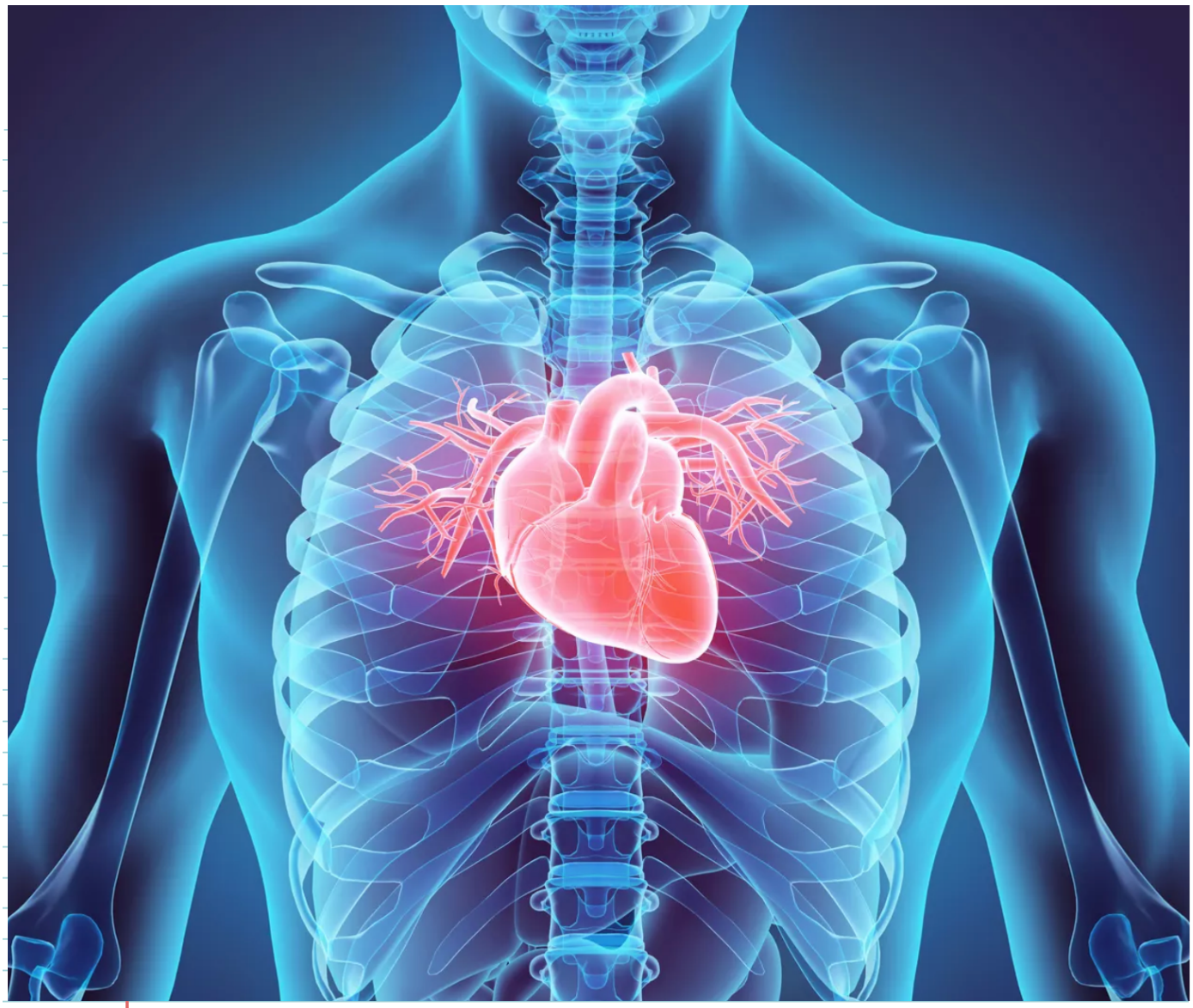
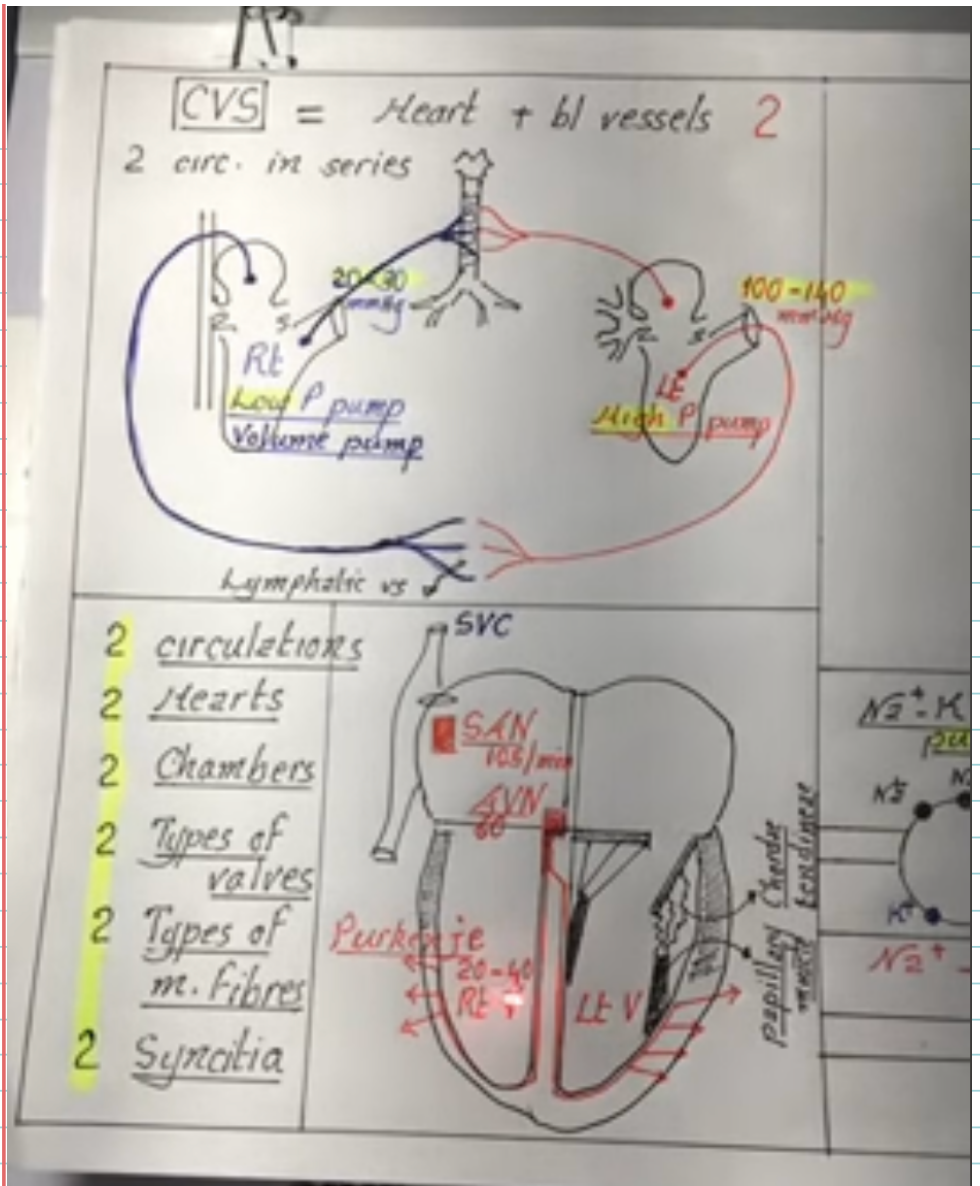


Cardio Vascular System physiology

DR. Nagi

- intro
- Contractility
- Excitability
- Conductivity
- ECG

Done by Dima AlRafaiah.



1 The Cardiovascular system consists of:

Ⓐ The Heart Ⓑ vessels

We have 2 circulations that are in series

2 Circulation: ① systemic circulation that pumps the blood with higher pressure to overcome the higher peripheral resistance from the left ventricle to the whole body and going back to the right atrium

② pulmonary circulation that pumps the blood with lower pressure from the right ventricle to the lungs and then to the left atrium

So I guess we can say that we have 2 hearts → The left heart (systemic)
→ The right heart (pulmonary)

in series = So both circulations are connected in sequence, one after the other

Cardic output (volume of the blood) is equal in both circulations

* what is different is the pump pressure which is higher in the left ventricle

Lt ventricle → high pump pressure 100-140 mmHg Rt ventricle → low pump pressure 20-30 mmHg

2) The heart consist of 2 types of chambers
- 2 to pump blood (Rt, Lt ventricles)
- 2 act as blood reservoir (Rt, Lt atrium)

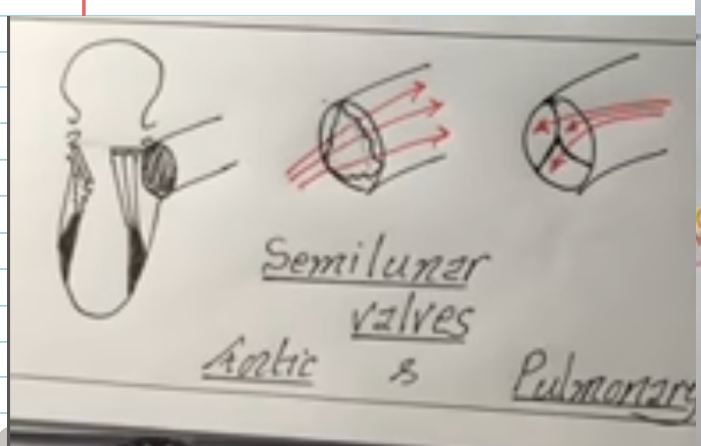
2 types of valves designed to prevent regurgitation of blood
- 2 atrioventricular (Rt tricuspid AV valve) (Lt bicuspid mitral AV valve)
- 2 Semilunars (Lt ventricle and aorta) Rt ventricle and pulmonary trunk)

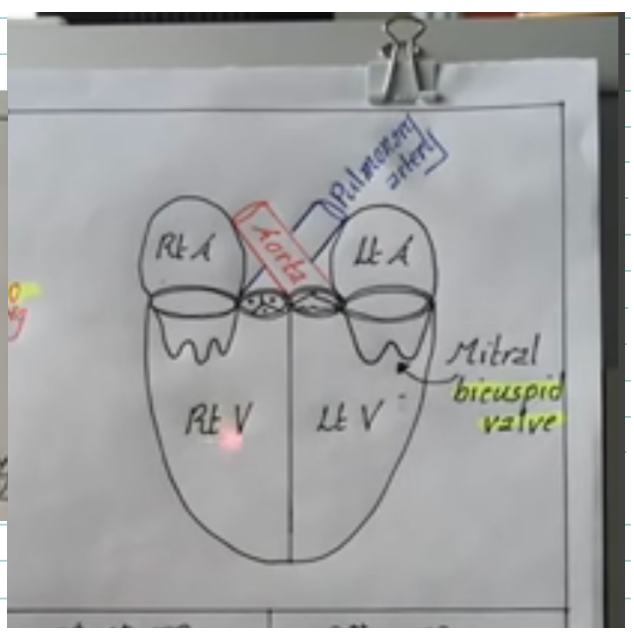
• The AV valves work passively meaning that their opening and closing is primirly influenced by pressure changes in heart chambers
→ The atria contracts, pressure there exceeds the pressure in ventricles so the valve opens

• The AV values cusps (leaflets) are connected to papillary muscles in the floor of ventricles via cord like structures called Chordae tendineae
→ as the ventricle relaxes, papillary muscle relaxes, chordae tendineae relaxes, the valve opens (This happens during Diastole)

→ as the ventricle contracts, p.muscle contracts, C.T contract and works to anchor the valve cusps closing them preventing backflow of blood (This happens during Systole)

⊛ While the p. Muscles and C.T provide structural support to the AV values, the actual opening and closing of the values are primirly passive responses to pressure changes. The p.m play crucial role in preventing the inversion of the valve cusps during closure





[3] In the heart, we find 2 types of muscle fibers
- The working contractile muscle fibers
- The conducting (pacemaker) muscle fibers

• The contractile myocardial cells, responsible for the forceful contraction of the heart.
(1) they're similar to the skeletal muscles as they exhibit a striated appearance due to the organization of their contractile proteins, actin and myosin.

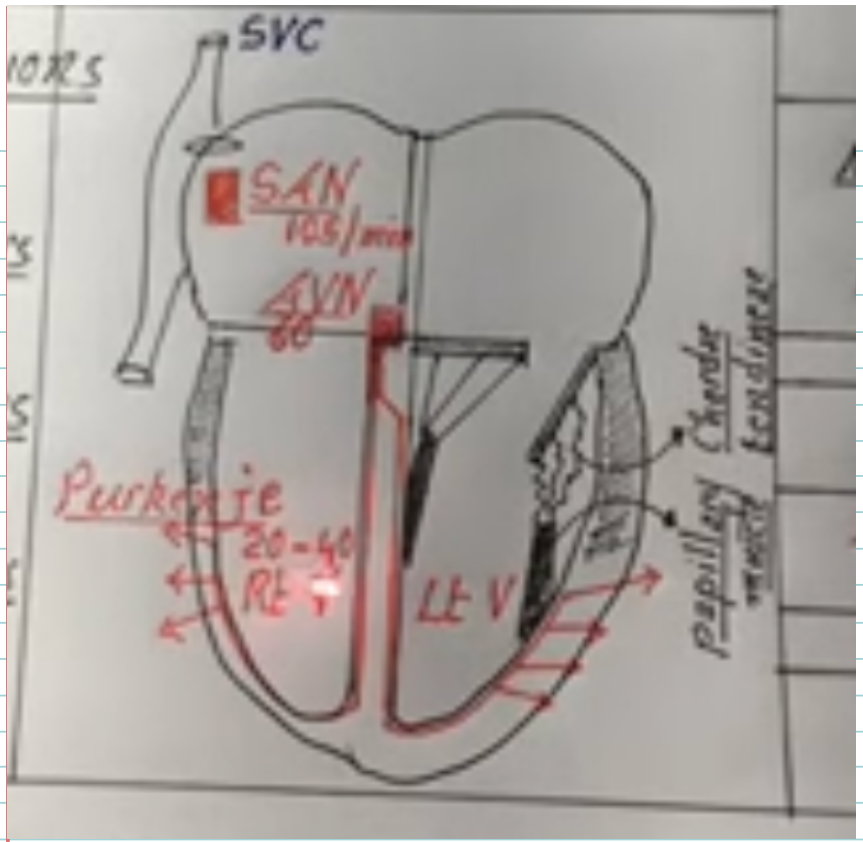
(2) they're connected end to end via specialized junctions called intercalated discs (functional syncytium)

(3) They use 2 sources of calcium as the heart is a non-stopping contractile muscle
- Extracellular calcium, enters via voltage gated calcium channels during depolarization (similar to smooth muscles)
- Intracellular calcium, stored and released from the sacroplasmic reticulum (similar to skeletal muscles)

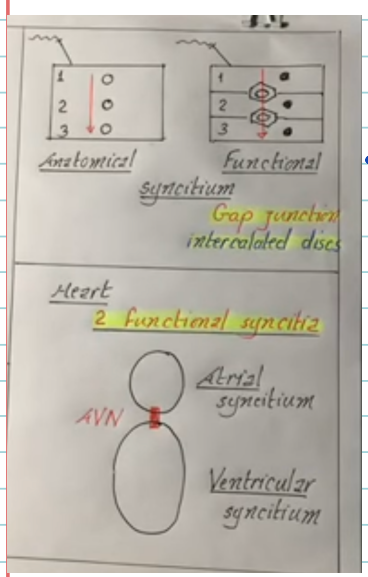
(4) They have transverse tubules, which are invaginations of the cell membrane, play role in transmitting the electrical signals.

• The conducting fibers are specialized cells that generate electrical impulses, there are two type
1. Sinoatrial (SA) node
   - → located in the right atrium, near the opening of SVC
   - → it's a natural pacemaker that initiates electrical impulses (around 105 in a minute) that spread through the atria, causing them to contract and pump blood into ventricles
   (They can spontaneously depolarize and reach a threshold potential without external stimuli)
   " pacemaker potential"
2. Atrioventricular (AV) node and bundle → in right atrium near the septum
   - → a bundle of specialized fibers located in the interventricular septum
   - → this bundle conduct the electrical impulses from the AV node to the ventricles transmitting the signal for ventricular contraction, HOW?
   - → through its branches, as it first divides into right and left branches which further divide into small purkinje fibers beneath ventricular endocardium extending outward to the pericardium

* purkinji fibers are specialized conducting fibers that spread throughout the ventricles, they transmit the electrical impulses to all parts, ensuring a powerful synchronized contraction (they have the highest conduction velocity)



4 In the heart, we have 2 functional Syncytium
- atrial syncytium
- Ventricular syncytium



- The functional syncytium in the heart refers to the coordinated and synchronized contraction of myocardial cells. Unlike skeletal muscle fibers that are stimulated independently, cardiac muscle cells function as a single interconnected unit during contraction

And that's achieved by the intercalated discs / gap junctions / desmosomes

5 In the heart we have 2 important pumps
- $3Na^+/2K^+$ Atpase pump in the sarcolemma of cardiac cells
- $3Na^+/1Ca^{++}$ exchanger also in the SL of cardiac cells

→ Na^+/K^+ actively transport ions across the membrane, maintaining a higher Na^+ conc outside and higher K^+ in
→ Na^+/Ca^{++} exchanger functions to regulate the intracellular conc of Calcium, it pumps Ca^+ both ways according to the need. e.g. During relaxation it removes Ca^{++} and lower its intracellular conc allowing the muscle to relax.

6 There are two primary types of action potentials in the heart:

- The ordinary action potential of the working myocardial cells
- action potential of the specialized conducting cells

Both of these play a crucial role in coordinating the rythmic contractions of the heart

(A) Action potential of the working atrial or ventricular myocardial cells (fast response)
- → responsible for the forceful contraction of the heart
- → characterized by a plateau phase and that's what distinguishes it from the typical A.P seen in neurons or skeletal muscle cells
- → Consists of 5 phases 0 – 4

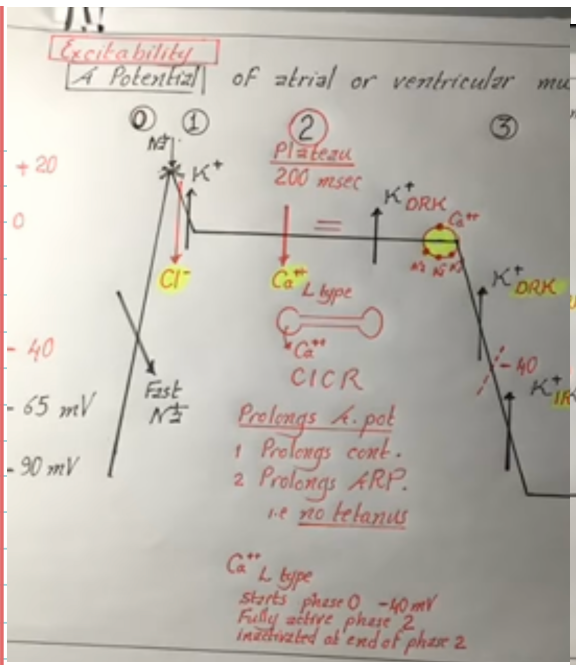
- Phase 0, Rapid ascending depolarization:
Here voltage-gated sodium channels open, allowing for rapid influx of Na^+ into the cells leading to rapid increase in membrane potential (→ with peak of +20), this happens in response to stimulus (from SA node or neighboring cells)
(That's why it's called fast response, because of the rapid influx of Na^+)

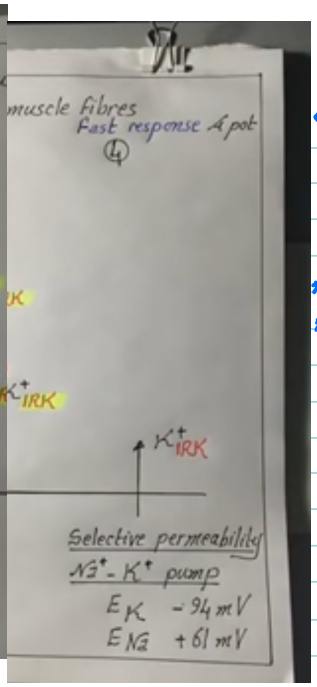
- Phase 1, Early repolarization (rapid)
- → Shortly after the peak of depolarization, Na^+ channels are closed and Transient Outward K^+ channels are opened
- → This allows the efflux of K^+ ions, leading to brief repolarization or a "dip" in the membrane potential
- → Simultaneously, there's also transient influx of chloride ions to accelerate the repolarization

- phase 2, Plateau (maintained depolarization)
- → In this phase there's balance between Ca^{++} and K^+
- → L-type calcium channels opens allowing the influx of Ca^{++}, which balances the efflux of K^+ through delayed rectifier channels (DRC)
- → So the influx of Ca^{++} prolongs depolarization, creating a plateau. Also this extracellular Ca^{++} induces the release of intracellular Ca^{++}, and together they cause contraction as we know
- → This plateau phase is unique to the cardiac action potential and is important to prolong A.P and the absolute refractory period, preventing premature depolarization, summation, tetanus

- Phase 3, final repolarization:
- → By the end of phase 2, L-type Ca^{++} channels close and Na^+/Ca^{++} exchanger is activated to play role in Calcium extrusion
- → Delayed rectifier K^+ channels remain open, allowing further efflux of K^+

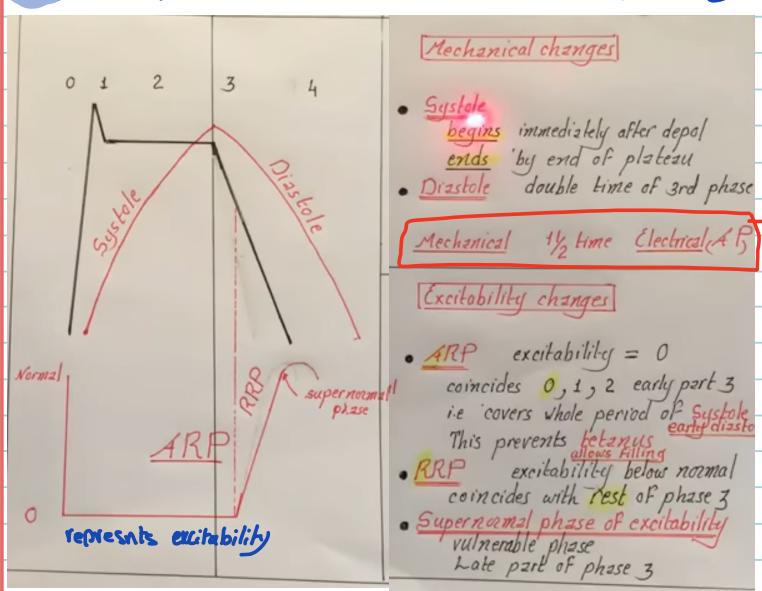
- Phase 4, Resting membrane potential (-90):
- → maintained by the membrane permeability and the activity of leak channels for K^+ and Na^+
- → the cell is stable and ready to the next stimulus





* longer action potential
* longer absolute Refractory period (to prevent tetanus)
* No hyperpolarization
* No Summation

Accompained Mechanical and excitability changes



Very important to:
1- prevent tetanic contraction
2- optimal filling of chambers

Mechanical

- **Systole** (Contraction):
begins right after depolarization lasts whenever there's intracellular calcium, so during phase 0,1,2 and closes when calcium channels are closed early at phase 3

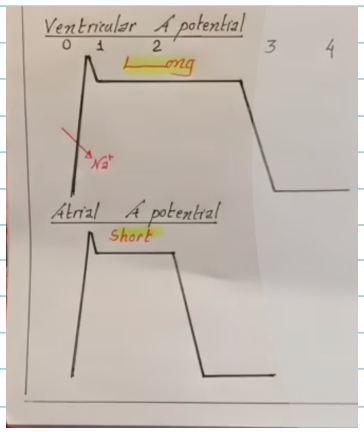
- **Diastole** (Relaxation)
begins at beginning of phase 3 when Ca^{++} channels are closed, lasts double the time of phase 3

Excitability

- **ARP** (very long compared to skeletal cells)
Cardiac cells can't be stimulated, excitability = 0. lasts during phase 0,1,2, early 3 covering systole and early diastole (to ensure filling before the next contraction) A long ARP prevents summation and tetanus

- **RRP** : excitability is not 0 but below normal, covers diastole

Ventricular Vs Atrial A.P



As you can see, Ventricular Myocardial cells go through longer action potentials in comparsion with atrial ones, and that's because they have distinct composition and behaviors of Ion channels.

Ventricular cells need a longer action potential to support the extended contraction required for pumping blood into the systemic circ. In contrast, atria primarily contribute to filling the ventricles (which 80% happens passively)

(B) PaceMaker potential, Slow response A.P

→ Auto-rythmic cells, meaning that they can generate spontaneous action potentials without external stimulation, particularly those found in (SA) node as they have the highest rythmicity.
→ They lack A stable resting membrane potential, they exhibit a gradual spontaneous depolarization during their diastolic phase (phase 4)
→ Consist of 3 phases 4-0-3
→ No plateau

- phase 4 (Diastolic depolarization)
→ unlike non-pacemakers, pace maker cells don't have stable resting membrane
→ Instead they undergo a spontaneous gradual depolarization during their diastolic phase
→ This depolarization is initiated by the slow influx of positive Ions

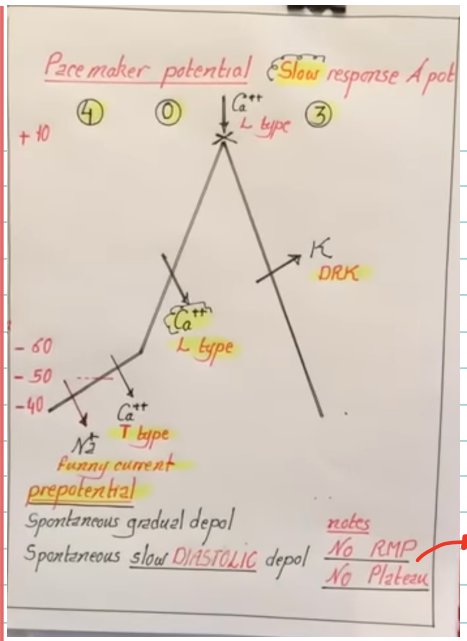
($-50 \rightarrow -45$) Ca^{++} through T-type Calcium channels

Na^{+} ($-60 \rightarrow -50$) Through funny channels (If)

→ This phase is depicted as a slope on a graph, it represents the gradual increase in membrane potential toward the threshold, the higher the slope the higher the rythmicity, and it's affected by the type of the pacemaker (higher slope in SA) and the Stimulation of autonomic nervous system

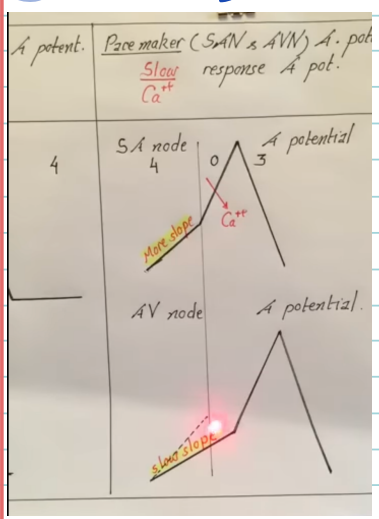
- phase 0 (upstroke)
→ Calcium influx due to opening of L-type Ca^{++} channels causing depolarization with peak of +10

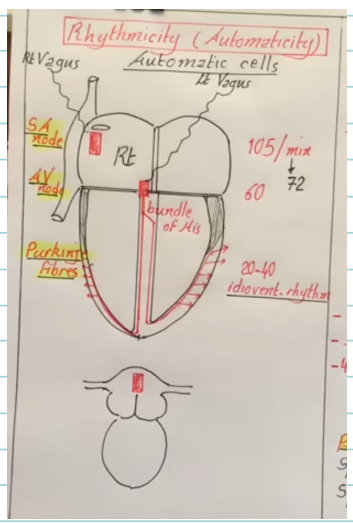
- phase 3 (Repolarization)
→ potasium efflux due to opening of DRK channels causing repolarization back to -60



→ No Stable resting membrane potential

✳ Rythmicity





→ The natural primary pacemaker of the heart is SA node, it has the highest rhythmicity, it can generate around 105 impulse/M (Tapered down to 75/m by vagal stim)

→ The electrical signals from SA node spread across the atria causing them to contract
→ due to the presence of fibrous rings (electrical insulators), the only connection between SA node and the ventricles is via AV node.
→ So the primary function of AV node is to conduct electrical impulses to ventricles
AV node – AV bundles – purkinje fibers
→ But under certain circumstances where SA Node is defective, it can serve as secondary pacemaker but with much lower rhythmicity (notice the lower Slope), it can generate 60 impulses/m (escape rythms)
* AV bundles and purkinje fibers can also generate escape rythms in cases where AV node is also impaired
→ 20-40 /M

* factors affecting rate of discharge of SA node (Rhythmicity or HR)

1. Autonomic nerves

Sympathetic → +ve chronotropy, tachycardia
Mech: Noradrenaline binds to B_1 leading to → ↑ cAMP, ↑ funny current, ↑ slope of phase 4, reach the threshold, phase 0 in shorter time

Parasympathetic → -ve chronotropy, bradycardia
Mech: Acetylcholine binds to Muscarinic receptors → ↓ cAMP
- Activates K_{Ach} channels → K efflux, ↓ funny current, lower slope

2. Catecholamines → Same effect as sympathetic N.S

3. Body temp → each 1 c° rise increases heart rate by 10 beats/min

4. Extracellular K
↓ K → ↑ HR (↑ slope of phase 4 by ↓ K conductance in SAN)
↑ K → ↓ HR

5. Calcium channel blocking drugs
↓ HR ↓ Contractility (inotropy) by inactivating Ca^{++} L-type channels.

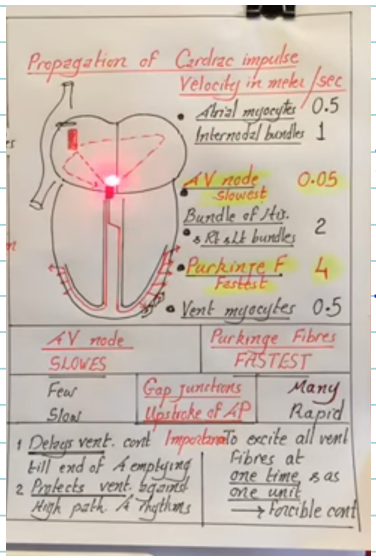
7. So we talked about 3 properties of the heart (Contractility, Excitability, rhythmicity) and the last one is ⇒ Conductivity (Note that they're all MYOGENIC)
→ Conductivity of the heart refers to its ability to transmit electrical impulses through its specialized conduction system allowing coordinated contraction and rythmic beating

→ The heart has a specialized conduction system composed of SA node, AV node AV bundle (bundle of his), purkinje fibers
→ each component is different in its velocity of conduction with purkinje fibers being the fastest, and AV node being the slowest. So what determines velocity of conduction?

Velocity of conduction depends mainly on
- Gap Junctions
  → Concentrated in the intercalated discs which are specialized regions connecting myocytes
  → the more gap junctions there are, the higher the conductivity
- Amplitude and speed of upstroke (depolarization)
  → a higher amplitude and a faster upstroke indicates a more rapid rise in membrane potential and thus a higher conductivity

- There's other factors affecting conductivity like O_2 (directly), intracellular Ca^{++} (inversly), fiber diameter (directly), Membrane resistance (inversly)

→ Propagation of Cardiac impulse



1) Begins with the initiation of electrical signals in the SA node

2) The electrical signals spread across both atria through atrial Muscle Cell with velocity of 0.5 m/sec and through internodal bundles 1 m/sec

3) The impulse reaches the AV node and experience a brief delay, making the velocity of AV node the slowest in the path 0.05 m/sec

4) from the AV node, the impulse travels down bundle of His and then divides into the left and right bundle branches which extend into the respective ventricles 1 - 2 m/sec

5) The impulse travels along the purkinje fibers, spreading throughout the ventricles 2 - 4 m/sec

6) The impulse spread across the ventricle through ventricular myocytes 0.5m/sec

* AV node (slowest), less gap junctions, Slow upstroke, to ensure complete filling of ventricles before they deliver them the signal to Contract
* purkinje fibers (fastest), Many gap junctions, rapid upstroke, to excite all ventricle fibers at one time as one unite (forcible Contraction)

✳ factors affecting velocity of Conduction

1. Autonomic nerves
   - Sympathetic → +ve dromotropy, ↑ rate of Conduction
     Noradrenaline (β_1) ⇒ faster upstroke
   - Parasympathetic → -ve dromotropy, ↓ rate of Conduction
     Acetylcholine (Muscarinic) ⇒ Slower upstroke

Mech

2. Drugs → Digitalis (Stimulates parasympathetic)
   +ve inotropic, -ve dromotropic, -ve Chronotropic
   ↳ used for heart failure
   ↳ used for tachycardia

18 ECG, ElectroCardioGram (Biphasic action potential recording)

→ A diagnostic tool in Cardiology, helping healthcare professionals asses the hearts electrical patterns and detect irregularities in rhythm and conduction

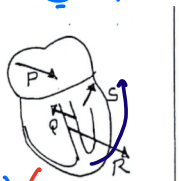
→ It records rapid voltage (summed electrical activity) changes of heart reading between 2 skin electrodes (body fluids are good conductor)

→ The Machine
- a monitoring Machine used in ICU
- The electrical signals from the heart are fed to a heat stylus (pen) that creates marks on a moving calibrated strip of paper producing the characteristic ECG waveform.

→ Vector (what we're trying to capture)
- An arrow that represents Sum of electrical activity

instantaneous vectors

Vector represents	Direction of vector (وجهة هاد وكتفق لوين اتجاهها)
Atrial activity	! Downward to the left
Septal activity	upward to the Rt
ventricular activity	! Downward to Lt
Base of Lt vent	upward to Lt

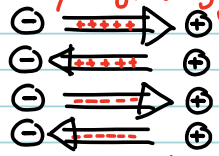


So 1 vector in the atria appears as 1 wave (P)
3 vectors in ventricles apears as 3 waves (Q, R, S) (our main vector is the ventricular one → (R) (axis of the hear))

Rules of waves

- Voltage, length of vector (وقوة الموجة وارتفاعها) ∝ Mass of tissue (ventricle > atria)
- Duration of wave (width) ∝ $\frac{1}{\text{velocity of conduction}}$
- polarity of wave (positive deflection OR negative deflection)

changes in intracellular charges during pathway of electrical signal



if depolarization ++ directed toward +ve electrode → +ve wave
if depolarization ++ directed toward -ve electrode → -ve wave
if repolarization −− directed toward +ve electrode → -ve wave
if repolarization −− directed toward -ve electrode → +ve wave

(احنا منقارن اتجاه الـ vector بكل ما كانو متوازيين وبنفس الاتجاه ، كل ما هاد الـ vector كان متوازي مع الـ lead (leads) positioning of electrodes بال (electrical activity) ما رح نقرأ اشي)
وازا كانت عمودية على الـ lead ، ما رح نقرأ اشي واعطاني موجة لوفخ ، وازا كانت عمودية على الـ lead ، ما رح نقرأ اشي

* No potential is recorded when the ventricular muscle is either completely depolarized or repolarized

Vec 1: ماشي بنفس اتجاه الـ lead (متوازي) اذا رح يكون عندو لوفخ واطول موجب ورح تكون لفوق

Vec 2: ماشي بعكس اتجاه الـ lead ولكنه موازي اذا رح يكون عندو موجه للتسفل

Vec 3: بنفس اتجاه الـ lead ولكنه عامل زاوية (منخليها ومناخد الحكية السيبنية) رصيك منعتبر الموجة لفوق

Vec 4: عمودي على اتجاه الـ lead اذا ما رح نقرأها .

متى حطينا صدول الـ 2 electrode بهاي الوضعية حوالين القلب ، وبدي احاول القط الـ vectors وارسملهم موجة ،
لازم اعرف : ١- بأي اتجاه ماشي الـ vector بالنسبة لـ lead ؟ عشان اقدر اتجاه الموجه
٢- كم قيمة هاد الـ vector ؟ عشان اقدر ارتفاع الموجه
٣- كم وقت استغرق هاد الـ vector للمرحلة اللي بدي عشانه اقدر عرض الموجة

→ lead
- is the position between 2 electrodes, or the actual reading between them
- we use 10 electrodes (6 on chest, 4 on limbs) and we gain 12 readings (12 leads)
- According to the TYPE of electrodes, leads may be:
  1. Bipolar leads → use 2 electrodes placed on the body (+ve, -ve), and the electrical activity (voltage differences) is measured between two points. (So bipolar leads are between 2 exploring electrode and they're 3 out of the 12)
     → the remaining 9 leads
  2. Unipolar leads → represents electrical activity between one active (exploring) electrode and a reference point (0 milli volt), the reference (ground) electrode is a combination of other electrodes or an average of electrical potentials

- According to the place of the electrode, leads may be
  1. Chest leads (6)
  2. limbs leads (4)

→ So the Machine have 10 electrode, we put 6 on the chest, 4 on the limbs (Not on a bony prominance) and the Machine generate 12 readings.
- Why putting 10 electrodes and having 12 different readings?
  to view the heart from every single angle

- The 10 electrodes in a standard 12-leads ECG are placed as:

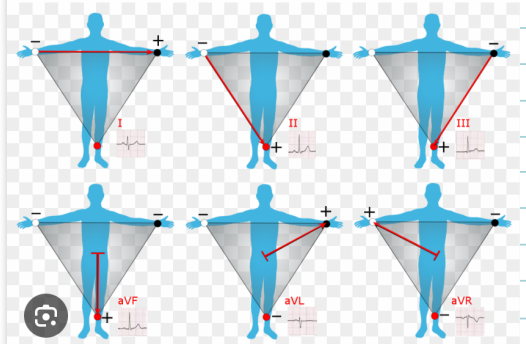
4 on the limbs → one on the Rt arm, one on the Lt arm, on the Lt leg, on the Rt leg

6 on the chest → V_1 in the 4th intercostal space right the Sternum
V_2 in the 4th intercostal space left the Sternum
V_3 between V_2 and V_4
V_4 in the 5th intercostal space at the midclavicula line
V_5 horizontally at the same level as V_4 in the left anterior axillary line
V_6 horizontally at the same level as V_4 and V_5 in the left Midaxillary line

- Those 10 electrodes eventually give us 12 leads (readings), of 3 types:

[1] Standard Bipolar leads (between 2 exploring, limbs)

	Exploring -ve electrode	Exploring +ve electrode
Lead I	Rt arm	Lt arm
Lead II	Rt arm	Lt leg
Lead III	Lt arm	Lt leg



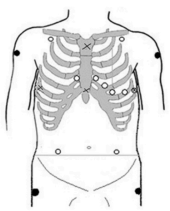
* we put +ve electrode on the Lt and down because we want +ve wave (remember direction of vectors and the polarity)

[2] Augmented (a) unipolar (V = unipolar) leads (one +ve electrode is attached to a limb, and the other is attached to the other two limbs)

	Exploring +ve electrode	other electrode
aVR	Rt arm	Lt arm and Lt leg
aVL	Lt arm	Lt leg and Rt arm
aVf	Lt leg	Rt arm and Lt arm

[3] unipolar chest leads (pericardial)

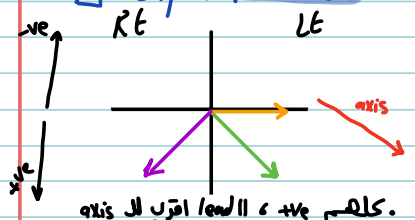
	Exploring +ve electrode	Exploring -ve electrode
V_1	4th space para sternal (Rt)	R L f
V_2	4th space para sternal (Lt)	R L f
V_3	between V_2 and V_4	R L f
V_4	5th space midclavicular	R L f
V_5	5th space ant. axillary line	R L f
V_6	5th space mid axillary line	R L f



R: Rt arm
L: Lt arm
f: Lt leg

Let's imagine those leads

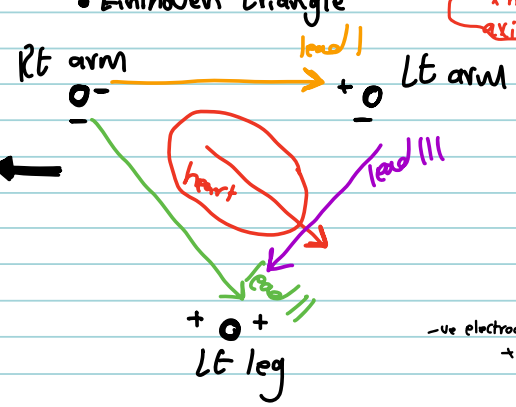
[1] Bipolar leads



axis اقرب لـ lead II و +ve كلهم.

• Einthoven triangle

* here our vector is the axis of the heart



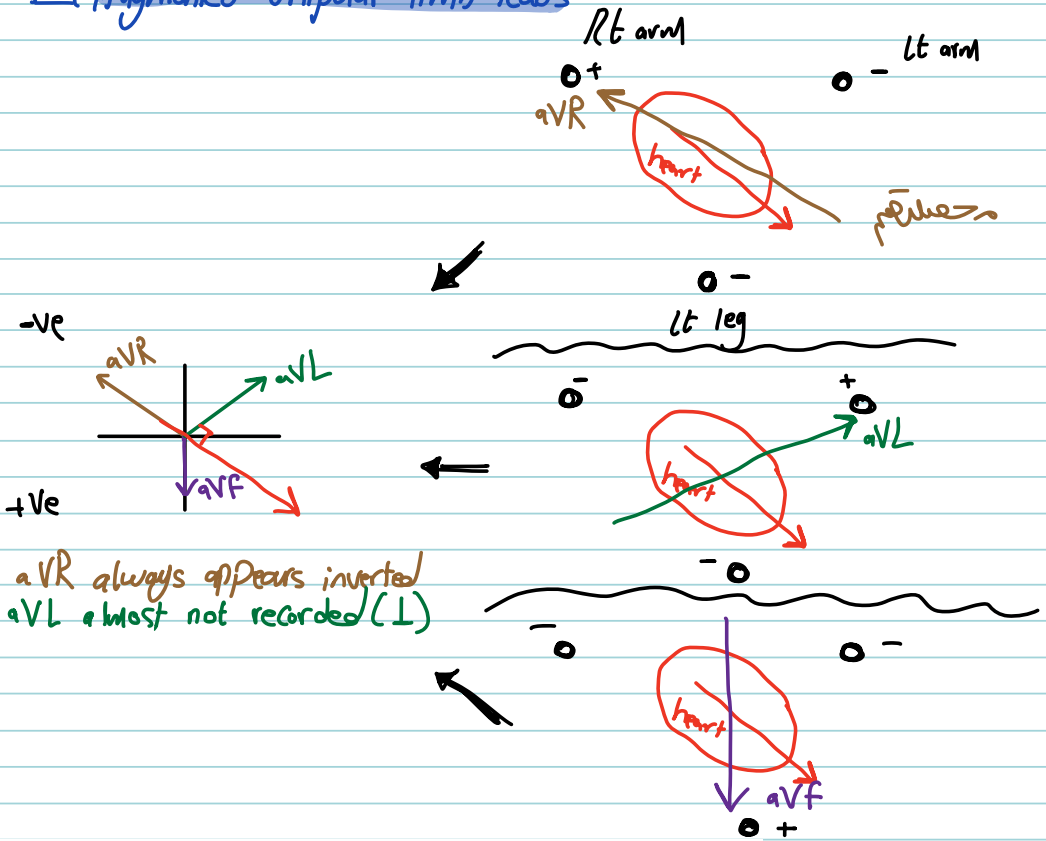
+ اتجاه نظراع للقلب هو -ve electrode و +ve الـ

حتى نعرف اتجاه الموجة لهاد الـ lead وارتفاعها ، ننظر لحالة قلبها بمحور القلب
That's why lead II appears. كل ما كانت اقرب لاتجاههم كل ما كانت اعلى.
The 1- highest 2- positive wave

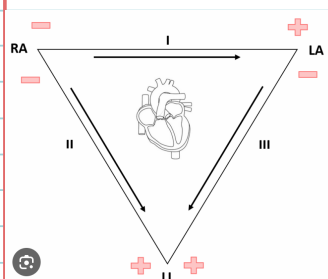
• we have a rule, The more the lead is parallel to the vector the maximal the effect (if it's perpendicular → not recorded)

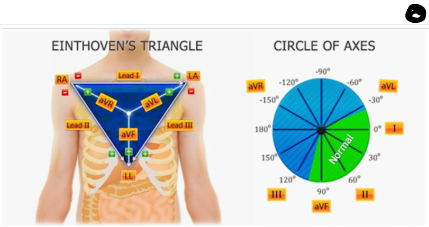
in this case our vector is (R) the axis of the heart

[2] Augmented unipolar limb leads



aVR always appears inverted
aVL almost not recorded (⊥)





* **Einthoven triangle**:
Helps to understand the orientation of the limb leads and how they contribute to the overall electrical axis of the heart in the frontal plane

* **Einthoven law**:
a mathematical relationship that holds true for normal electrical axis in the heart, any deviation from this relationship may indicate abnormalities in cardiac conduction or the presence of pathologies.

lead III + lead I = lead II

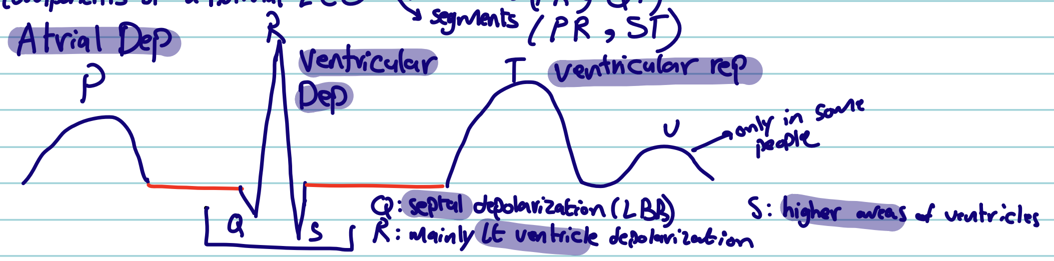
- Views of the heart
  - Lateral view : lead I, V_5, V_6, aVR, aVL
  - Inferior view : lead II,
  - Inferior lateral view : lead III, aVF
  - Septal view : V_1, V_2
  - Anterior view : V_3, V_4

- Important to Identify where the problem is exactly;

e.g if the ST segment of lead II is deviated ⇒ MI in inferior of the Heart

- Normal ECG

→ Components of a normal ECG
- waves (P – Q – R – S – T – U)
- intervals (PR, QT)
- segments (PR, ST)



* Before going into details, remember the electrical activity that is being recorded is either Repolarization or Depolarization

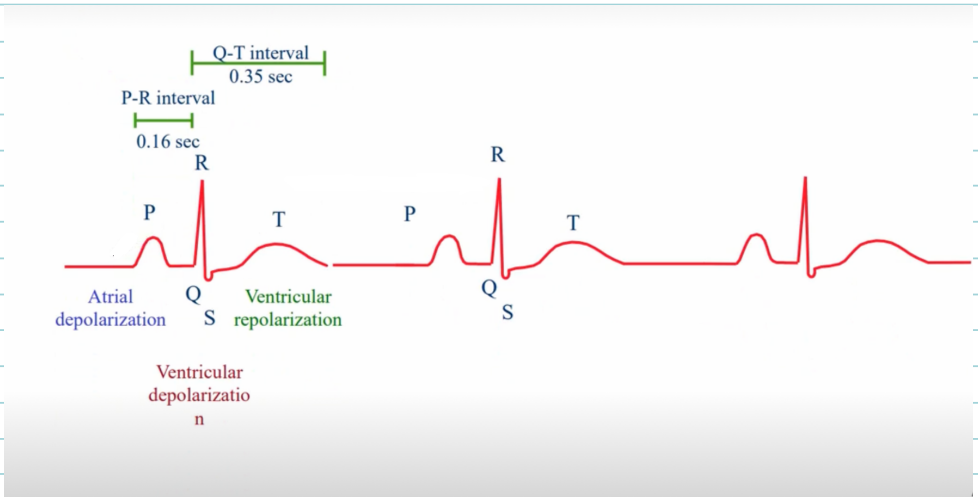
happening in either atria or ventricle

Also remember the order
Depolarization of At – Repolarization of At – Dep of vent – Rep of vent

(Repolarization of At: happens at the same time, so you won't see the wave of atrial repolarization as it masked by ventricular depolarization)

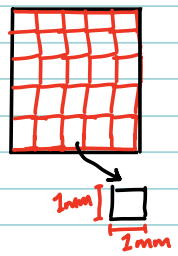
	• P wave	• QRS complex	• T wave	• U wave
Description	Atrial Depolarization	Ventricular Dep	ventricular Rep	papillary Rep
Duration	0.08 – 0.15 sec (1.5 to 3 small squares)	0.08 sec (3 small squares)	0.16 sec	0.05 sec
Amplitude	less than 2.5 mm 0.25 MV	More than 5 mm (1 MV) if it's chest lead it's higher 3-4 MV	less than 5 mm 0.5 MV	
Shape	1st half for Rt atrium, 2nd half for Lt atrim (حسب شكل ال wave بتعرفي وين المشكلة بال atria)	V_1, V_2 small R, large S; V_3, V_4 moderate R and S; V_5, V_6 large R, small S	slightly rounded and asymmetrical	
Direction (normally) when the heart axis isn't deviated	Inverted in aVR	inverted in aVR	inverted in aVR	

- Intervals : Segment + wave
  - **PR interval** : from the beginning of the P wave to the beginning of QRS complex (0.16 sec) <.20 عند الطفل
  - **QT interval** : from the beginning of QRS complex to the end of T wave (0.35 sec)
- Segments :
  - **PR segment** : baseline segment between the end of P wave and the beginning of QRS complex.
    it represents the delay in electrical conduction between atria and ventricle (AV node)
  - **ST segment** : baseline (isoelectric segment of ECG between the end of QRS and beginning of T wave.
    it represents plateau and any deviations from baseline may indicate MI.



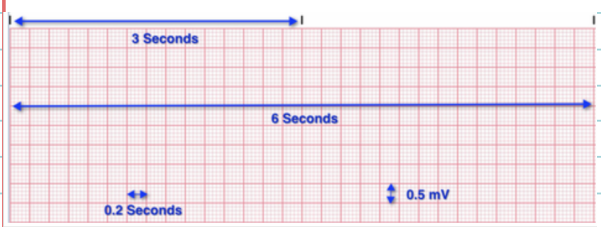
→ The ECG paper consists of a grid of small and large squares each representing a specific duration and voltage

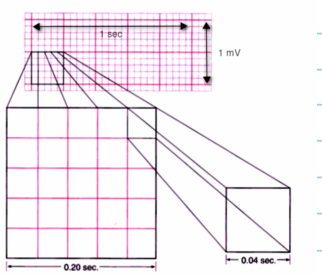
- Large Squares: each is comprised of 5 by 5 (25) small squares
  when a wave walks one large square it takes a duration of 0.20 sec (200 milliseconds)
  Amplitude vertically is 0.5 mV (5mm)



- Small Squares: each small square is 1 mm by 1 mm
  Duration horizontally is 0.04 sec (40 milliseconds)
  amplitude vertically is typically 0.1 mV (1mm)

← اذا موجة مستيت مربع كبير كامل يعني مستيت 0 مربعات صغيرة يعني 0.2 = 5 x 0.04





→ Calculating Heart beats:

We measure HR from RR interval, between 2 R waves that represents one complete Cardiac cycle (one heart beat)

e.g in our ECG, RR interval = 2 large squares (0.2 sec + 0.2 sec) = 0.4 sec

1 hear beat → 0.4 sec
X → 60 sec ?

HR = 150 beat/M

* In cases of irregular HR, where RR intervals are not constant through ECG, we can calculate an average RR to estimate an average HR.

we usually calculate average of HR rate in 6 sec, 30 Large squares
we count num of R waves within 6 sec, within 30 large squares

(e.g)

6 sec → 15 beat (15 R wave)
60 sec → ?

6 x ?

Average HR = 150 B/M

← قبل ما نتعرف على المشاكل اللي ممكن تصير على القلب وكيف رح نشوفها بال ECG، لازم نتعلم الوضع الطبيعي ، كيف الـ leads بترتتب بالوضع الطبيعي حوليه محور القلب ، نحتاج Reference.

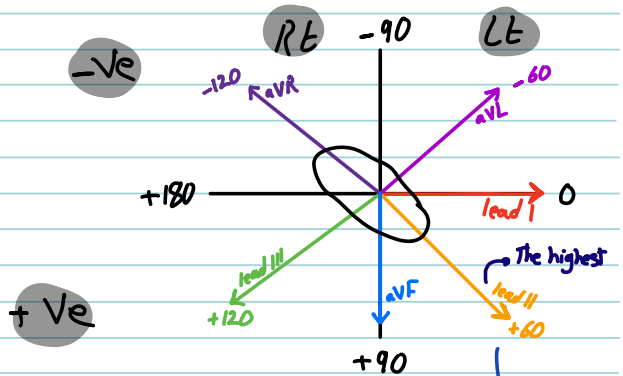
* Note that we chose our vector to be (R) ⇒ The Mean Cardiac vector representing the axis of the heart

→ **The axial and hexaxial reference :**

- Refere to the different planes in which electrical vectors (direct of depolarization) are measured, they help to create a three-dimensional representation of the heart's electrical activity.
- These different leads and angles provide a comprehensive view, helping to identify the location and nature of cardiac abnormalities
- practically, when clinicians evaluate ECG, they assess deviations, ST-segment changes and other abnormalities in specific leads,
  ← مقارنة بوضعهم الطبيعي

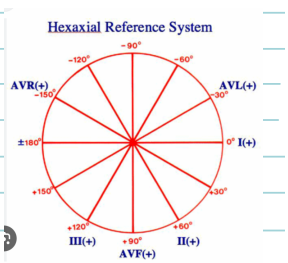
So first we need to explore the normal directions of the leads represente on 2 types of planes, where the heart is the center.

[1] **Axial plane** : it's a two dimensional (x, y) plane that passes through the body dividing it into upper and lower halves, it's used to represent the different electrical vectors in the frontal plane (includes lead I, II, III, aVL, aVF, aVR)

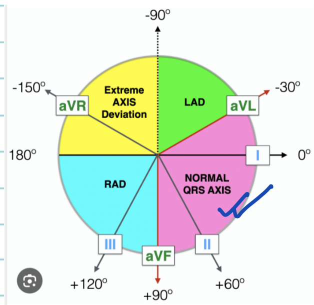


The Mean Cardiac vector is normally situated here

[2] **Hexaxial plane** : expands on the axial reference by introducing additional leads allowing for a more comprehensive evaluation of electrical vectors in both frontal and horizontal planes, here it includes all the leads



→ looking at the ECG, How Can we know the axis direction to decide wether it's in its normal place or not ?

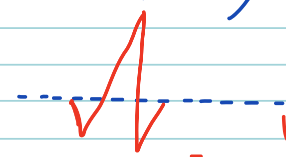


حتى نعرف اتجاه القلب ، ننظر لـ QRS في lead I و aVF

وهم أسهل تنتين موجودين على المحور السيني والصادي

يا اما بتستخدم الحسابات يا اما بتحفظ

طريقة الحساب ← vector analysis



١- منحسب عدد المربعات لارتفاعها

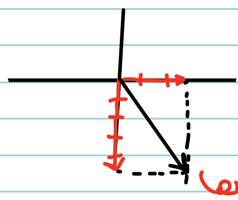
مثلاً R ← ٦ مربعات صغيرة فوق +٦

Q ← ٢ مربعين صغار تحت -٢

S ← ٢ مربعين صغار تحت -٢

= ②

٢- منروح منحطها عى المحاور (٢ للطول ، اتجاهها باتجاه lead I كونو +ve QRS)

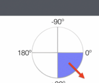
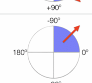
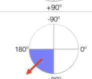
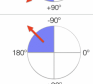


٣- منعمل نفس الخطوات لل aVF ، وخلينا نعتبر انو QRS = ٤+

٤- ال axis رح يكون التقاء مسقط الأسهم بزاوية معينة مش مهم حسابها

المهم طلع Normal axis

طريقة الحفظ ← ننظر لل QRS في lead I و aVF

Lead 1	Lead aVF	Quadrant	Axis
POSITIVE	POSITIVE		Normal Axis (0 to +90°)
POSITIVE	NEGATIVE		**Possible LAD (0 to -90°)
NEGATIVE	POSITIVE		RAD (+90° to 180°)
NEGATIVE	NEGATIVE		Extreme Axis (-90° to 180°)

- Abnormal ECG

[1] Normal Heart rate 60 - 100

- Tachycardia HR > 100
  - Sinus Tachycardia → ↑ SA node firing in response to a stimulus like ↑ symp. ↑ Temp ↑ toxin
- Bradycardia HR < 60
  - Sinus bradycardia → ↓ SA node firing in response to parasymp.

[2] Heart block: blockage in the electrical conduction → abnormal delay or complete block of the transmission of electrical impulses from atria to ventricles

- First degree: Prolonged PR interval (>0.20 sec), usually asymptomatic
- Second degree → Type 1: gradual increase in PR interval until a P wave is not conducted (missing QRS complex), due to delay in AV node

  Type 2: Consistent PR interval, but occasional P waves are not followed by QRS, may progress to complete heart block
- Third degree (complete block): NO association between P waves and QRS. Atrial and ventricular rythms are independent. Life-threatening