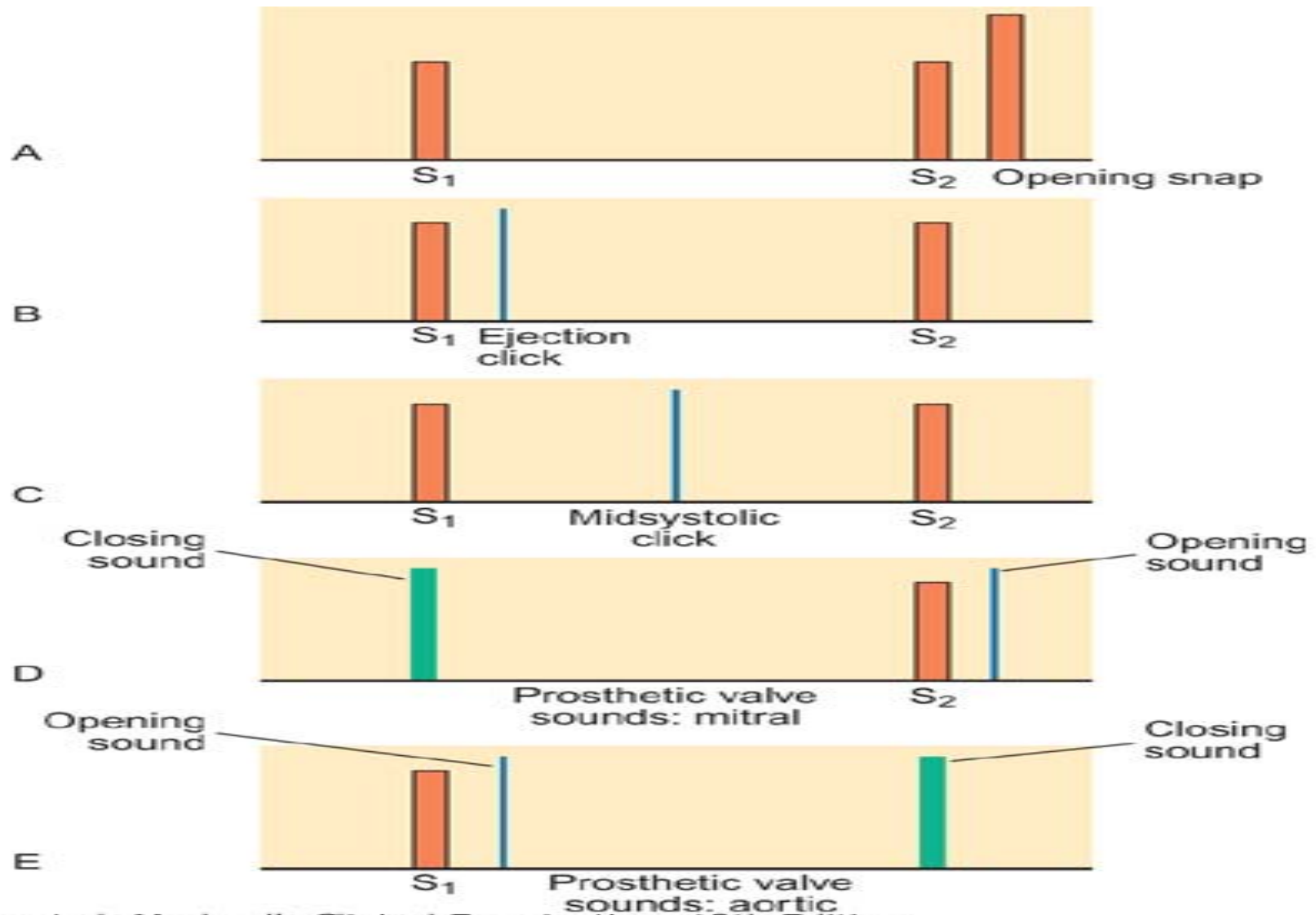


# Mechanical Heart Sounds

- High-pitched **metallic** and often palpable.







# Pericardial Friction Rub

- Coarse scratching sound.
- With the **diaphragm**, hold breath in expiration and lean forward.

• Causes:

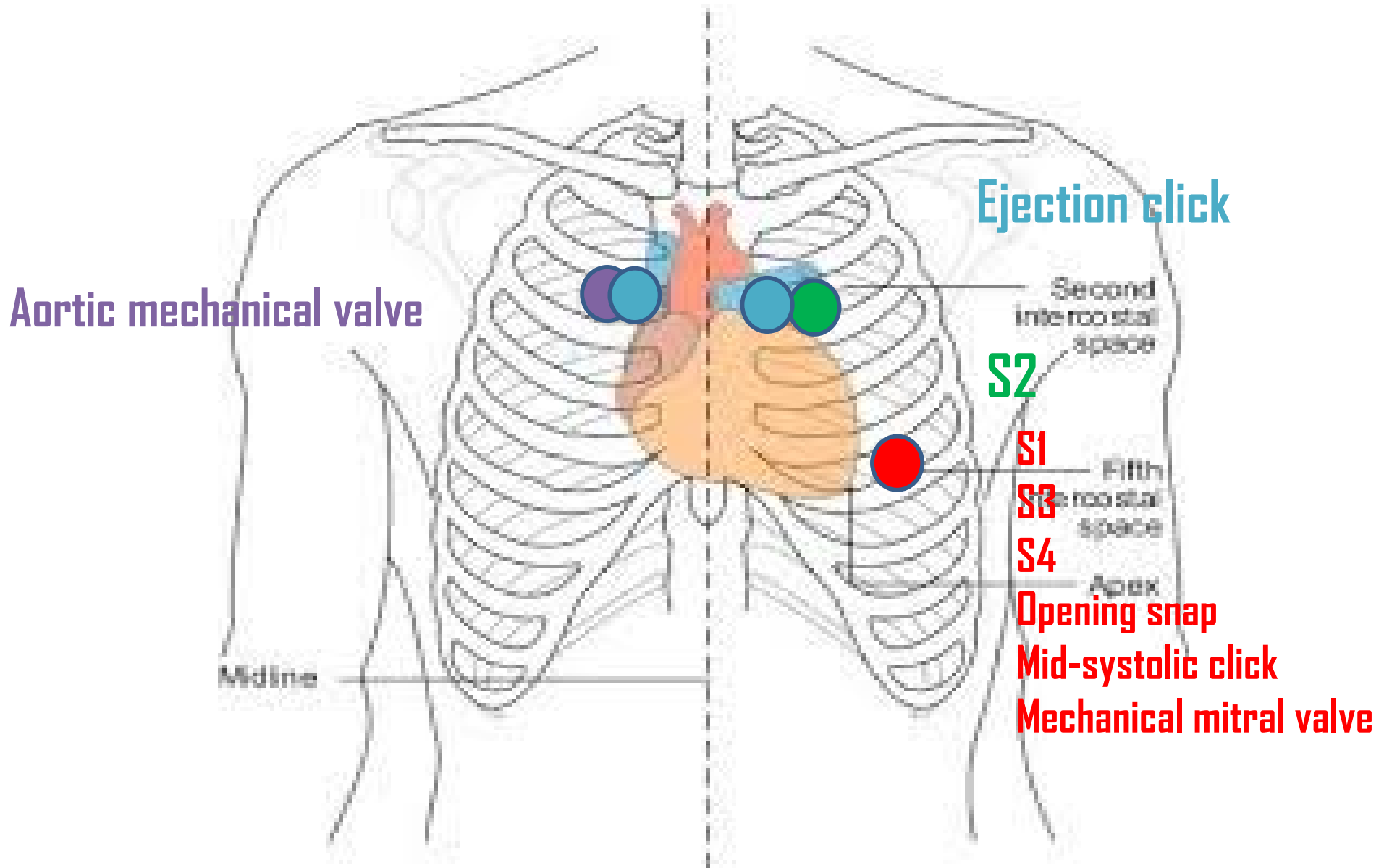
1) Acute pericarditis

2) Few days post-extensive myocardial infarction

\*\* Pleuropericardial rub

\*\* Pneumopericardium

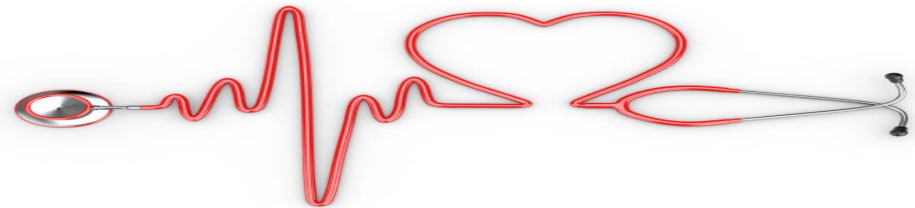






# Murmurs

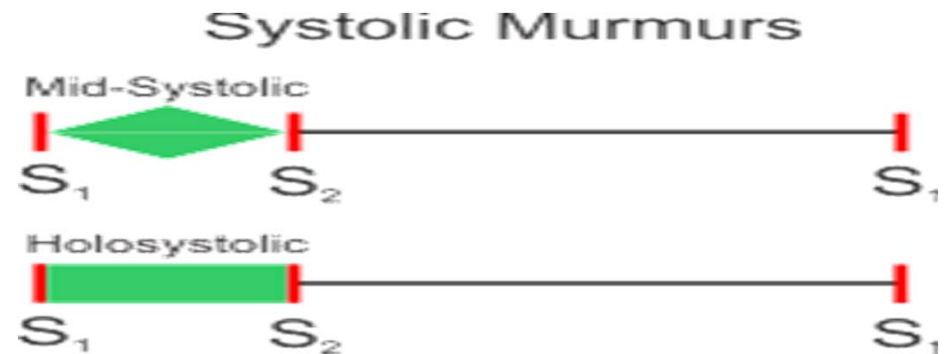
- **Examination includes:**
  - **Timing and duration**
  - **Character/pitch and intensity**
  - **Location and radiation**



# Murmurs/Timing

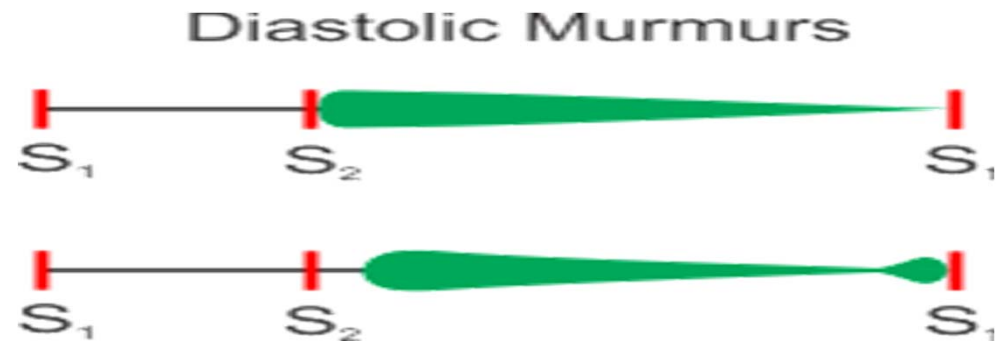
- **Systolic** murmurs

The interval between S1 and S2

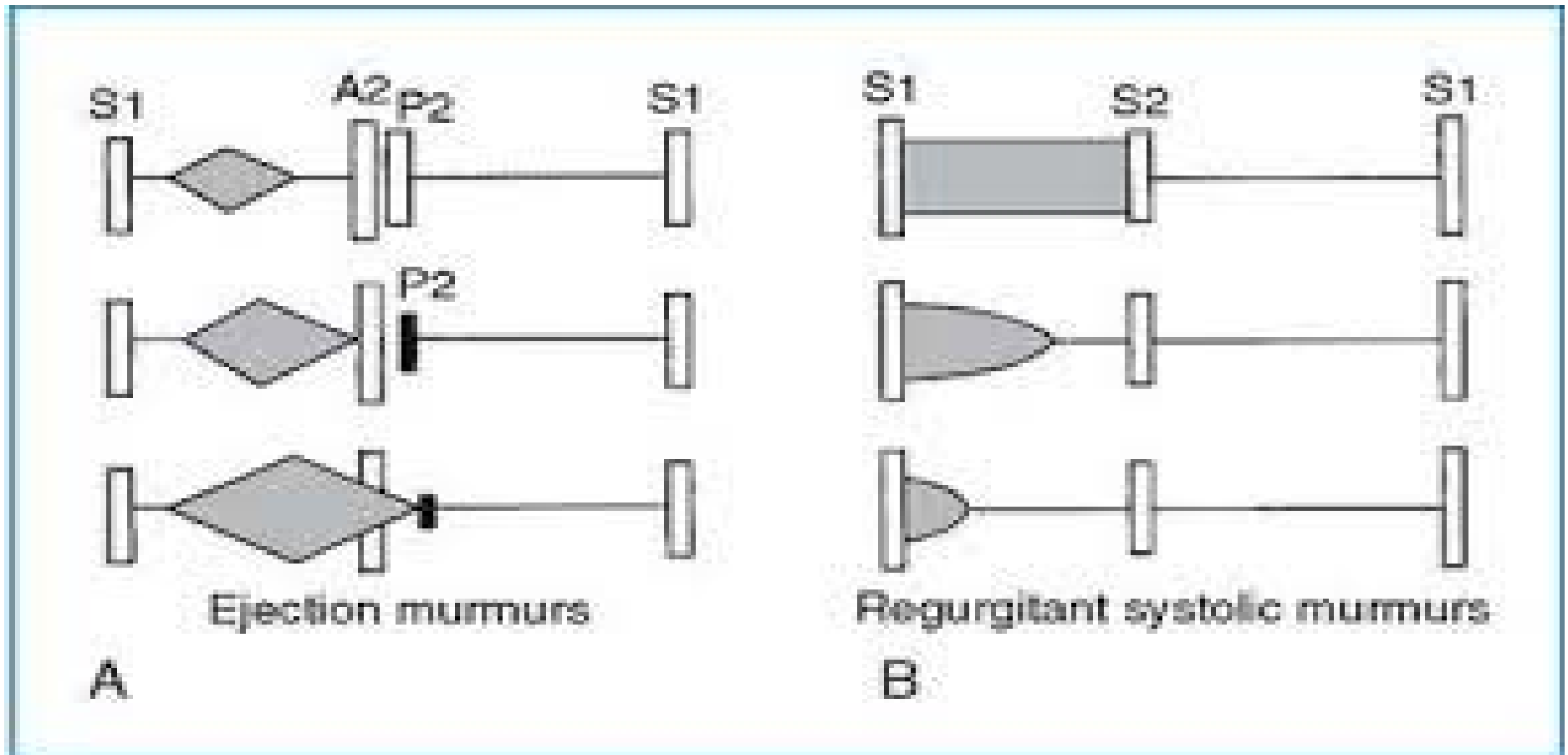


- **Diastolic** murmurs

The interval between S2 to S1

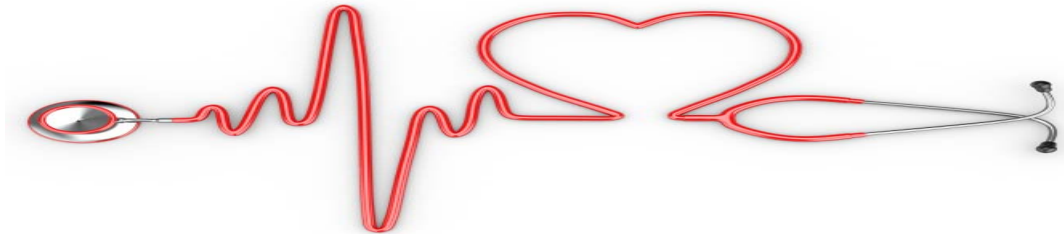


# Murmurs/Duration



# Murmurs/Character and Pitch

- **Harsh: AS**
- **Blowing: MR**
- **Musical: AS in children (still's murmur)**
- **Rumbling: MS**
  
- **High-pitched: high pressure gradient**
- **Low-pitched: low pressure gradient**





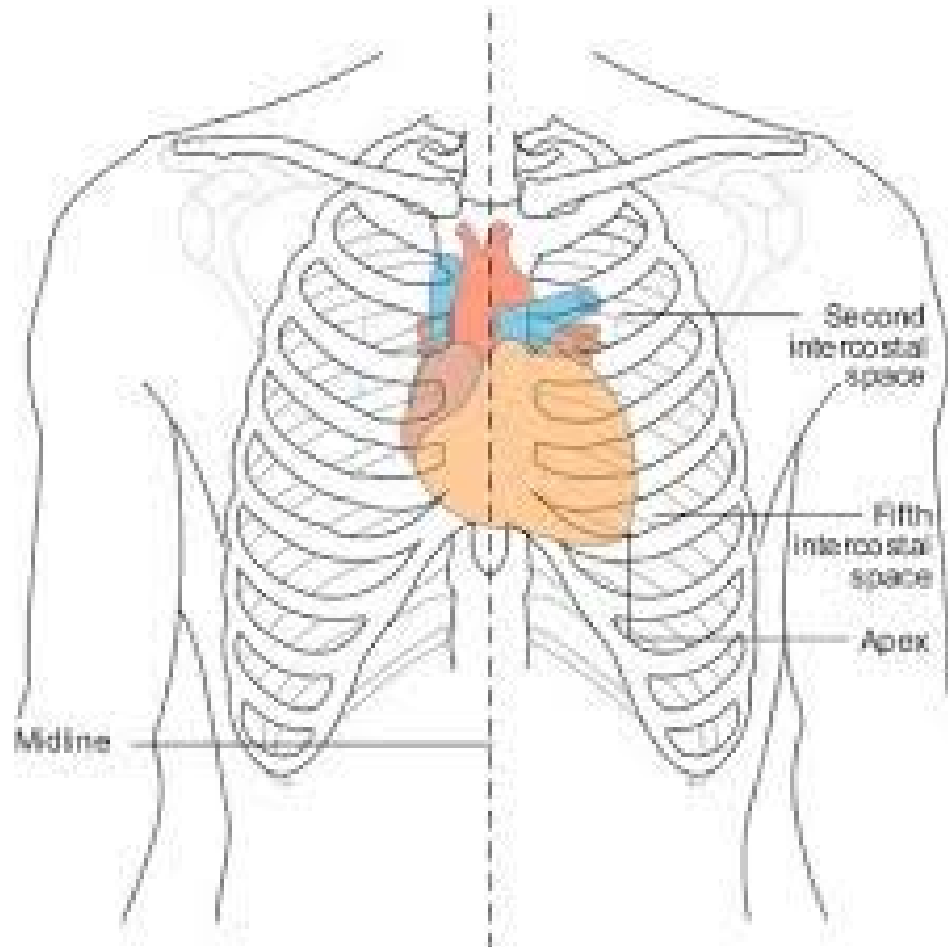
# Murmurs/Intensity

- **The intensity of the murmur does not correlate with the severity of the valve of valve dysfunction**
- **Change in intensity with time is important , as they can denote progression of a valve lesion**
- **Rapidly changing murmur can occur with infective endocarditis**

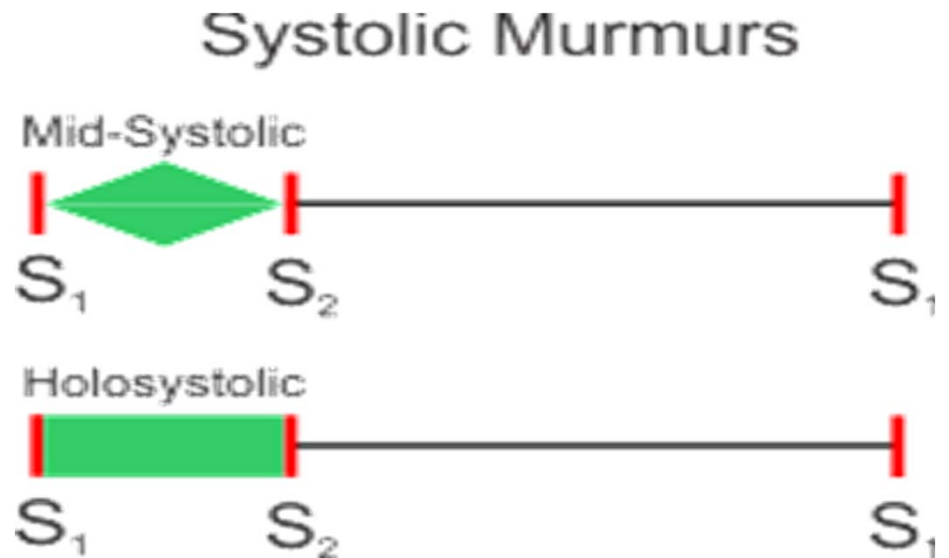
## Grades of intensity of murmur

Grade 1	Heard by an expert in optimum conditions
Grade 2	Heard by non-expert in optimum conditions
Grade 3	Easily heard, no thrill
Grade 4	A loud murmur, with a thrill
Grade 5	Very loud, over large area, with thrill
Grade 6	Extremely loud, heard without stethoscope

# Murmurs/Location, Radiation



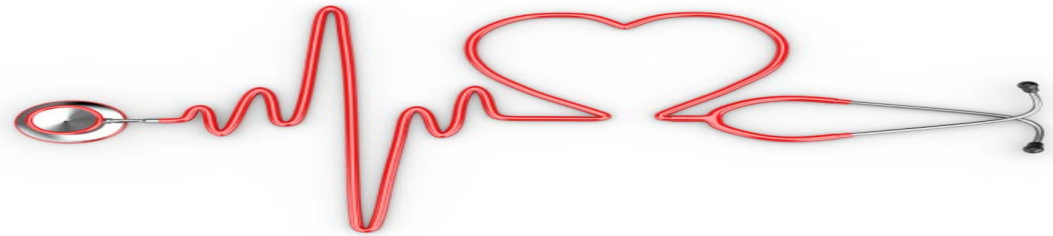
# Systolic Murmurs



- Ejection systolic murmurs

Caused by increased flow through a normal valve (flow or innocent murmur), or by turbulent flow through an abnormal valve.

- Pansystolic



# Ejection systolic murmurs

- Increased flow through a normal valve

**Sever anemia/ fever/ athletes/ pregnancy**

**ASD (pulmonary flow murmur)**

**Increased stroke volume (aortic regurgitation)**

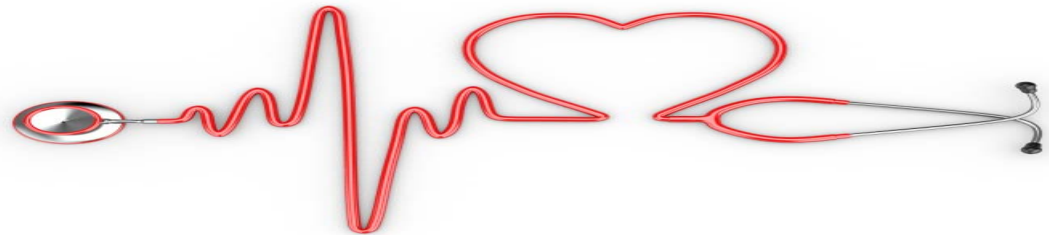
- Normal or reduced flow through a stenotic valve

**Aortic stenosis**

**Pulmonary stenosis**

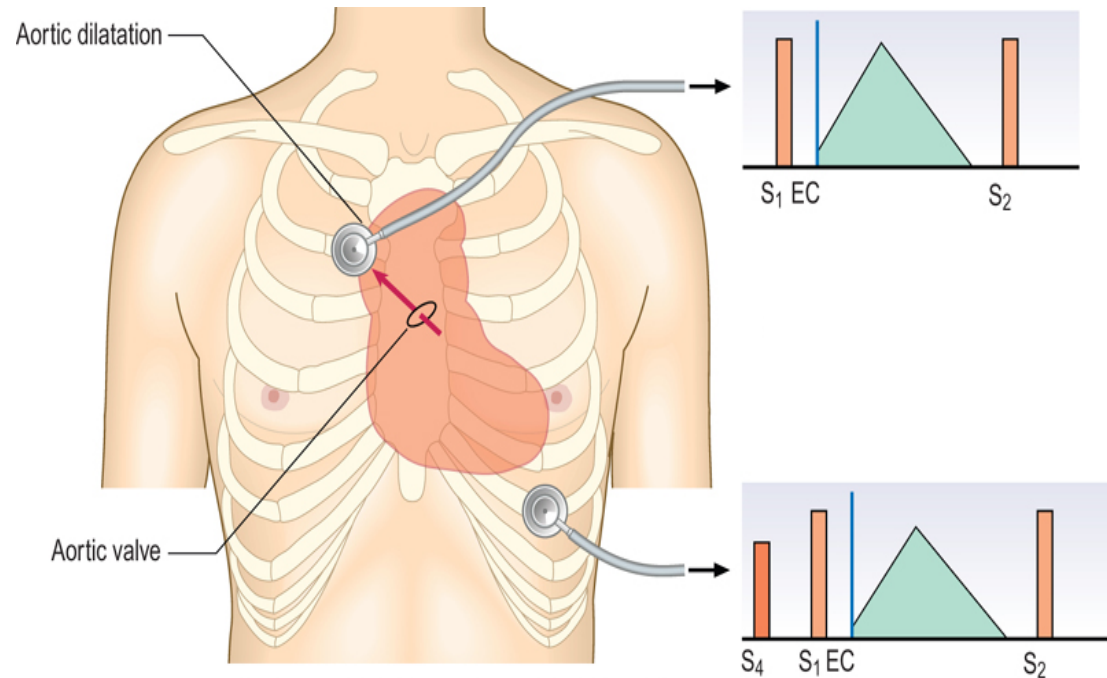
- Subvalvular obstruction

**HOCM**



# Aortic stenosis Murmur

- **Timing: systolic**
- **Duration: after S1, peaks mid systolic, decrease before S2 (Crescendo-decrescendo murmur)**
- **Character: Harsh, Musical in children**
- **Pitch: high (Audible all over the precordium)**
- **Intensity: May be associated with thrill**
- **Location: Right 2<sup>nd</sup> ICS**
- **Radiation: carotids, suprasternal notch**



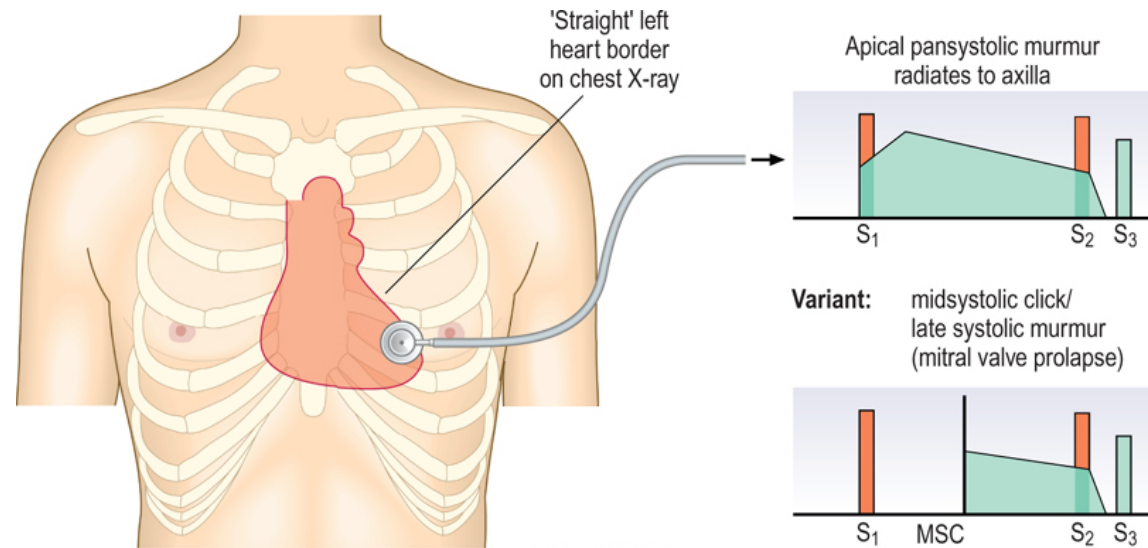
Douglas et al: Macleod's Clinical Examination, 12th Edition.  
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❖ **May follow ejection click**

# Mitral Regurgitation murmur

- **Timing: systolic**
- **Duration: pansystolic**
- **Character: blowing**
- **Pitch: high**
- **Intensity: may feel a thrill**
- **Location: apex**
- **Radiation: Left axilla**

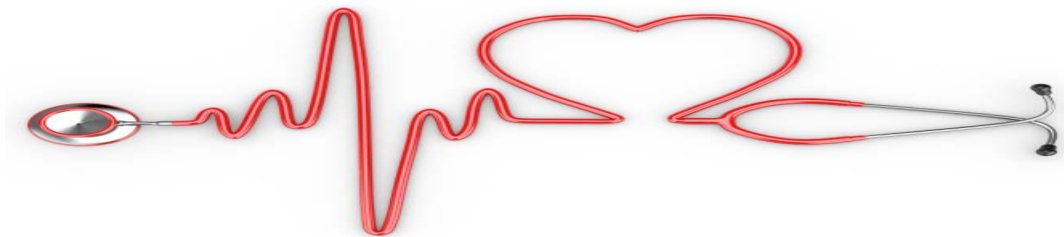
**In mitral valve prolapse, regurgitation begins in mid-systole producing a late murmur**



Douglas et al: Macleod's Clinical Examination, 12th Edition.  
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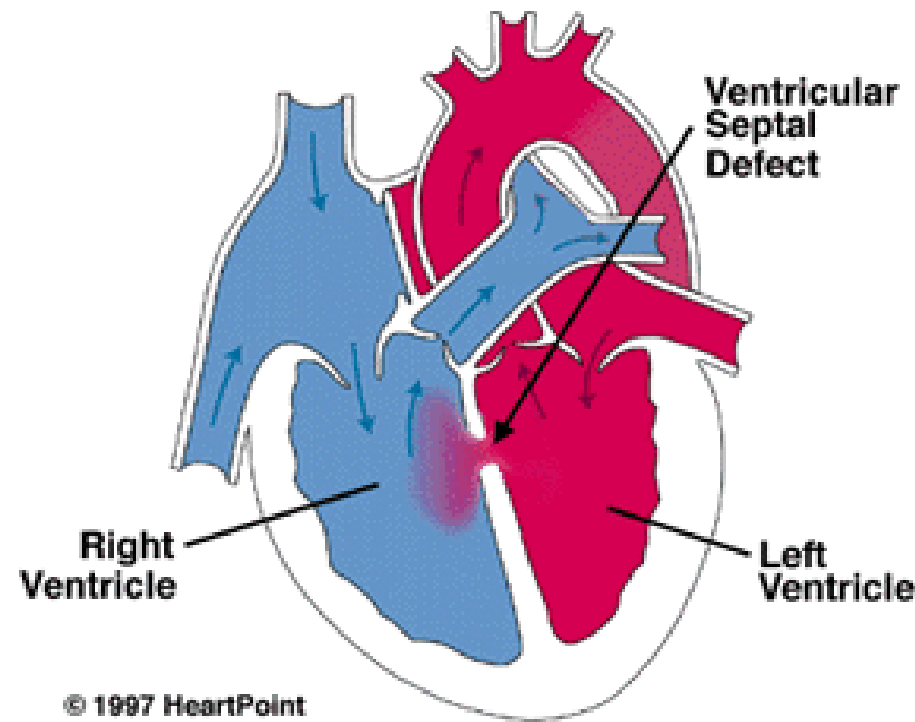
# Tricuspid regurgitation

- Heard at the **lower left sternal edge**
- Prominent **V wave** in the JVP
- **Pulsatile** liver



# Ventricular Septal Defect

- Loud murmur
- At the **left sternal border**
- Radiates to the **right sternal border**
- Associated with **thrill**
- Pansystolic
- Acquired VSD in septal rupture post-MI





# Diastolic Murmurs



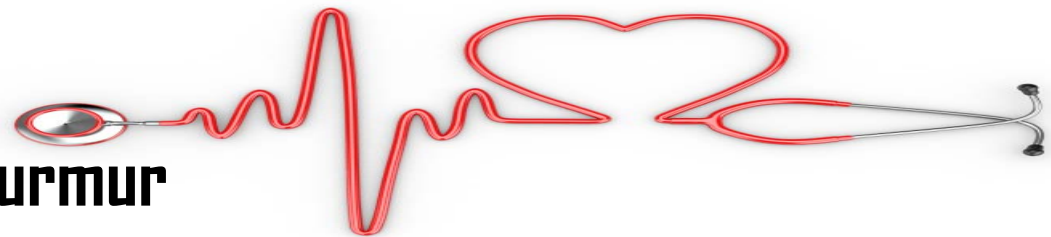
- Early diastolic murmurs

Usually lasts throughout the diastole but are loudest in early diastole

Aortic and pulmonary regurgitation

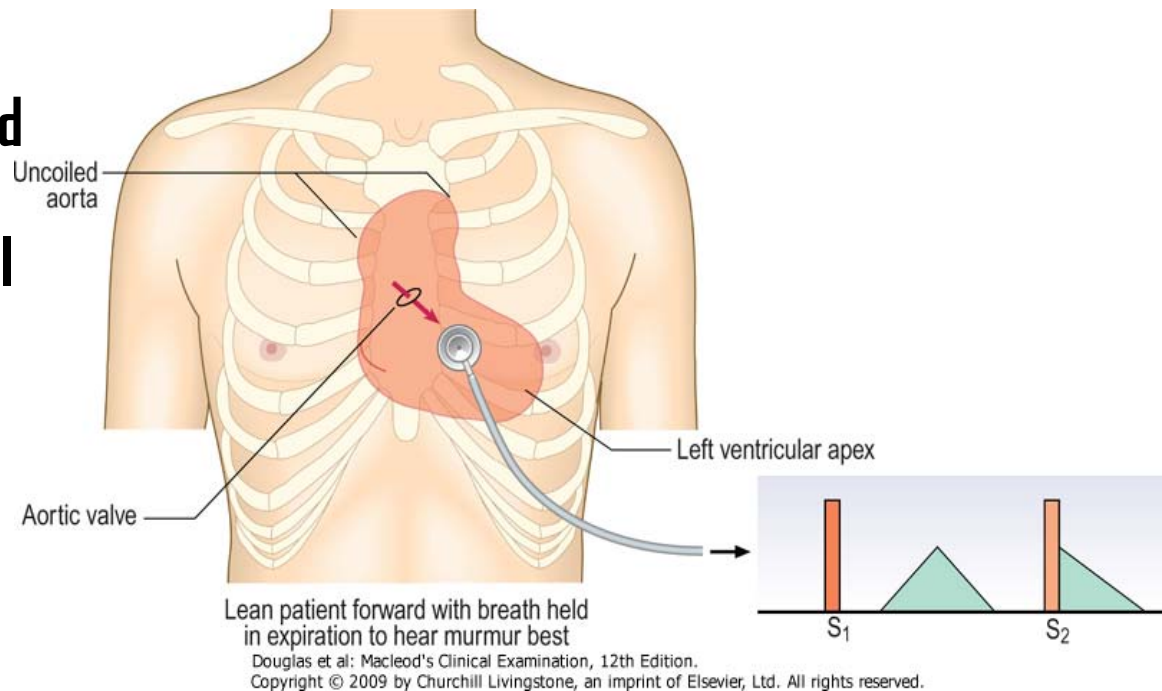
- Mid-diastolic murmurs

Mitral stenosis and Austin flint murmur



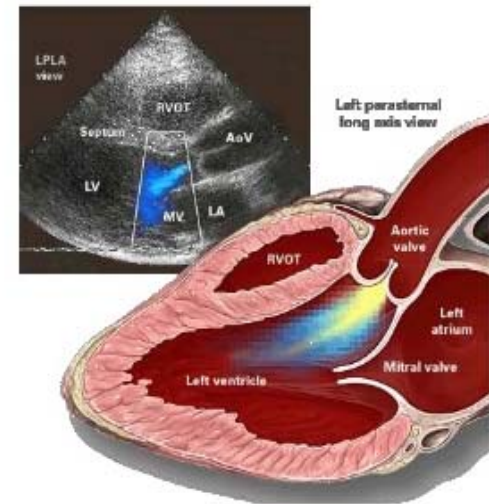
# Aortic Regurgitation

- **Timing: early diastolic**
  - **Pitch: low (ask the pt to lean forward and hold his breath in expiration)**
  - **Location: 2 areas ( Rt 2<sup>nd</sup> intercostal space, Lt third intercostal space- Erb's area)**
- 
- ❖ **The duration of the murmur is inversely proportional to the the severity**
  - ❖ **Can be associated with systolic flow murmur**



# Austin Flint Murmur

- **Mid-diastolic murmur that accompanies aortic regurgitation**
- **Caused by regurgitant jet striking the anterior leaflet of the mitral valve, restricting the inflow to the left ventricle**

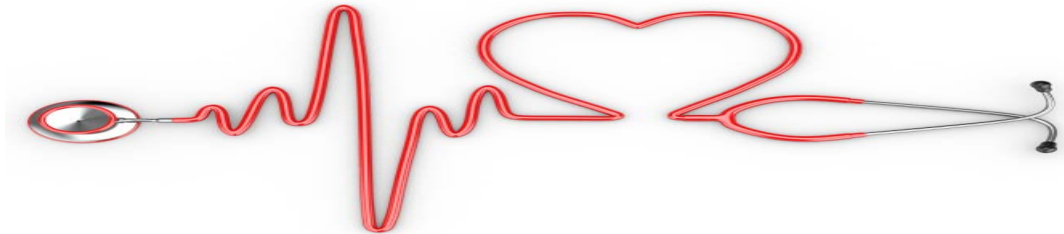


# Pulmonary Regurgitation

- Pulmonary regurgitation caused by pulmonary dilatation in pulmonary hypertension

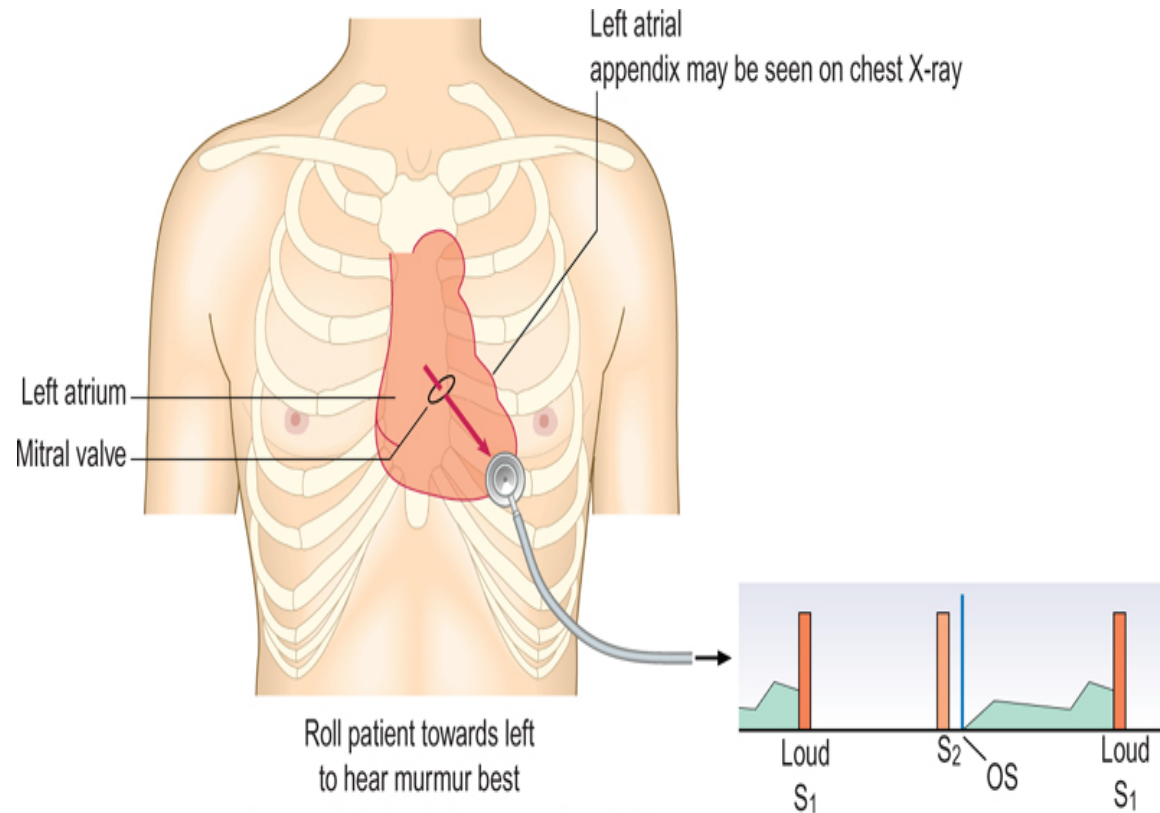
## Graham Steel murmur

- Congenital defect of the pulmonary valve



# Mitral Stenosis

- **Timing: late diastolic**
  - **Character: blowing**
  - **Pitch: low (ask the pt to turn to the left)**
  - **Location: apex**
- 
- ❖ **May follow opening snap**
  - ❖ **The murmur is accentuated by exercise**

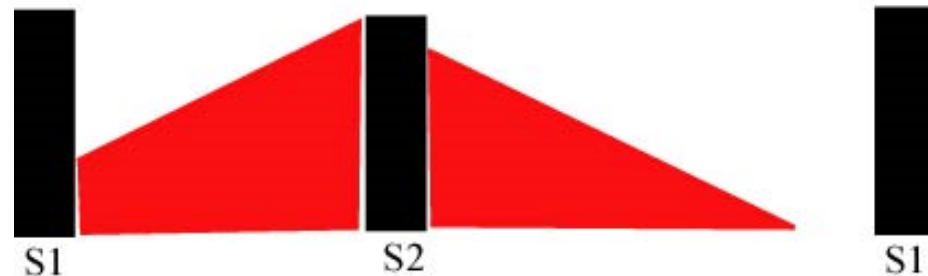


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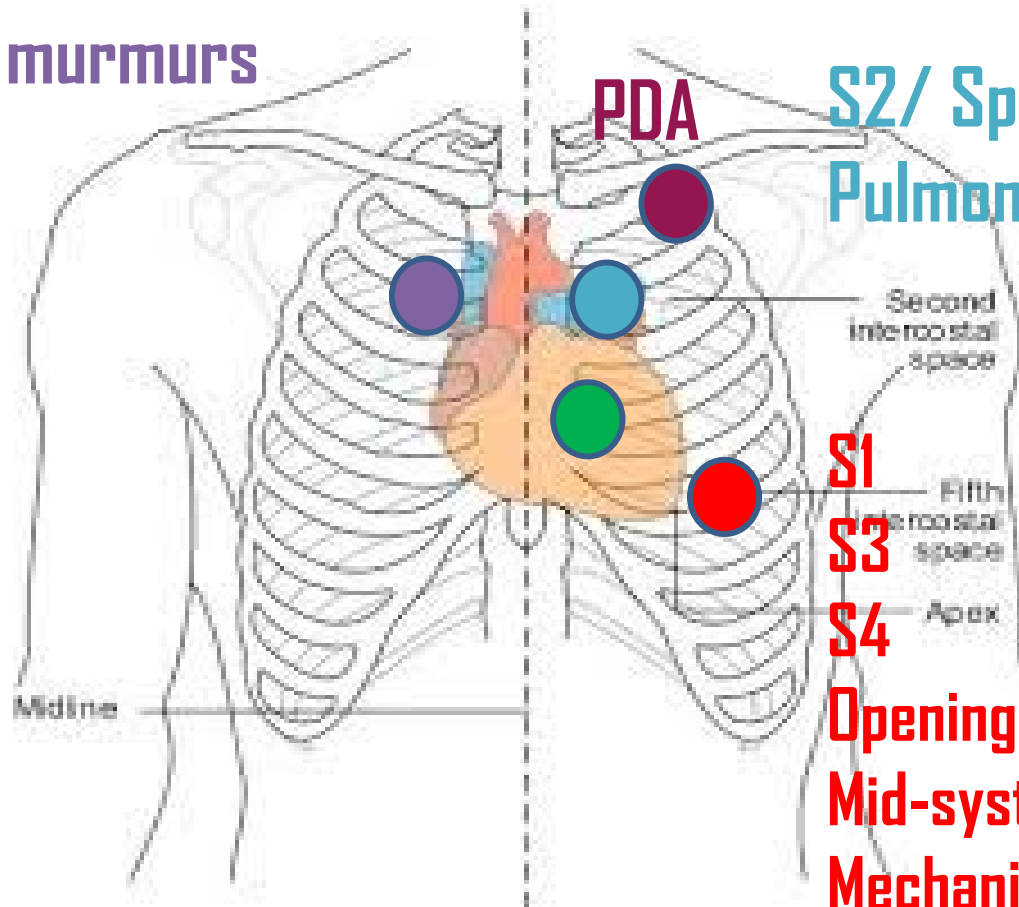
# Continuous Murmurs

- Rare in adults
  - **Patent ductus arteriosus** is the most common cause
  - Timing: systolic and diastolic
  - Duration: continuous
  - Character: machinery-like
  - Pitch: high pitch, louder in systolic
  - Location: left infraclavicular
  - Radiation: left scapula
- ❖ Aortic pressure always exceeds pulmonary pressure , there is continuous ductal flow with the greatest pressure difference in systole resulting in a louder systolic component

Patent Ductus Arteriosus



Mechanical aortic valve  
Systolic Ejection murmurs  
HOCM  
Aortic regurg



S2/ Split

Pulmonary murmurs

Tricuspid Regurg/stenosis

Aortic regurg

S1

S3

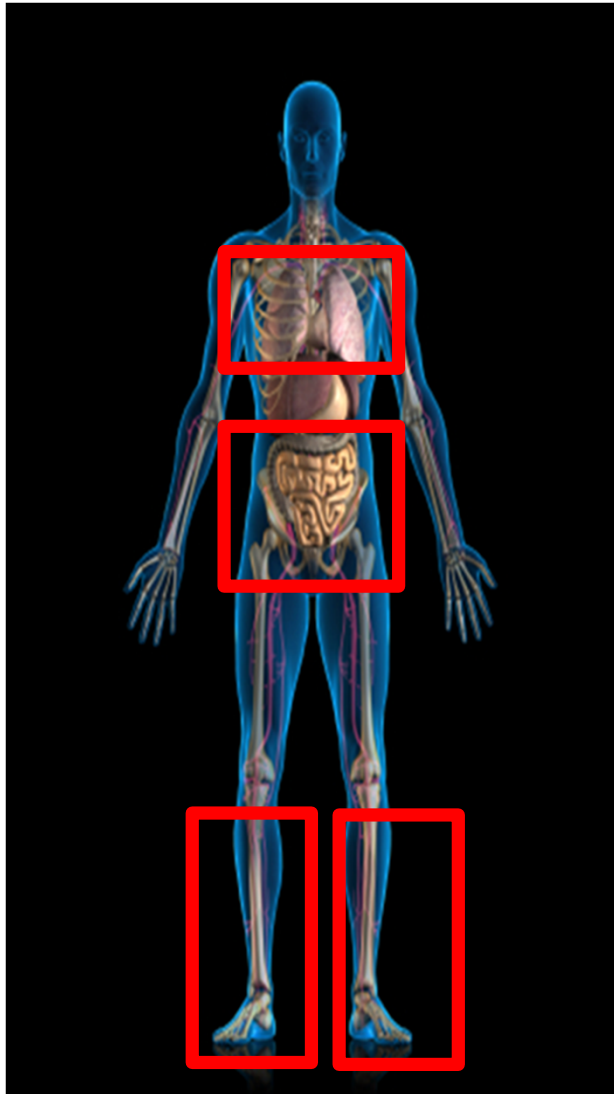
S4

Opening snap

Mid-systolic click

Mechanical mitral valve

Mitral Regurg/Stenosis



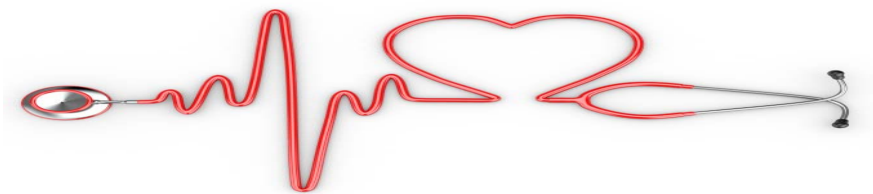
## Complete your examination

- Auscultate the lung for crackles and pleural effusion
- Examine the abdomen for ascites
- Auscultate for Bruit
- Examine lower limb/sacrum for edema



# Aortic Stenosis

- **Slow rising pulse**
- **Displaced apex beat, S4**
- **Apical heave**
- **Thrill over the apex and right upper sternal boarder**
- **Ejection systolic murmur right upper sternal boarder radiating to the carotids**
- **Ejection click**
- **Reversed splitting S2**



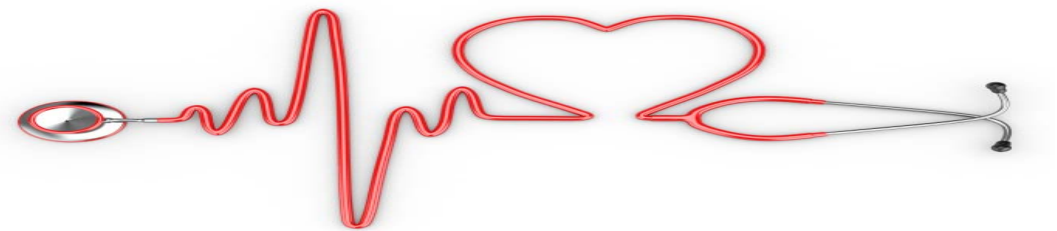
# Mitral stenosis

- Tapping apex beat
- Opening snap
- Mid-diastolic murmur at the apex
- Loud S1



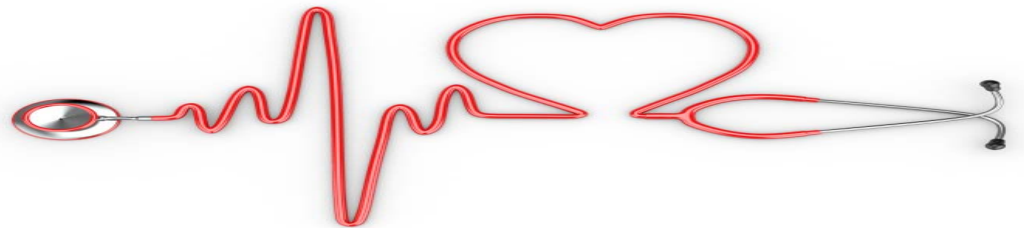
# HOCM

- **Bisferiens pulse**
- **Double apical impulse**
- **Ejection systolic murmur**
- **Reversed splitting S2**



# VSD

- **Right and left sternal border thrill**
- **Pansystolic murmur left sternal border**
- **Wide splitting S2**



# Tricuspid Regurgitation 2<sup>nd</sup> to pulmonary HTN

- **Giant V wave in JVP**
- **Left parasternal heave**
- **Wide splitting/ loud S2**
- **Graham steel murmur (if pulmonary artery dilates)**

