Frank- starling mechanism and mechanical efficiency

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Clinical topography of the heart

Holotopy

intercostal middle of mediastinum

Syntopy

Anteriorly sternum

Inferiorly diaphragm

Laterally pleural of the lung

Posteriorly esophagus and vasculature

Superiorly great blood vessels

Sceletopy right to lift

Upper border

3rd rib horizontal

Right border

1.5cm 3rd to 5th rib parasternal

Lower border

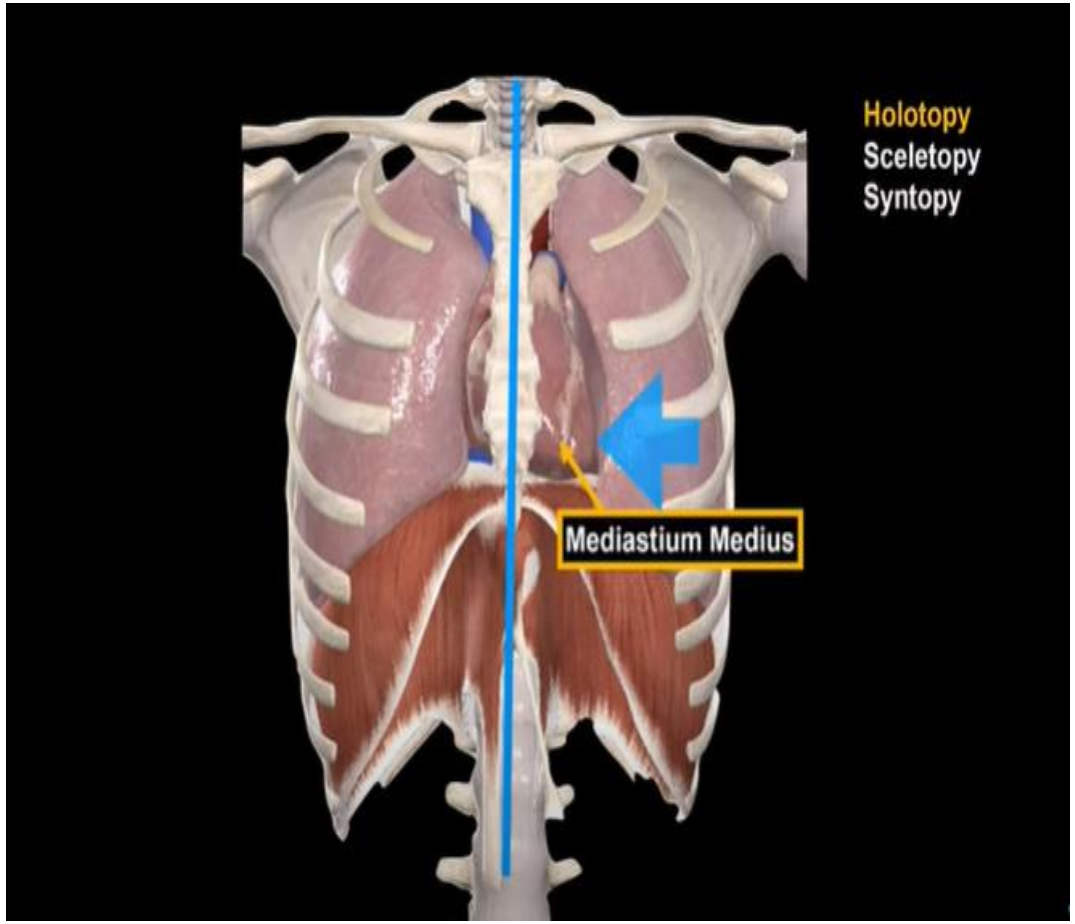
5th rib cartilage to 5th intercostal obliquely

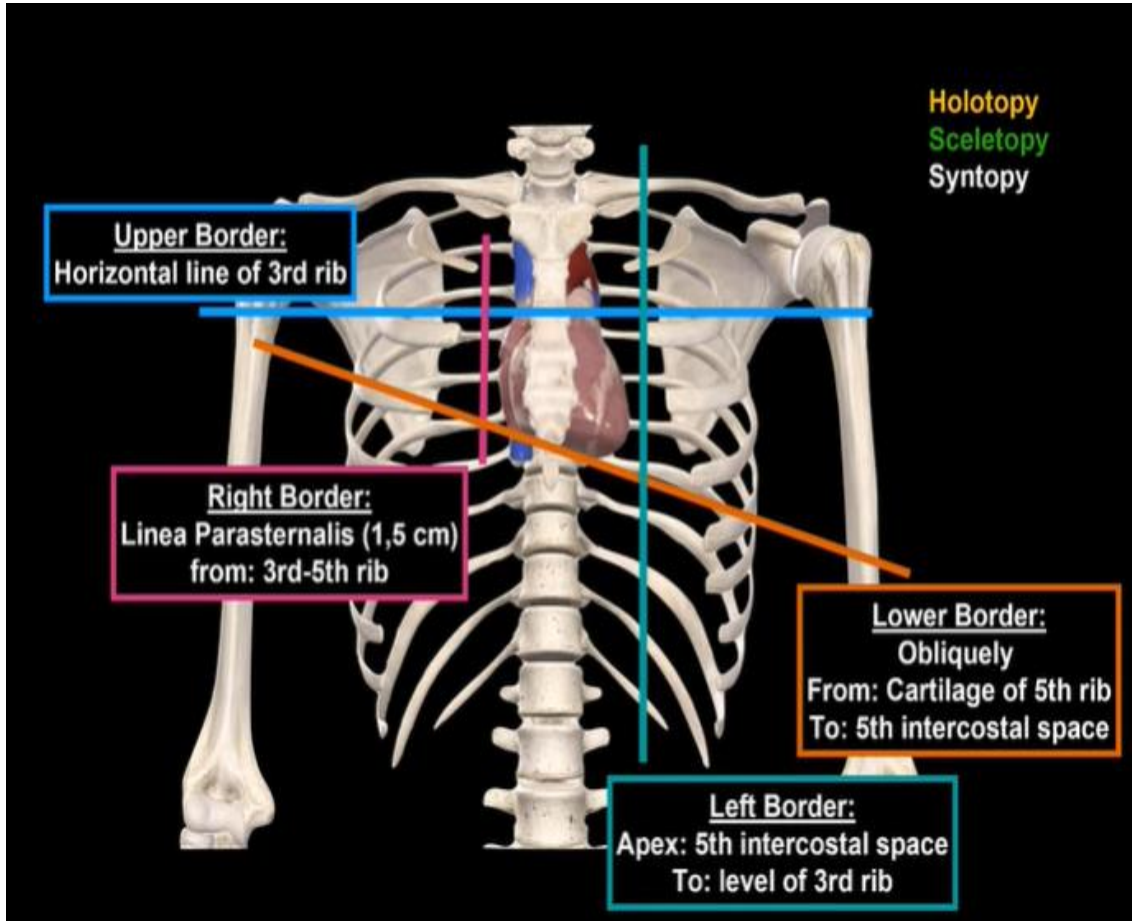
Left border

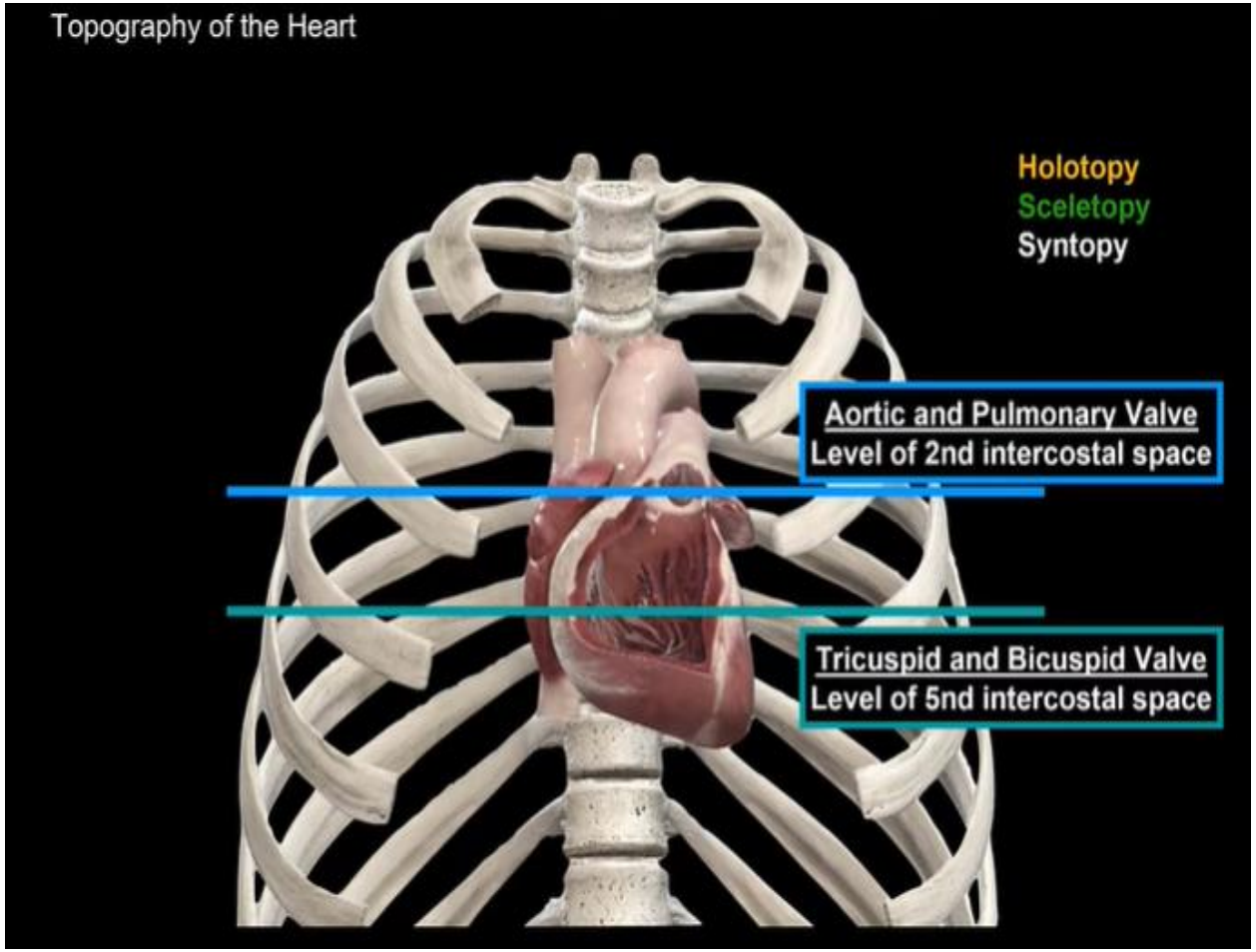
5th intercostal Apex to 3rd rib

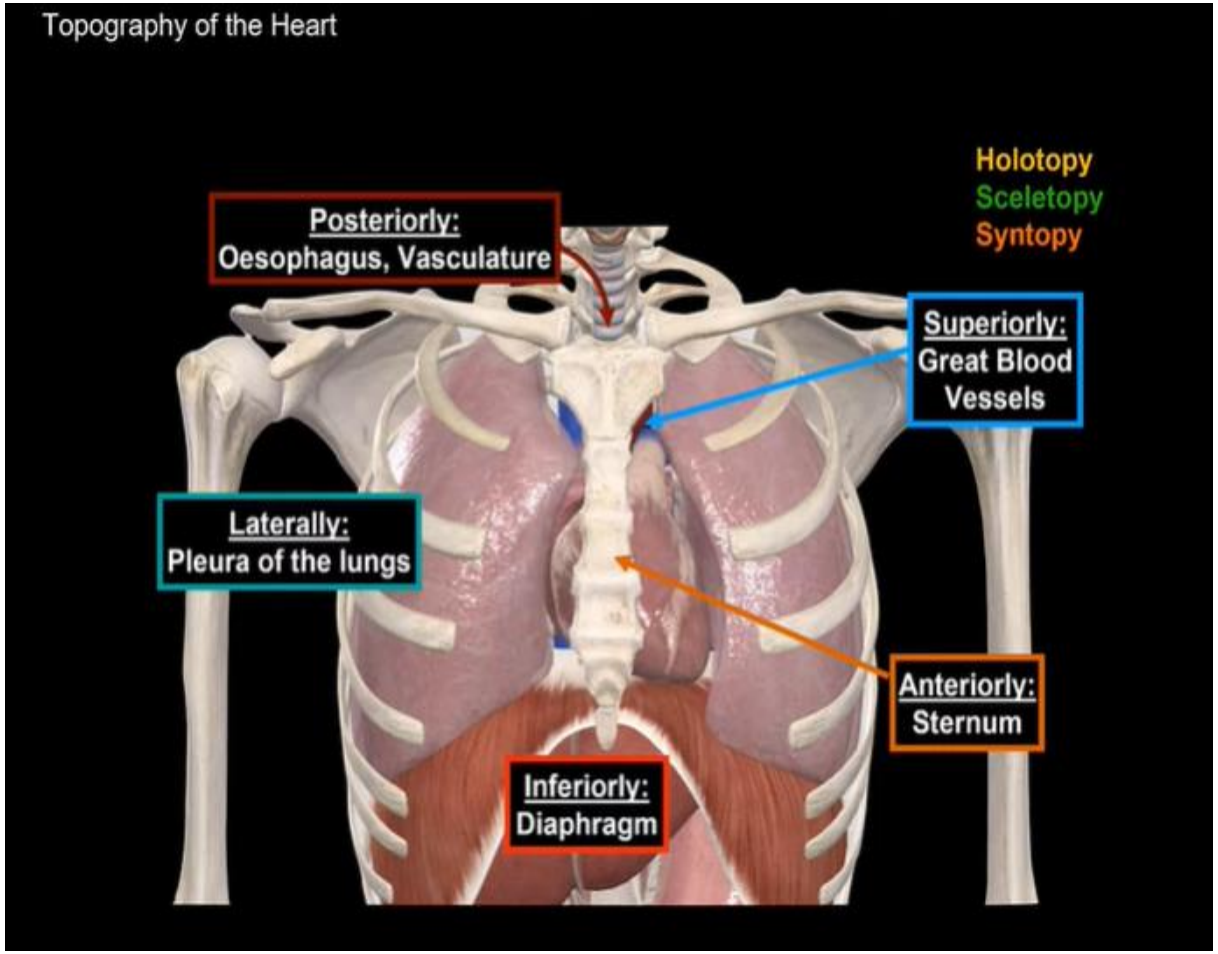
Left to right ; AV openings 3rd to 6th rib sternal junction 5th stethoscope

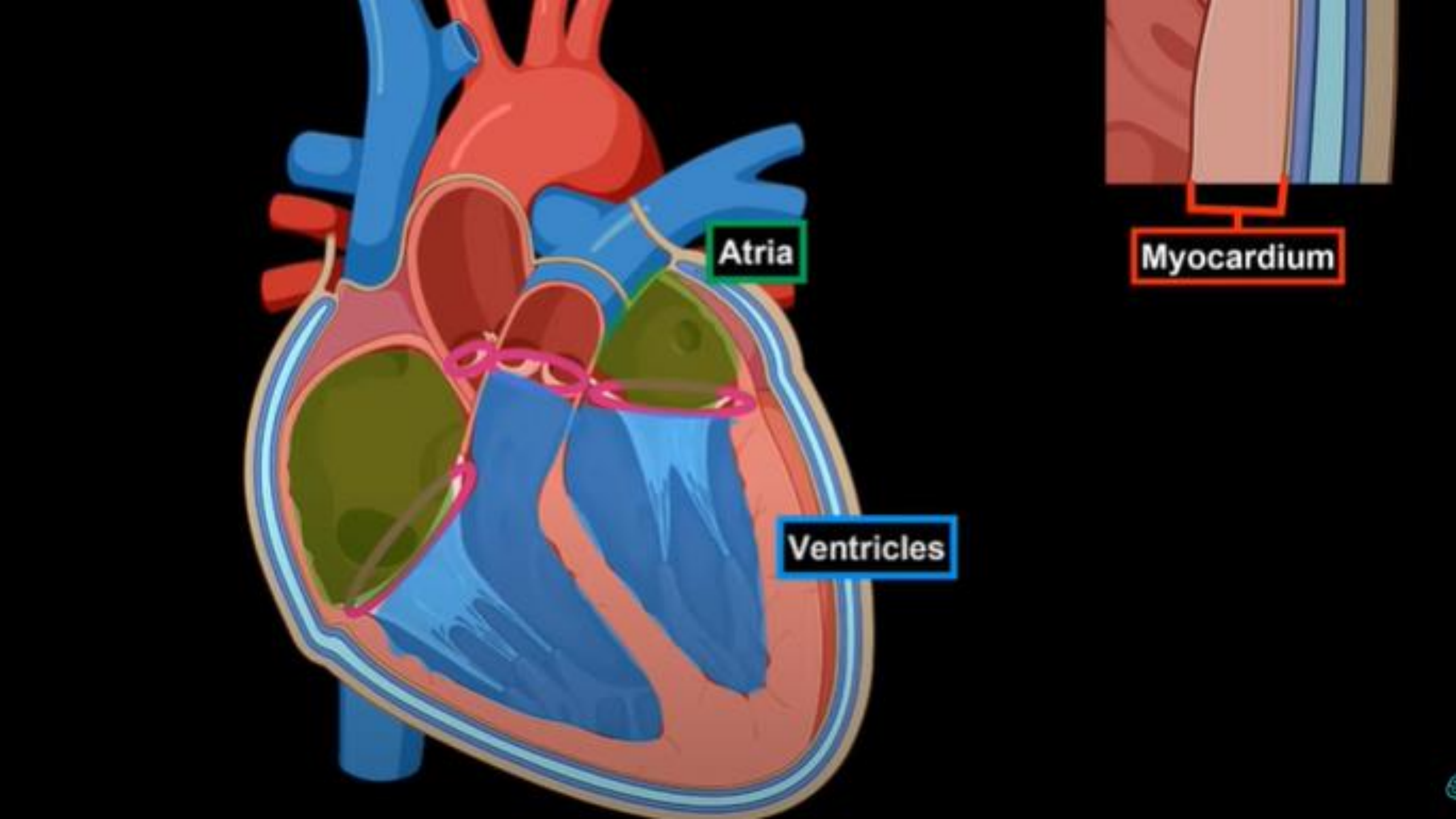
aortic and pulmonary 3rd to 4th sternal junction 2nd stethoscope


Holotopy
Sceletopy
Syntopy
Mediastium Medius


Holotopy
Sceletopy
Syntopy
Upper Border:
Horizontal line of 3rd rib
Right Border:
Linea Parasternalis (1,5 cm)
from: 3rd-5th rib
Lower Border:
Obliquely
From: Cartilage of 5th rib
To: 5th intercostal space
Left Border:
Apex: 5th intercostal space
To: level of 3rd rib


Topography of the Heart
Holotopy
Sceletopy
Syntopy
Aortic and Pulmonary Valve
Level of 2nd intercostal space
Tricuspid and Bicuspid Valve
Level of 5nd intercostal space


Topography of the Heart
Holotopy
Sceletopy
Syntopy
Posteriorly:
Oesophagus, Vasculature
Superiorly:
Great Blood
Vessels
Laterally:
Pleura of the lungs
Anteriorly:
Sternum
Inferiorly:
Diaphragm


Atria
Ventricles
Myocardium

Types of heart cells

Two different types of myocardium

- Nodal cells are non contractile cells these are the ones that generates automaticity set a rhythm or the base (SA, AV, AV Bundle(His), Bundle branches (left and right), Purkinje fibers)
- Contractile cells(actin and myosin, troponin and tropomyosin, sarcoplasmic reticulum) those ones that force and pushing the blood out of the heart

Intrinsic cardiac conduction system

- Electrophysiology of the heart is so special it had the ability to intrinsically depolarize itself it doesn’t really depend upon the nervous system

- The heart exhibits was called automaticity (the heart has its intrinsic ability on tis own to spontaneously depolarize itself and then trigger action potentials to send it out to all other parts of the heart)

Heart cells : contractile cells

“Worker bees” of the hear

- Don’t pace the heart
- Very slow compared to nodal cells
- Cell to cell spread not so fast
- The wave of depolarization “ knock out”

Overdrive suppression the crushing of the worker bee’s dream

Electrophysiological system

SA node

Superficial under visceral epicardium

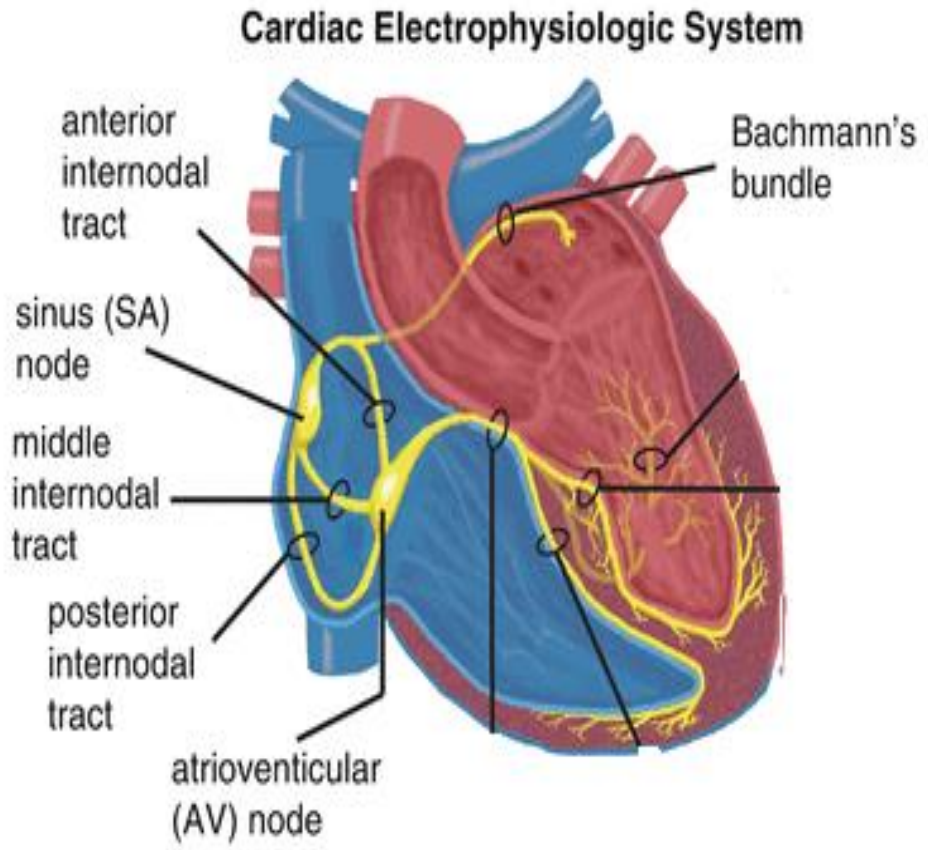
Crescent shape structure ;Superior component of the right atrium just beneath the large vessel here called superior vena cava;

Easily damaged by

Sets the pace at around 60 to about 80 beats per minute (normal heartbeat) on its own without any extrinsic innervation and this is called sinus rhythm

Speed up: Sympathetic and angiotensin II

Slow down : vagus



Pacemaker cells

- SA node “ Champion” Sinoatrial node
- AV node
- Bundle of his

Conduction system

- 1%
- Bachmann’s bundle
- Atrial internodal conduction pathways
- Bundle branches
- Purkinje

Bachman's bundle (superfast conduction highway) Saves the day

The electrical potential conducted from the right atrium by SA node to the left atrium through Bachman's bundle

Make sure the right and left atrium contract simultaneously

Internodal pathway

This will supply all the other parts of the right atrium but eventually all this internodal pathways converge on this second important structure to the AV node

AV node

Runs from the actual right atrium to the interventricular septum so it is acting as a connection, the gateway between the atria and the ventricles because what happened is some potentials of Bachman's bundle can make their way over here to the AV node also

So, all the action potentials that are coming from the SA node that are being spread out to the internodal pathway or the Bachman's bundle are converging to the AV node

40-60b/m

Can permanently takeover

Bundel of his

20-40b/m

Starting dizzy

Bundles branches and distal purkinje fibers

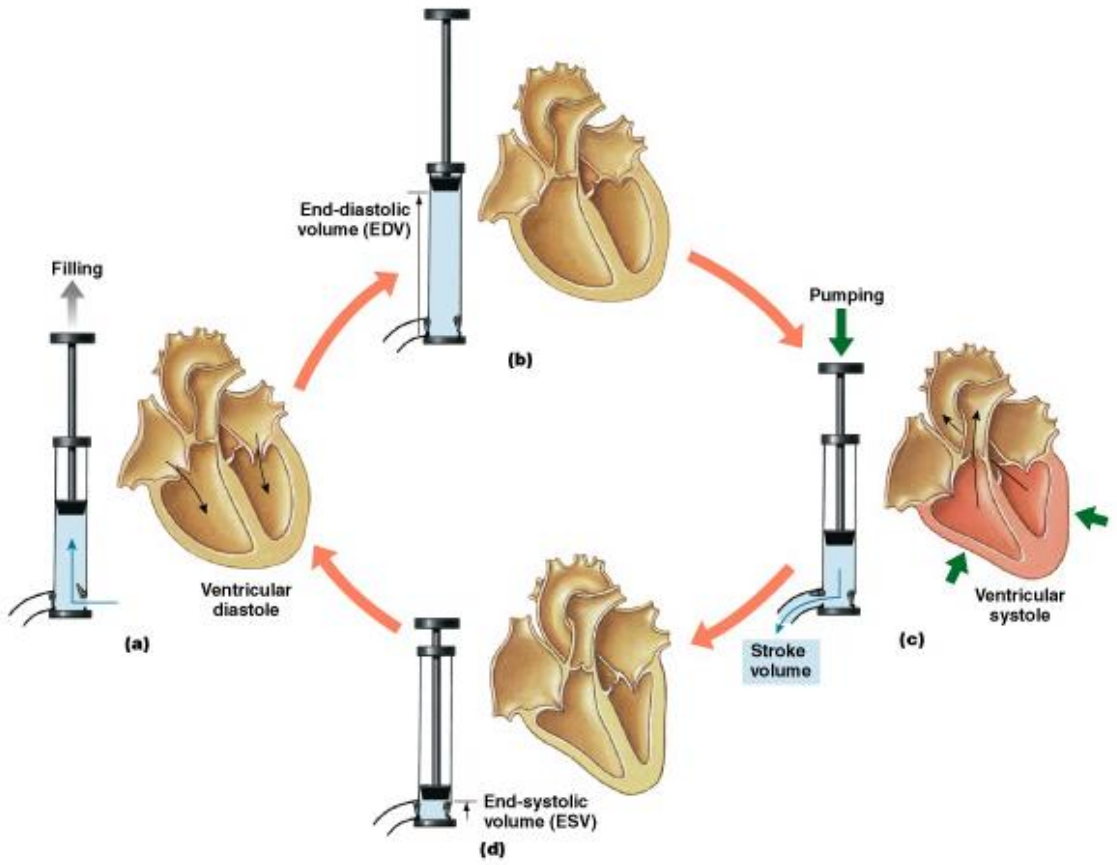
15-40b/m

Pacemaker potential not enough to sustain life very long

- Atrial myocardial cells (45-65b/m)

Rebels' missy and driving crazy don’t permanently takeover the duties

Stroke Volume(SV)



Frank – Starling Principle

- End diastolic volume: volume: The amount of blood that remains in the ventricle just before ventricular early systole is the EDV
- End systolic volume: The amount of blood that remains in the ventricle at the end of ventricular systole is the ESV

$$SV = EDV - ESV$$

Ventricular volumes

Stroke volume

- In a healthy 70-kg man, ESV is approximately 50 mL and EDV is approximately 120mL, giving a difference of 70 mL for the stroke volume.

SV = EDV - ESV

Ejection fraction (EF)

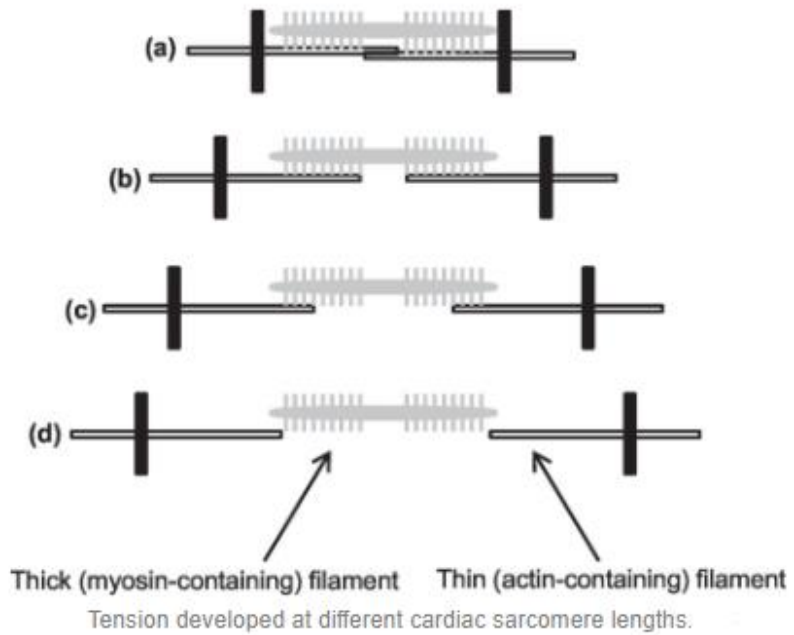
- Volumetric fraction of blood ejected from a heart with each contraction (heartbeat).
- EF is widely used as a measure of the pumping efficiency of the heart and is used to classify heart failure types.

- . The EF of the right heart, or right ventricular ejection fraction (RVEF), is a measure of the efficiency of pumping into the pulmonary circulation

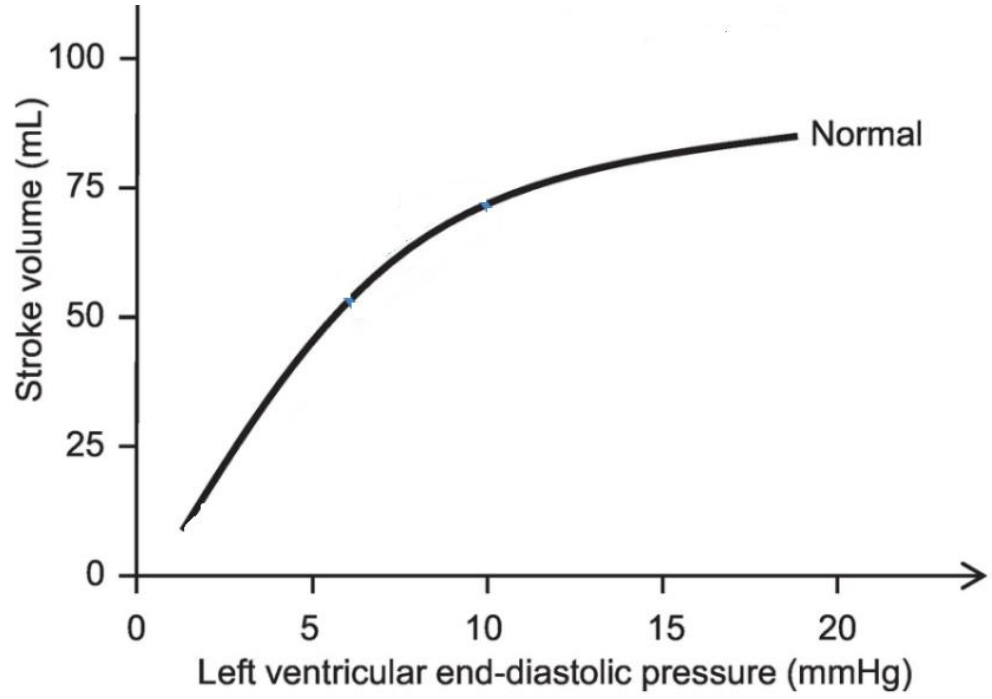
- The EF of the left heart (LVEF) is an indicator of the effectiveness of pumping into the systemic circula

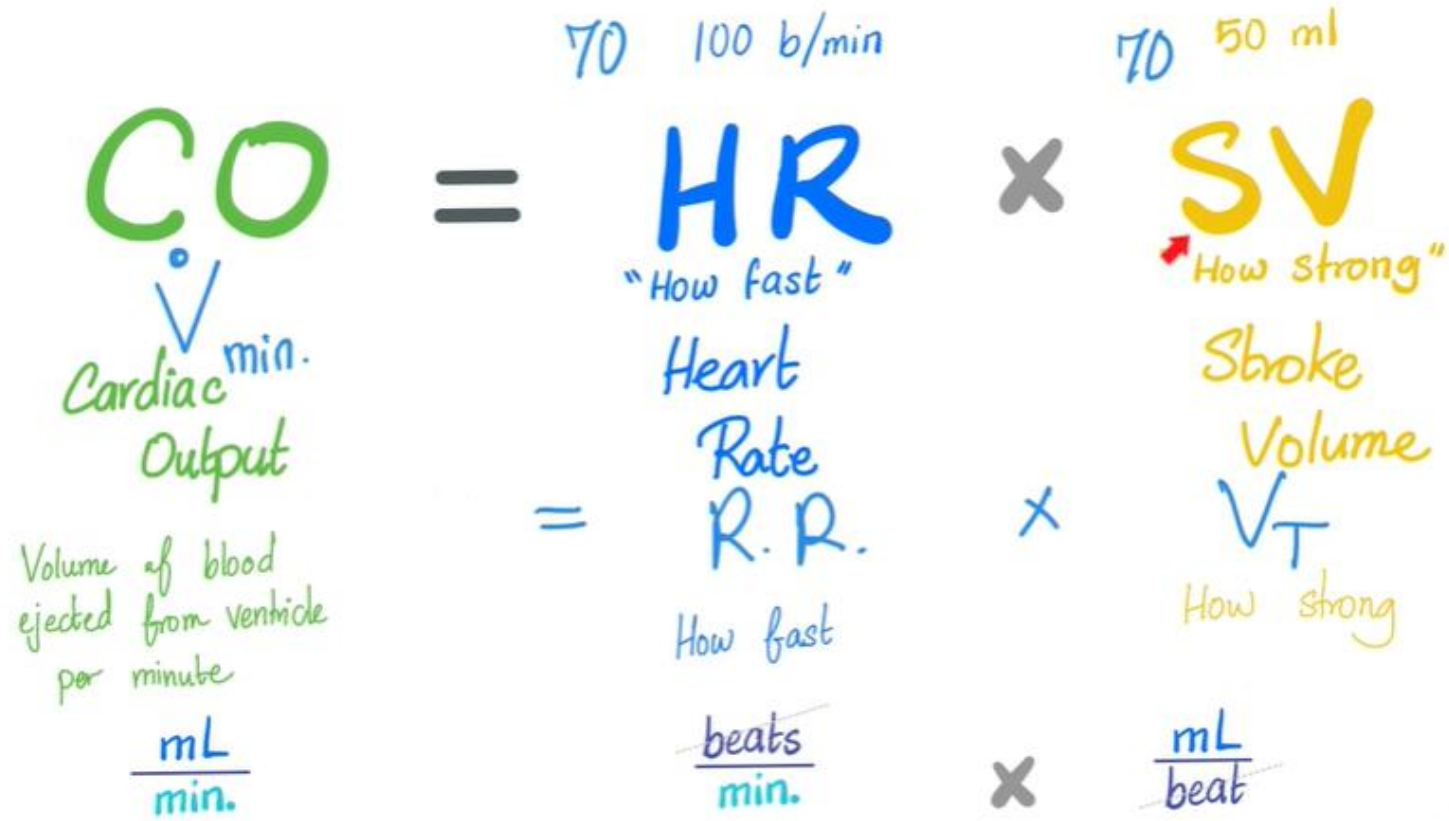
$$EF(\%) = \frac{SV}{EDV} \times 100$$

Frank starling mechanism



Tension developed at different cardiac sarcomere lengths.




70
100 b/min
70
50 ml
CO
=
HR
×
SV
"How fast"
"How strong"
V̇ Cardiac min. Output
Heart Rate
Stroke Volume
Volume of blood ejected from ventricle per minute
= R.R. × VT
How fast
How strong
mL/min.
beats/min.
× mL/beat

Frank – Starling Principle

- The Frank–Starling law of the heart (also known as Starling's law and the Frank–Starling mechanism) represents the relationship between stroke volume and end diastolic pressure
- This principle illustrates the relationship between cardiac output and left ventricular end diastolic volume
- The law states that the stroke volume of the heart increases in response to an increase in the volume of blood in the ventricles, before contraction (the end diastolic volume), when all other factors remain constant.
- As a larger volume of blood flows into the ventricle, the blood stretches the cardiac muscle fibers, leading to an increase in the force of contraction.
- The Frank-Starling mechanism allows the cardiac output to be synchronized with the venous return, arterial blood supply
- The physiological importance of the mechanism lies mainly in maintaining left and right ventricular output equality
- If this mechanism did not exist and the right and left cardiac outputs were not equivalent, blood would accumulate in the pulmonary circulation (were the right ventricle producing more output than the left) or the systemic circulation (were the left ventricle producing more output than the right).

Cardia out put

- **HR**
- PSNS -
- SNS +
- Hormones (EPI, NE) +
- IONS: Ca++, Na+ , K+ dependents on their level increase or decrease

SV

+ Preload ; Increase the blood volume returns increase diastolic volume

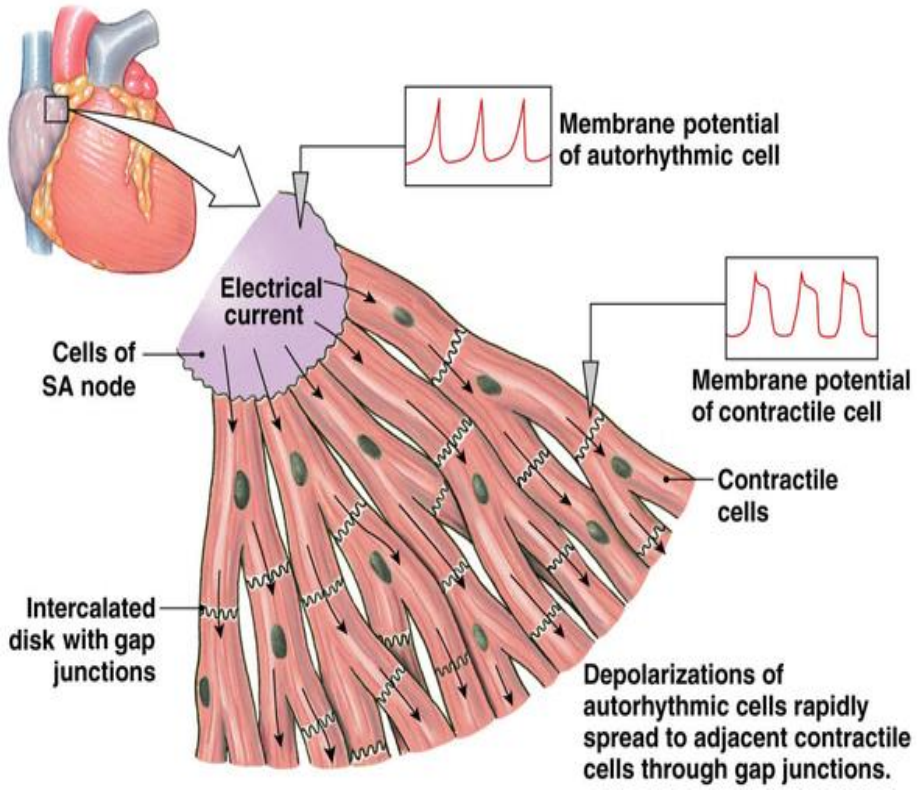
+ Contractility ; SNS (EPI,NE+), Hormones (glucagon,T3 and T4), IONS like Ca++

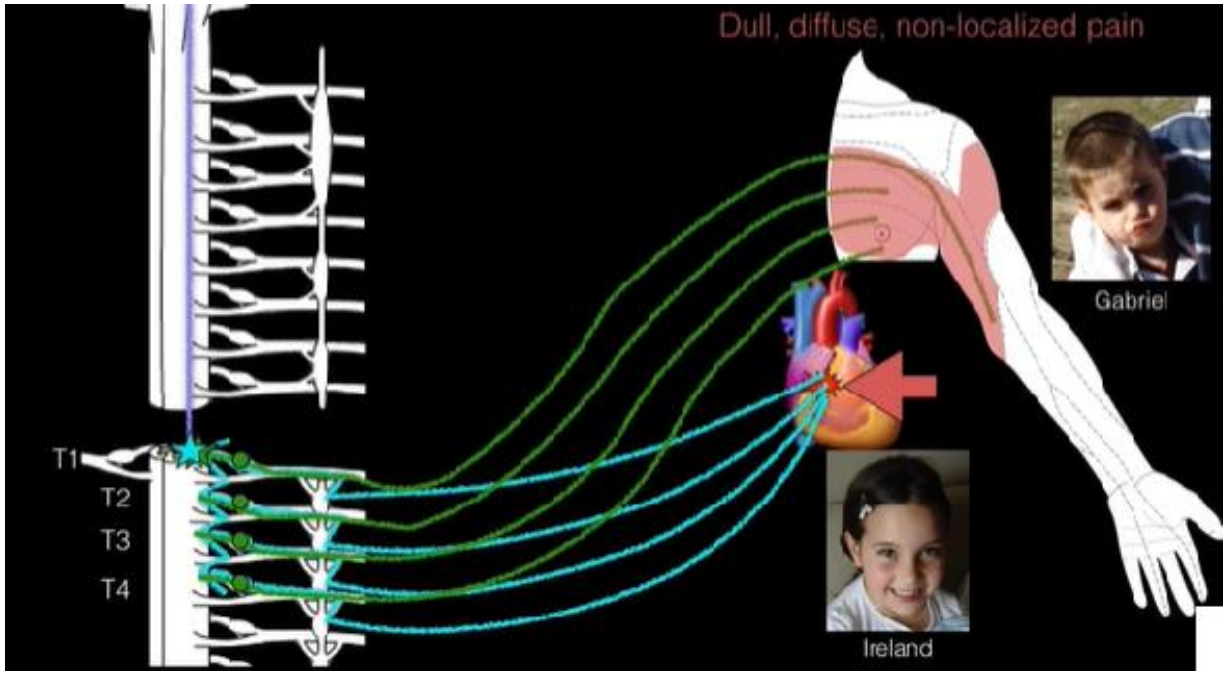
- Afterload; Hypertension, Atherosclerotic plaques , TPR

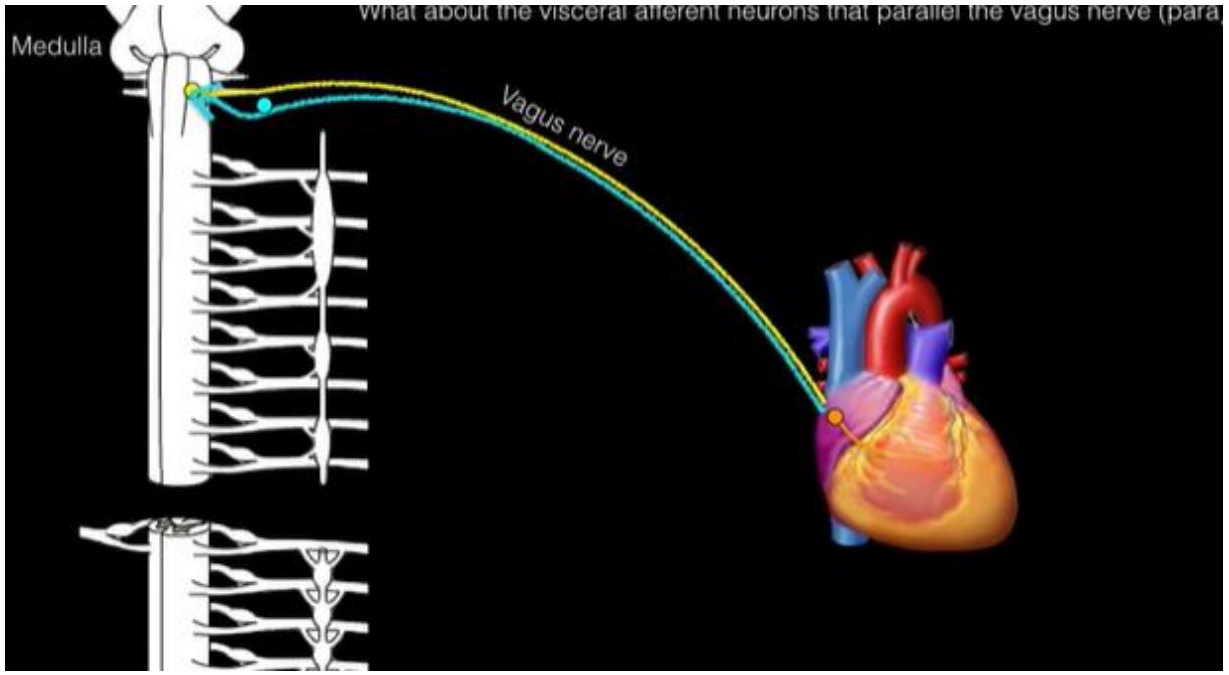
functional syncytium

- Desmosomes is basically acting like adhesion and tighten molecules

- **Intercalated disks are basically a bunch of gap junctions and desmosomes connecting the actual cardiac cells together**







Take home message:

- T1-T4 (T5)
- Sympathetic chain
- Cervical and Thoracic sympathetic ner
- Increase heart rate and contraction

Take home message:

- Medulla
- Vagus
- Intramural ganglion
- Decrease heart rate and contraction

Myocardium clinical disorders

Angina pectoris

Myocardial infarction (heart attack)