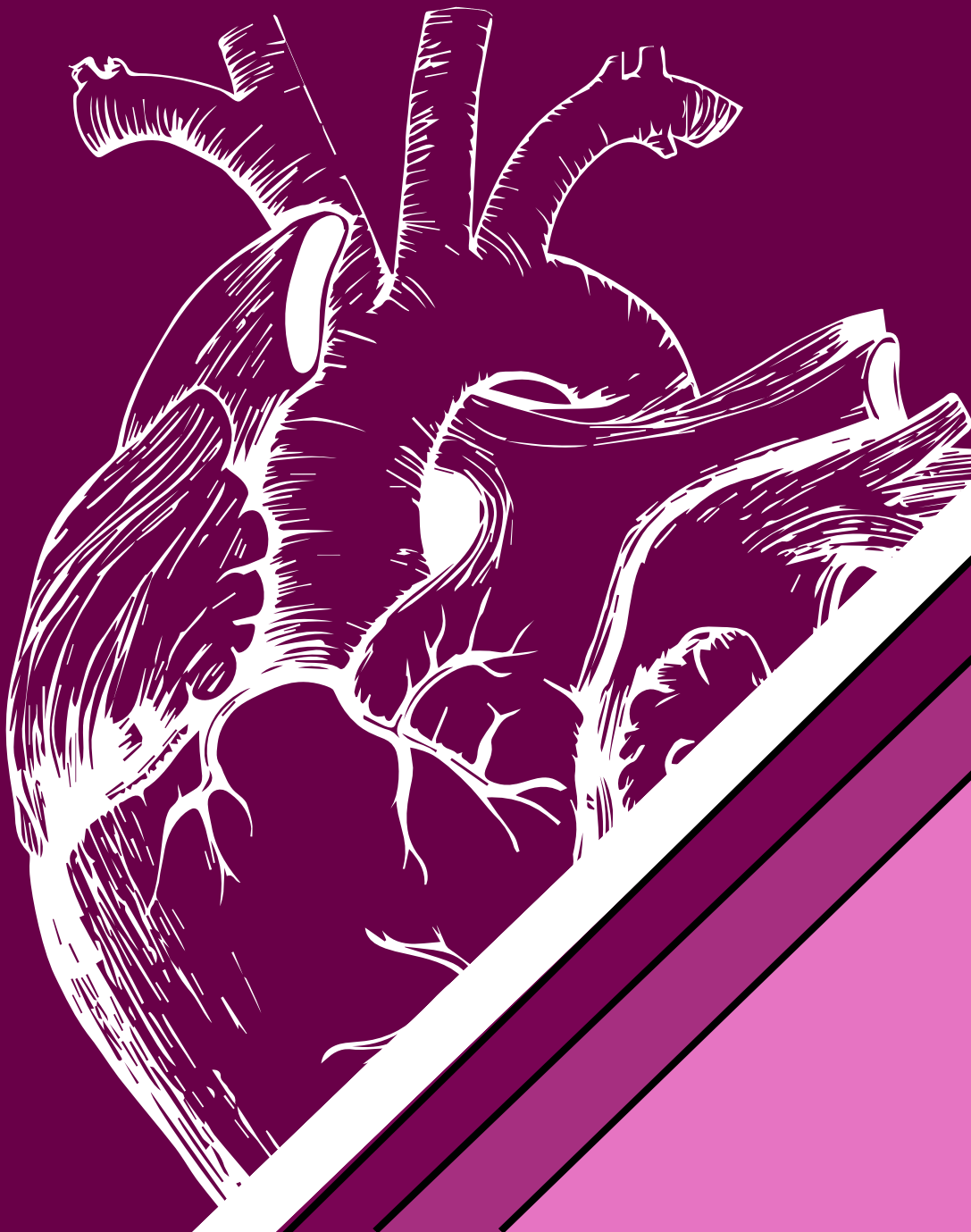
physiology

Dr.yanal –final summary – CVS

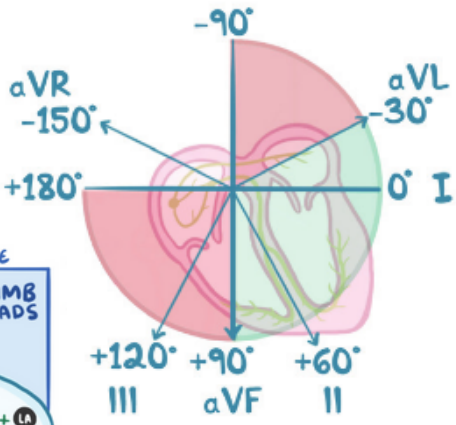


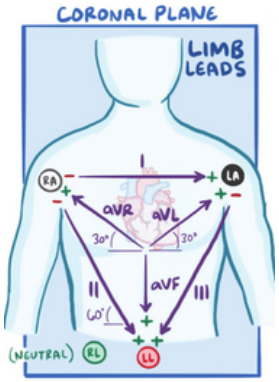
EBAA ALZU'BI

2023 / 2024

cardiac axis deviation

these are the positive leads vectors , if the lead is upward deflection on ECG , then it is positive vector , if the lead is downward on ECG , its vector is 180 degree opposite the positive one .





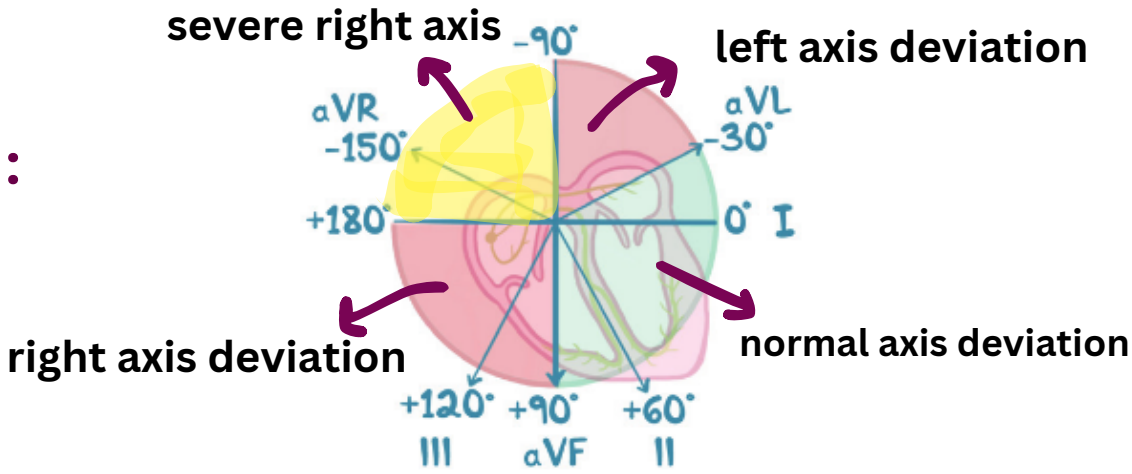
lead 1 = negative right arm to positive left arm
lead 2 = negative right arm to positive left leg
lead 3 = negative left arm to left leg

1 + 3 = 2

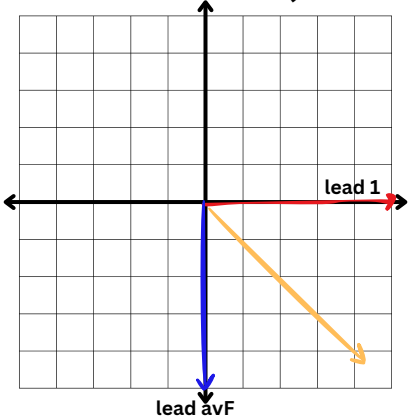
lead 1 + lead 3 = lead 2

to find cardiac axis , we find the mean axis , by choosing 2 leads , then locate them on axis , the mean axis is between them and closer to the higher voltage “bigger wave”.

if the mean is :



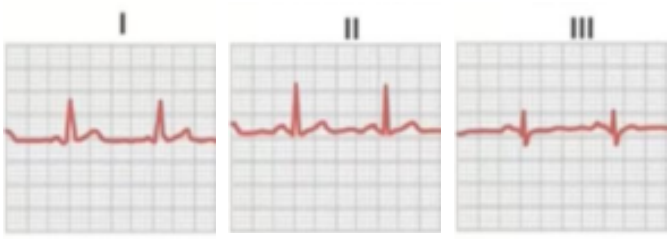
Q: if lead 1 is : + 5, lead avF : +5 , then the mean axis is ?



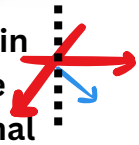
it is normal axis , 45 degree

	lead 1	lead avF
normal	+	+
right axis deviation	−	+
severe right axis	−	−
left axis deviation	+	−

in this case , if lead avF is bigger then it is left axis , if it lead 1 is bigger or lead2 is positive , then it is normal

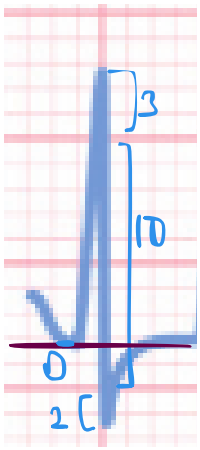


lead 1 + lead 3 are positive ,120 degree in between , and lead 1 is bigger , then the mean is closer to lead 1 , then it is normal



if lead 1 positive , lead avF negative , lead 2 positive , lead 3 negative , then the mean is ?

voltage

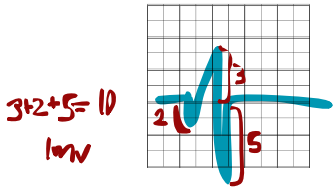


to calculate voltage , count small square number in Q, R, S , then multiply it with 0.1 , this is the voltage .

voltage is : 1.5

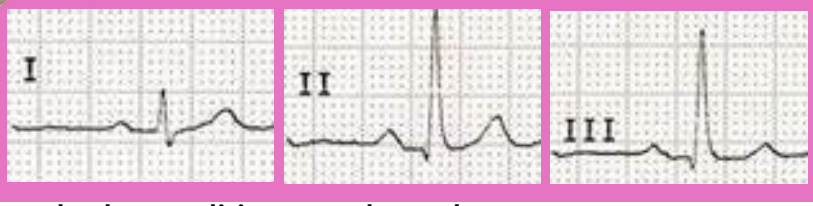
>4mV = high voltage

<1.5mV = low voltage



Increased voltage can indicate left ventricular hypertrophy, right ventricular hypertrophy, may be normal

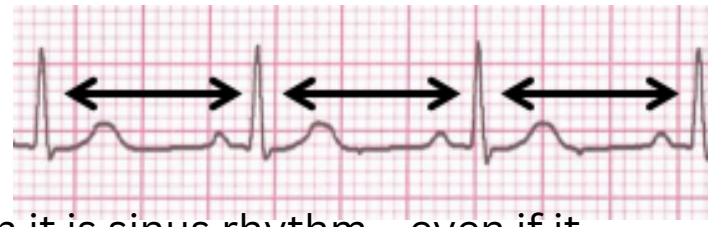
Determine the Lead with Greater Voltage



fluid in pericardium, pleural effusions, emphysema,muscle abnormalities cause low voltage

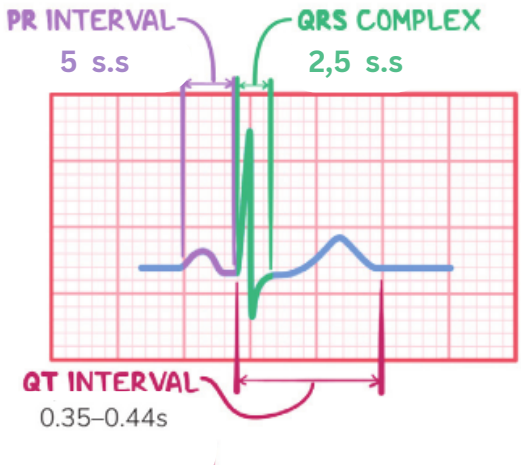
normal sinus Rhythm

- Constant RR interval
- Predictable recurring wave pattern (P-waves, QRS, T waves)



as long as it is predictable rhythm , then it is sinus rhythm , even if it is irregular , then it is regular irregularity .

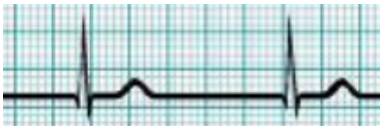
if it is unpredictable then it is arrythmia, to estimate heart rate count R waves , then multiply by 6 .



ASSESSMENT FOR NORMAL REGULAR RHYTHM

- Is there a P before every QRS complex?
- Is there a QRS after every P?
- Are the P waves normal?

IF YES THEN NORMAL SINUS



NO P WAVE – NOT SINUS = NODAL

Sinus Tachycardia

- Sinus tachycardia > 100bpm

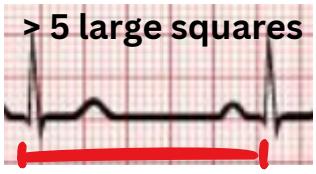
FAST beat , due to increased temperature (each Celsius = 13 more beats) , sympathetic stimulation , thyrotoxicosis (hyperthyroidism = more norepinephrine)

Sinus bradycardia

- Conventionally defined, sinus bradycardia <60bpm

sick sinus syndrome (ectopic pacemaker) , Drugs (like beta-1 blockers, calcium channel blockers, Digoxin that increases the vagal stimulation) ,Vagal stimulation

athletes have normal (physiological) bradycardia (50) , because of large stroke volume , they don't need faster pumping .



- If SA node fails, other latent ectopic pacemakers capable of automaticity
  - Atria, AV junction, His bundle, bundle branches can set heart rate
  - Each foci has unique rate (atrial foci 60–80bpm; junctional foci 40–60bpm; ventricular foci 20–40bpm)

Heart blocks

- Sinus block
  - SA node temporarily ceases to conduct impulse; usually resumes, may cause escape rhythm **no p waves , predictable rhythm**

A-V node InCharge -bradycardia

- AV block **due to Ischemia ,Compression , inflammation , excessive vagal stimulation**

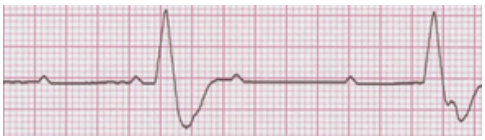
CAUSES ARRHYTHMIA

  - *First degree:* prolonged PR interval > 0.2s

  - *Second degree:* some P waves conducted to ventricles, followed by QRS complex while some not

Irregularly irregular : 2 p waves=1 qrs

  - *Third degree:* atria, ventricles beat asynchronously with no conduction through AV node (complete dissociation between P, QRS complexes) **, 15-40 bmp**



(e) Ventricular fibrillation

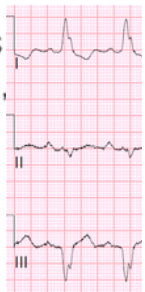
Stokes-Adams Syndrome

Complete A-V block=no Ventricles contraction for 30 min , Patient faints because of poor cerebral blood flow

Bundle branch blocks **due to ischemia , vagal stimulation ,compression , inflammation**

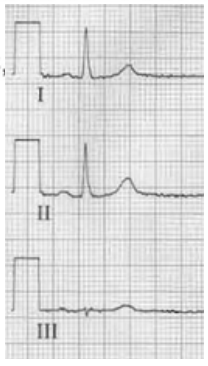
- Left bundle branch block (LBBB)
  - Activation of left ventricle delayed causing left ventricle to contract later than right ventricle
  - Broad QRS **>0.12**
  - Secondary repolarization abnormalities in right precordial leads (ST depression, T wave inversions)

left axis deviation



- Right bundle branch block (RBBB)
  - Activation of right ventricle delayed causing right ventricle to contract later than left ventricle
  - Absence of Q waves,

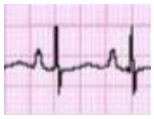
right axis deviation



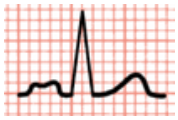
ECG CARDIAC HYPERTROPHY

ATRIAL DILATION/ENLARGEMENT

- Initial component of wave larger
  - Right atrial enlargement



- Terminal component of wave larger
  - Left atrial enlargement



- Amplitude of P wave in any limb lead > 2.5mm
  - Probable right atrial enlargement

RIGHT VENTRICULAR HYPERTROPHY

- Possible right axis deviation

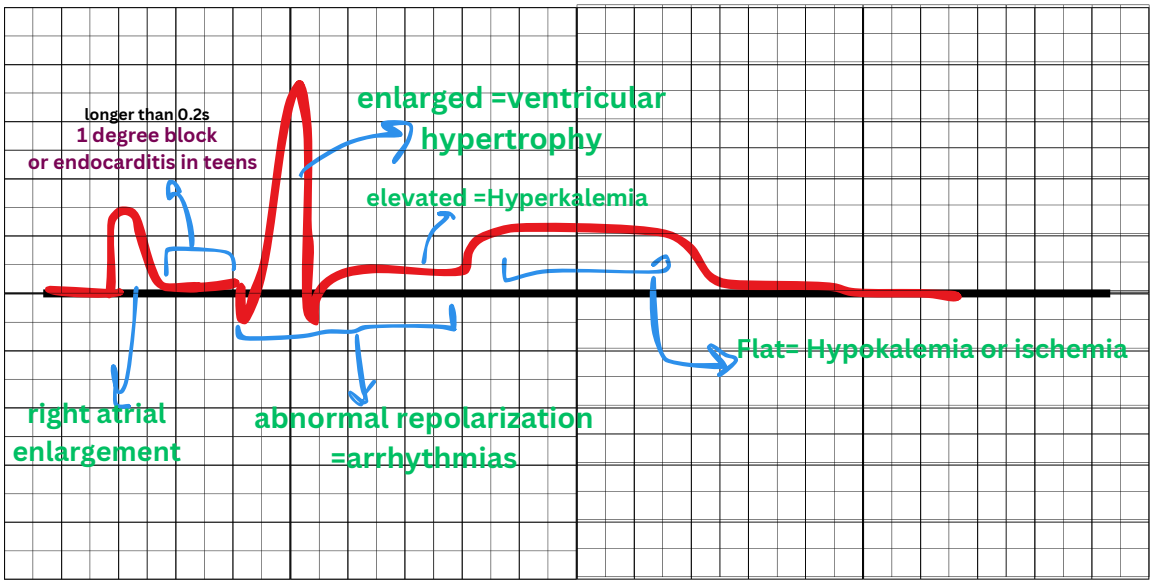
LEFT VENTRICULAR HYPERTROPHY

- Possible left axis deviation

- Changes in heart position: left shift caused by expiration, lying down and excess abdominal fat, short and obese (in obese people the abdominal content pushing against the heart causes LAD).
- Right shift caused by thin and tall person, also inspiration.

ventricular hypertrophy we look at :

1. **Heart's axis**
2. **duration of QRS complex**
3. **Voltage** >4mV = high voltage
   <1.5mV = low voltage



CALCULATIONS

pbo

$$SV = EDV - ESV$$

$$EF = SV / EDV$$

$$CO = SV * HR = EF * EDV * HR$$

SV

- Volume of blood (mL) ejected from ventricle with each contraction
- Calculated as difference between volume of blood before ejection/EDV, after ejection (ESV)
- EDV (120mL) - ESV (50mL) = 70mL
- SV affected by preload, afterload, inotropy

EF

- Fraction of EDV ejected with each contraction
- SV (70)/EDV (120) = 58 (EF)
- Average = 50–65%

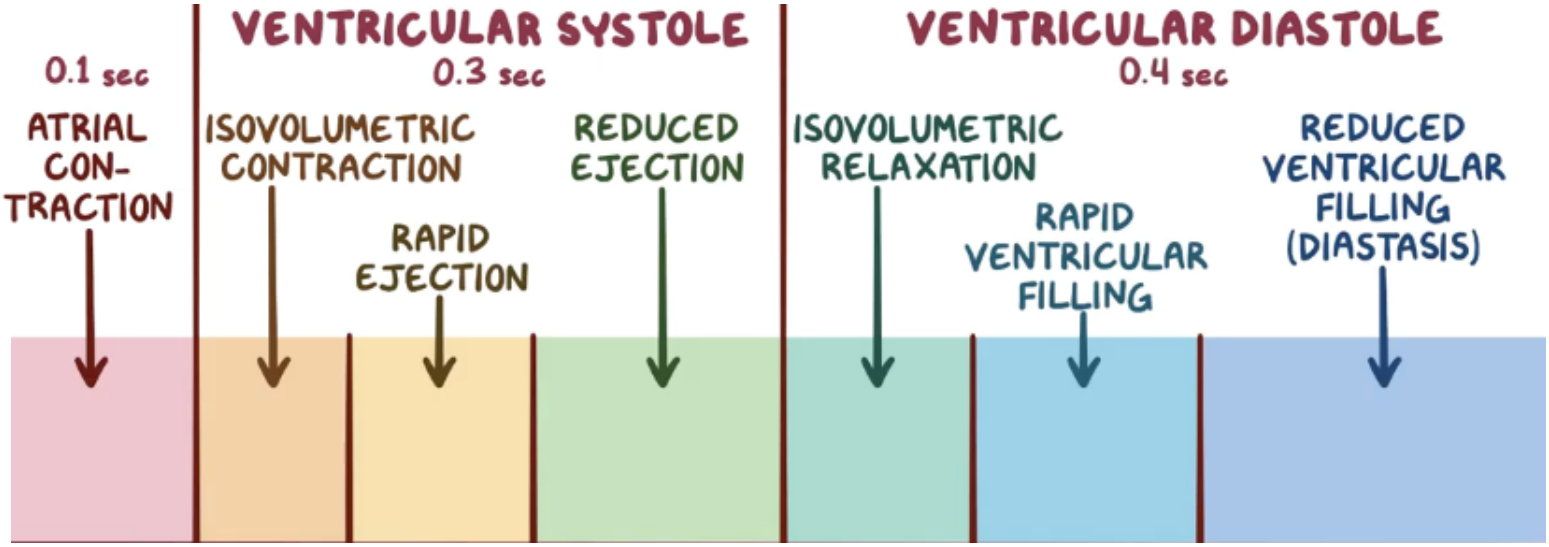
to classify heart failure degree

preserved ejection fraction (HFpEF) - >50%)
mildly reduced ejection fraction (>40% - 49%)
reduced ejection fraction (<40%)

CO

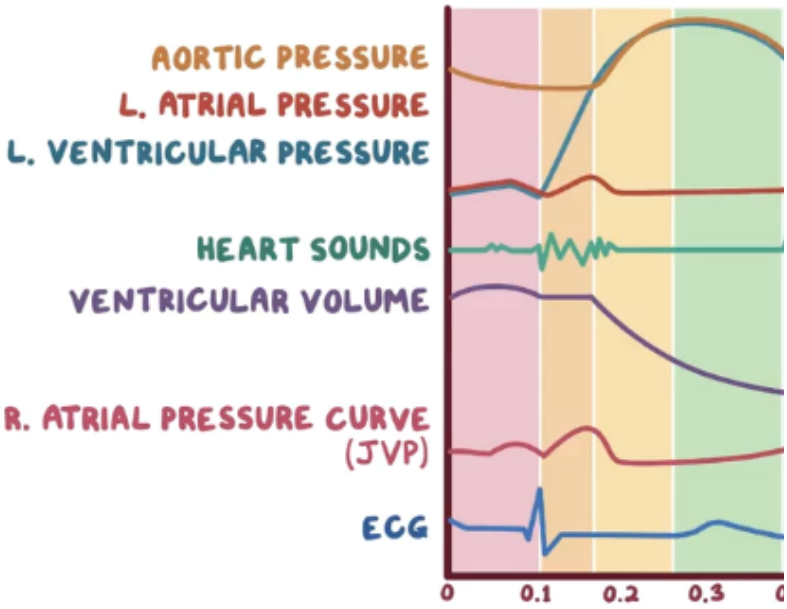
- Volume of blood ejected by ventricles per minute
- SV (120) x HR (70) = 4900mL/min

CARDIAC CYCLE



atrial contraction

from SA node , P wave , increase in atrial & ventricular pressure ,(S4)



isovolumetric contraction

CLOSED CHAMBER

ventricular depolarization QRS, high ventricular pressure , atrioventricular and aortic and pulmonary valves are closed , mitral closing cause S1 =lubb , C wave= contraction

rapid ejection

starts when ventricular pressure exceeds aortic pressure , aortic & pulmonary valves open, reach maximum pressure, ST segment

reduced ventricular ejection

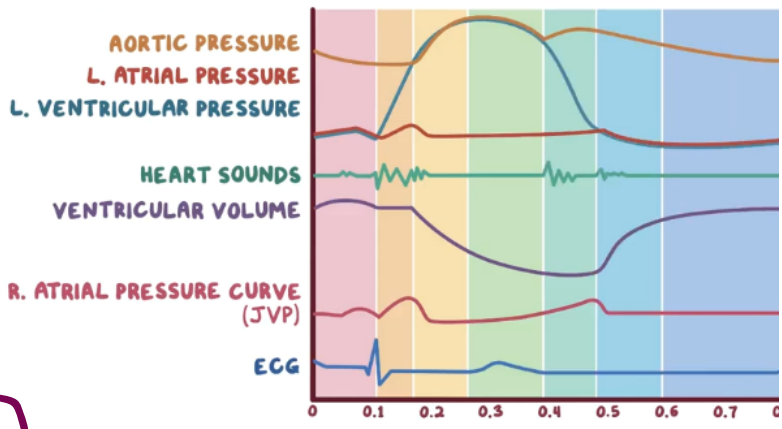
begins T wave ,ventricular repolarization, decreased ventricular volume & pressure.

isovolumetric relaxation

ventricular diastole begins ,end of the T wave , relaxed ventricle , low pressure , closed aortic valve = S2 =dupp , all valves are closed , "v" wave= no ECG changes.

CLOSED CHAMBER

Dicrotic notch due to backflow of blood= incisura



rapid ventricular filling

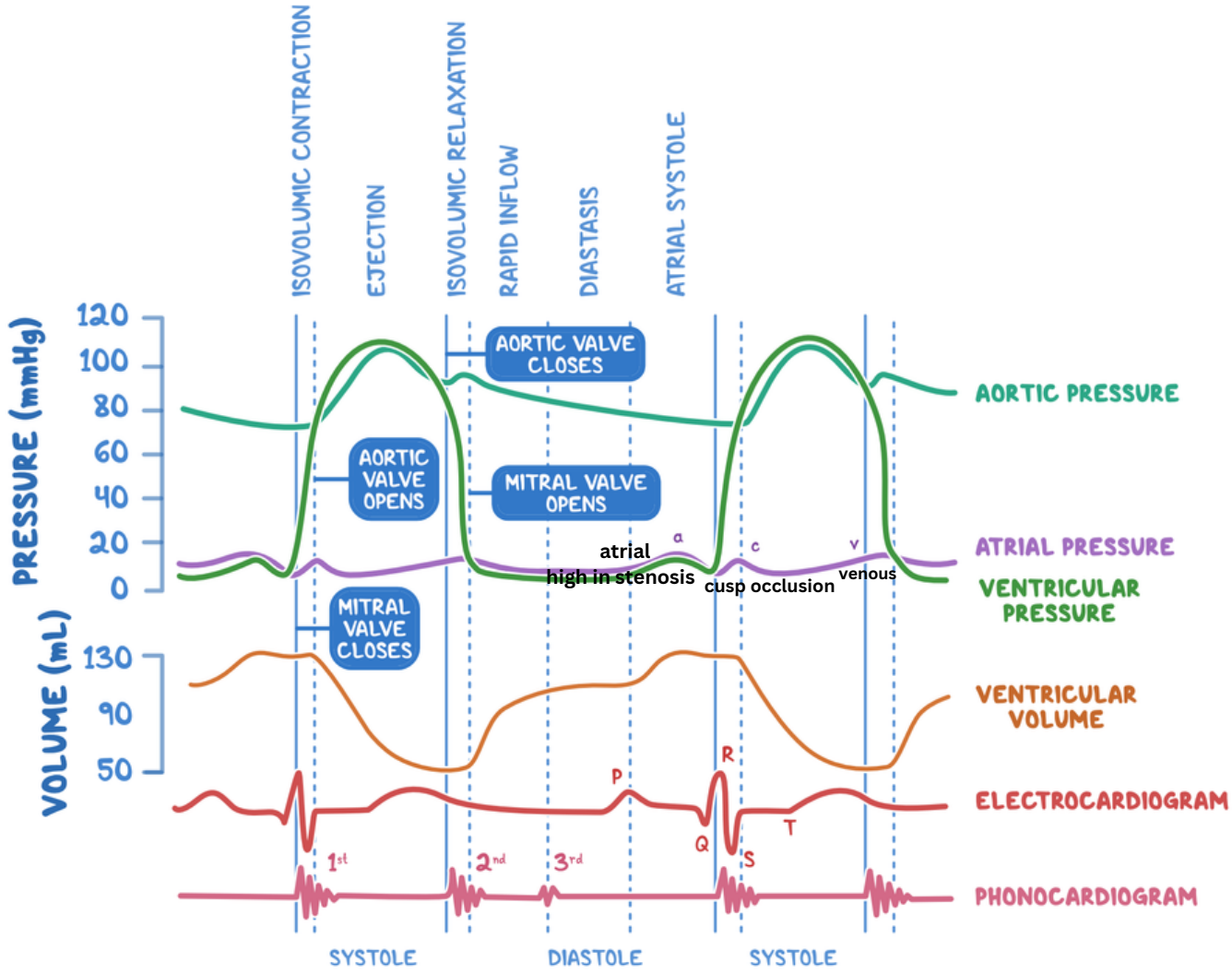
two atrioventricular valves open ,rapid filling .

VENTRICLES GET ABOUT 90% OF THE BLOOD

Reduced ventricular filling

longest phase ,called diastasis

THE CARDIAC CYCLE



SOME NOTES : BEST THINGS ARE IN THE MIDDLE

hypoxia cause increase in membrane permeability , increase Na + Ca inside , inducing ectopic pacemaker of the ischemic region , to send new impulse causing tachycardia , (less filling - less ejection - more ischemic) , same story again and again causing fibrilation then death .

tachycardia cause more tachycardia , ischemia cause more ischemia

cardiac output = heart rate x stroke volume

decrease HR = decrease CO , also high increase in HR = decrease in filling time = decrease in CO . so the max HR , or the suitable is =(220- age) X 75%

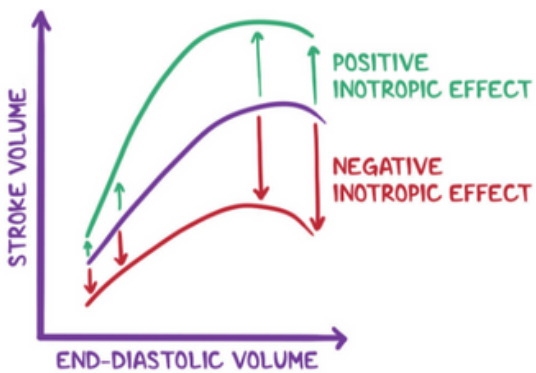
sympathetic = increase in funny Na , Ca+ L-type = increase HR =Positive chronotropic

parasympathetic = acetylcholine , K efflux = decrease HR

increase in preload "end- diastolic"+ contractility = increase in Stroke volume

increase afterload decrease stroke volume

- Extrinsic control through sympathetic stimulation, hormones (e.g. epinephrine), medications (e.g. digoxin) → ↑ contractility (positive inotropy), SV
- Negative inotropic agents (e.g beta-blockers) → ↓ contractility → ↓ SV

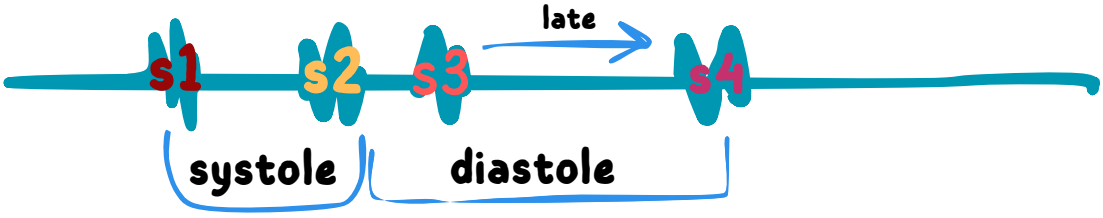


S1 = LUB , mitral close

S2= DUB , aortic close

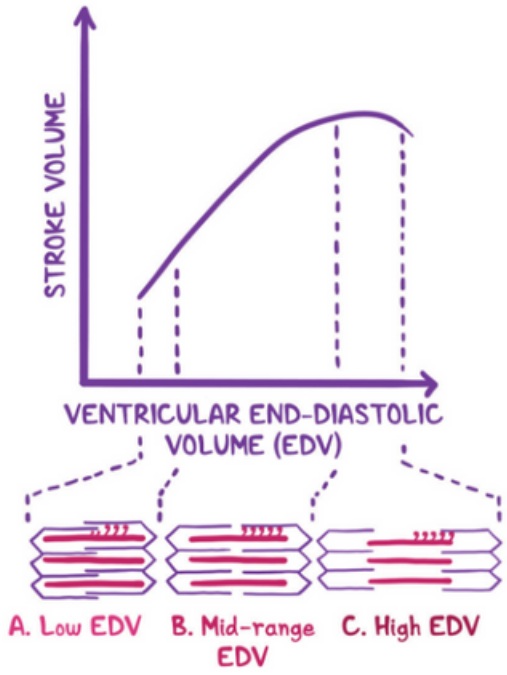
S3= Gallop Rythm , normal in children , pathological in adults= ventricular dilation

S4= pathological - ventricular stiff & hypertrophy .



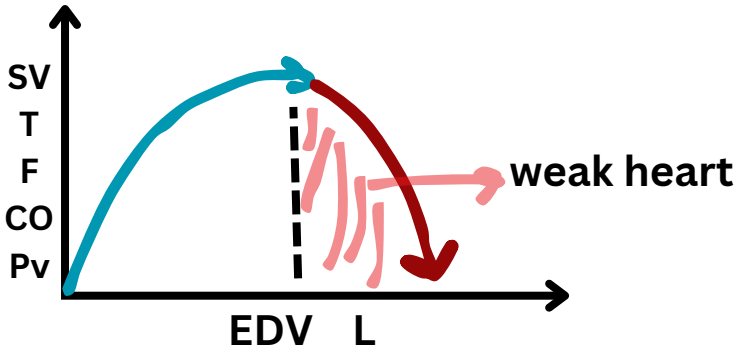
FRANK–STARLING RELATIONSHIP

- Loading ventricle with blood during diastole, stretching cardiac muscle → force of contraction during systole
- Length-tension relationship
  - Amount of tension (force of muscle contraction during systole) → depends on resting length of sarcomere → depends on amount of blood that fills ventricles during diastole (EDV)
  - Length of sarcomere determines amount of overlap between actin, myosin filaments, amount of myosin heads that bind to actin at cross-bridge formation
  - Low EDV → ↓ sarcomere stretching → ↓ myosin heads bind to actin → weak contraction during systole → ↓ SV
  - Too much sarcomere stretching prevents optimal overlap between actin, myosin → ↓ force of contraction → ↓ SV



- ↑ afterload → ↓ velocity of sarcomere shortening
- ↓ afterload → ↑ velocity of sarcomere shortening

increase in EDV = increase in length of sarcomere = increase of force and tension = increase in SV & CO to physiological limits .

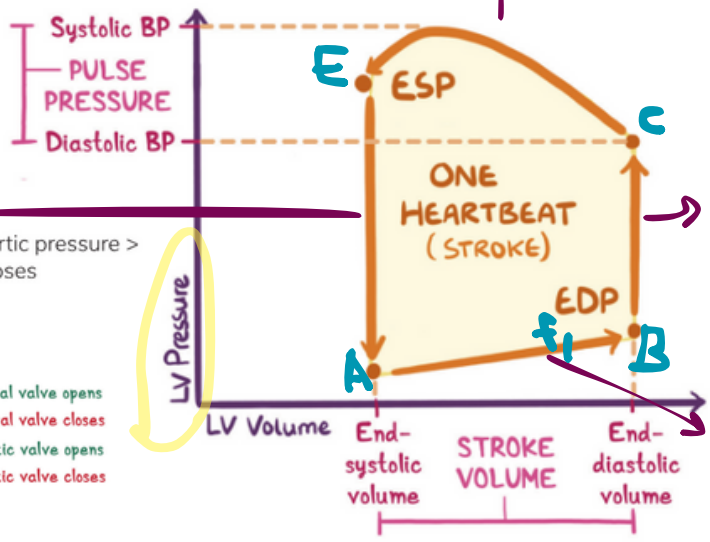


pericardium is there to prevent heart from further expansion (physiologically - NO ELASTIC)

L max = L rest in skeletal but heart can adopt : L max > L rest

PRESSURE-VOLUME LOOPS

- Graphs represent pressure, volume changes in LV during one heartbeat (one cardiac cycle/"stroke work")
- Pressure in left ventricle on y axis, volume of left ventricle on x axis



Ventricular ejection
- Pressure in left ventricle > aortic pressure → aortic valve opens → blood ejected

0.25s

Isovolumic contraction
- Systole begins (ventricular contraction)
- No changes to ventricular volume (mitral, aortic valve closed)
- Pressure builds

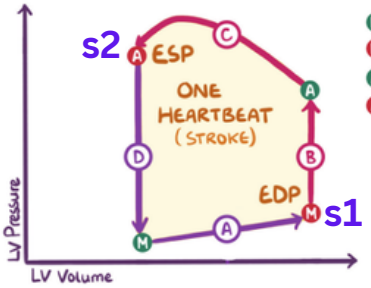
0.05s

Isovolumic relaxation
- Ventricle starts relaxing → aortic pressure > LV pressure → aortic valve closes
- End of systole
- ESV = 70mL

0.15s

Ventricular filling during diastole
- At end of this phase:
  - Mitral valve closed
  - Left ventricle filled (EDV) distended
  - EDV = 140mL

0.35s

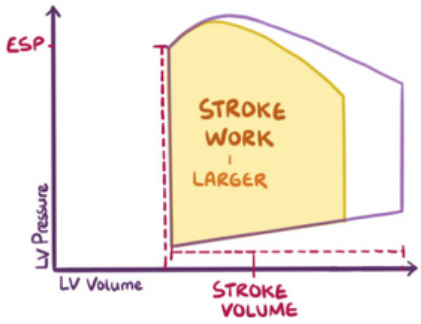


you can find :
EDV , SV , ESV , pulse pressure , mean pressure , EF , QRS wave (B to C) , P wave (f to b) ,shorter (b to c) , longer (a to b)

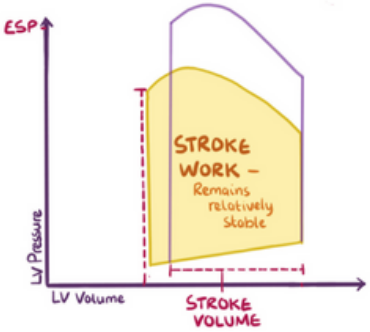
sound is cuz of closing not opening

- Cardiac parameters change → volume-pressure loops change
- ↑ preload (↑ EDV) → ↑ strength of contraction → ↑ stroke volume → larger loop
- ↑ afterload → ↑ ventricular pressure during isovolumetric contraction → ↑ less blood leaves ventricle → ↑ end-systolic volume (ESV) → ↓ SV → loop narrower, taller (smaller SV, higher pressure; stroke work remains relatively stable)
- ↑ contractility → blood under ↑ pressure → longer ejection phase → left ventricular pressure = aortic pressure → ↑ SV, stroke work, ↓ ejection fraction (EF), EDV → loop widens

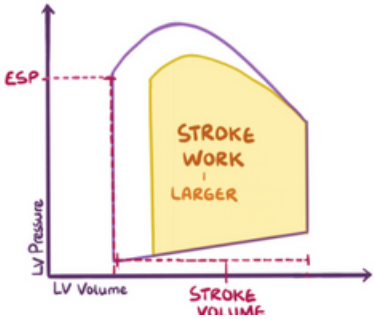
B. INCREASED PRELOAD



C. INCREASED AFTERLOAD

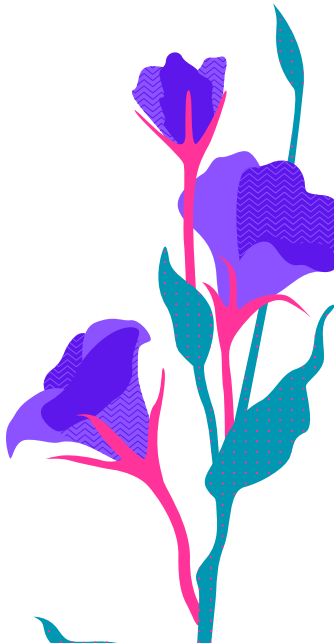


D. INCREASED CONTRACTILITY



WORK (PHYSICS) = FORCE x DISTANCE

External work is area of Pressure-Volume curve



FICK PRINCIPLE

oxygen consumption for each organ

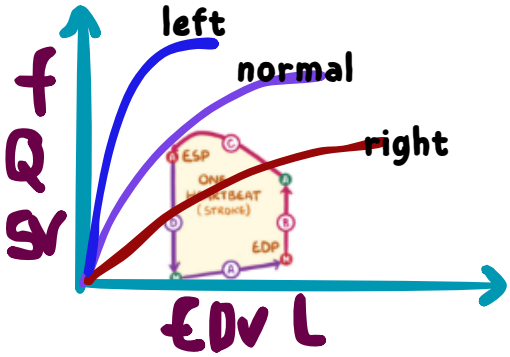
O2 CONSUMPTION = BLOOD FLOW X (ARTERIAL O2 - VENOUS O2)

$$\text{Cardiac Output} = \frac{O_2 \text{ consumption}}{[O_2] \text{ pulmonary vein} - [O_2] \text{ pulmonary artery}}$$

$$R = \frac{8\eta l}{\pi r^4}$$

- R = resistance
- η = blood viscosity
- l = length of blood vessel
- r^4 = radius (diameter) blood vessel raised to fourth power

Cardiac and vascular function curves



the curve shifts upward to left :

- **positive inotropic = more contractility = more SV**
- **digoxin**
- **epinephrine , thyroxine (sympathetic)**

the curve shifts downward to right :

- **parasympathetic**
- **beta blockers – Calcium channel blockers**
- **Acidosis**

increase in circulating blood(arterial , afterload)= shifts to right = decrease in venous retain

decrease in circulating blood = shifts to left = increase in venous retain"preload"

increase TPR = shifts downward

decrease TPR = shifts upward

اللهم إني أسألك أنك العليم الكريم أن توفقنا لما تحبه وترضى، وأن تكتب لنا النجاح والفلاح والتوفيق في تحصيل كل علم نافع، اللهم مالك السماوات والأرض ومنزل الكتاب لا تنزع العلم من بيننا وارفعنا به، وارزقنا يا الله فهم أنبيائك وحفظ الملائكة ورسلك