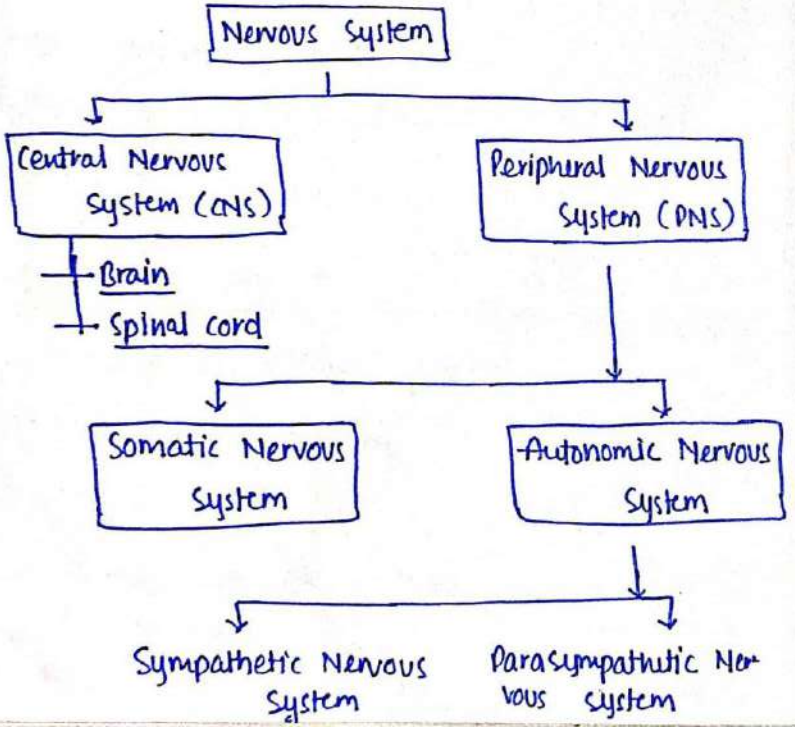
UNIT-III[rd] PHARMACOLOGY

Pharmacology of drugs acting on Peripheral Nervous System

- Nervous System :- It control and co-ordinati the human body and it gives the quick response to our body.



- CNS → It is a main system of our body which consist of brain and spinal cord. It coordinati the body.
- PNS → It consist all nerves of our body which transmit information from body to brain or brain to body. It consist the somatic and autonomic nervous system.
- Somatic Nervous System → In this system, voluntary movement happend (which we can control). eg. Hand movement, walking etc
- Autonomic Nervous System → In this system, involuntary movement happened (which we can't control). eg. Breathing, Digestion, Heart rati etc..

Now, we have to study about the action of that drugs which act on PNS.

Syllabus :-

a) Organization and function of ANS.

b) Neurohumoral transmission, Co-transmission and classification of neurotransmitter.

c) Parasympathomimetics, Parasympatholytics, Sympathomimetics, Sympatholytics.

d) Neuromuscular blocking agent and skeletal muscle relaxants (peripheral).

e) Local anesthetic agents

f) Drugs used in myasthenia gravis and glaucoma.

Organisation and function of ANS

ANS :- Autonomic Nervous System

It involves involuntary responses (movement) of our body.

- It further divided into two parts :-

i) Sympathetic N.S.

ii) Parasympathetic N.S.

i) Sympathetic Nervous System :-

- fight/flight situation [Abnormal]

- Activate in condition of three f (3F)

fear fight flight

- Those system which active in abnormal situation of body and maintain the body

example → Increases heart rate, decrease digestion rate etc.

ii) Parasympathetic Nervous System :-

- Rest and Digest condition

- In this, our body come back to normal condition after any abnormal situation.

- Also help to maintain the homeostasis of body.

example → increase digestion rate and normal heart rate etc...

Both system are motory function of body

Both system are important to maintain the homeostasis and work of our body.

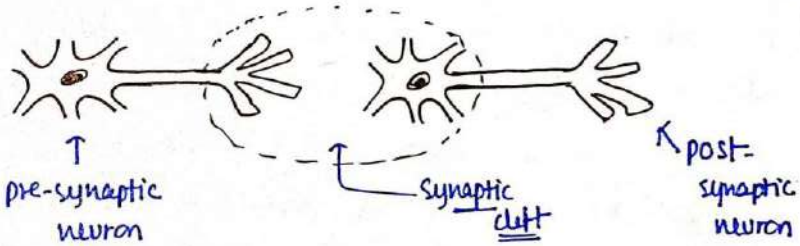
• Different b/w Symapathetic & Parasympathetic system

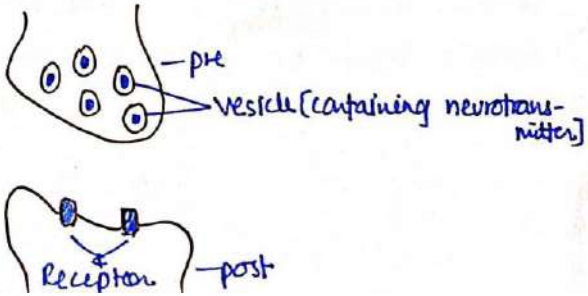
Sympathetic Nervous System	Parasympathetic Nervous system
• Involved in the fight or flight response • prepare the body for any potential danger. • increase heartbeat, muscles tense up. • pupil dilate • Saliva secretion is inhibited	• Involved in maintaining homeostasis and also, permits the rest and digest response • to bring the body to a state of calm. • Reduces heartbeat, muscle relaxes. • pupil contract • Saliva secretion increases, digestion increases
Neurotransmitter release ↓ Adrenaline and nor- adrenaline	Neurotransmitter release ↓ Acetylcholine.

NEUROHUMORAL TRANSMISSION

Neurohumoral
→ Neuro → Nerve / Neuron
+
Humoral → chemical messenger

• It is the process of transfer of any message or signal from one neuron to another neuron with the help of any chemical messenger (neurotransmitter, harmony)





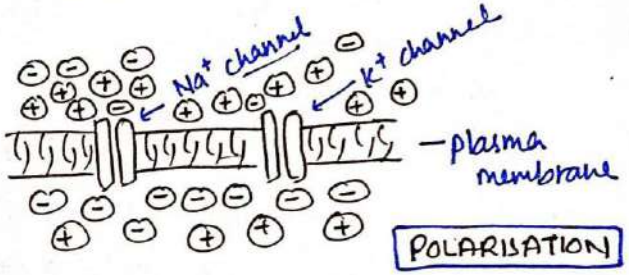
- for this purpose, firstly neurotransmitter is synthesized and stored in vesicles in nerve terminals.
- Now, Neurohumoral transmission involves following steps :-

i) Impulse conductance
ii) Transmitter release
iii) Transmitter action on post junctional membrane
iv) Post junctional activity
v) Termination of transmitter action.

i) Impulse conductance :-

In this step, firstly impulse is generated by the process of "Action potential".

- At resting state [when nerve impulse is not transmitted from neuron], Resting trans-membrane potential is -70 mV.
- Na^+ ion have high concentration at outside the cell and more (+)ve charge at outside the plasma membrane
- K^+ ion have high concentration at inside the cell and more (-)ve charge at inside the plasma membrane.

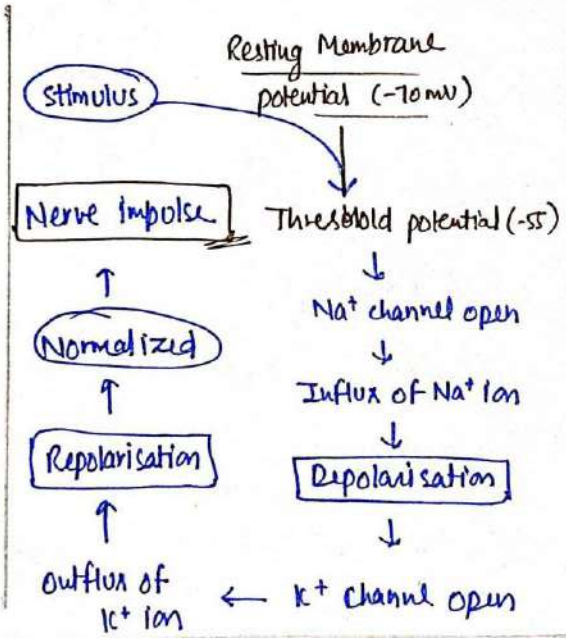


- Resting membrane potential → -70 mV.
- Depolarisation :- When any kind of stimulus detected, then it changes the Resting Membrane potential to less potential. (increases).
- If stimulus change resting potential (-70mV) to (-55mV) then it is called threshold potential.
- Threshold potential open Na^+ ion channel. So Na^+ ion enters inside the cell and (+)ve charge produce inside the cell and (-)ve at outside the cell and it is called depolarisation.

- **Repolarisation** :- Stimulus continues increase the potential, now when potential reach at (+20 mV to +30 mV) it open K^+ ion channel and K^+ ion move outside the cells.
- The ionic distribution is normalized during the refractory period by the activation of $Na^+ K^+$ pump.

The cycle of depolarisation and repolarisation is called Action Potential.

- These action potential works 1000 times in one second.



ii) Transmitter release → Nerve impulse promotes fusion of vascular and axonal membrane, through Ca^{++} entry which fluidized membranes.

- This promotes exocytosis (transmitter release from vesicle) in synaptic cleft.

iii) Transmitter action on postjunction membrane :→

The transmitter release and attached with specific receptor on postjunctional membrane and depending on nature it induce two types of action

EPSP [Excitatory post-synaptic potential]
↓
Increase in permeability to all cation → Na^+ or Ca^+ influx cause depolarisation followed by K^+ efflux
↓
iv) Nerve impulse, contraction in muscle, secretion in glands

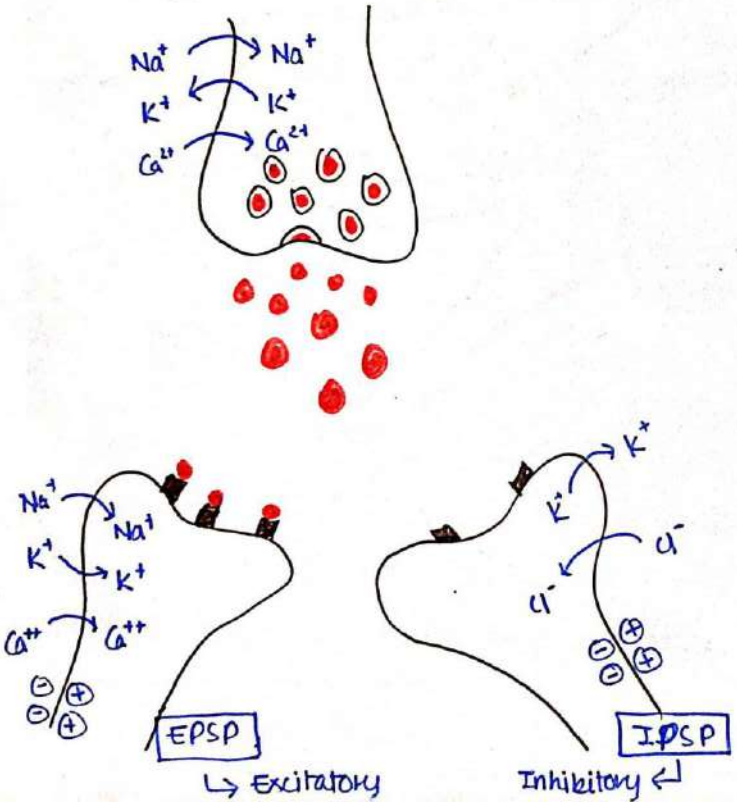
IPSP [Inhibitory post synaptic potential]
↓
If inhibitory neurotransmitter act increase in permeability to smaller ion or anions.
K^+ and Cl^- moves in, resulting Hyperpolarisation.
(move out)
↓
Resist depolarizing stimuli

v) Termination of transmitter action :-

Neurotransmitter is degraded locally or any other mechanism.

- It can also be degraded by enzymatic action.

eg. Acetylcholine degraded by cholinesterase



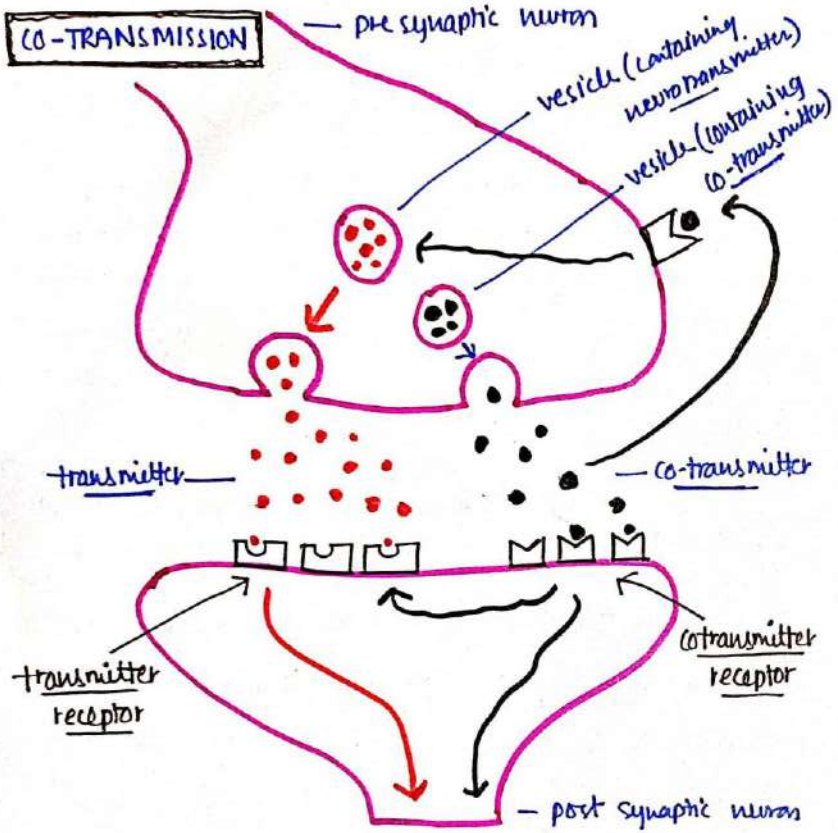
Co-transmission :-

- Peripheral and central nervous system release more than one active substance when stimulated.
- Definition :- Cotransmission is the release of several types of neurotransmitter from a single nerve terminal.
- Co-transmitter :- It is a chemical substance that is released along with primary neurotransmitter.

examples → In autonomic nervous system

- Primary neurotransmitter → Ach , NA
- Co-transmitter are → Purines :- ATP , Adenosine
  Peptides :- Vaso intestinal peptide (VIP)
  Nitric oxide
  Prostaglandings (PGs)

On release of Acetylcholine (Ach) , Glutamate , Vasoactive intestinal peptide (VIP) co-transmitter release



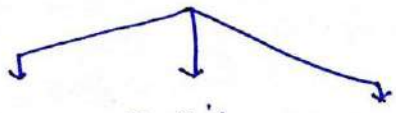
- Cotransmitter is stored with primary transmitter vesicle or in different (separati) vesicle.
- function :— i) enhance or regulate presynaptic release of primary neurotransmitter.
ii) Modulate post-synaptic sensitivity of primary neurotransmitter
iii) serve as a alternate to primary neurotransmitter.

Classification of Neurotransmitters :—

- Neurotransmitter :— These are chemical messenger that transmit signal from a neuron to a target cell across a synapse.
- target cells may be a other neuron or some other kind of cell such as muscles or glands.
- These are stored into synaptic vesicle in pre-synaptic neurons.

Neurotransmitter → bind → RECEPTOR → ACTION

- They can be classified as either excitatory or inhibitory.
- Excitatory :— Activate receptor on post-synaptic membrane and enhance the effect of the action potential (increase activity).
- Inhibitory :— decrease the activity of transmitter or receptor. (decrease the effect of the action potential.
- Some neurotransmitter show both type of activity.



Excitatory	Inhibitory	Both
• Acetylcholine	• GABA	• Acetylcholine
• Adrenaline	• Serotonine	• Dopamine
• Nor. adrenaline		• Nor-adrenaline
• Dopamine		
• Histamine		
• Glutamate		

– following major neurotransmitter with their functions:—

• Acetylcholine → (learning) → Involved in thought, learning, and memory. It activate muscle contraction in the body and is also associated with attention and awekening.

• Adrenaline → (fight or flight) → It is primarily released by the adrenal gland, but some neurons may secrete it as a neurotransmitter.
– It is produced in stressful situation, increase heart rate and blood flow.
– leading to physical boost and aware heightened awareness.

• Nor-adrenaline ⊢ (concentration] → It improve attention and responding actions in the brain.
– Contracts blood vessels, increasing blood vessels.

• Dopamine :– (pleasure] → feeling of pleasure, also addiction, movement and motivational.
– people's repeat behaviours lead to dopamine release

• Serotonin → (mood] → contributes to well beings and happiness.
– Helps sleep cycle and digustive system regulation.

• GABA → (calming] → Calm firing nerves in the CNS.
– High level → improve focus
– Low level → cause anxiety.
– Also contributes to motor control and vision.

• Histamine → Released by mast cells.
– involved in local immune responses
– Contraction of smooth muscle tissue of the lungs, uterus and stomach.

• Glutamate → (memory) → Involved in learning and memory. It regulates development and creation of nerve contacts.

C. Parasympathomimetics, Parasympatholytics, Sympathomimetics, Sympatholytics.

Drugs acting on Autonomic Nervous System.

These all are those drugs which act on Autonomic Nervous system and produce effect on it.

- Adrenergic drugs [Sympathomimetics] } Sympathetic Nervous system
- Antiadrenergic drugs [Sympatholytics] }
- Cholinergic drugs [Parasympathomimetics] } Parasympathetic Nervous system
- Anti-cholinergic drugs [Parasympatholytics] }

What we have to study ??

- Introduction / Defination
- Synthesis, storage, Release, Degradation
- Receptor and Mechanism
- Classification – Drugs
  - Name
  - MOA (Mechanism of Action)
  - Uses

  (examples)
- Blocking Agents and Pharmacological action.

CHOLINERGIC SYSTEM

– Also known as Parasympathomimetics system.

Parasympathomimetics – parasympatho → Parasympathetic Nervous system; mimetic (mimic) → copy the action.

- These are those chemical agents or drugs which copy the action of parasympathetic Nervous System.
- These drugs bind with cholinergic receptors [Muscarinic & Nicotinic] and give their action.

Why??

- When the neurotransmitter of parasympathetic nervous system (Acetylcholine) in body is less as per demand, then we use drugs externally which act as a cholinergic neurotransmitter. i.e.. parasympathomimetics.

eg: Acetylcholine, Carbachol, Physostigmine, Neostigmine etc.

Pharmacological action of parasympathomimetics.
(Acetylcholine)

i) Cardiovascular system :- (M_2 receptor)

- depress auricular muscles
- ↓ contraction of bundle of his AV node
- BP ↓ due to vasodilation.

ii) Eye :-

- Constrict the pupil and cause pupil.
- Intraocular pressure decreases.
- used in Glaucoma

iii) Skeletal muscles :-

- Constraction of skeletal muscle
- Useful in myasthenia gravis

iv) Respiratory system :-

- Bronchoconstriction
- Induce Asthma.

v) Gastrointestinal system :-

- Contract smooth muscle of GIT.
- increase tone, motility and peristalsis movement. (constriction & relaxation)
- Increase salivary, pancreatic, liver, gall bladder and intestinal secretions.

- therapeutic uses :-
  - Glaucoma (physostigmine, Pilocarpine)
  - Myasthenia gravis (Neostigmine)
  - Paralytic ileus and Post-operative urine retention (Bethanechol, Carbachol)
  - Atropine poisoning (Physostigmine)
  - Curare poisoning – neuromuscular junction block (Neostigmine)
  - Alzheimer's disease (cholinesterase inhibitor)
- Adverse effects :-

Parasympathomimetics may cause –

- Nausea
- Vomiting
- Bradycardia / Hypotension (higher doses)
- Cause Asthma

FOR MORE DETAILS NOTES ON THIS TOPIC, CHECK MEDICINAL CHEMISTRY UNIT-2 & 3 NOTES.

Parasympatholytics

Parasympatho — paracympathetic Nervous System

Lytics — Lysis (breakdown) → oppose the effect

- These are those drugs which inhibit the effect of acetylcholine or parasympatho-mimetics by blocking the cholinergic receptors.

-FOR MORE DETAILS NOTES ON THIS TOPIC, VISIT MEDICINAL CHEMISTRY NOTES- UNIT 2/3

- MUST WATCH VIDEO - L-3, Unit-3, Pharmacology, Carewell pharma

Pharmacological action of Parasympatholytics (Atropine).

i) CNS :- It can cross BBB (Blood brain barrier) so it can produce their effect CNS.
- It cause respiratory depression
- cause drowsiness and sedative effects.

ii) Cardiovascular system :- It block M receptors and cause :-
- increase the heart rate
- Increase the conduction from SA node

iii) Eye :-
- Cause mydriasis

iv) Exocrine gland :-
- decrease secretion of salivary, bronchial, gastric, pancreatic, lacrimal and sweat-gland.
- rise in body temp.

v) GIT :—

- Reduce motility of GIT.
- Gastric juice secretion reduced↓
- Used to treate peptic ulcer.

• therapeutic uses :—

- mydriasis
- Anti-parkinsonism agent
- Pre-anaesthetic medication
- Motion sickness
- peptic ulcer
- Bronchial Asthma [Ipratroplum]
- Anti-cold tablets.

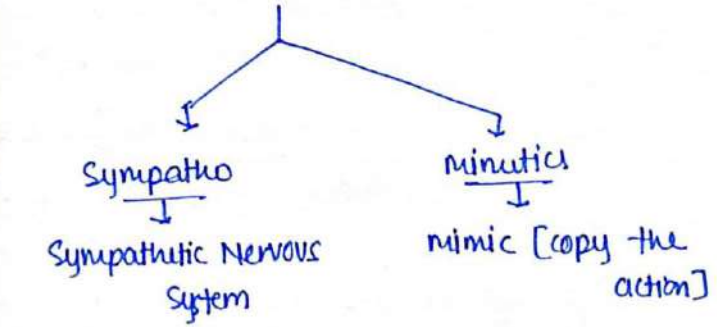
• Adverse effects :— may cause

- can cause glaucoma in some patients.
- Tachycardia

ADRENERGIC SYSTEM

— It involves Sympathomimetics drugs

Sympathomimetics



• These are those chemical agents or drugs which copy the action of sympathetic nervous system.

• These drugs bind with adrenergic receptors [Alpha & Beta] and give their action.

eg. Adrenaline [Epinephrine]
Nor-adrenaline [Nor-epinephrine]
Phenylephrine etc.—

Pharmacological action of Sympathomimetics [Adrenaline]

i) Cardio vascular system :—

- increase force of contraction
- Increase in Heart rati
- increase in cardiac output

} Heart

— Blood vessels

- on skeleton muscles — vasodilation (coronary arteries)
- on smooth muscles — vaso constriction

ii) Respiratory system :—

- on β_2 receptor → Bronchodilation
- α_1 receptor present in the blood vessels of nasal mucosa.
  ↓
  cause vasoconstriction of nasal mucosa

iii) Eye (α_1 receptor) :—

- Contraction of radial muscle of iris
  ↓
  cause mydriasis — dilation of pupil of the eye

iv) GIT (Gastro intestinal tract) :—

- Relaxation of GI smooth muscles.

v) Urinary tract :—

- relaxation of urinary bladder (β_2) and closure of sphincter (α_1).
- Urinary retention.

- therapeutic effects :—

— Bronchial asthma (salbutamol)
— Nasal decongestant
— as a cardiac stimulant in case of sudden cardiac arrest.

- Adverse effects :— may cause
  - Restlessness
  - Anxiety
  - Insomnia
  - Blood pressure ↑
  - Contra-Indicated in
    - Hypertension
    - Diabetes
    - Arteriosclerosis.

Sympatholytic

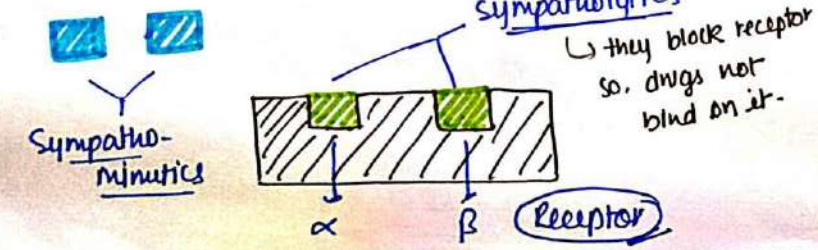
- Sympatho → Sympathetic Nervous System
- Lytic (Lysis) → oppose (breakdown)

These are those drug which inhibit the effects of sympathomimetic drug by blocking the receptor.

— Also known as:—

- Anti-adrenergic drugs
- Adrenergic antagonic
- Adrenergic blocker

Mechanism



- pharmacological action of sympatholytics
- α_1 blockers → vasodilation and ↓ BP.
- α_2 blockers → stimulates release of Nor-adrenaline → ↓ tachycardia
- Non-selective α-blockers → produce Hypotension, tachycardia, Increased cardiac output.
- β_1 blockers → decrease heart rate
- β_2 blockers → cause Bronchoconstriction
- β_3 blockers → block lipolysis & glycogenolysis.
- therapeutic uses:—

i) α- blockers →
- Hypertension
- Congestive heart-failure
- Peripheral vascular disease

ii) β- blockers →
- Angina pectoris
- Myocardial infarction
- Cardiac arrythmias
- Glaucoma

NEUROMUSCULAR BLOCKING AGENTS

SKELETAL MUSCLE RELAXANTS (peripheral)

- **Neuromuscular blocking agents :–**

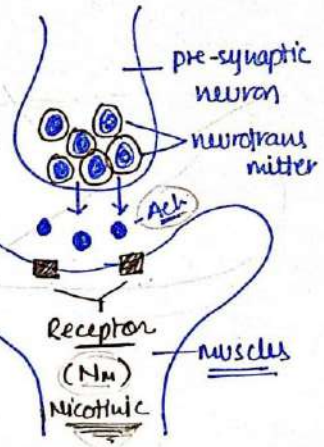
These are those agents or drugs which are used to block the Neuromuscular junction (NMJ) and inhibit the contraction of muscle and cause relaxation of muscles.

- They are also known as skeletal muscle relaxants

— Neuromuscular Junction (NMJ)

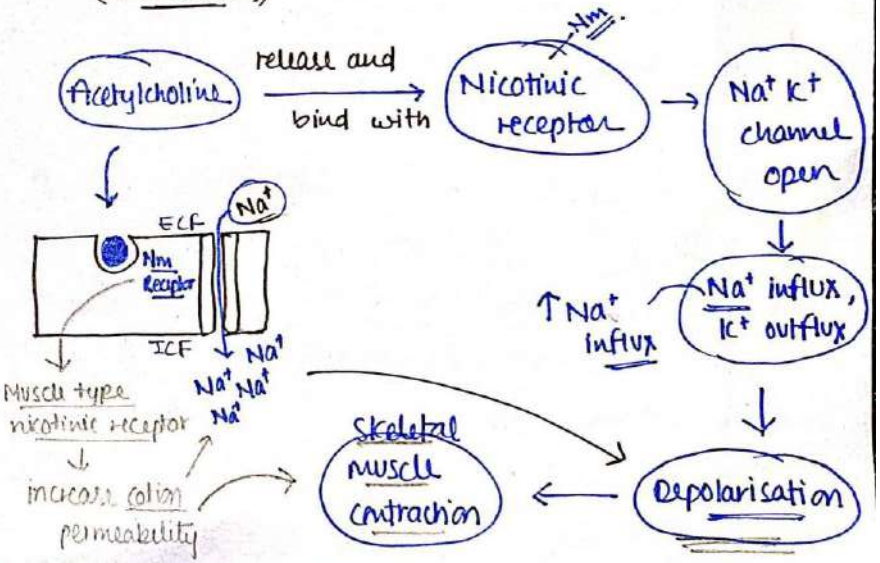
- It is the junction b/w the neuron and muscles, in which, neurotransmitter release from neuron and bind with receptor (nicotinic) present on muscle and cause contraction & movement.

Neuromuscular blocking agent blocks this NMJ.

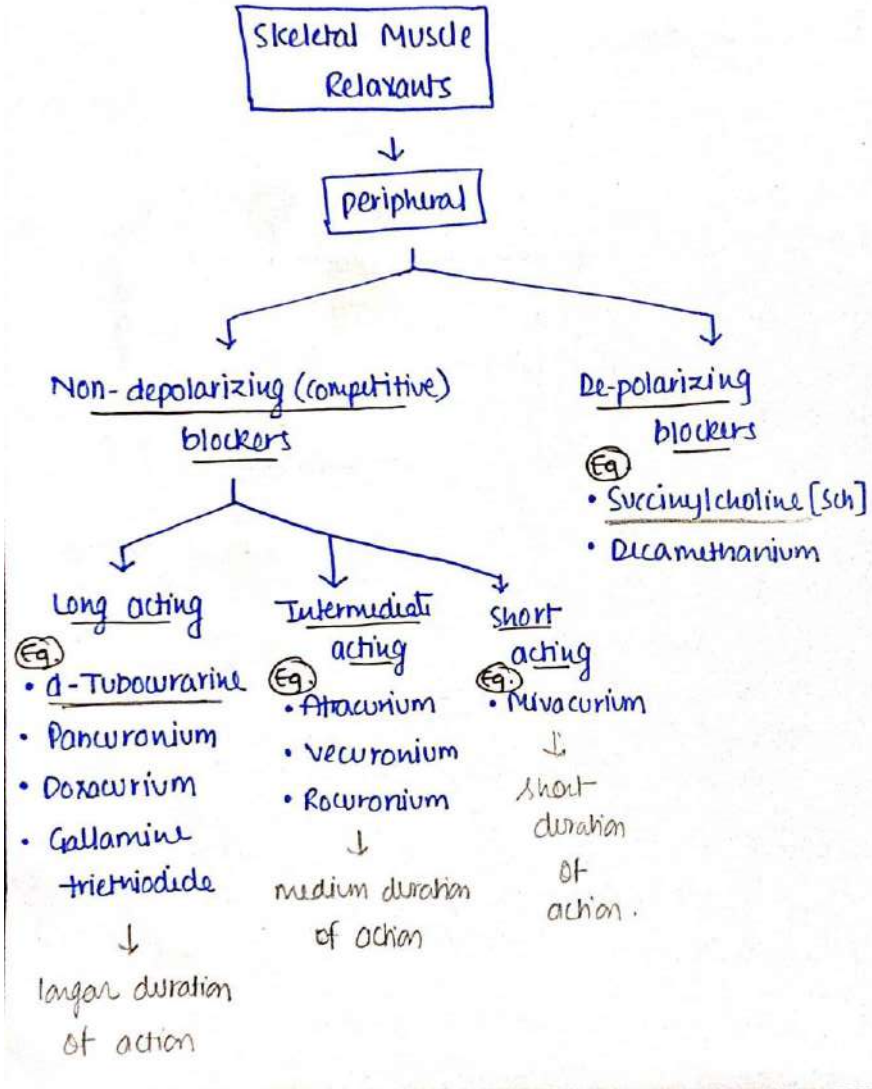


Uses :–

- generally used by Doctor for relaxation of muscle during operation
- used to improve symptoms such as muscle spasm (Tetanus), pain and hyperreflexia (over-responsive)
- used as a curare (skeletal muscle relaxants) — for hunting animals.
- used as a alternate of anaesthetic.
- General procedure of Muscle contraction (mechanism)



- **Classification**



1) **Non-depolarizing blockers** :—

- The first neuromuscular blocking drug was found - curare - used by south american hunter to paralyse the animals
- Most used → d-tubocuranine [d-TC]

- **Mechanism** →

- they are competitive antagonist and structural analogs (similar structure) of acetylcholine

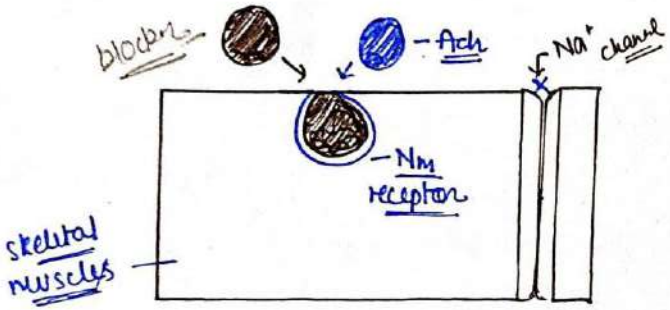
Tubocuranine and other non-depolarizing blockers have quaternary compounds with two positively charged nitrogen

So, they have affinity to bind N_M (Nicotinic) receptor but have no intrinsic activity.

- Acetylcholine and these blockers fight for one place (receptor) and finally these blockers bind with receptor in place of acetylcholine
- So, Ach not bind with receptor → Sodium channel not open → Depolarisation not

occur

- therefore, the motor nerve impulse cannot transmit and contraction is stopped (prevent).
- finally, skeletal muscle relaxation occurs.



- Pharmacological action :—

i) Skeletal muscles :—

- induced flaccid paralysis
- paralyse acc to this order —

  muscles of face → eye → finger → limb → neck

  (arm & legs)
- recovery occurs in reverse order.

ii) Histamine release :—

- d-TC has a greater tendency to liberate histamine from mast cells

iii) Cardiovascular system :—

- d-TC produce hypotension due to histamine release
- Gallamine cause tachycardia.

iv) Respiratory effect :—

- Bronchospasm

v) Autonomic ganglia :—

- produce some degree of ganglionic blockade.

It can overcome or reverse by use of Neostigmine and Pyridostigmine which increase the availability of Ach (increase concentration) by inhibiting Acetylcholinesterase (enzyme).

- Adverse effect :—

— Hypoxia
— Respiratory paralysis
— Hypotension
— Constipation
— Tachycardia etc.

eg: d-Tubocurarine, Pancuronium, Atracurium, Rivacurium etc.

2) Depolarizing blockers :—

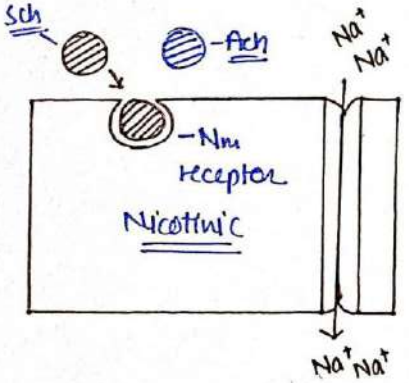
- these are non-competitive antagonist.
- Mostly used → succinylcholine (sch) as general clinical use.
- Succinyl choline does not hydrolyzed by Acetylcholinesterase

- Mechanism :-
- They have similar action as acetylcholine

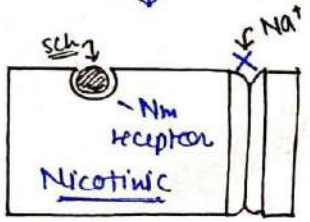
⁂ In case, when Ach bind on receptor, it cause depolarisation but after some time Ach hydrolysed by cholinesterase enzyme
↓
normal sufficient response (contraction)

⁂ Sch bind in place of Ach with Nm receptor.
↓
Na^+ channel open → Na^+ ion influx → Depolarization → Skeletal muscle contraction.

- But Sch does not hydrolyzed or terminate so. cause continuous depolarization.
↓
Repetitive excitation of muscles ← Desensitization of receptor due to excessive impulse
↓
Nerve conduction terminate → Relaxed (Fatigue)



→ Continuous depolarization
↓
Receptor desensitization
↓
also Na^+ channel blocks
↓



Repolarisation ← fatigue ← Relaxed

- Pharmacological action :—
- muscle twitching → small muscle contraction
- muscle soreness → pain
- Apnoea
  - Muscle and tissue in the throat relax (total blockage of airway that last for 10 sec).

- therapeutic uses :—
- adjuvants (initial ingredient) to general anaesthesia
- prevent trauma during electroconvulsive therapy (used in severe depression).
- to control ventillation.

- Adverse effects :—
- muscle rigidity
- prolonged apnoea
- Nausea & Vomitting.
- Muscle soreness

Example :— Succinylcholine (sch), it can degrade by butyrylcholinesterase

LOCAL ANAESTHETICS - LA

- **Basic terms :–**
- **Pain** → A symptom of being hurt or sick.

  OR

  An unpleasant sensation.

  – It can be mild, discomfort but sometime it is out of control.

- **Sensation** → It is a feeling which you feel when you come in contact with anything. [hot, cold, soft, hard].

- **Analgesic** → those drugs which reduce pain and produce analgesia effect.
  (used as a painkiller)

- **Anaesthetic** → those drugs which reduce sensation which further reduce pain.

  – Reversible loss of sensation. (sensation back after some time / work).

Anaesthetic

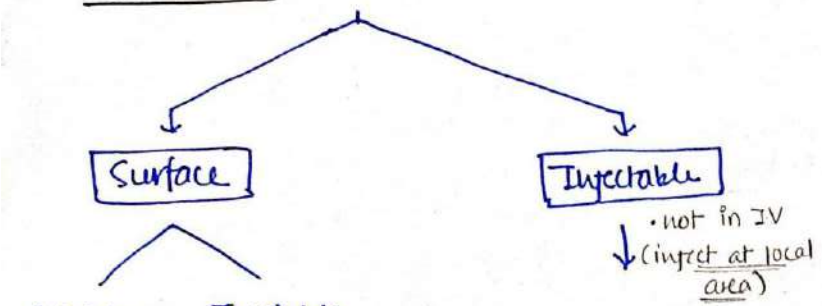
Local anaesthetics	General anaesthetics
• loss of sensation & pain at any particular area or a local area.	• loss of sensation in complete full body.
• used for a minor surgery.	• used for a major operation / surgery
eg. removal of tooth, eye surgery etc–	eg Heart surgery, organ transplant etc–
– Drugs → Lidocaine, Procaine etc–	– Drugs → opioids, diazepam isoflurane etc–

Definition :–

These are those drugs which blocks the neuronal conduction at any particular area in body.

- they produce reversible loss of sensation.
- also cause muscular paralysis.

Classification :-



- Low potency / short duration
  - procaine, -chloroprocaine
- Intermidiati potency / duration
  - Lidocaine, - Prilocaine
- High potency / long duration
  - Tetracaine, - Ropivacaine

- Surface anaesthesia :-
  - Apply topically on skin
  - Sensory nerve ending are affected
  - Limited to surface (mucous) membrane
  - It can damaged skin surface, wound or burns

  eg. Benzocaine, Lidocaine etc..

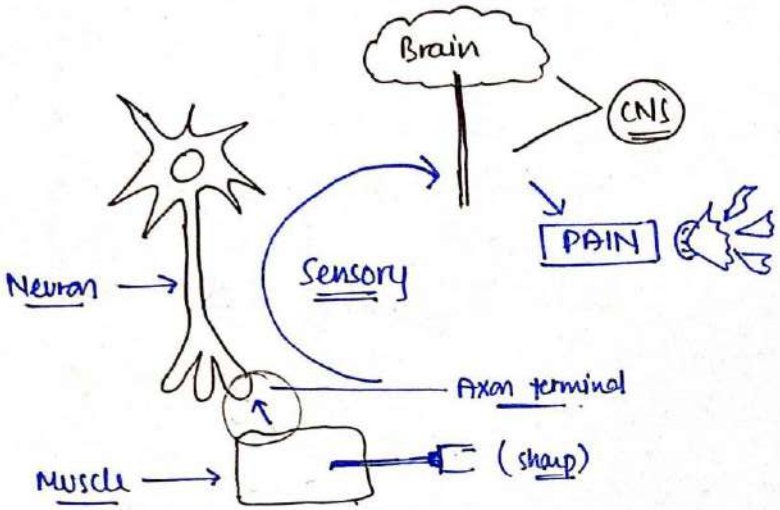
- Injectable anaesthesia :-
  - those drugs which inject under the skin (not through I.V.)
  - Used for minor surgery
  - Skin cut etc..

eg. Procaine, Bupivacaine etc..

⁂ All types of local anaesthetic drugs contain tertiary amine structure. So, they are basic in nature.

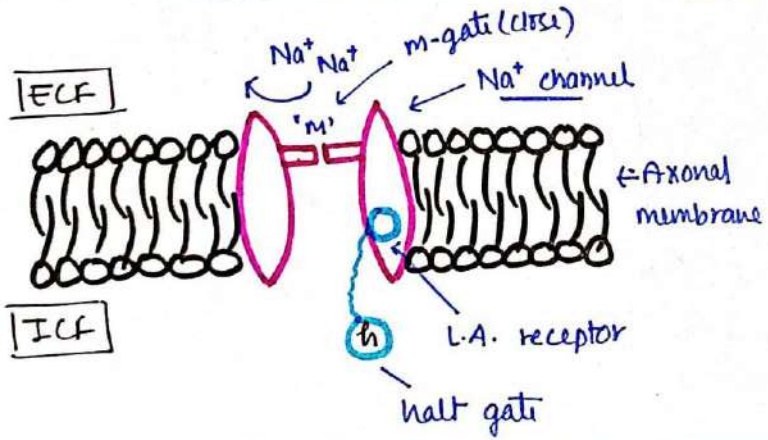
- they are lipophilic in nature. so they can easily cross membrane (mostly).
- L.A. contain two types of bond
  - Amide bond → Lidocaine, Bupivacaine, tetracaine etc
  - Ester bond → procaine, cocaine, Benzocaine etc..

- Mechanism of Action



① Normal (Resting condition) →

When no impulse generati. → no sensation.

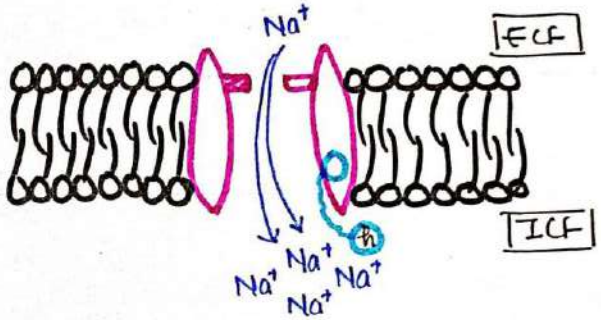


② Activated →

Any signal (impulse) generati → m-gati open

↓

Na+ channel open → Na+ ion influx → Depolarisation → sensation OR Response.



③ Inactivated → (Blocked state).

Now, L.A. drugs introduced into body. (BH)

↓

which can easily cross membrane (axonal)

↓

Now in intracellular it dissociate into ionized form → $BH \rightleftharpoons B^- + H^+$

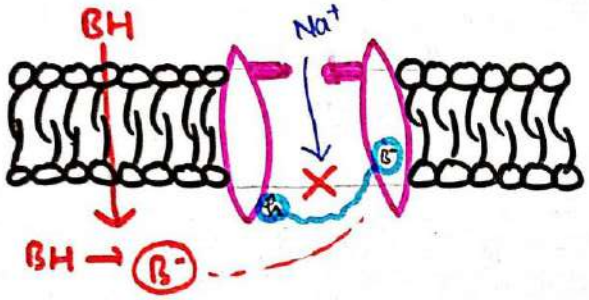
↓

Now, B^- bind with LA receptor which close (block) the Na^+ channel with the help of

halt(n) gati.

↓

Depolarisation stop → Reversibly → Loss of sensation



Adverse effects :—

- Tounge numbness
- Muscle twitching
- Hypotension
- Redness of skin
- Asthma

You can write about some drugs in details.

eg. Cocaine, procaine, Lidocaine, Benzocaine etc

Uses :—

- loss of sensation (reduce pain).
- nerve block (block voltage gated Na^+ channel).
- Used as ointment, injection etc.

DRUGS USED IN MYASTHENIA GRAVIS

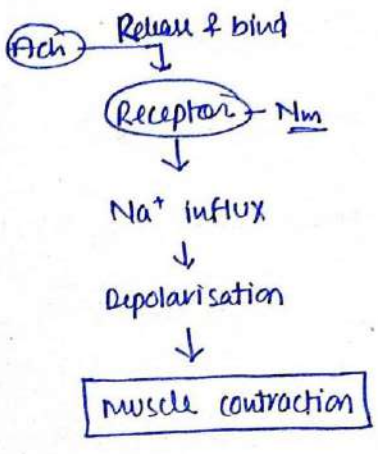
- Myasthenia Gravis :- A weakness and rapid fatigue of muscle.
- It is an auto-immune disorder, in which our immune system produce antibodies that block or destroy muscle's receptor.
- Breakdown in communication b/w nerves and muscles.

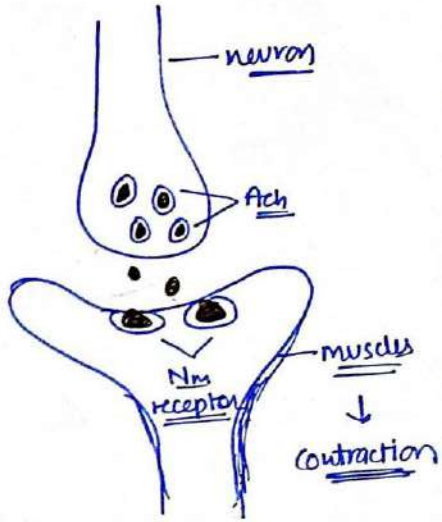
— Symptoms :—

- weakness in the arms and leg muscles.
- Double vision
- Difficulties with speech and chewing
- fatigue, shortness of breath.

— Mechanism :—

- Normal :→ When myasthenia gravis not occured and our body behave normal and contraction and relaxation happened normally in muscles



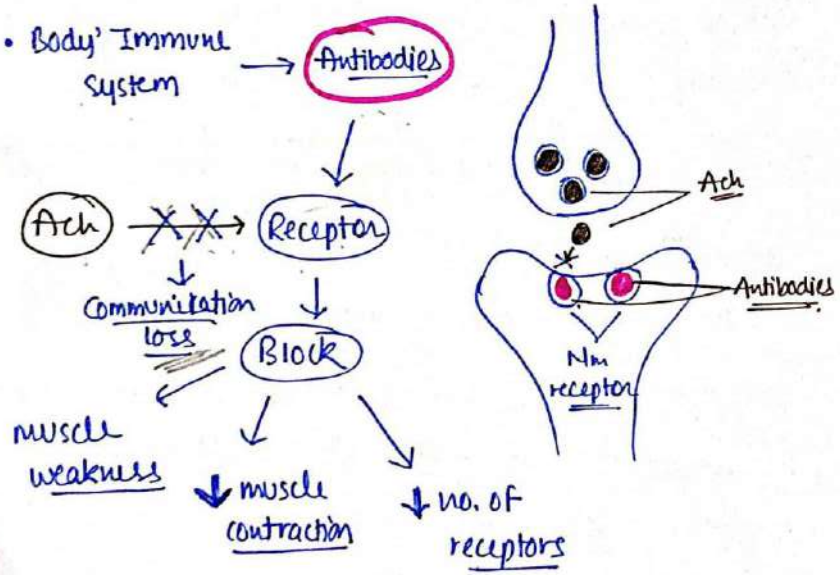


- M.G. →

It is an auto-immune disorder

- In this disorder, our immune system produce antibodies to block/destroy the Nicotinic receptor.
- Because, Acc to immune system these receptors are harmful for body.
- So, these antibodies bind with these receptor and block them.
- Now due to blockage of receptor, acetylcholine (Ach) does not bind on receptor.

- Due to this, there are loss of communication b/w nerves (ach) and muscle.
- which further decrease the contraction of muscles.
- Also muscle become weak & fatigue
- these antibodies also destroy or kill the receptor. Due to this there are also decrease in the no. of receptors.



TREATMENT – Drugs Used.

1) Anticholinesterase :–

Use these drug to treat myasthenia gravis.

Anticholinesterase drugs → inhibit → Cholinesterase enzyme

- cholinesterase inhibit the acetylcholine by hydrolysis.
- So, when these drugs inhibit (stop) this enzymes, concentration of Ach increases.
- By ↑ ach release → can replace antibodies.

eg: Pyridostigmine, Neostigmine etc...

11) Immunosuppresant :–

Use these drugs to suppress the immune system to decrease the formation of antibodies.

eg: Cyclosparine A, Methotrexate, Azathioprine, Cyclophosphamide etc...

iii) Corticosteroids :–

- Decrease Antibodies
- Increase synthesis of nicotinic receptor.

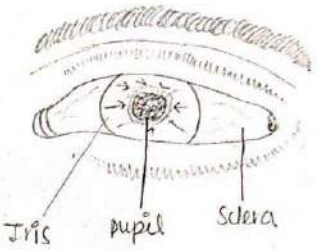
iv) Plasmapheresis :– (plasma exchange)

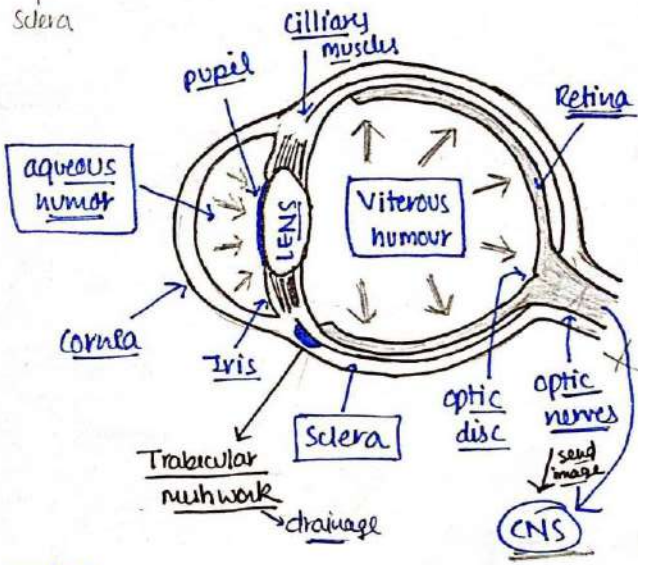
- It is a technique used to treat myasthenia gravis.
- the plasma of the blood is exchange with substitute plasma, so Antibodies (Ab) remove from body and immune system does not attack the body's own tissue

DRUG USED IN GLAUCOMA

- Glaucoma :– A group of eye condition that (गोलियाबिंद) can cause blindness (loss of vision).
- In this, the nerve connecting the eye to the brain (optic nerves) is damaged due to high eye pressure (intra ocular pressure).
- Intra-ocular pressure is more than ↑ 21 mmHg

- mechanism :–





- two main reason
  - 1) – ↑ production of aqueous humour
  - 2) – ↓ drainage of aqueous humour

→ I.O.P. ↑

- ↓ mid-dilated pupil
- eye pain
- optic nerve damage
- loss of Vision (Blindness)

- Symptoms :-
  - eye pain
  - mid - dilated pupil
  - redness of the eye
  - Vision loss , blurred vision
- Risk factor :-
  - increased pressure in the eye
  - due to genetic factor (family history)
  - High blood pressure
  - excessive use of liquid diets (such as alcohol etc).
  - excessive use of steroids.
- Diagnosis :-
  - Dilated eye examination.
- Types :-

I) Open angle glaucoma

II) closed angle glaucoma

I) Open angle glaucoma

- Also known as chronic and wide angle glaucoma
- symptoms → gradual vision loss.
- Optic nerve damage
- most common type of glaucoma

II) Angle closure glaucoma

- Also known as acute and narrow angle glaucoma.
- flow of Aqueous humor blocked → drainage ↓
  - ↓
    - Severe
    - Quick
    - painful increase in pressure
- It is an emergency condition
- Symptoms → . severe pain , . Nausea , Blurred vision.

Treatment →

- By ↓ I.O.P (Intraocular pressure)
  - ↓ production of aq. humor
  - ↑ drainage of aq. humor

i) α- agonist ⇒ ↓ I.O.P by increasing the uveoscleral outflow.
↳ ↑ drainage of aq. humor.

eg. Brimonidine, Apraclonidine, Dipivefrine etc..

ii) β- blocker ⇒ ↓ I.O.P by decreasing the formation of aq. humor.

eg. Timolol, Betaxolol, Levobunolol etc...

iii) Prostaglandin (P.G.) analogues ⇒ same as α-agonist
↓ I.O.P by ↑ uveoscleral outflow.

eg. Latanoprost, Travoprost, Bimatoprost etc....

iv) Carbonic anhydrase inhibitors ⇒ used orally
↓ aqueous formation by ↓ bicarbonate ion in cilliary epithelium.

eg. Acetazolamide, Dorzolamide etc...

v) Miotic agent ⇒ ↓ I.O.P by increasing ciliary muscle tone. (used rarely for glaucoma).

eg. Pilocarpine etc..

COMPLETE

THANK YOU