General Physiology

14 February 2024 13:56

⇒ General Physiology ✓

→ Nerve Muscle

→ Digestive System GIT ✓

→ Cardio Vascular CVS

→ Respiratory system RS

→ Renal system ✓

→ Endocrine ✓

→ Central Nervous system

→ Special Senses ✓

→ Hematology

→ Reproductive systems. ✓

General Physiology

→ Homeostasis = Walter Canon = Term

→ Cell membrane & Transport across membrane

→ Membrane Potentials.

⇒ Maintainance of constant & internal Environment

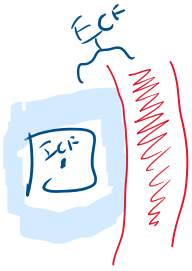
= Claude Bernard = Concept

→ Total Body water

→ ECF — Interstitial fluid, Blood

→ ICF

→ CSF



Mechanisms of Homeostasis

→ Feedback = ① Negative fb. = Body works against the change. 95%.

② Positive feb. = Body works towards the change = 5%.
"Vicious cycle"

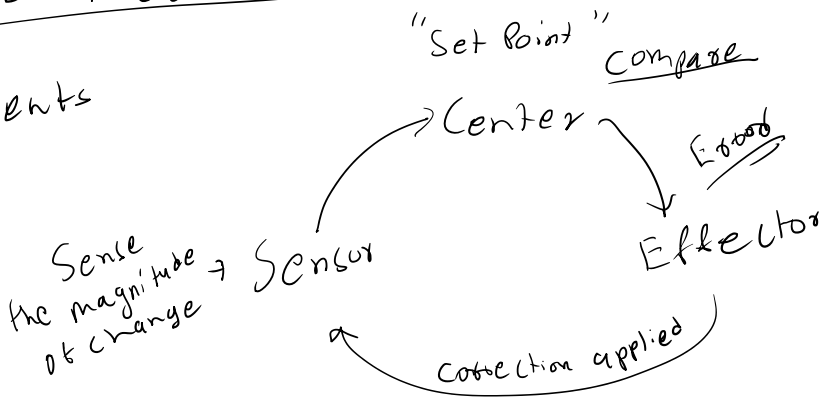
CLAS Parturition
cloth LH AP Shock

⇒ Feed Forward ⇒ Body works in anticipation for the change.

eg - Gastric secretion before meal
- Cardiovascular changes before any exercise
- Regulation of muscle tone

Negative feed back

- Components



Gain of feed back mechanism

$$\text{Gain} = \frac{\text{Correction applied}}{\text{Residual error}}$$

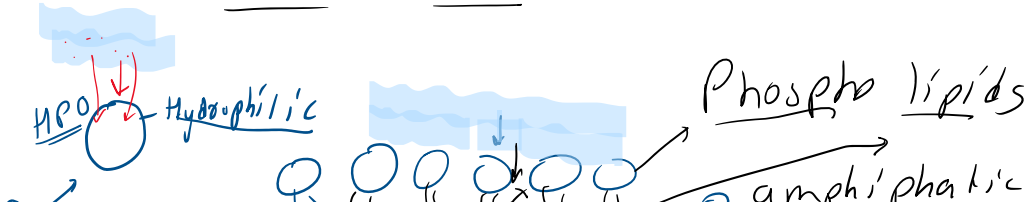
Air Condition room = 20°C
Record temp = 36.5°C
Normal = 37°C

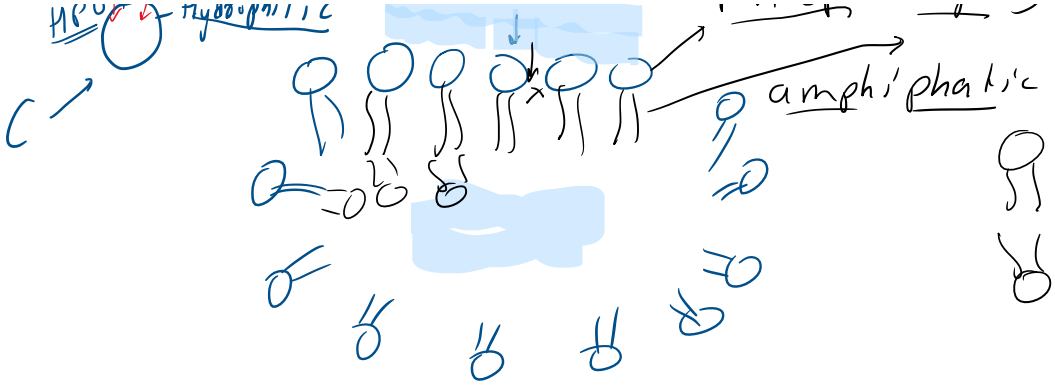
$$\text{Gain} = \frac{CA}{Er.} = \frac{16.5}{0.5} = 33$$

SBP = 160
Corr. = 130
Nor = 120

$$\text{Gain} = \frac{30}{10} = 3$$

Cell membrane



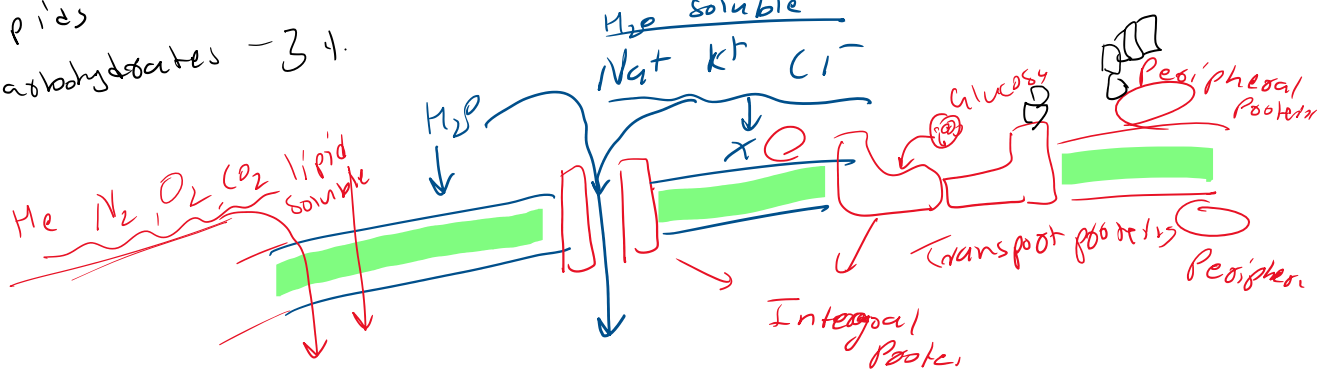


→ lipid bilayer

→ Proteins – 55%

→ Lipids – 42%

→ Carbohydrates – 3%



Transport across membrane

ICF / Inside	ECF / outside
K^+	Na^+
	Cl^-
	Ca^{++}
Mg^{++}	
HPO_4^-	
	Glucose
	O_2
CO_2	
H^+	HCO_3^-

Passive Transport Across Membrane

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Transport across

Passive

1. Higher Conc^n → lower Conc^n
2. No energy is required
3. Channels, pores, through lipid bilayer, carrier proteins.

types
1. Simple diffusion
2. Facilitated diffusion

Active

1. Lower conc^n to → Higher conc^n
2. Energy is utilized directly or indirectly
3. Pumps, ATPase, Exchangers, Counter transport, Co-transport, Symports, antiports,

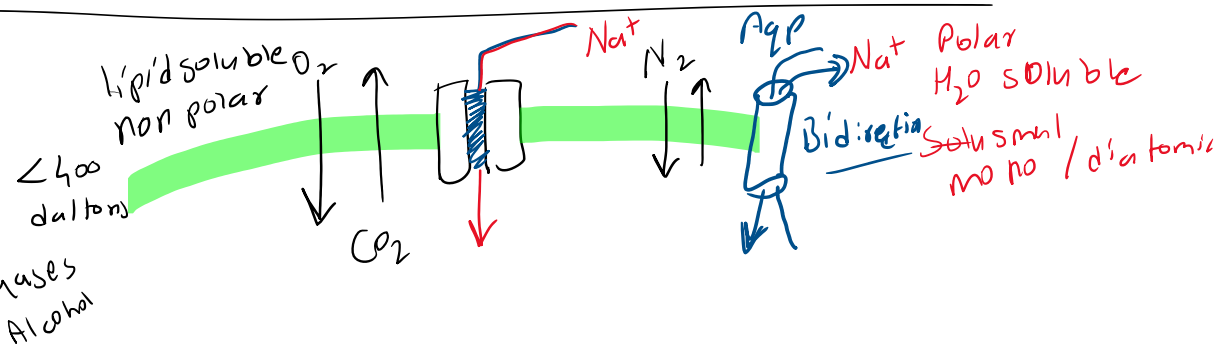
1. Primary active [ATP]
2. Secondary active [indirect energy]

Simple Diffusion

→ H → L

→ No energy is used

- Channels, pores, lipid bilayers

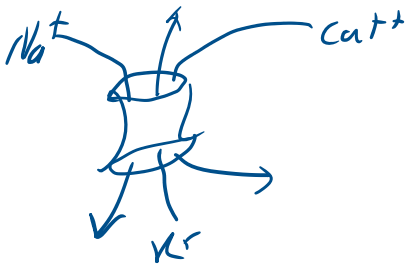


- Gases
- Alcohol

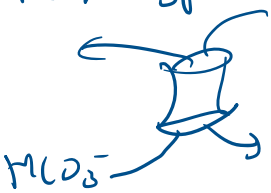
Types of Channels

(1) leaky channels = always open

(2) Non specific cation Ch.

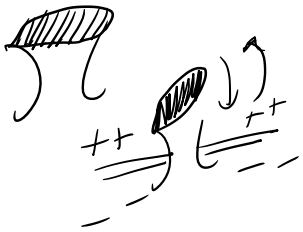


Anion Ch.



Diffusion

(3) Gated Channels



(1) Voltage gated Channels.

eg. Na.V. voltage gated Na^+ Channels

Blocked by lignocaine, xylocaine, Novocaine.

Na^+V, K^+V., $Ca^{++}V$.

(2) ligand gated ch. / chemically

eg. - olfaction → smell
- Taste → gustation
- neurotransmitters = Ach, Adrenaline ligand gated Channels

(3) Mechanically gated Chan =

movement / stretching / pressure etc on cell memb will open the Ch.

eg touch, pressure.

(4) Time gated Channels

Rhythm

eg Heart rate = Pace make cells

Behaviour

eg. Heart rate = Pace make cells

Aquaporins = Water channels

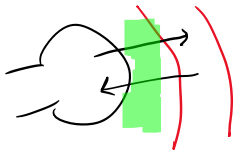
Factors Affecting Rate of Diffusion [ROD]

1) Temp ∝ ROD eg warm up exercise, fever.

2) Surface area ∝ ROD eg. alveoli in lungs
Nephrons in kidney
Villi in intestine.

3) Solubility ∝ ROD eg. CO_2 >> O_2 in lipid bilayer.

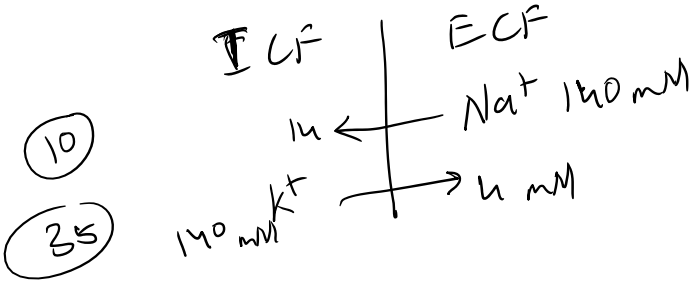
4) Thickness of membrane $\frac{1}{\propto}$ ROD eg pulmonary edema
Diffusion distance



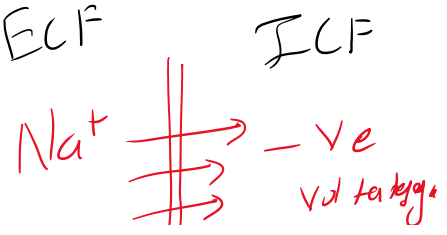
5) Size of molecule/ion $\frac{1}{\propto}$ ROD eg. Ca^{++} glucose

6) Charge on the molecule/ion $\frac{1}{\propto}$ ROD Na^+ >> charge the K^+

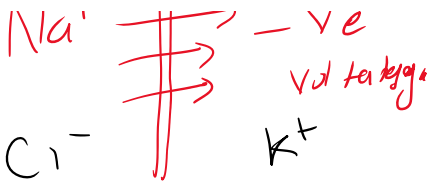
7) Conc^n gradient ∝ ROD



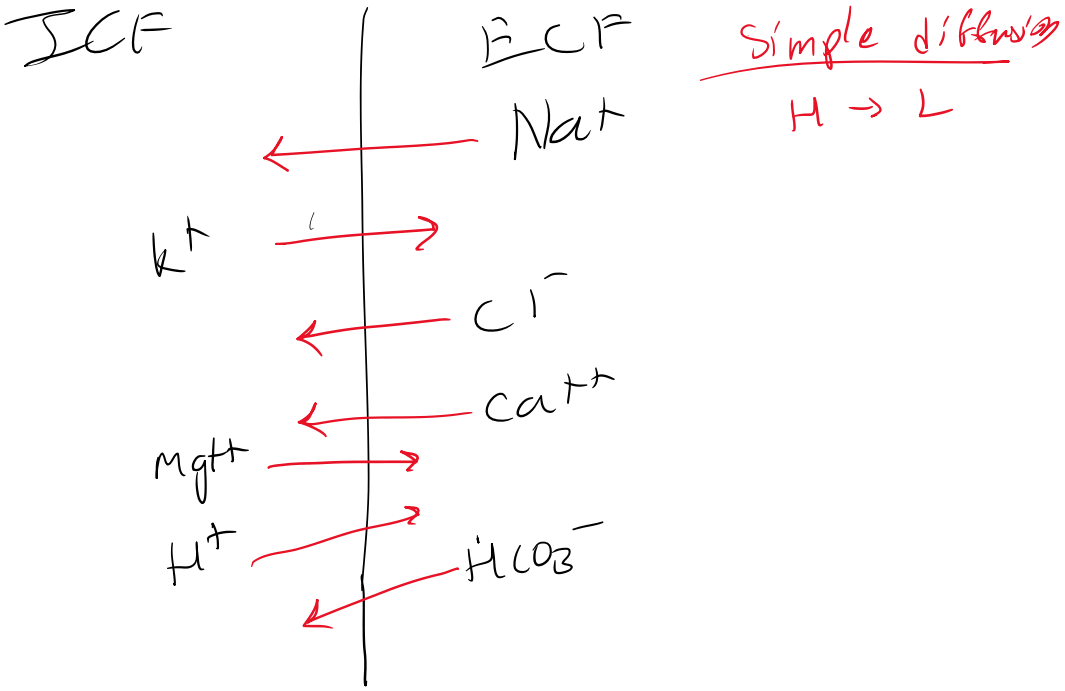
8) Charge on the memb. ∝ ROD



8) Charge on the memb. ∝ RPD
(Voltage difference across the memb)



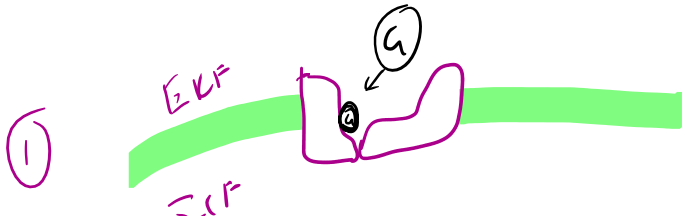
9) Pressure ∝ RPD



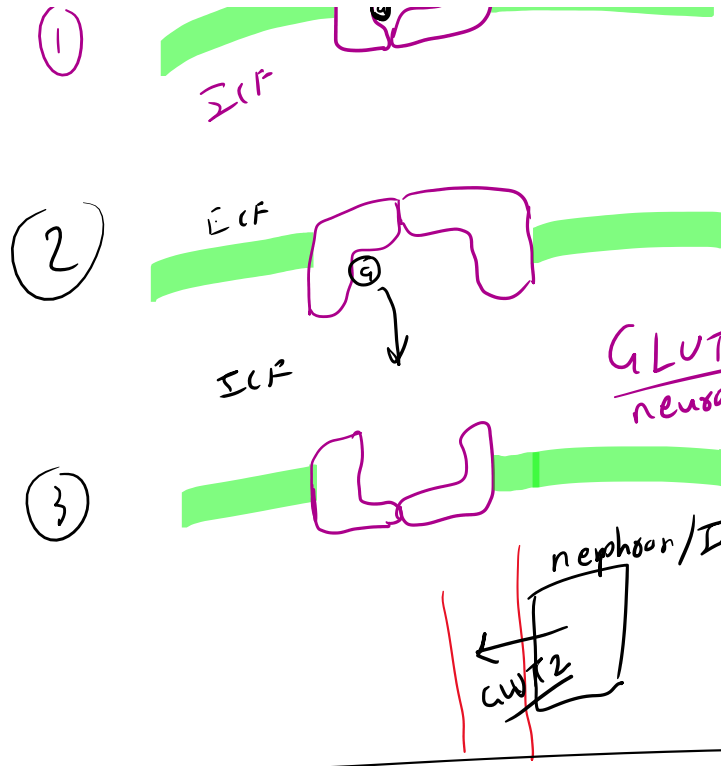
Facilitated Diffusion

→ large molecules eg Glucose, Urea, a.a. etc.
→ With the help of carriers proteins / transport proteins

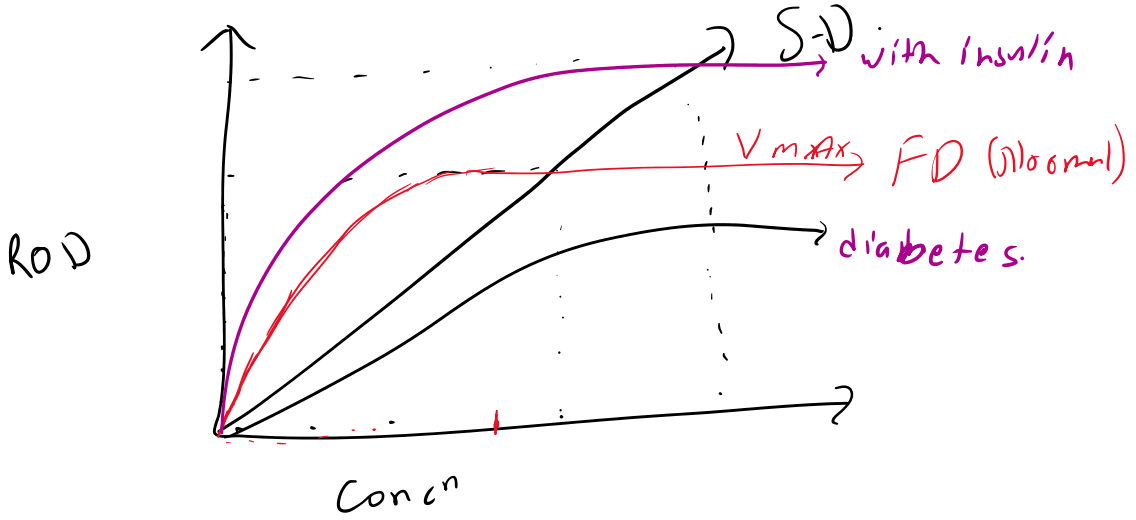
→ Higher to lower
→ No energy is required
→ Transporters, carriers, facilitated



GLUT
Flip
AAT UT = Urea trans



Concn Grad. SD Vs F.D.



Active Transport

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ACTIVE Transport

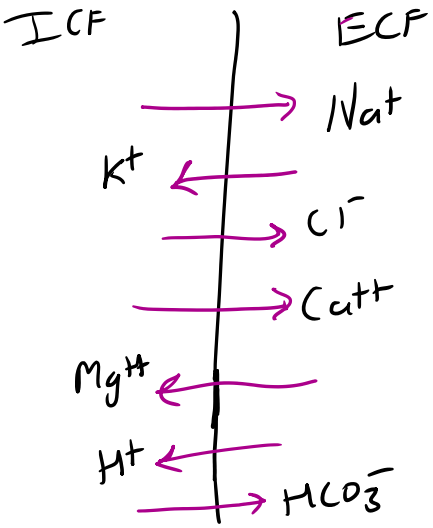
(1) L → H

(2) Energy is used

(3) Pumps, ATPase exchangers

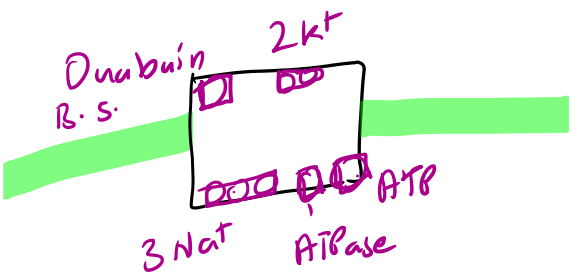
(1) Primary Active Transport → Uses ATP

Lower → Higher



Example Na+ K+ Pump = Carrier / Transport protein

L → H

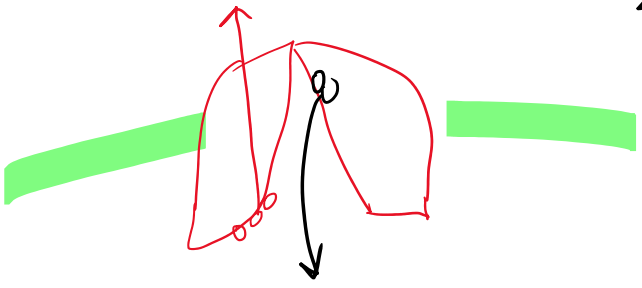


NOKIA

3 Na+ out

2 K+ in

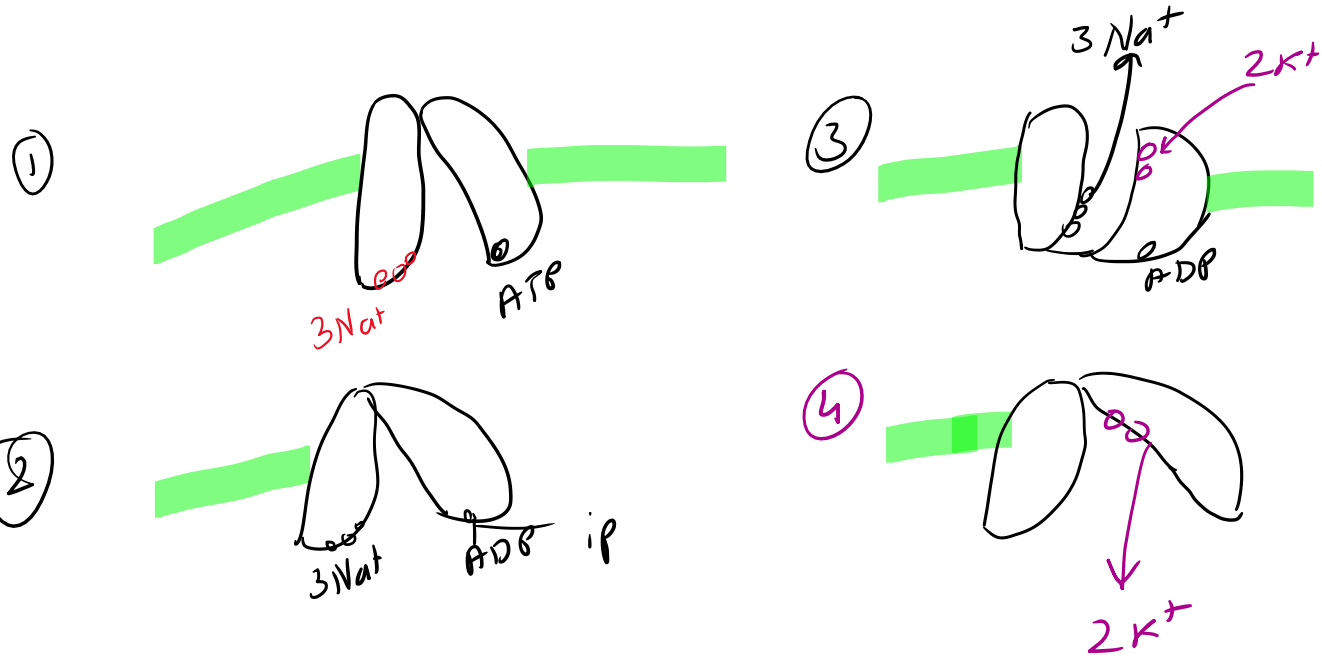
1 ATP



Na+K+ Pump Japanese Scientist

Ubiquitor Pump, Na^+K^+ Pump, Japanese Scientist / Noble "Janes Skou"

Present on all cells

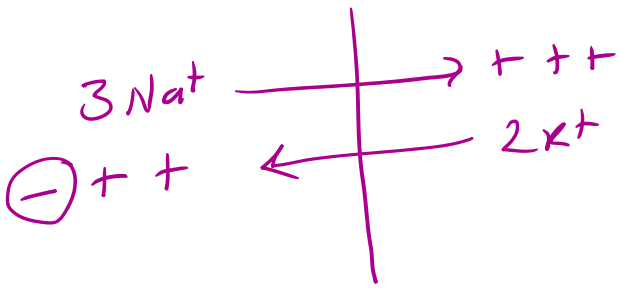


Advantages

(1) Maintains Na^+ K^+ gradient across cell memb.

(2) Prevents the cell from Osmolysis.

(3) Generates -ve voltage (Potential difference) inside the cell. Due to unequal distribution of Na^+ & K^+ ions.



eg. 2- H^+ Pump = Stomach

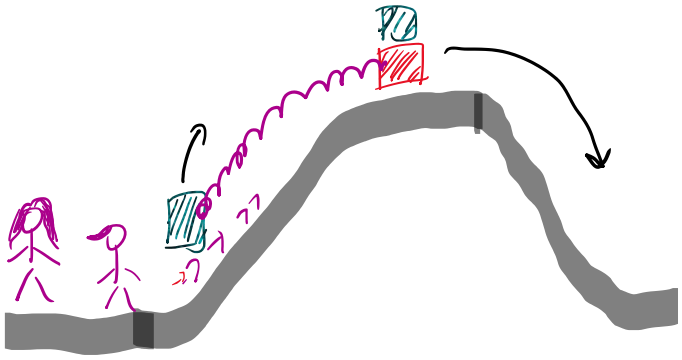
Ca^{++} Pump = Muscles

Secondary active transport

→ Carrier mediated transport

L → H.

Energy Indirectly - without ATP



→ In Secondary A.T. one substance moves from Higher conc^n to Lower conc and other substan move from Lower conc to Higher conc^n

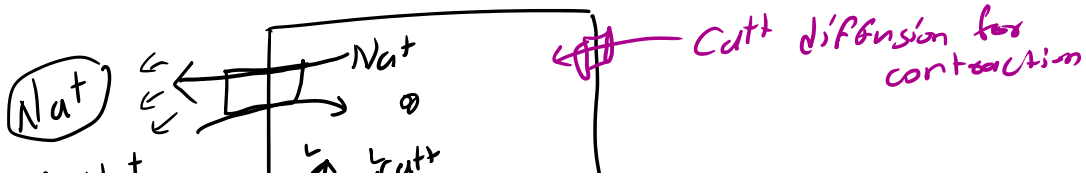
→ It works on the potential energy generated by primary active transport

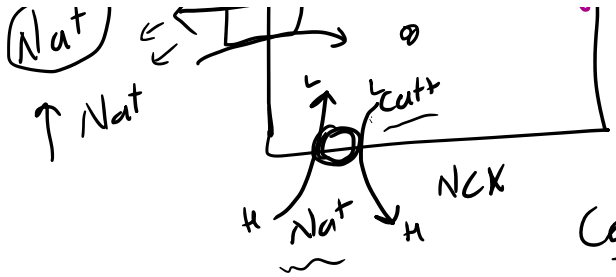
eg. Exchangers, Countertransport, antiport

Symport, Cotransporters.

eg. ① Na^+ Ca^{++} exchanger = NCX

Card. muscle cell





Counter transport

N : C X

3 : 1

(2) Co transport = Both the substance move in same direction -

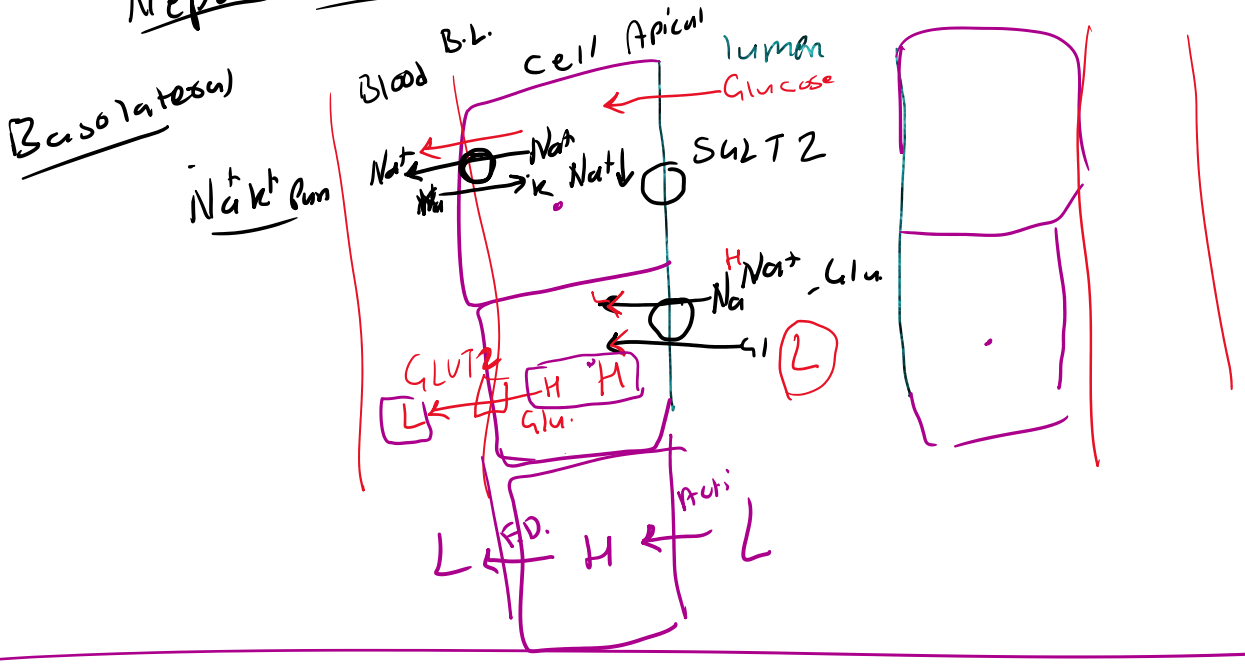
But One moves H → L

other moves L → H

eg SGLT = @ Na+ Glucose linked Transporter

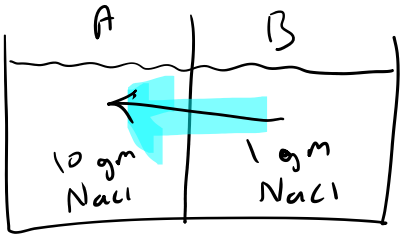
Na+ Gl. co transporter

Nephron kidney Reabsorption



Osmosis

Def.- Movement of H_2O molecules from higher H_2O concn to lower H_2O concn



→ Osmolarity - W/V eg $\frac{\text{gm solute}}{\text{litres of } H_2O}$ = temp dependent

→ Osmolality - W/W $\frac{\text{gm solute}}{\text{Kg of } H_2O}$ = temp independent

Plasma osmolality = 290 - 300 mOsm/Kg

Normal Saline = 0.9 gm% NaCl

$0.9\text{gm} \frac{1}{100}$ $\frac{\text{gm}}{100}$

When we take 0.9 gm % NaCl we make 150 mMol of NaCl solution

56 gm / 1000 ml 1 M

58gm	1000	mMol
x	150	mMol

$\frac{5.6 \times 1.5}{10} = \frac{8.4 \text{ gm}/1000 \text{ ml}}{150 \text{ mMol NaCl}}$

χ 150 mMol | 1 ppt

150 mMol NaCl

150 mMol NaCl

150 Nat

150 Cl^-

300 particle

300 mOsm

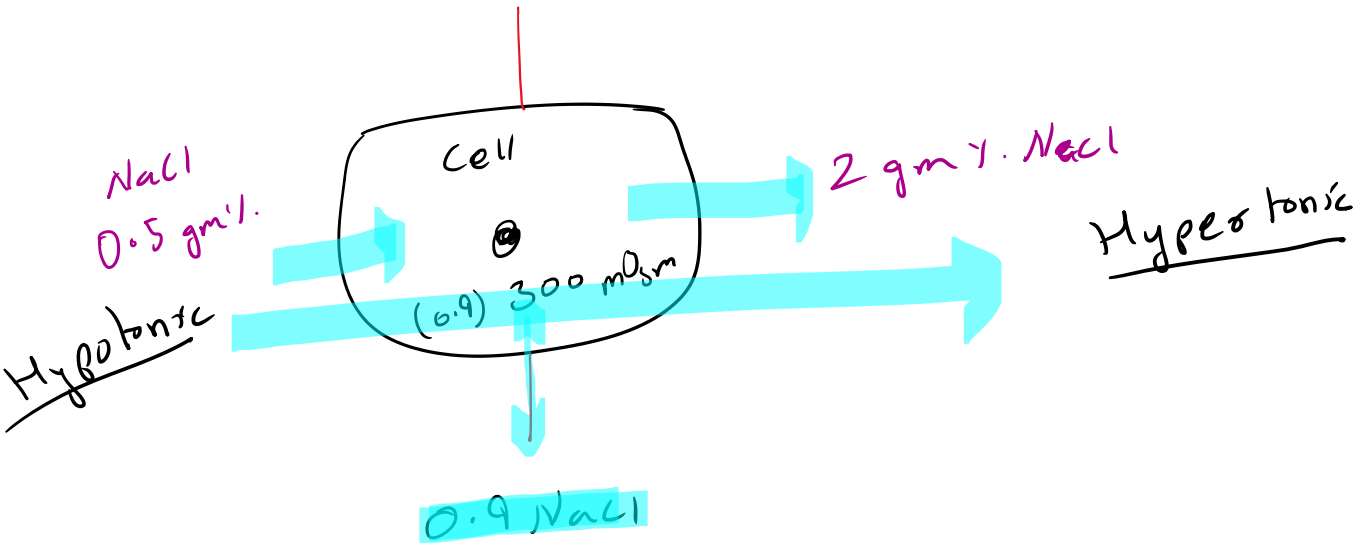
$\therefore$ 0.8% ≈ 0.9% (0.8/100 ≈ 0.9/100)

150 KCl

$CaCl_2$ 100 mMol

300 mOsm

Plasma osmolarity is primarily dependent on Nat, glucose & Urea



70 kg Body fluid Compartments

70 Kg Healthy Adult Male

TBW = 60 % Body weight = 42 l

ICF = 40 % " = 28 l

+

" = 14 l

ECF = 20 % of TBW = 14 l

- ① Plasma 5% = 3.5 L
- ② Interstitial fluid = 10.5 l

Females = 50 % to 55 %. Because of more Fat content

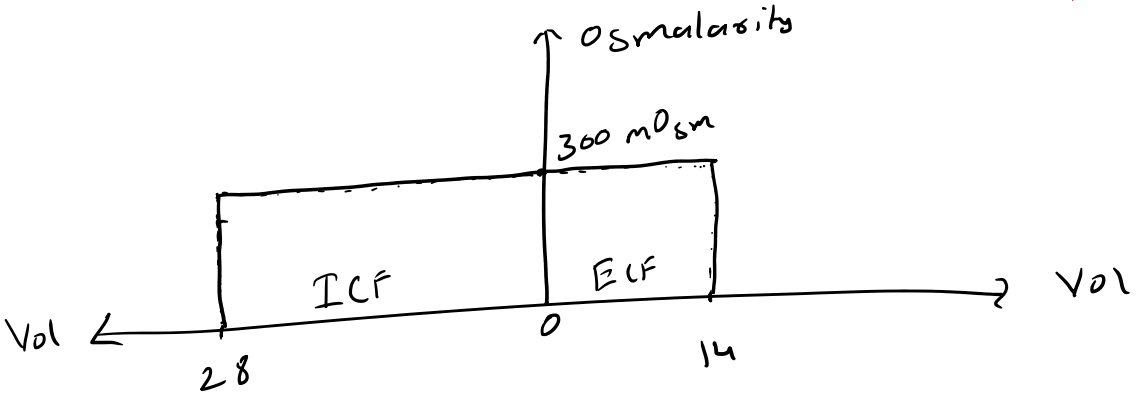
Children & neonates = 70 to 80 %.

Muscles are known as Fat free Mass

70 % H_2O

Correlation Between Body fluids & Osmolarity

Darrow Yannet Plot



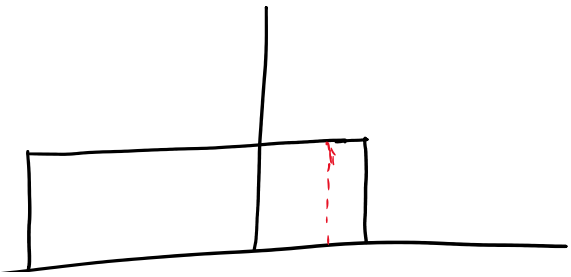
Gain

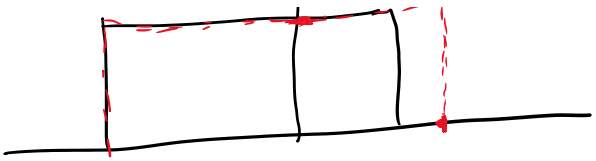
Isotonic fluid

eg. IV NS.

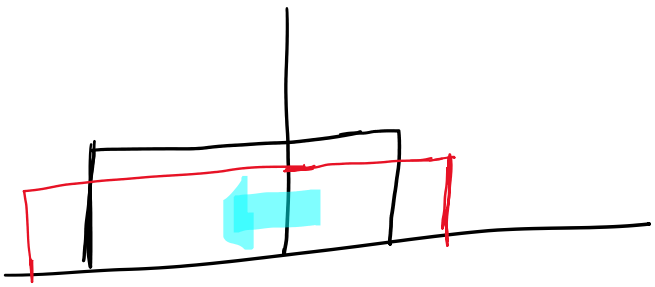
↑ECF
↑BP

loss of Isotonic

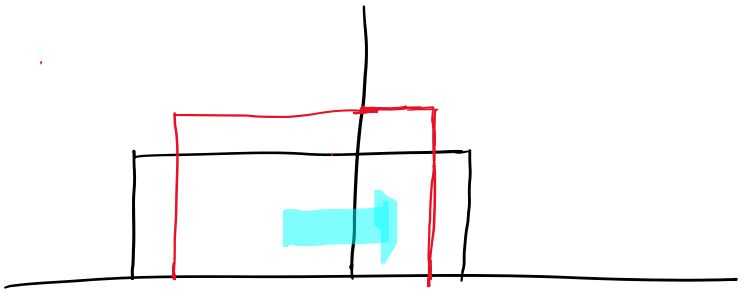




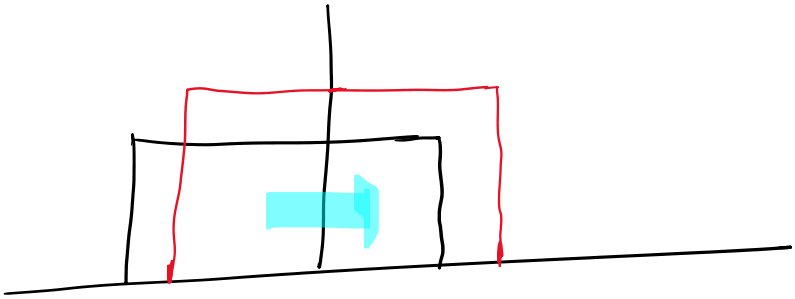
Gain → Hypotonic



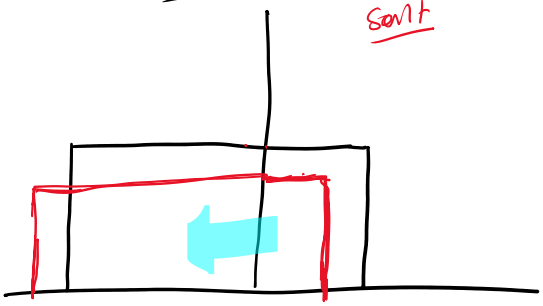
loss of Hypotonic



Gain of Hypotonic



loss of Hypotonic

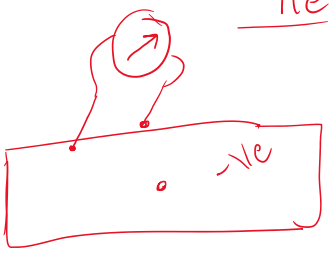


Membrane Potentials

23 February 2024 14:56

Resting Membrane Potential

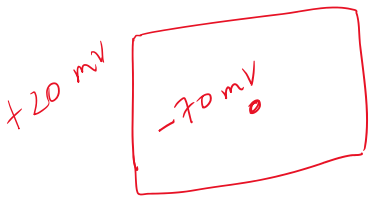
RMP



A -ve potential maintained inside the membrane of an excitable cell unless or otherwise it is excited. When the cell is in resting condition

① RMP - -70 mV (considered outside 0)

② Transmemb. Potential - = 90 mV = The distance between the Potentials



Skeletal Muscle = -90 mV

Neuronal cell = -70 mV

Cardiac myocytes = -89 mV

Pace makers = -65 mV

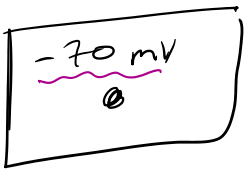
Smooth muscle = -45 -50 mV

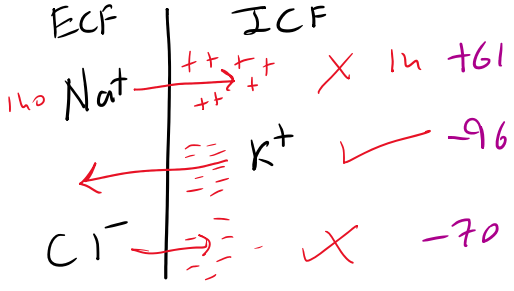
Genesis of RMP

1) Conc^{n} gradient ions

2) Permeability of ions

3) Na^{+} K^{+} pump.





Begining of memb pot is due Donnan Memb. equili.

Nernst equation = Electro Chemical Equiv

(1) A charge / potential at ~~with~~ which the chemical diffusion is balanced

$$ECE = \pm 61 \log \frac{Co}{Ci}$$

Na^+ $\frac{Co}{Ci}$ $\frac{1kg}{1k}$

$$= \pm 61 \log \frac{1kg}{1k} = \pm 61 \log 10$$

$$= \pm 61 \times 1 = +61$$

Na^+

$Na^+ = +61$ mV

$K^+ = -96$ mV

$Cl^- = -70$ mV

$Ca^{++} = +125$ mV

---

(2) $K^+ >> Na^+ >> Cl^-$ Permeability

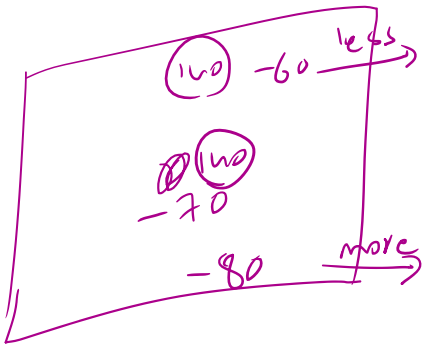
∴ according GHK equation Goldmann Hodgekin katz

RMP due to ECE & Permeability of Na^+ K^+ & Cl^- was -66 mV

(3) Na^+ K^+ Pump -4 mV using ATP ∴ -66 Diffusion, -4 Active transport

-70 mV

1kg -60 lgs → 6 mM/l Hyperkalemia ↑ K^+



6 mM/L Hyperkalemia ↑ K^+

Normal = 4 to 4.2

2 Hypokalemia ↓ K^+

ECF

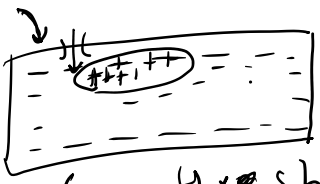
Nerve Muscle Physiology

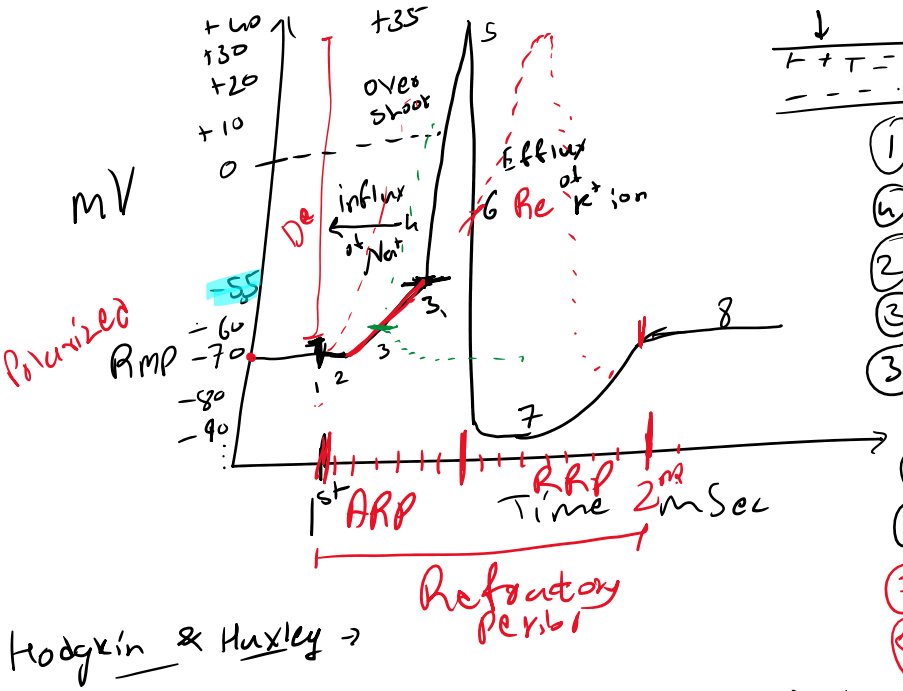
09 March 2024 09:54

- Action potential
- Nerve fibres
- Neuro muscular Junction
- E-C coupling
- Mechanism of Muscle contraction
- Properties of muscle contraction
- Wallerian degeneration
- Energetics of muscle contraction.

Action Potential

- Change in the potential of the memb. from -ve to +ve and back to -ve, on application of a threshold stimulus, for a very brief period. This leads to the excitation of the cell.

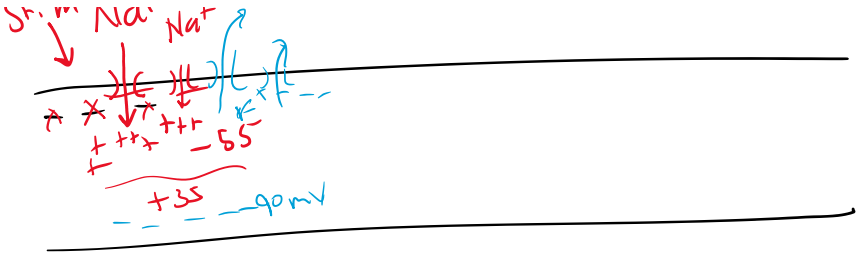




1. Point of Stimulus
4. Depolarization
2. latent phase
3. Slow rising phase
3, Firing level / Threshold point
5. Spike
6. Repolarization
7. Hyperpolarization
8. RMP / polarized

Hodgkin & Huxley →

→ Squid Giant → Axon of a tentacle



Na^+ V. Blockers

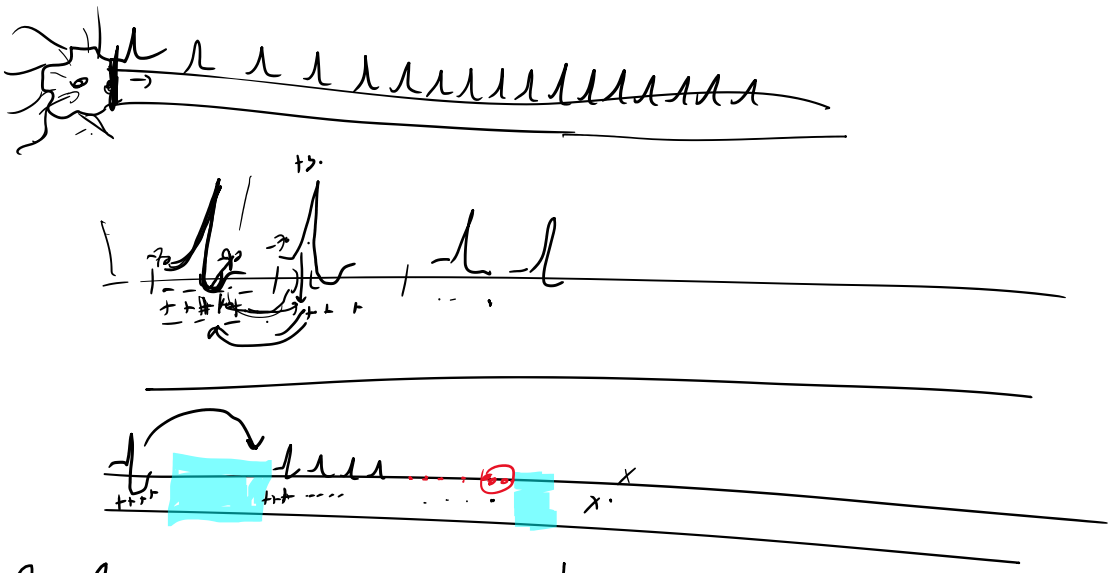
1. TTX — Tetraodotoxin = Puffer fish
2. STX — Saxitoxin = Clam fish
3. BTX — Batrachotoxin = Toad (frog)
4. lignocaine
5. Xylocaine
6. M conotoxin

K^+ V Blocker

1. TEA — Tetra ethyl amine

Properties of Action potentials

1. All or none principle = on threshold stimulus stimulus
   there will be complete A.P or it won't occur at all.
2. Threshold point = An Action potential always consist of threshold point beyond which the Na.V become independent of stimulus energy.
3. Depolarizing = always depolarizing the potential from -ve voltage to +ve voltage.
4. Propogatory = It can travel throughout the cell membrane



(5) Refractory period

Def. =

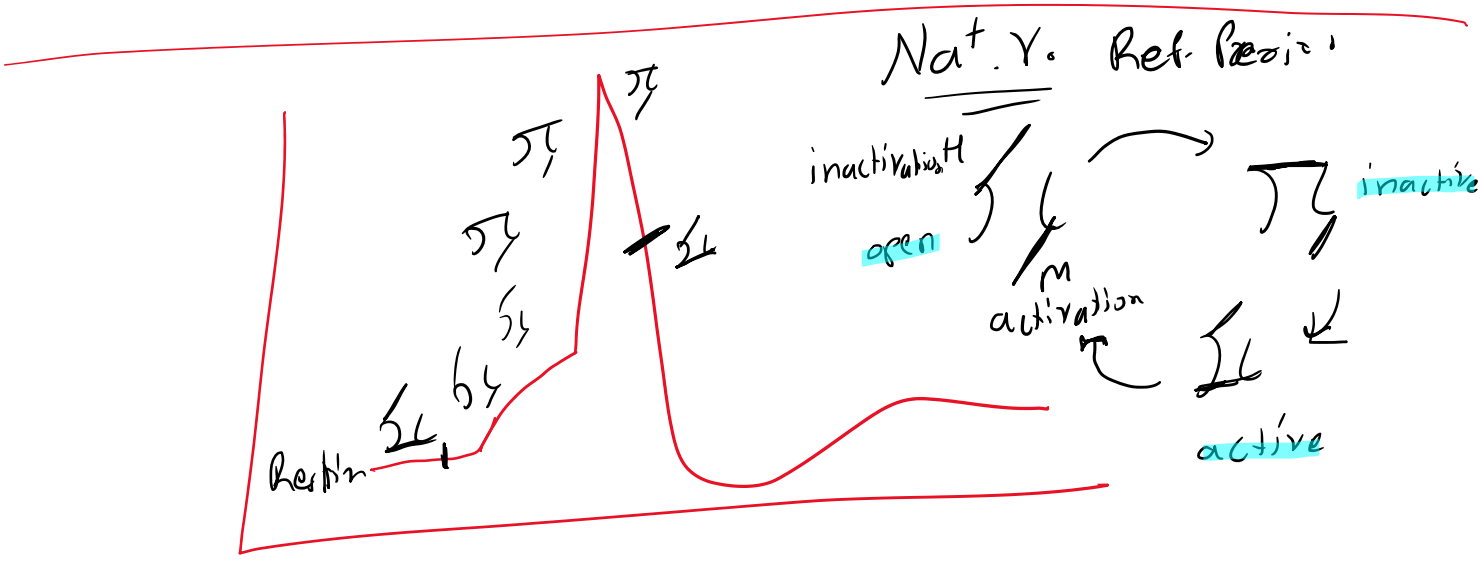
It is a period during which the second action potential cannot be generated

(1) ARP = Absolute Ref. Per.

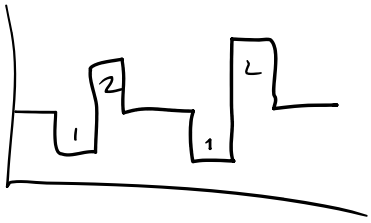
It is a period during which the second action potential can never be generated whatever may be the strength of stimulus

(2) RRP

It is a period during which the second action potential can be generated only when the strength of second stimulus is much more higher than the first stimulus it can be given in the later half of the repolarization



(3) It is Biphasic



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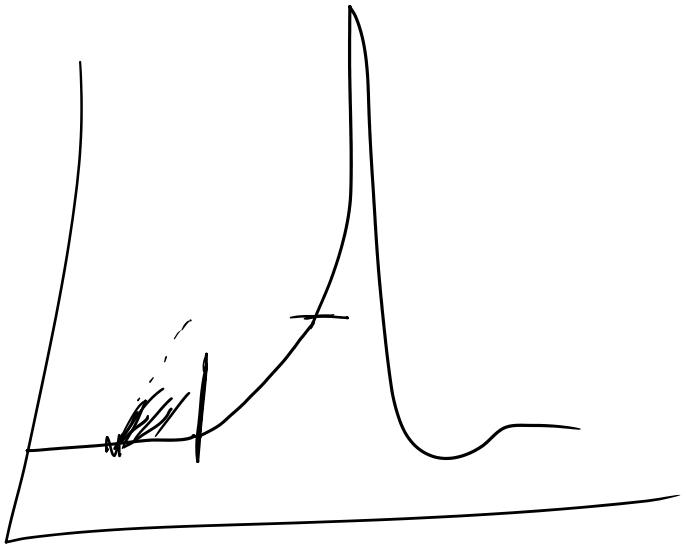
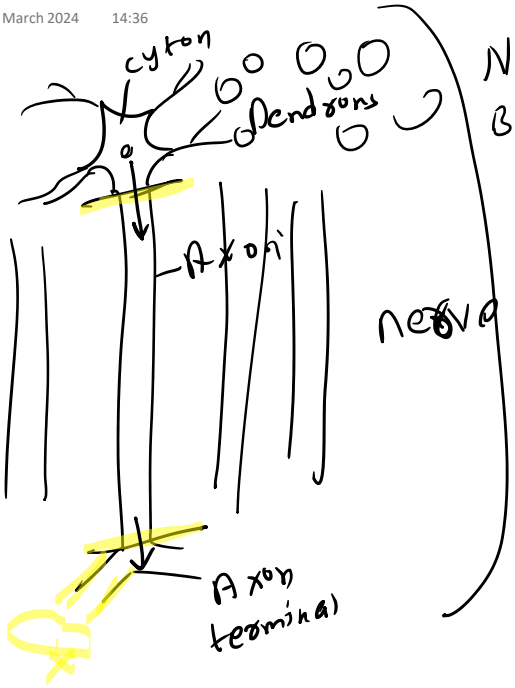
large accumulation of

(7) Accomodation = accumulation of large number of sub threshold stimulus will lead to inactivation of all the Na.V. and at this point the threshold stimulus will not cause Action potential.





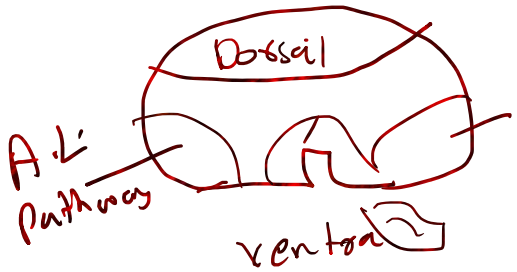
Nuclei, Ganglia
Brain Body

nerve Tracts

Classification of nerve fibers

Erlanger & Gasser Classification

	Myelin	Thickness / Thickest 20 MM	Velocity of AP / Fastest 70 m/s 120 m/s	Functions
Aα	M			Motor = Skeletal muscle
β	M			Sensory = Fine, (Dorsal column) Pres. Prop., Vib
γ	M			Motor = Muscle spindles
δ	M			Sensory = Ant. lat. pathway Pain [Fast]
B →	M			Autonomic
C →	Non M	Thinnest 1 µm	Slowest 0.5 m/s	Sensory = Slow pain



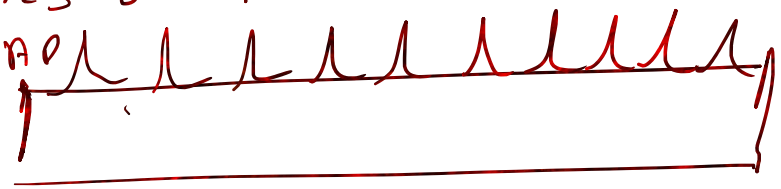
Sensitivity of nerve fibers

Most | Least

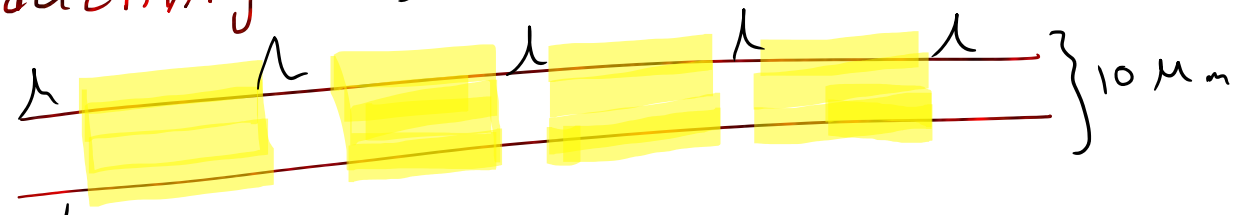
	Most susceptible	Inter	least
Hypoxia	B →	A	C
Pressure	A →	B	C ✓
Local Anaesthesia	C →	B	A ✓

Properties of nerve fibers

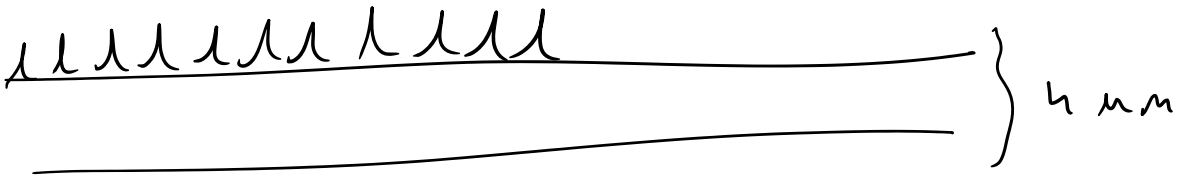
① All or none



② Conductivity m/s



D x 6
60 m/s



√4
= 2 m/s

③ Excitability = nerve fiber is excitable tissue

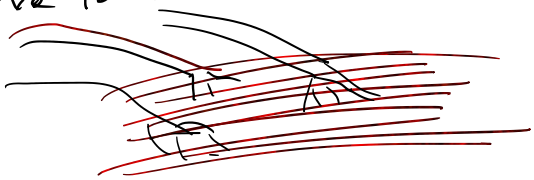
④ Non-fatigue

⑤ Refractory period = minimum - Ad $\frac{1000}{sec}$

C = $\frac{10-20}{sec}$

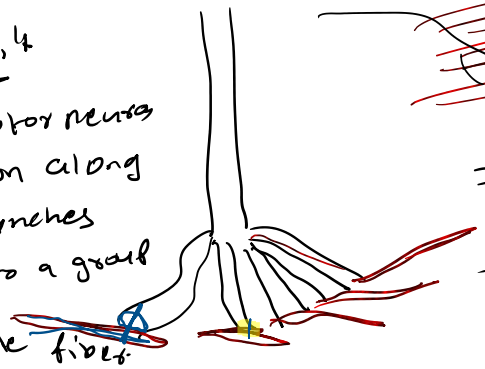
NMJ ★★★

It is a functional junction between neuron and a muscle fibes for transmission of impulse from nerve to muscle.

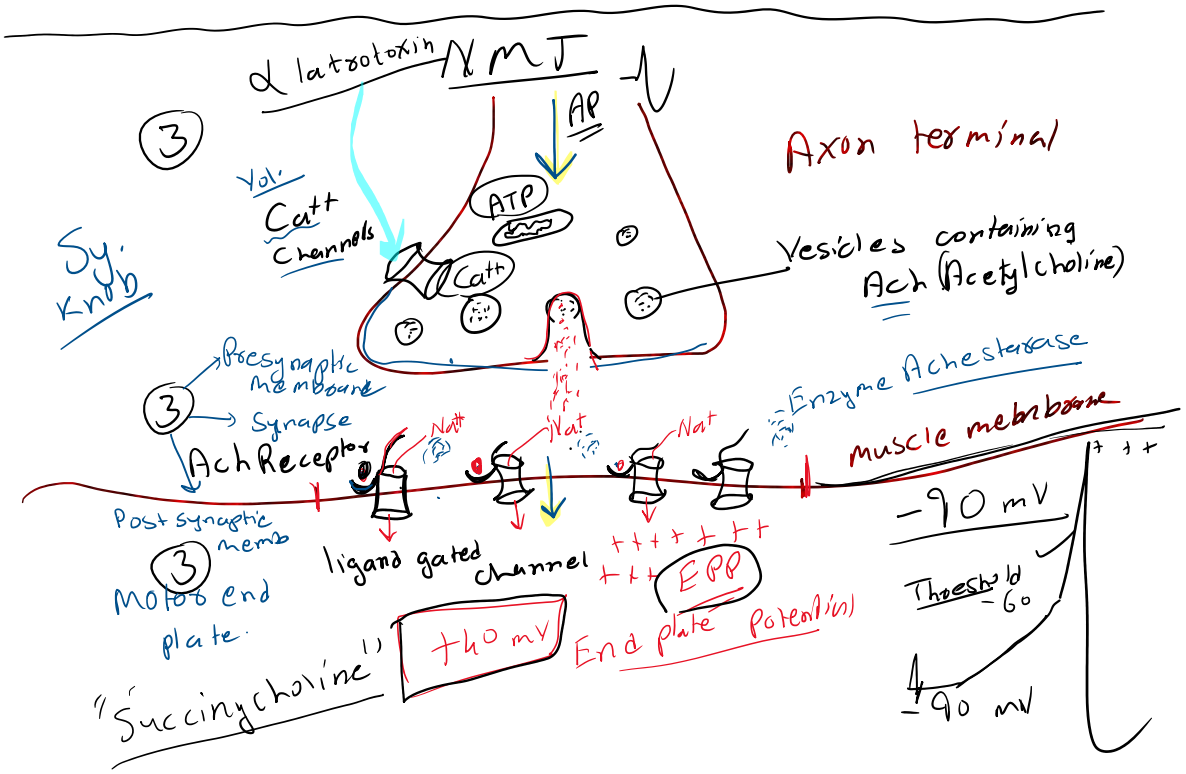


Motor unit

An Aα motor neuron and its axon along with its branches supplies to a group of muscle fibes.



= Recruitement of motor units



Events

A.P arrives

Action potential arrives at nerve terminal
Opening of calcium ions channels and entry of calcium ions in the nerve terminal
Calcium and ATP causes exocytosis of acetylcholine in synapse
Acetylcholine diffuses in the synapse and binds onto the acetylcholine receptor on the motor end plate of muscle membrane.
The opening of ligand gated channels at motor end plate causes entry of sodium ions in the membrane
These sodium ions develop the local positive voltage known as End Plate Potential
The end plate potential causes development of action potential in the sarcolemma or muscle membrane
The action potential leads to excitation of the muscle fibre and hence leads to the contraction of muscle fibre by the mechanism of
Excitation-contraction coupling

For relaxation of the muscle fibre the action potential at nerve terminal is stopped. The enzyme acetylcholinesterase destroys the Ach in the Synapse
The ligand gated channels close at Motor End Plate leading to cessation of EPP and hence AP does not develop muscle does not excite does not contract and hence relaxes.

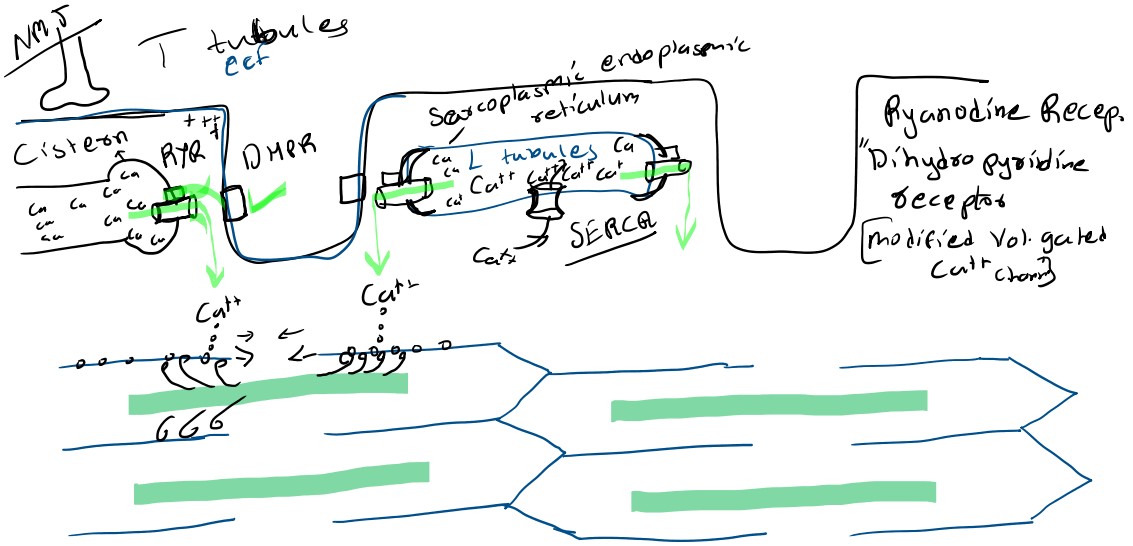
Myasthenia Gravis = It is

It is an autoimmune disorder which causes destruction of acetylcholine receptor ligand gated channels this leads to excessive usage of acetylcholine from the pre synaptic terminal and hence early fatigue due to the depletion of ach in the pre synaptic terminal
The common symptoms are early fatigue unable to maintain long duration work hours ptosis of the eyelids.
The treatment involves inhibition of acetylcholinestarase enzyme by the certain drugs like Neostigmine, physiostigmine and pyridostigmine also in certain cases the treatment involves usage of steroids.

Botulinum toxin

Botox inhibits the release of Ach by blocking "SNARE" [Proteins for exocytosis of Ach] There by inhibiting the ~~cont~~ excitation & contraction of muscle fiber.

Excitation - Contraction coupling



① Malignant Hyperthermia

Mutation of RYR

② Central core disease

Mutation of

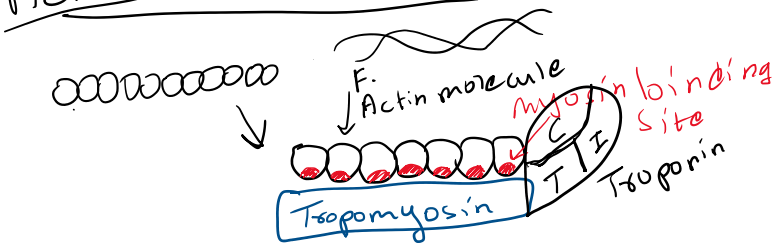
(2) Central core disease
Mutation of Mitochondria.

Molecular Basis of Muscle Contraction

Mechanism of Muscle contraction

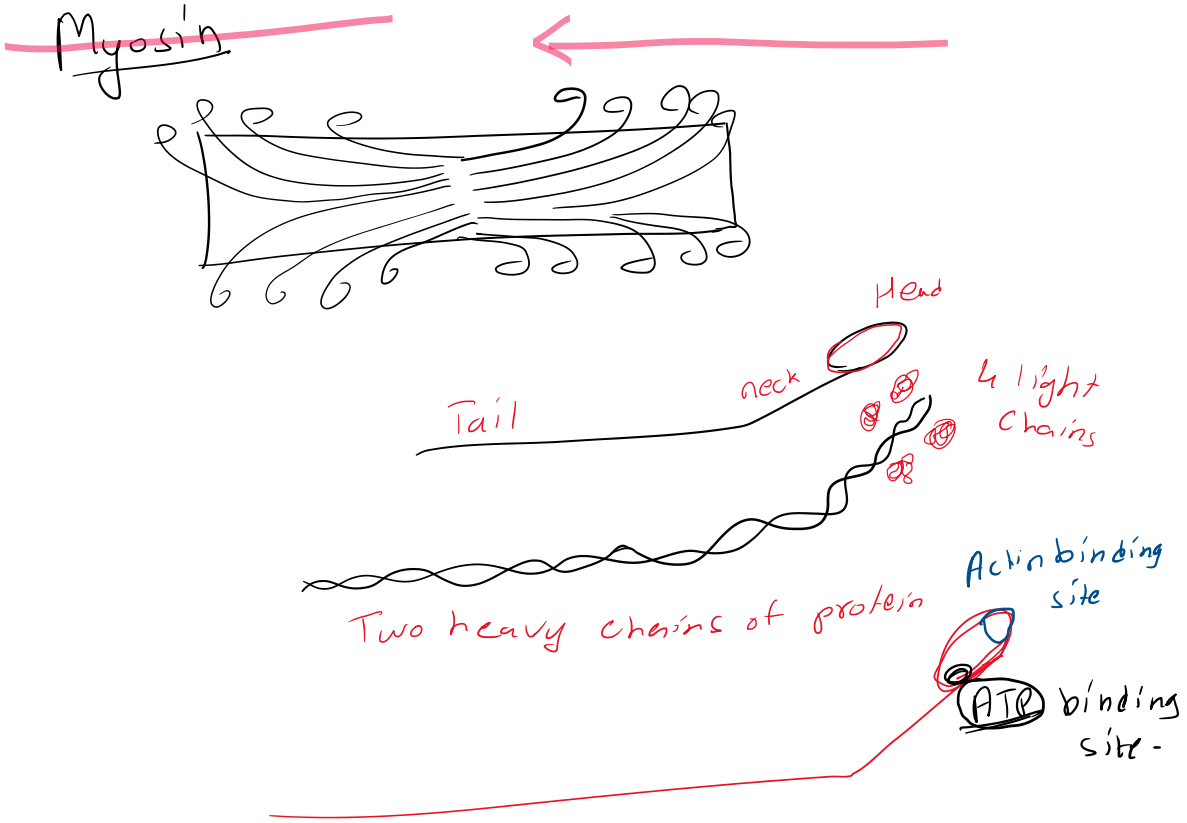
Walk along theory / Cross bridge cycling / Sliding filament Theory

Actin



C = Ca^{++} binding Site
I = Inhibitory site
T = Troponin - tropomyosin binding site.

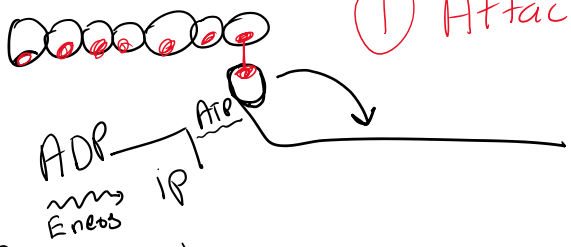
Myosin



Cross bridge cycle

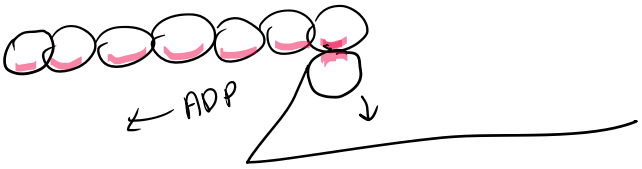
Cyclic event of attachment and detatchment
... actin & myosin molecules.

Cyclic event of
of Actin & myosin molecules.

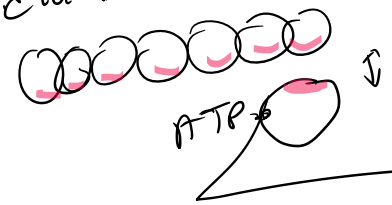
→ 

① Attachment.

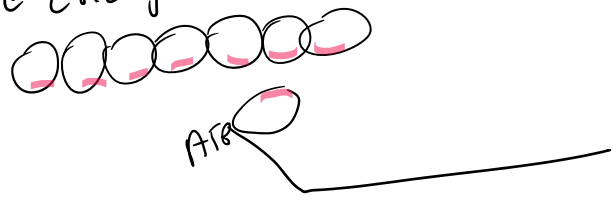
② Power stroke



③ Detatchment → Rigor Mortis
New ATP is not available for detatchment



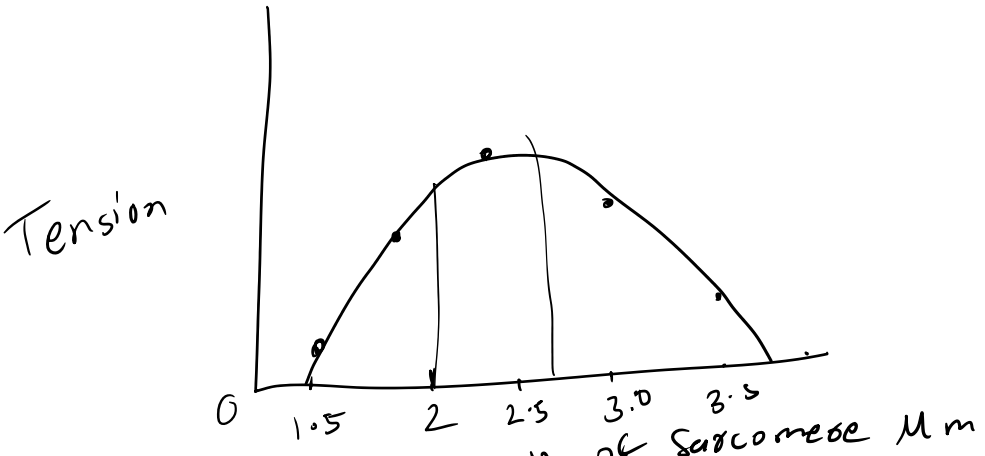
④ Re energization

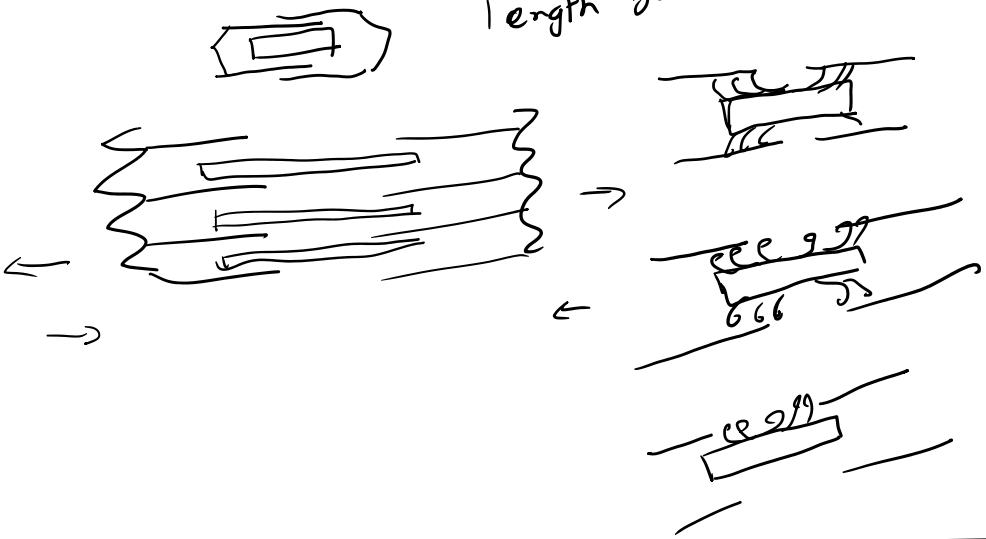


Muscle Contraction

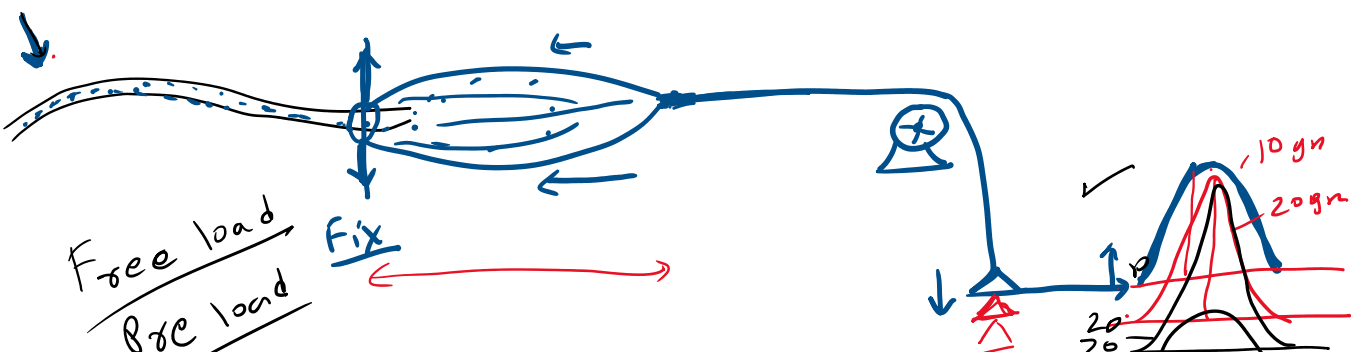
Frank Starling's law = Within physiological limits, the force of contraction is proportional initial length.

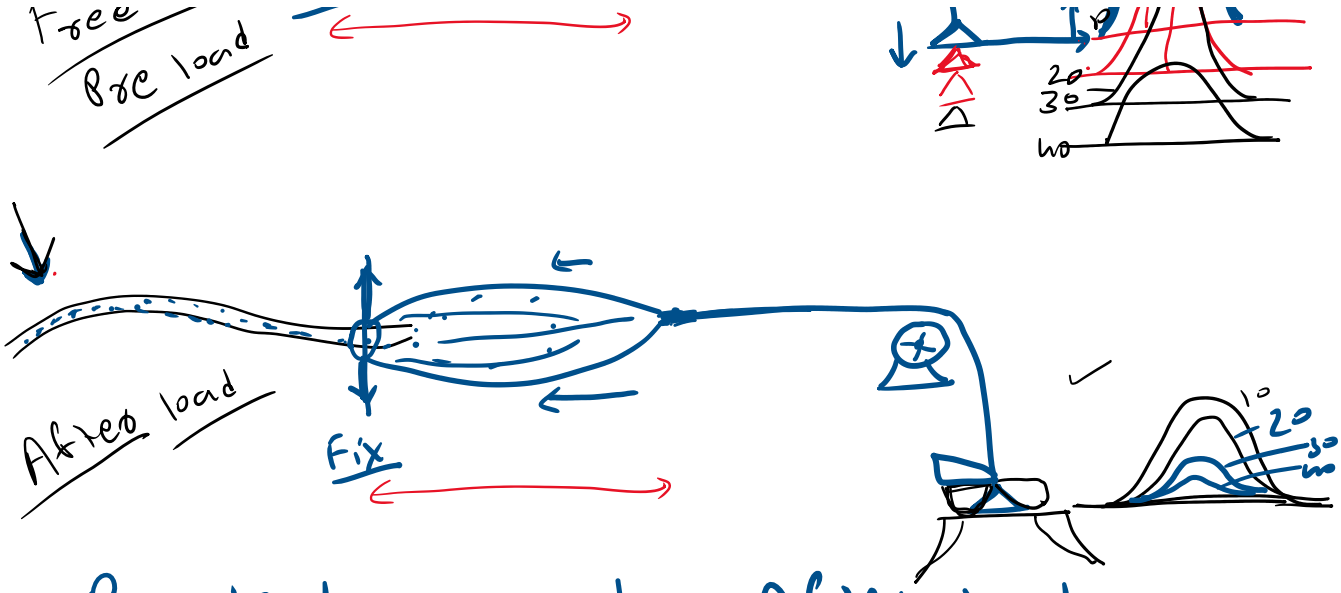
→ length tension Relation ship





Frog

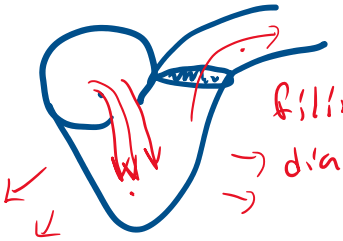




Pre load

(1) load acts before contraction

(2) FOL $\propto$ load

(3) 

(4) increase in venous return ↑ Preload

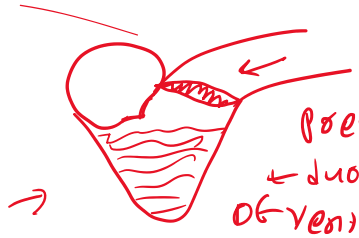
(5) ↑ Pre load heart ↑ Cardiac Output

(6) eg. dynamic exercise, javeline throw, discus throw, shot put

After load

(1) load acts during contraction

(2) FOL $\frac{1}{\propto}$ load

(3) 

(4) decrease in venous return) increase in back Pressure of aorta ↑ after load

(5) ↑ After load ↓ Cardiac Output

(6) eg arm wrestling, tug of war, deadlift, Spring exercise.

(2) Isometric

↑ After load

↓ Venous return

↓ Card. out.

Isotonic

↑ Pre load

↑ Venous return

↑ Card. output

Types of muscles

Red

(1) Endurance

(2) High amount of myoglobin

(3) long duration work output low intensity

eg. Marathon runner, Boxer, foot ball,

White

(1) Intensity

(2) low amount myoglobin

(3) Short duration work output with high intensity.

eg. sprint runner, power weigh lifter.

Energetics of Muscle contraction

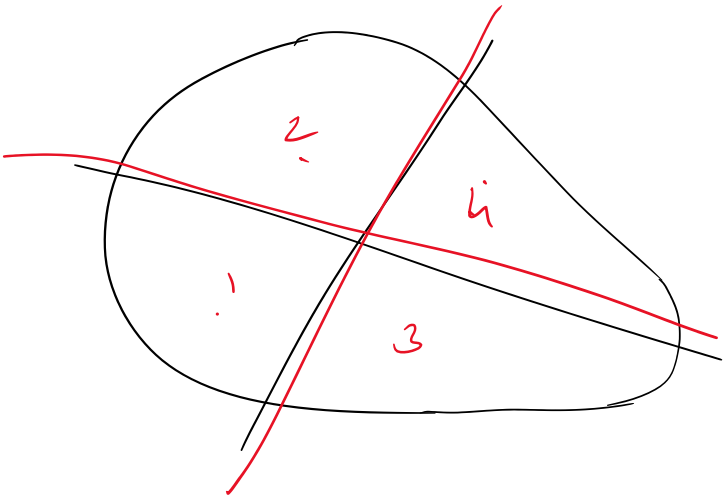
(1) Stored ATP → 2-3 sec. → eg Power lifters

(2) Phospho Creatine → 9-10 sec → eg sprint runner

(3) Aerobic Glycolysis (Glycogen) ⇒ 20-30 mins ⇒ Jogging

(4) Oxidative phosphorylation (fatty acids, glycerol, Anaerobic glycolysis) ⇒ hours ⇒ Marathon runner, Boxer, foot ball,

→ Properties of Cardiac muscle
→ Conducting system of heart
→ Cardiac cycle
→ Blood pressure
→ Cardiac Output
→ Hemodynamics
→ Shock
→ Regional circulation.



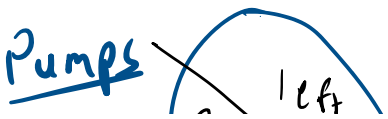
Properties

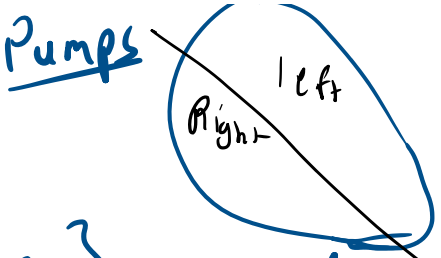
(1) **Excitability** = Cardiac muscles are an excitable tissues which shows action potential as a part of depolarization and repolarization

(2) **Conductivity** = The action potential in cardiac muscles it travels from SA node to AV node and other junctional tissues including cardiac muscles hence conduction of action potential occurs in the heart

(3) **Contractility** = On excitation the cardiac muscles undergoes contraction due to excitation contraction coupling

(4) **Syncitium** = On excitation the cardiac muscles act like a one unit and undergoes excitation and contraction like a single unit of the heart





(5) Refractory period = Absolute, Relative } long refractory period

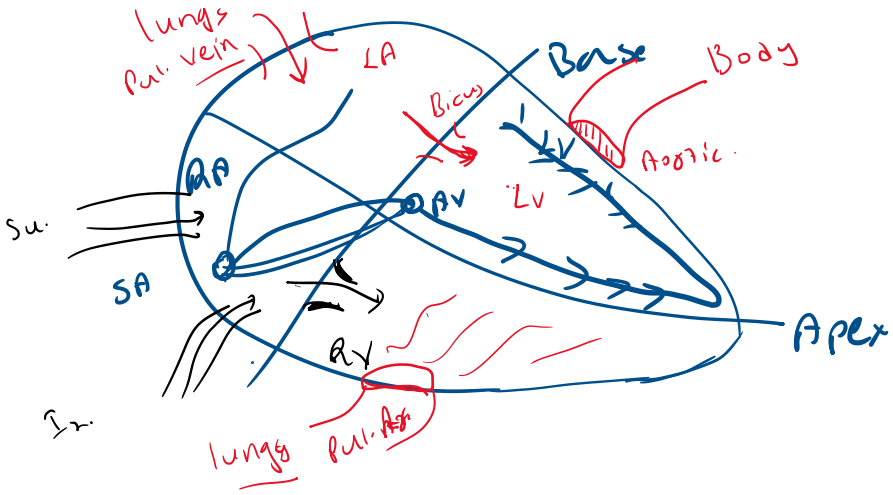
(6) All or none principle =

(7) Auto rhythmicity =

Every cardiac tissue has an ability to generate its own rhythm of excitation and contraction but in normal condition the SA node presides the all other tissues for excitation and contraction only under pathological conditions like ischemic heart disease the other tissues of the heart behave like a pacemaker cells

Cardiac Cycle

07 April 2024 11:56



Def. = A cyclic event in the heart comprising one systole & one diastole of the complete heart.

Duration = $\frac{\text{BPM}}{\text{Heart Rate}}$

C.C. = $\frac{60}{70} = 0.8$ sec

duration sec
60
x
70
1

As As the heart rate increases the duration of cardiac cycle decreases since we cannot have zero seconds for cardiac cycle duration there has to be a upper limit of maximum heart rate and this halt rate is the rate at which the duration of cardiac cycle will be minimum for that particular patient or a person with a particular age

Rules = Principles

1. Systole & diastole are time dependent
2. Opening of valve & movement of Blood are pressure dependent.

0.8 sec

Atrial Systole = 0.1 sec

Atrial diastole = 0.7 sec

0.8

Ventricular syst = 0.3 sec

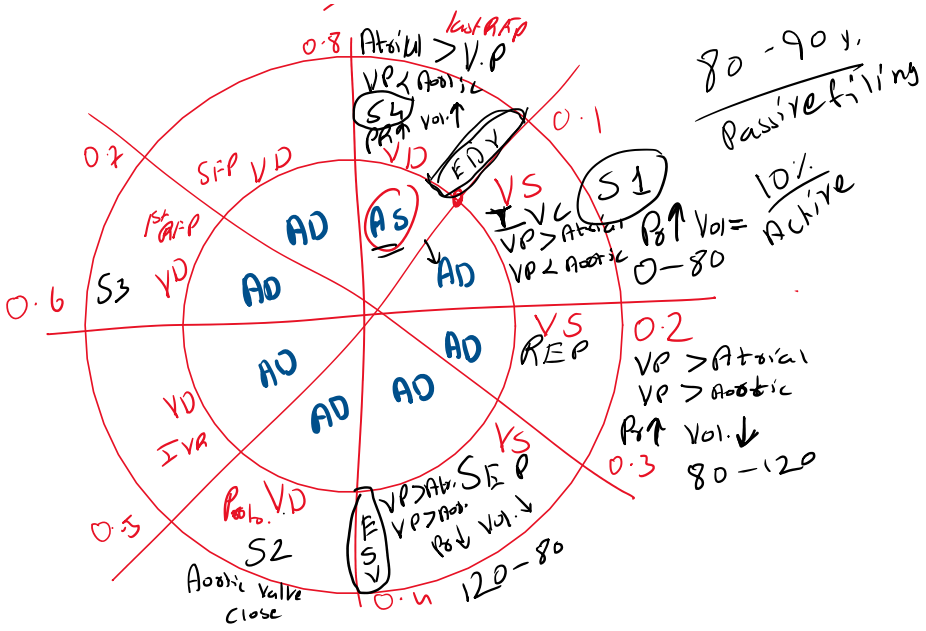
Ventric = 0.5 sec

0.8

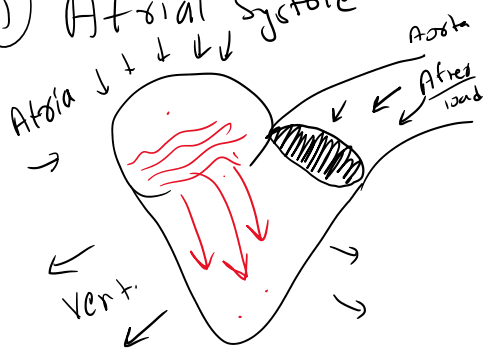
0.8 | Atrial > V.P | last AVP

V.P > Aortic

80 - 90 y.



(1) Atrial Systole



(1) Atria excites & contracts

Atrial Pressure > Vent. Pressu

Vent press < Aortic press

Blood moves from Atria → Vent.

4th Heart sound

Amount of in vent at the end of this phase is known as

" End Diastolic Volume "

" Pre load of the heart "

(2) Atrial Diastole = Atria Relaxes (0.7) and receives the blood from Subsequent Blood vessels

Ventricular systole (0.3)

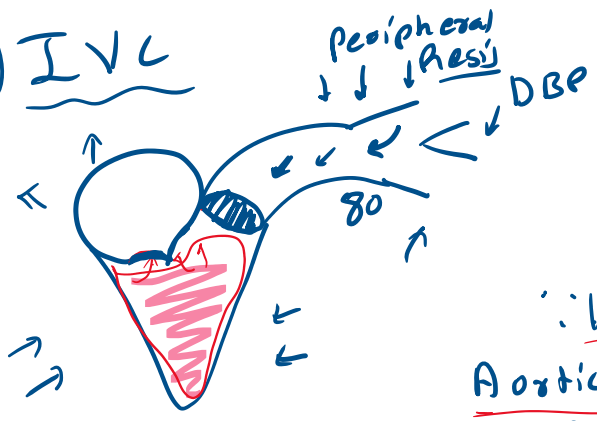
(1) IVC Iso volumetric Contraction

(2) REP Rapid ejection phase

(3) SEP Slow / Reduced ejection phase

Ventricles starts Contracti

① IVC



Ventricles starts contraction

V.P > Atrial Bp.

AV valve closes.

H.S. 1.

∴ VP < Aortic pressure.

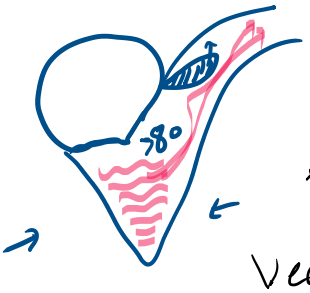
Aortic valve has not yet opened.

Vent. contracts as a closed chamber.

Known as Iso volumetric contractn

Tremendous increase in a pressure in a very short time. 0 → 80 mm Hg

② Rapid ejection phase



VP > Atrial press.

VP > Aortic press

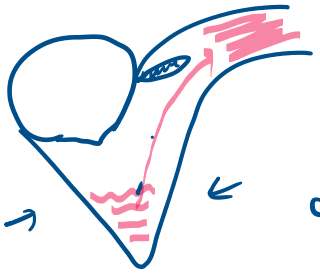
Aortic valve opens. Blood jumps Rapidly from ventricle to aorta.

∴ REP

Ven. Bp. ↑ Vent. vol. ↓

Pressure in the vent. increases from 80 to 120 mm Hg

③ Slow ejection Phase



VP > Atrial pressure

VP > Aortic pres

As the vol of Blood in vent. decreases the pressure in the vent also decreasses gradually

120 → 80

The blood moves slowly from vent. to aorta as VP > Aortic pres

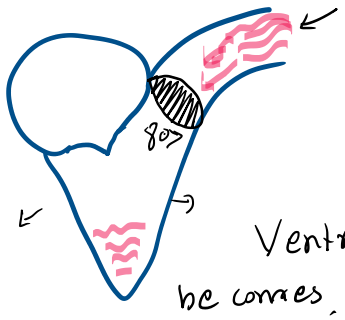
At the end of systole the blood remains in the Vent. is known as "End Systolic Volume"

At the end of systole the blood remains in the vent is known as "End Systolic Volume"

Ventricular Diastole [0.5] sec

1) Proto diastole
2) IVR Iso Volumetric Relaxation
3) 1st Rapid filling phase
4) Slow filling phase
5) last Rapid filling phase.

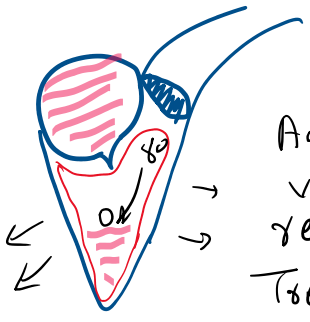
(1) Proto diastole



Vent. Pr. > Atrial Pr.
Vent Pr. < Aortic Pr.
Aortic valve is closed
H.S. 2

Ventricles begin to relax and their pressure becomes less the aortic pressure.
∴ Closure of Aortic occurs due to back pressure from aorta.

(2) Iso Volumetric Relaxation



VP. > Atrial pressure
VP < Aortic pressure
Aortic valve is closed and AV valve has not yet opened vent. relax as a closed chamber.
Tremendous decrease in pressure in a very short time.

(3) 1st RFP Rapid Filling phase

VP < Atrial pressure
AV valve opens. Blood jumps rapidly from Atria to ventricles

AV valve ... rapidly from Atria to ventricles
This (turbulance of Blood causes

H.S.3

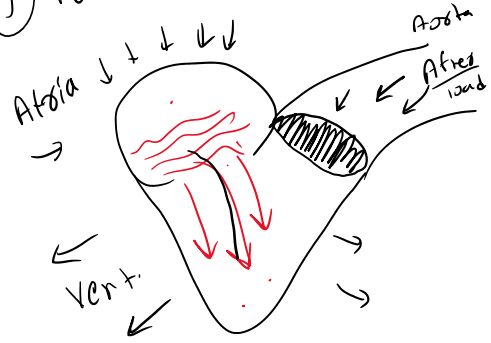
VP < Aortic pressure ∴ Aortic valve remains closed.

(4) Slow filling phase "Diastasis"

VP ≈ Atrial pressure
VP < Aortic pressure
Since the pressure gradient → between Ventricles & Atria is negligible.
The rate flow of blood from Atria to vent. is slow and gradual due to momentum of the blood.

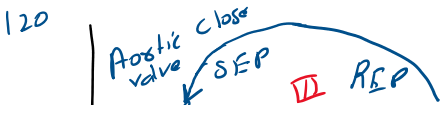
(5) last Rapid filling.

(1) Ventricular diastole

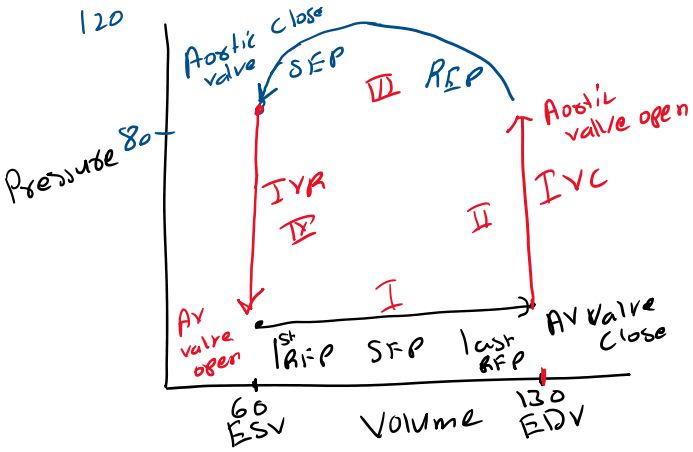


(1) Atria excites & contracts
Atrial Pressure > Vent. Pressu
Vent press < Aortic press
Blood moves from Atria → Vent. Rapidly
4th Heart sound
Amount of in vent at the end of this phase is known as
"End Diastolic Volume"
"Pre load of the heart"

Pressur - Volume loop in the left vent.



S.V. = EDV - ESV
= 130 - 60



S.V. = EDV − ESV
= 130 − 60
= 70 ml

Ejection fraction = $\frac{(EDV - ESV)}{EDV} \times 100$

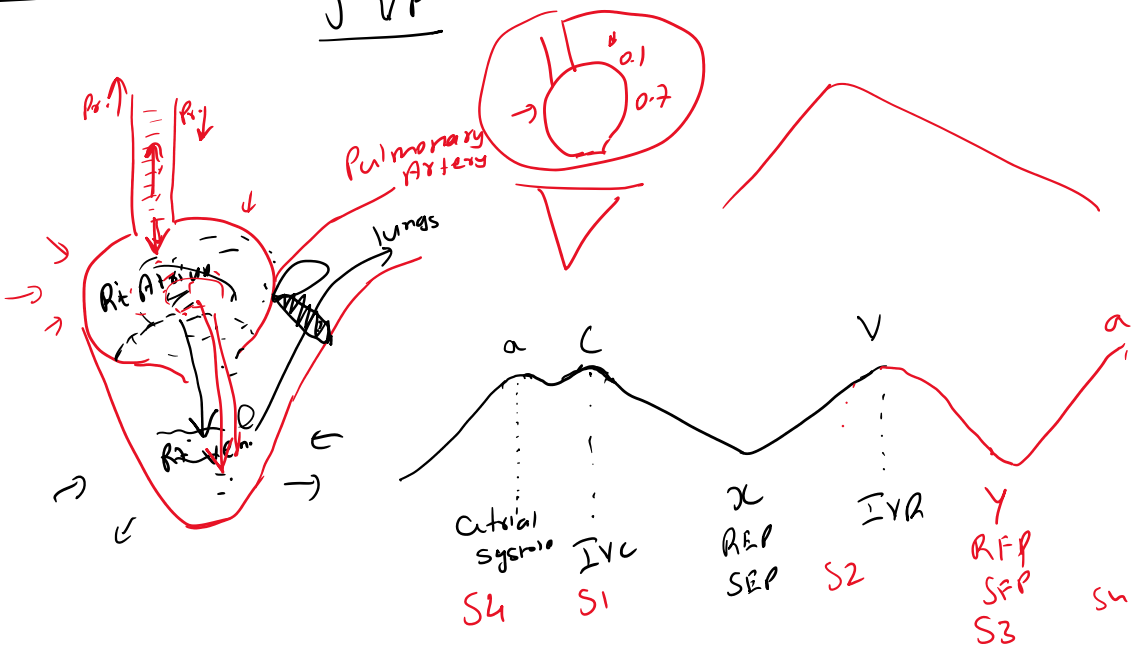
EDV 130 ↔ SV 70 (EDV − ESV)
100 ↔ x

$\frac{130 - 60}{130} \times 100$

EDV = 130
ESV = 50

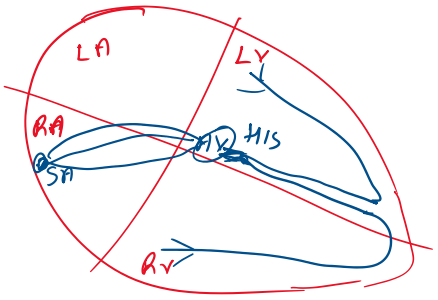
$= \frac{70}{130} \times 100$
$= 53.\ldots$
$\simeq 55\%$

JVP

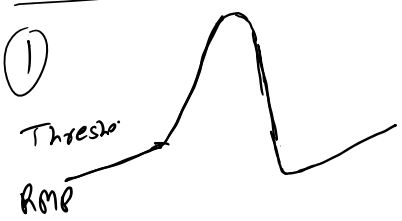
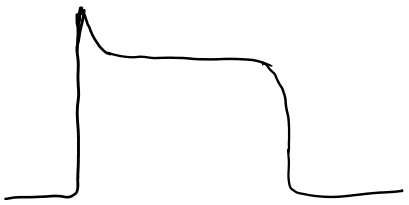


Conducting System of Heart

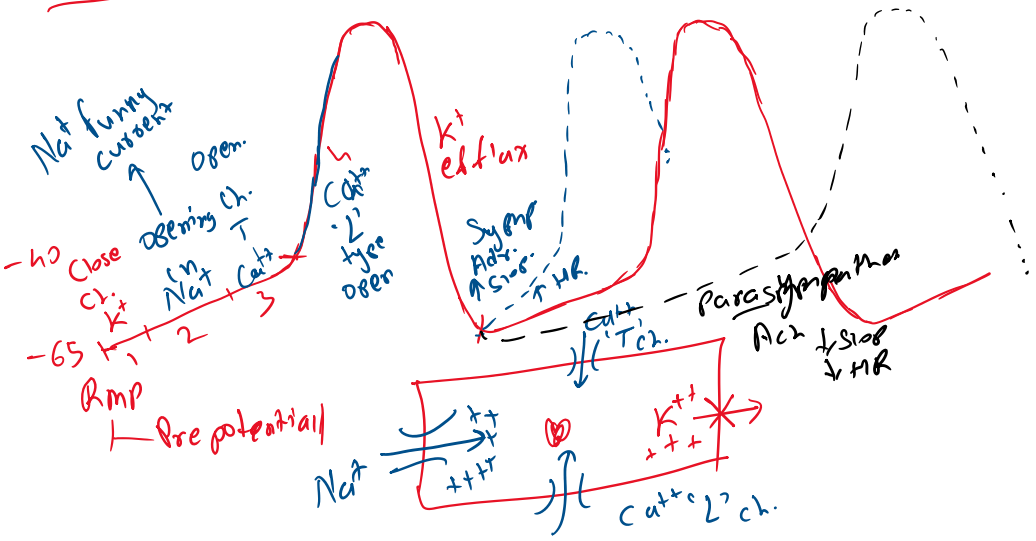
- Electrical impulse generation and conduction of this through the heart is known as conducting sy. of heart.
- The conducting system of Heart comprises of
  - SA node
  - Internodal pathways
  - AV node
  - Bundle of His
  - Purkinje fibers
  - Atrial & Ventricle myocytes.



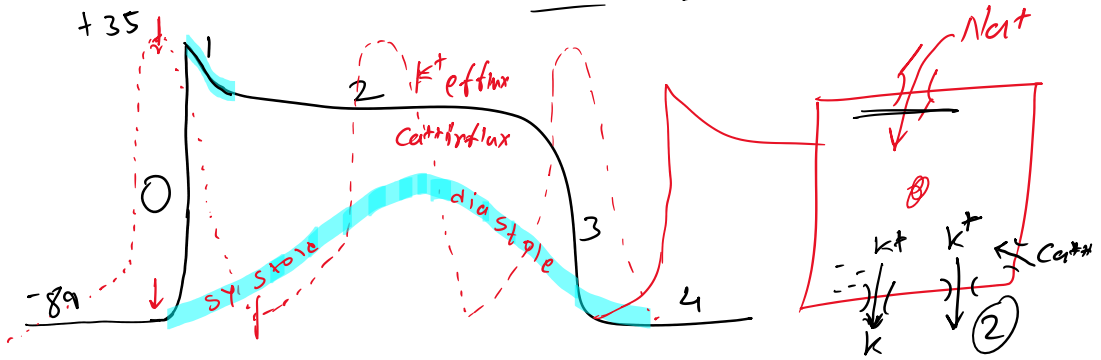
The action potential in the heart is divided in two types One type of action potential is in the pacemaker cell that is sino atrial node and atrial ventricular node these cells show a slow type of excitation known as slow type fibres and the other action potential is seen in the rest of the cardiac cells like internodal pathways, Purkinje fibres, bundle of His and ventricular and atrial myocytes these cells show fast action potential and hence they are known as fast response type cells

Slow response type fibers	Fast response type fibers
(1) 	(1) 
(2) SA, AV node	(2) His, I.N.P, Purkinje, Atrial & Vent. myocytes.
(3) Depolarization is Ca^{++}	(3) Dep.^{n} is Na^+
(4) Depo. is Slow Rep. is Rapid	(4) Depo is Rapid, Rep'n is Slow.
(5) They have automaticity normally	(5) They show automaticity in Pathological conditions.

Pacemaker potential



Action potential in Fast Response cells



0 = Opening Vol. gat Na+ Ch.

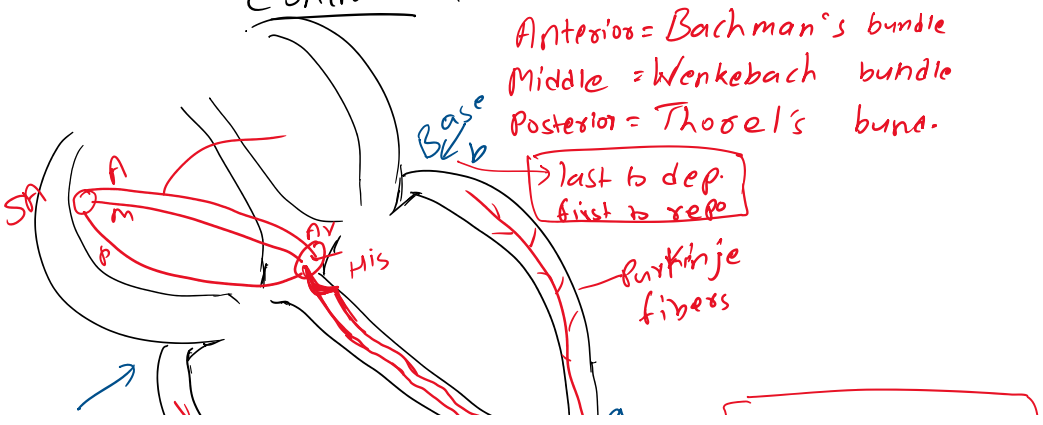
1 = a closure Na+ Ch.
b Opening of K+ Ch. efflux

2 = a opening of K+ Ch efflux
b opening of Ca++ Ch influx

3 = a Ca++ Ch. close
b K+ Ch remain open

4) = RMP factors → leaky channels
Na+ K+ Pump

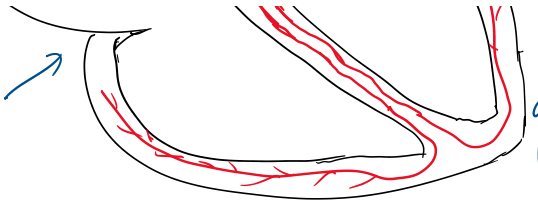
Conduction



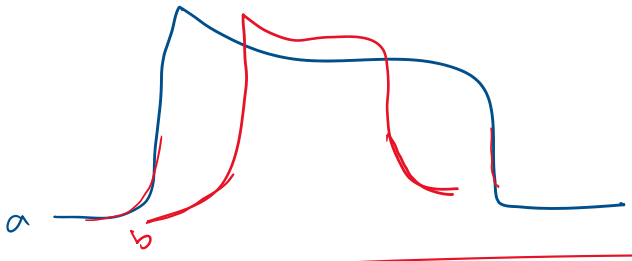
Anterior = Bachman's bundle
Middle = Wenkebach bundle
Posterior = Thorel's bund.

→ last to dep.
first to repo

Purkinje fibers



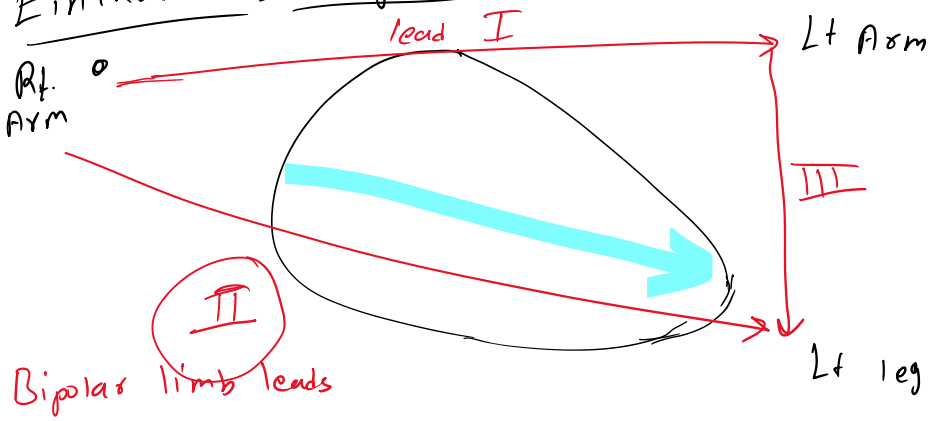
Apex → 1st to depolarize
last to repolarize



ECG :

Electrocardiogram = Recording of electrical activity of Heart.

Einthoven = Physics



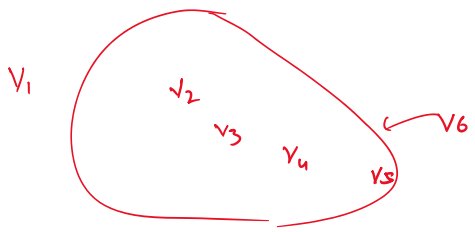
Bipolar limb leads

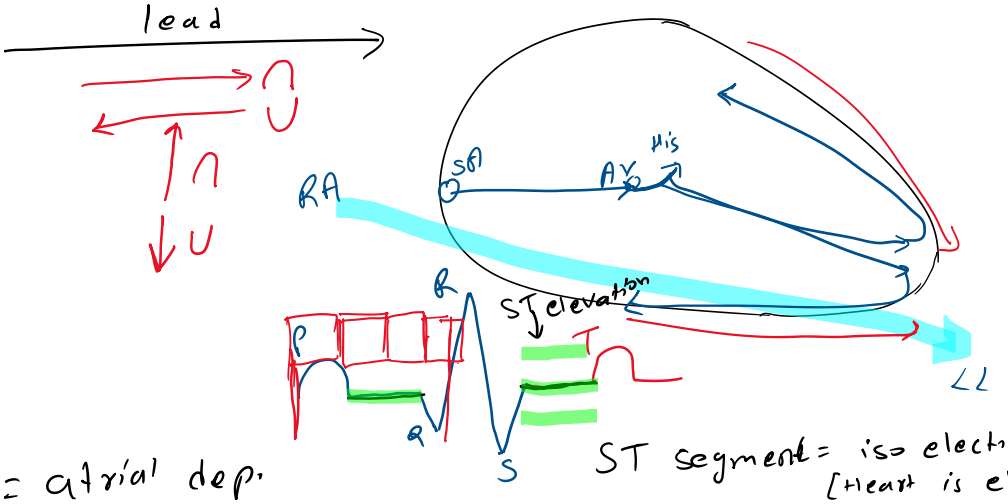
Unipol limb leads / Augmented limb leads

aVR : augmented limb lead of Right arm
aVL : " " left arm
aVF : " " left foot

6 chest leads / Precordial leads

V_1 V_2 V_3 V_4 V_5 V_6





P = atrial dep.
QRS = Vent dep
T = Vent Rep

ST segment = iso electric line
[Heart is electrically neutral]
" ST elevation = Ischemic heart disease
ST depression = Myocardial infarction"

1 mm = 0.04 Sec
Speed of Paper = 25 mm/sec

∴ P–R = 4 mm.
∴ PR int = 4 x 0.04
= 0.16 sec

∴ 0.12 sec to 0.20 sec

Heart Block

SA node → late/no → AV node.

1st degree every P — QRS complex

P—QRS (0.30) P—QRS (0.30) P—QRS (0.30)

2nd degree
1st stage Type I = P—Q (0.3) P—Q (0.32) P—Q (0.36) P—P

Type II P—Q (0.36) P—Q (0.36) P—Q (0.36) P—P

Ratio P—Q P—Q P—Q P—P

3rd degree

P–P –P –Q –P Q– P –P –Q –Q