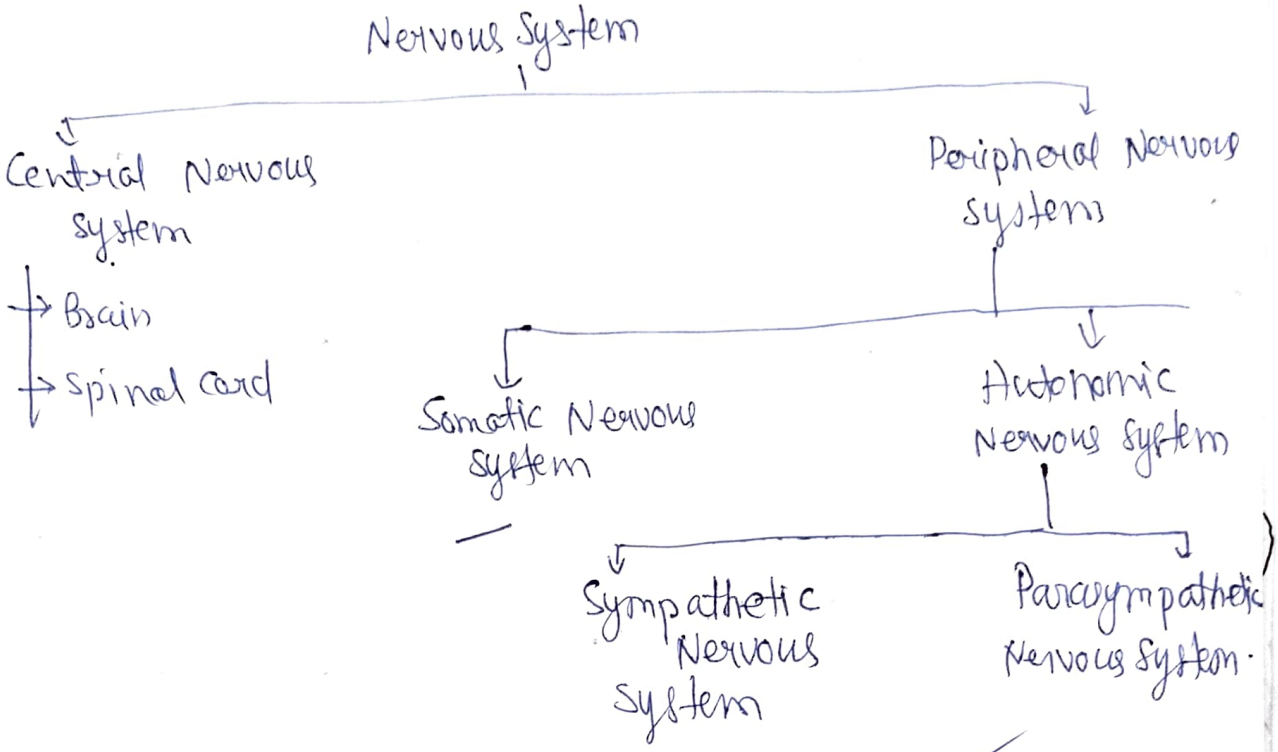
Unit-III Pharmacology of drugs acting on P.N.S.



Organization and function of ANS-

ANS- It involves involuntary responses (movement) of our body.

- It further divided into two parts -

(1) Sympathetic N.S.

(2) Parasympathetic N.S.

(1) Sympathetic Nervous System - (Adrenaline)
↓ fight / flight situation (Abnormal)

- Activate in condition of 3F — Fear
— fight
— flight

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

eg: increase heart rate, decrease digestion rate etc.

② Parasympathetic Nervous System – (Acetylecholine)
– Rest & Digest condition.

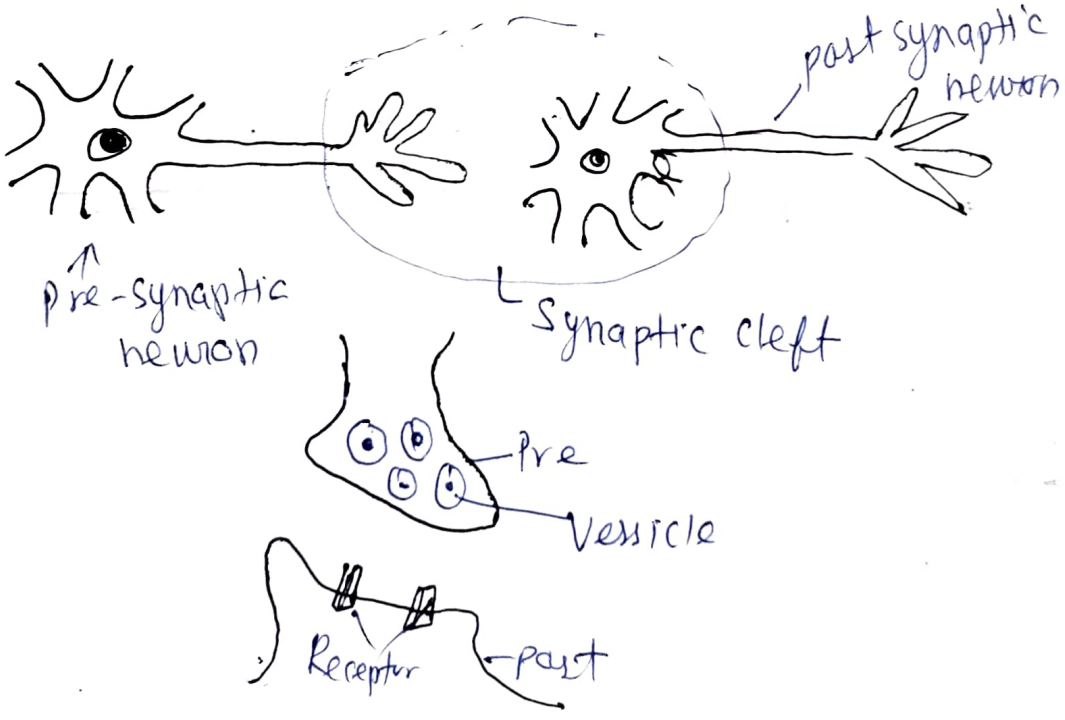
- In this our body come back to normal condition after any abnormal situation.
- Also help to maintain the homeostasis of body.

ex – increase digestion rate and normal heart rate etc.

Neurohumoral Transmission

Neurohumoral → Neuro → Nerve/Neuron
→ Humoral → chemical (messengers)

⇒ It is the process of tranfer of any message or signal from one neuron to another neuron with the help of any chemical messangers (neurotransmitter, hormones).



⇒ For this purpose, firstly neurotransmitter is synthesized and stored in vesicles in nerve terminals.

- Now, Neurohumoral transmission involves following steps –

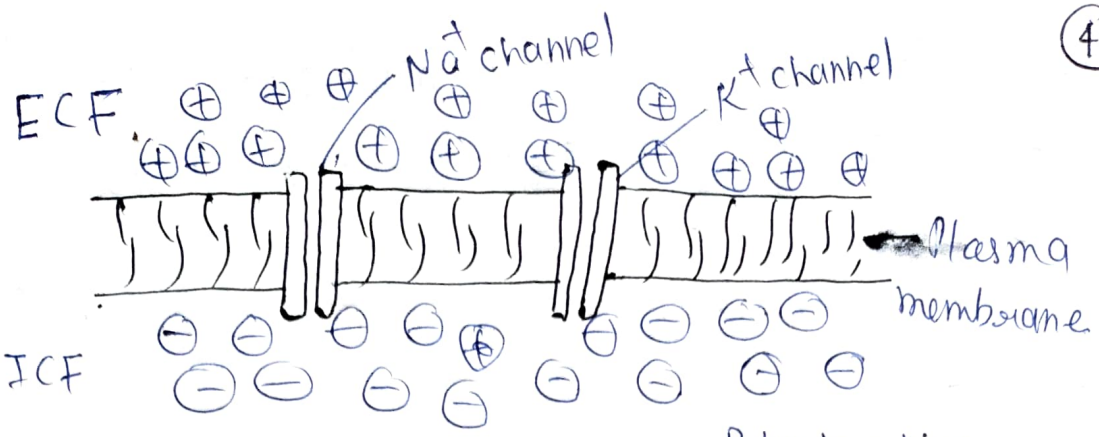
(i) Impulse conduction
(ii) Transmitter release
(iii) Transmitter action on post-junctional membrane
(iv) Post juctional activity –
(v) Termination of transmitter action.

(i) Impulse Conduction –

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

⇒ At resting state (when nerve impulse is not transmitted from neuron), Resting transmembrane potential is −70 mV.

- Na^+ ion have high concentration at outside the cell and move +ve charge at outside the plasma membrane.
- K^+ ion have high conc^n at inside the cell and move −ve charge at inside the plasma membrane.



Polarization

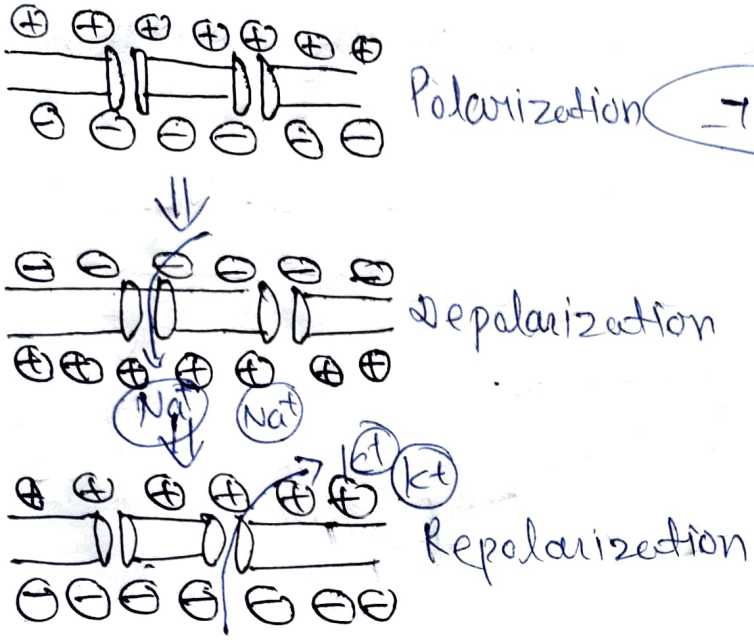
- Resting membrane potential → -70 mV.

- Depolarization – When any kind of stimulus detected then it changes the Resting membrane potential to less potential (increase)
- If stimulus changes resting potential (-70mV) to (-55 mV) then it is called threshold potential
- Threshold potential open Na^+ ion channel, so Na^+ ion enters inside the cell and -ve at outside the cell and it is called as depolarization.
- Repolarization – Stimulus continues increase the potential. now when potential reach at (+20mV to +30 mV) it open K^+ ion channel and K^+ move outside the cells.

⇒ The ionic distribution is normalized during the refractory period by the activation of Na^+-K^+ pump.

★ The cycle of depolarization and repolarization is called Action potential.

- These action potential works 100 times in one second.



Stimulus → Resting membrane potential (-70 mV)

↓

Threshold potential (-55 mV)

↓

Na^+ channel open

↓

Influx of Na^+ ion

↓

Depolarization

↓

K^+ channel open

↓

Outflux of K^+ ion

↑

Repolarization

↑

Normalized

↑

Nerve impulse

(ii) Transmitter release –

The transmitter (excitatory or inhibitory) is stored in prejunctional nerve ending within 'synaptic vesicles'.

Nerve impulse promotes fusion of vesicular and axonal membranes through Ca^{2+} entry which fluidizes membranes.

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

(iii) Transmitter action on postjuctional membrane –

The transmitter release and attached with specific receptor on postjuctional 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 depolarization followed by K^+ efflux
↓
Nerve impulse, contraction in muscle, secretion in glands.

IPSP
(Inhibitory post synaptic potential)
↓
If inhibitory neurotransmitter act increase in permeability to small ion or anions. K^+ & Cl^- moves in, resulting hyperpolarization
↓
Resist depolarization stimulus

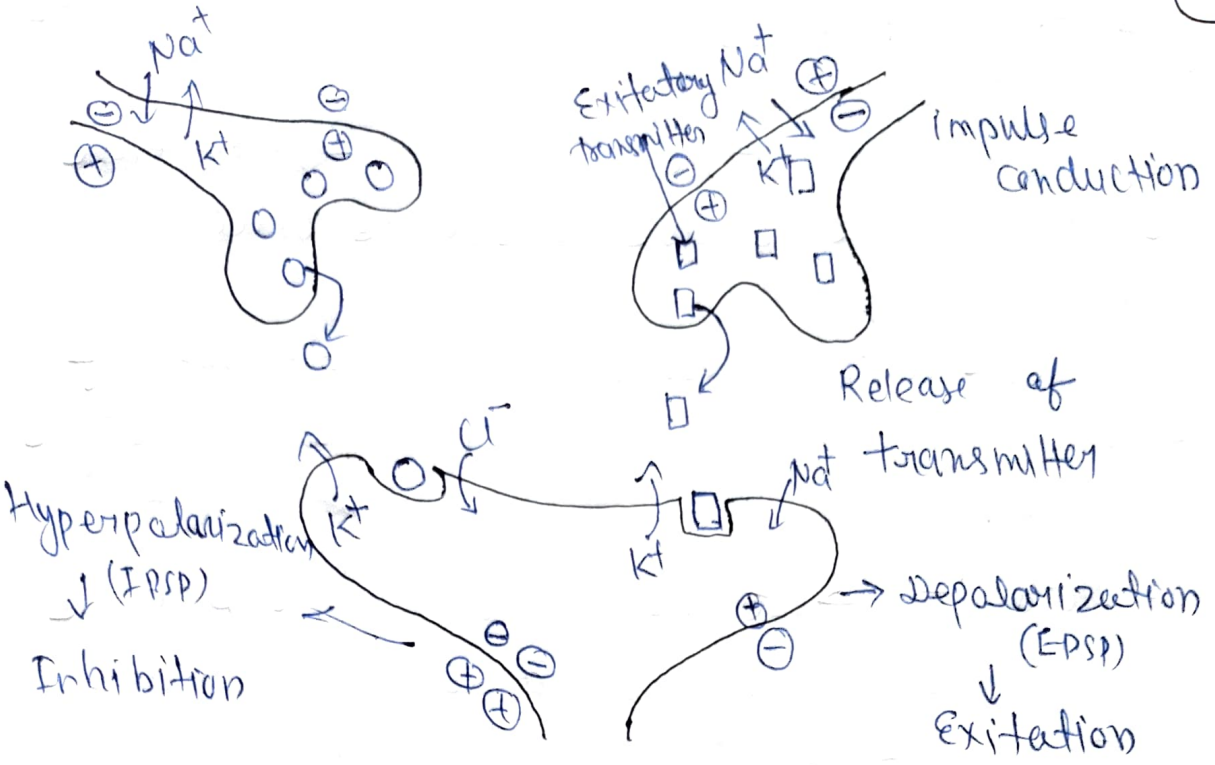


Fig- Diagrammatic representation of steps in excitatory and inhibitory nemohumoral transmission.

(iv) Postjunctional activity-

A superthreshold EPSP generates a ~~progated~~ propagated postjunctional activity which result in nerve impulse contraction (in muscle) or secretion (in gland).

- An IPSP stabilizes the postjunctional membrane and resist depolarization stimuli.

(v) Termination of transmitter action-

Neurotransmitter is degraded locally or any other mechanisms.

- It can also be degraded by enzymatic action.

eg- Acetylcholine degraded by Cholinsterase.

Co-transmission—

- Peripheral and central nervous system release more than one active substance when stimulated.

→ Definition → Co-transmission 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.

Eg - In autonomic nervous system

⇒ Primary neurotransmitter → Ach, NA

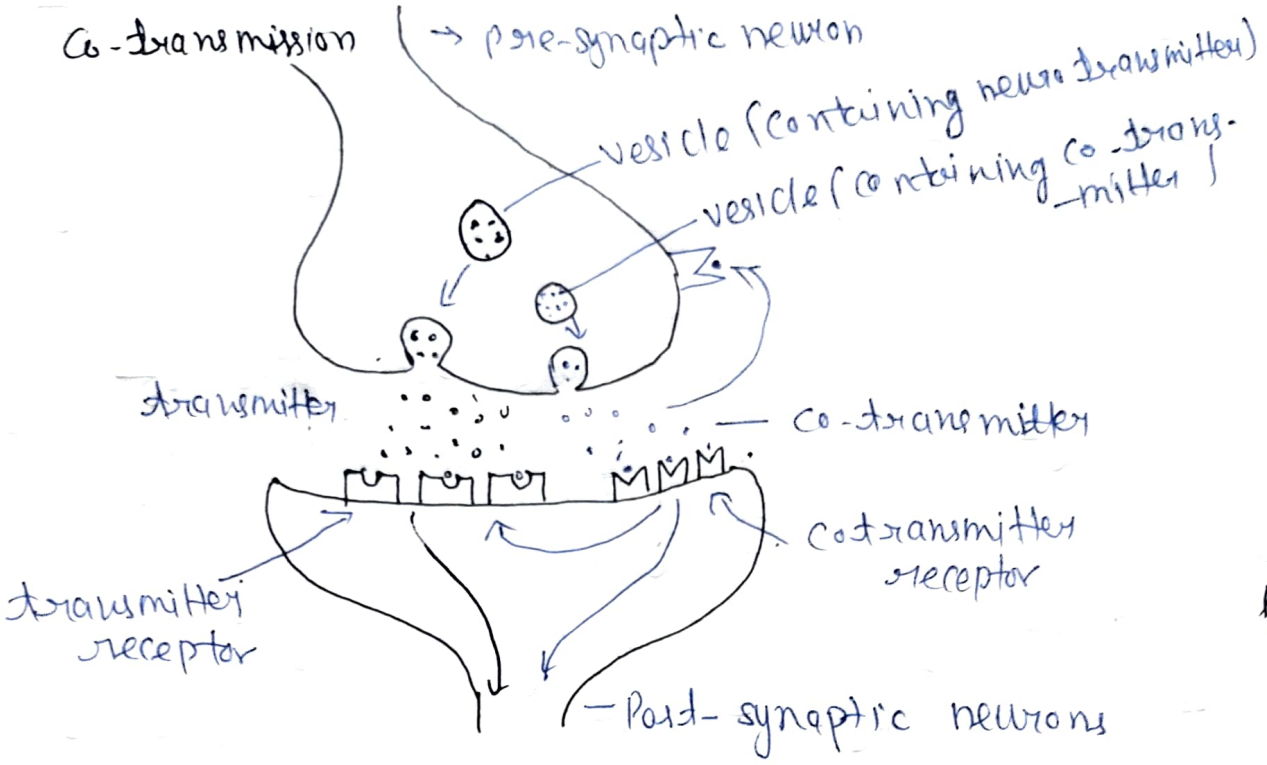
- Co-transmitter are → Purines — ATP, Adrenosine
  - Peptides → Vaso intestinal peptide (VIP)
  - Nitric oxide
  - Prostaglandings (PG)

★ On release of Acetylcholine (Ach), Glutamate, Vasoactive intestinal peptide (VIP), Co-transmitter release.

⇒ Co-transmitter is stored with primary transmitter vesicle or in different (separate) vesicle.

- Function - (i) enhance or regulate neurotransmitter
(ii) modulate post-synaptic sensitivity of primary neurotransmitter

(iii) Serve as a alternate to primary neuro-transmitter.



Classification of Neurotransmitter –

- **Neurotransmitter** – These are chemical messenger that transmit signal from a neuron to a target cell across a synapse.
- target cells may be a othe 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 inhibitatory.

- Excitatory - Active 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 -

Neurotransmitters

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 through, learning and memory. It activate muscle contraction in the body and is also associated with attention and awekening.
- Adrenaline - (flight or fight) - 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 heightened awareness

• Nor-adrenaline (concentration) - It improve attention and responding action in the brain.
- contracts blood vessels ~~its~~ increasing and vessels.

• Dopamine (pleasure) - feeling of pleasure, also addition, movement and motivational.
- people's repeat behavious lead to dopamine release.

• Serotonin (mood) - contributes to well beings and happiness.
- Helps sleep cycle and digestive 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.
- involves 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.

⇒ Drug 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] → Sympathetic Nervous system
→ Cholinergic drugs [Parasympathomimetics] → Parasympathetic Nervous system
→ Anti-cholinergic drugs [Parasympatholytics] → Parasympathetic Nervous system

Cholinergic Drugs

Parasympathomimetics

→ Parasympatho ⇒ Parasympathetic Nervous system
→ mimetic (mimic) → copy the action.

These are drugs which produce similar action to that of Ach, either by directly interacting with cholinergic receptors (cholinergic agonists) or by increasing availability of Ach at these sites.

* Cholinergic receptor → muscarinic (M) & Nicotinic (N)

Pharmacological Action

A. Muscarinic Actions (M2 receptor)

① Heart- Ach hyperpolarizes the SA ~~node~~ nodal cells and decreases their rate of diastolic depolarization.
As a result, rate of impulse generation is reduced- bradycardia occur.

② Eye- contraction of circular muscle of iris causes miosis.
- contraction of ciliary muscle causing spasm of accommodation, increased aquous outflow facility.

③ Glands- Secretion of glands is increased e.g. Sweating, salivation, lacrimation, increased tracheo-bronchial and gastric secretion.
- not effect on ~~pancr~~ pancreatic & intestinal gland
- not effect on secretion of milk and bile juice.

④ Blood vessels- All blood vessels are dilated.
→ Fall in BP. M_3 receptor are present on vascular endothelial cells.

B. Nicotinic Action-

① Sketetal muscles- Ach on the muscle end plate cause contraction of the fibre.

② Autonomic ganglia- Both sympathetic & parasympathetic ganglia are stimulated. High dose of Ach given after atropine cause tachycardia and rise in BP.

© CNS Action :-

Ach injected i.v. does not penetrate blood-brain-barrier and no central effects are seen.

Therapeutic uses -

- Glucoma (physostigmine, pilocarpine)
- Myasthenia gravis (Neostigmine)
- Paralytic ileus and post operative urine retention (Bethanichal, Carbachal ..
- Atropin poisoning (Physostigmine)
- Curare poisoning - neuromuscular junction block (Neostigmine)
- Alzheimer's disease (Cholinesterase inhibitor)

Adverse effects .

- Nausea
- Vomiting
- Bradycardia / Hypotension
- Cause Asthma.

Parasympatholytics – (Anti-cholinergic Drugs)

Parasympatho + lytics

Parasympathetic Nervous system

lysis (breakdown)

oppose the effect.

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

Antichalinergic Drugs
(Muscarin antagonists, Atropinic drugs, Parasympatholytics)

Natural Alkaloids
- Atropine
- & Hyoscine

Semi-synthetic derivatives
- Atropine methonitrate
- Homatropine
- Hyoscine butyl bromide
- Ipratropium bromide
- Tiotropium bromide.

Synthetic Compounds
- Vesicoselective
  - Oxybutynin
  - Flavoxate
  - Tolterodine
  - Darifenacin
  - Solifenacin

Anti-secretory-anti-spasmodics
- Quaternary Comp.
  - Propantheline
  - Oxyphenonium
  - Chiclinium
  - Isopropamide
  - Glycopyrrolate
- Tertiary Comp. Amine
  - Dicyclomine
  - Valethamate
  - Pirenzepine

Mydriatics
- Cyclopentolate
- Tropicamide

Antiperkinson's
- Trihexyphenidyl
- Procyclidine
- Biperiden.

Pharmacological Action (Atropine)

(i) **CNS** - Atropine stimulates many medullary centres - vagal, respiratory, vasomotor.
- It depresses vestibular excitation and has anti-motion sickness property.

(ii) **Heart** - Causes tachycardia on the heart, due to blockade of M_2 receptors on the SA node.

(iii) **Eye** - the autonomic control of iris muscles and action of mydriatics as well as miotics.

(iv) **Smooth muscles** - All visceral smooth muscles that receive parasympathetics motor innervation are relaxed by atropine (m_3 receptor).

(v) **Glands** - Atropine markedly decreases sweat, salivary, tracheobronchial and lacrimal secretion

Use — (I) As antisecretory
- (a) Preanaesthetic medication
- (b) Peptic ulcer
- (c) Pulmonary embolism

(II) As antispasmodic

(III) Bronchial asthma, asthmatic bronchitis, COPD.

(IV) As mydriatic and cycloplegic

(v) For central action -

① Parkinsonism ② motion sickness.

⇒ Adverse effects -

- Dry mouth, difficulty in swallowing and talking.
- Dry, flushed and hot skin (face & necks), fever, difficulty in micturition, decreased bowel sounds.

Adrenergic System (Sympathomimetic drugs)

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

⇒ These drugs bind with adrenergic receptors (α & β) and give their action.

Adrenergic Agonists

- **Direct-acting**
  - Nor-epinephrine
  - Epinephrine
  - Phenylephrine
  - Dopamine
  - Naphazoline
  - methyldopa
  - oxymetazaline
  - Clonidine
  - Xylometazoline
  - Dobutamine
  - Isoproterenol
  - Terbutaline
  - Salbutamol
  - Bitolterol
- **Indirect-Acting**
  - Hydroxyamphetamine
  - Pseudoephrine
  - Propylhexedine
- **Mixed**
  - Ephedrine
  - metaraminol.

Pharmacological Action

① **Heart-** · increase heart rate.
· Its increasing the slope of diastolic depolarization of SA node.

② **Blood vessel-** Both vasoconstriction and vasodilation can occur depending on the drug, its dose and vascular bed.

③ **Respiration-** Adrenaline (Adr) and isoprenaline are potent bronchodilators.

④ **Eye-** Mydriasis occurs due to contraction of radial muscles of iris.

⑤ **Urinary tract-**
· relaxation of urinary bladder (β_2) and closure of sphincture (α_1).

⇛ Therapeutic effects-

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

Adverse effect-

- Restlessness, headache, anxiety, tremor, palpitation, BP ↑,

Those drugs which produce the opposite effect of sympathomimetic system.

also known as — Anti-adrenergic drugs
- Adrenergic Antagonists
- Adrenergic blockers.

Pharmacological Action:

α- Receptor Antagonists

- **Non-selective**
  - Ergotamine
  - Ergotoxine
  - Dihydroergotamine
  - Dihydroergotoxine
  - Phentolamine
  - Chlorpromazine
- **α_1-selective**
  - Prazosin
  - Terazosin
  - Doxazosin
  - Alfuzosin
  - Tamsulosin
  - Silodosin
- **α_2-selective**
  - Yohimbine

Pharmacological Action

α - Receptor Antagonists-

1. B.P - cardiac output reduce → fall in BP.
2. Nose — Nasal stuffiness occures due to blockadge of α receptors in nasal blood vessel.

Sympatholytic Agents (Anti-Adrenergic drugs) (57)

Those drugs which produce the opposite effect of sympathomimetic system.

also known as – Anti-adrenergic drugs
- Adrenergic Antagonist
- Adrenergic blockers.

Pharmacological Action :-

α- Receptor Antagonists

- **Non-selective**
  - Ergotamine
  - Ergotoxine
  - Dihydroergotamine
  - Dihydroergotoxine
  - Phentolamine
  - Chlorpromazine
- **α_1-selective**
  - Prazosin
  - Terazosin
  - Doxazosin
  - Alfuzosin
  - Tamsulosin
  - Silodosin
- **α_2-selective**
  - Yohimbine

Pharmacological Action

α - Receptor Antagonists –

(1) B.P - cardiac output reduce → fall in BP.

(2) Nose – Nasal stuffiness occures due to blockadge of α receptors in nasal blood vessel.

③ GIT- loose motion may occur due to increased intestinal motility by partial inhibition of relaxant sympathetic influences.

④ Kidney- reduce renal blood flow.

Use

- Hypertension (α_1)
- Congestive heart failure (α & β)
- Pheochromocytoma → catechalamine secreting ↑ (α_1 & α_2)

Adverse effects

① Cardiovascular side effects - ↓ → hypotension, orthostatic, heart palpitations.

② Genitourinary side effect ↑ urination
- edema

③ weakness, fatigue, etc psychiatric depression and dry mouth.

⇒ β- Receptor Antagonist

Non-selective (β_1 & β_2)
- Propranolol
- ~~Methylpropranolol~~
- ~~Atenolol~~
- ~~Betazedol~~
- Sotalol
- Timolol
- Pindalol
- Labetalol
- carvedilol

β_1- selective
- metoprolol
- Atenolol
- Acebutolol
- Bisoprolol
- Esmolol
- Betaxalol

Pharmacological Action

① **Heart** - Propanolol decreases force of contraction, heart rate and cardiac output at higher doses.

② **Blood vessels** - Propanolol may increase B.P.

③ **Skeletal muscle** - It decrease exercise by β_2 mediated increase in blood flow to the exercising muscles.

Use - Hypertension
- Thyrotoxicosis
- migraine
- Cardiac arrhythmias
- Angina pectoris
- Congestive heart failure.

Neuromuscular blocking Agents

Those 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.

Peripherally Acting Muscle Relaxants

(Skeletal Muscle relaxants)

Neuromuscular blocking agents

Nondepolarizing (Competitive) blocker

Long acting

- d-Tubocurarine
- Pancuronium
- Doxacurium
- Pipecuronium

Intermediate acting

- → Vecuronium
- → Atracurium
- → Cisatracurium
- → Rocuronium
- → Rapacuronium

Short acting

- → Mivacurium

Depolarizing blockers

- → Succinyl choline (Suxamethonium)
- ⇒ Decamethonium

Direct Acting agents

- → Dantrolena sodium ~~Quinine~~
- → Quinine

1. Non-Depolarizing Blockers-

Mechanism of Action-

The site of action of both non depolarizing and depolarizing blockers is the end plate of skeletal muscle ~~fif~~ fibres

⇓

The competitive blockers bind to the nicotinic (Nm) cholinergic receptors at the muscle end plate

⇓

but have no intrinsic activity.

⇓

The Nm receptor have 5 subunits ($\alpha 2, \beta, \varepsilon$ or γ and δ) which are arranged like a rosette surrounding the Na^+ channel

⇓

The two α subunits carry two Ach-binding sites; Combine with the Ach ! Opening of Na^+ channel

⇓

Antagonist molecule does not allow conformational changes in the subunits needed for opening the channel

⇓

Ach released from motor nerve ending is not able to combine with its receptors to generate end plate potential (EPP).

⇓

thus reduces the frequency of channel opening

⇓

magnitude of EPP falls below a critical level, it is unable to trigger propagated muscle action potential (MAP)

⇓

muscle fails to contract in response to nerve impulse.

② **Depolarizing Blockers -**

SCh and other depolarizing drugs have affinity to bind the Nm cholinoreceptors.

⇓

They depolarize muscle end plates by opening Na^+ channel by entry of Na^+

⇓

Initially produce twitching and fasciculations

⇓

These drugs do not dissociate rapidly from the receptor and are not hydrolysed by AchE.

⇓

They induce prolonged partial depolarization of the region around muscle ~~en~~ end plate

⇓

Na^+ channels get inactivated and potential is drops to about -50 mV.

⇓

Ach released from motor nerve ending is unable to generate propagated muscle action potential

⇓

Flaccid paralysis in the end muscle plate.

Pharmacological Action :-

① Skeletal muscle
- induced flaccid paralysis.
- paralyse acc. to this order -

  muscle of face → eye → finger → limb → neck.
- recovery occur in reverce order.

② Histamine release -

d-TC stimulate release of histamine from mast cells.

⇒ increased respiratory secretion.

⇒ Heparin may also be simultaneously released from mast cells.

③ Autonomic ganglia - Competitive neuromuscular blockers produce some degree of ganglionic blockade due to Nn receptor present there. d-TC produces maximum effect.

⇒ Side effect -

- Hypoxia
- Respiratory paralysis
- Hypotension
- Constipation
- ~~Ca~~ Tachycardia etc.

Local Anaesthetics

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.

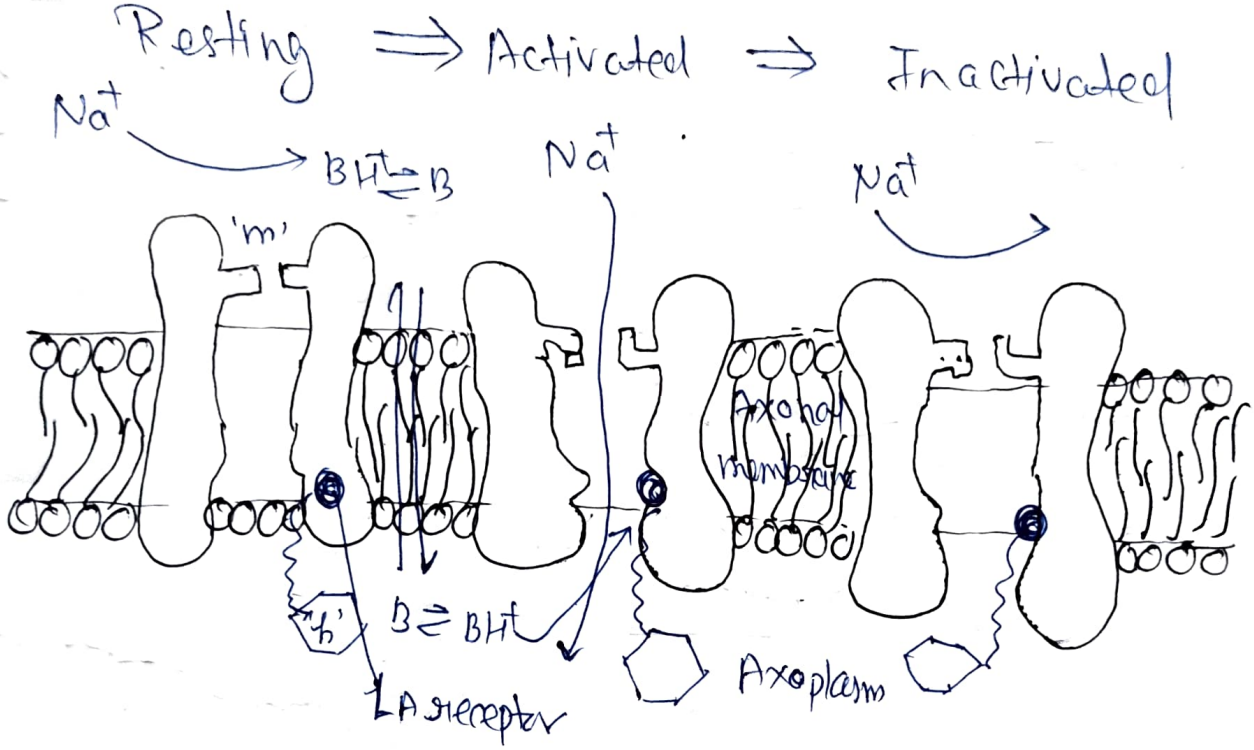
Local Anaesthetics

- **Injectable anaesthetic**
  - Low potency, short duration
    - Procaine
    - Chloroprocain
  - Intermediate potency and duration
    - Licocain
    - Prilocain
  - High potency long duration
    - Tetracaine
    - Bupivacaine
    - Ropivacaine
    - Dibucaine
- **Surface anaesthetic**
  - Soluble
    - Cocaine
    - Licocaine
    - Tetracaine
    - Propacaine
  - Insoluble
    - Benzocaine
    - Butylamino benzoate
    - Oxetha- -zaine

Mechanism of Action :-

Local anesthetics act at the cell membrane to prevent the generation and the conduction of nerve impulse. This action of local anaesthetics is due to their direct interaction with voltage-gated Na^+ - channels.

⇒ As the anaesthetic action progressively develops in the a nerve, the threshold for electrical excitability gradually increases, the rate of rise of the action potential decline, impulse Local anaesthetics block conduction by decreasing or preventing the large transient increase in the permeability of excitable membrane to Na^+ that normally is produced by a slight depolarization of membrane.



~~Acal~~ Local Action

LAs produce minimal local irritant action and block sensory nerve endings, nerve trunks, & neuromuscular junction, ganglionic synaps and receptors. LAs reduce release of acetylcholine from motor nerve endings.

Systemic Action

① Central Nervous System - Local anaesthetics may cause CNS stimulation, producing restlessness and tremor that may progress to clonic convulsion.

⇒ Cocaine is a powerful CNS stimulant causing in sequence euphoria → excitement → mental confusion restlessness → tremor and twitching of muscle → convulsion → unconsciousness → respiratory depression → death in a dose-dependent manner.

② Heart - decrease → electrical excitability
→ conduction rate
→ force of conduction or contraction

Adverse effect

→ Hypersensitivity reaction.
→ Cardiovascular ~~asthma~~ toxicity.
→ bradycardia, hypotension, cardiac arrhythmias
- light - headedness, mental confusion, ~~sat~~ respiratory arrest.

Drugs used in myasthenia gravis & glaucoma

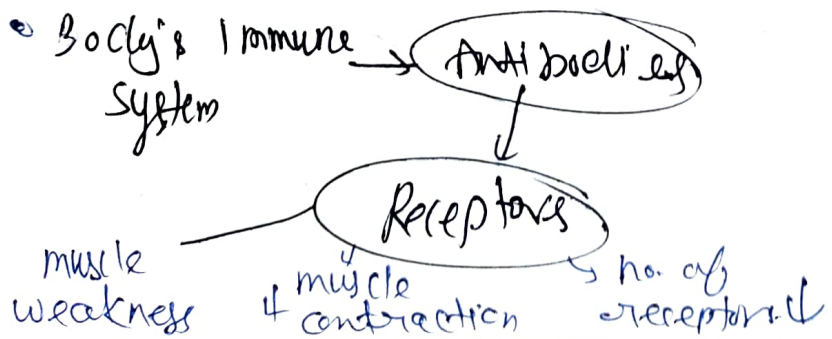
Myasthenia gravis-

It is an autoimmune disorder.

- In this disorder, our immune system produce antibodies to block/destroy the Nicotinic receptor
- Because, According 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 does not bind on receptor
- due to this, these are loss of communication b/w nerve and muscle.
- Also further decrease the contraction of muscle
- ~~these antibodies~~

→ Also muscle become weak & fatigue.

- these antibodies also destroy or kill the receptor, Due to this there are also decrease in the no. of receptor.



Treatment → Drugs used

① Anti chalinesterase –

use these drug to treat myasthenia gravis.

Anti chalinesterase drugs → inhibits → Chalinesterase enzyme

→ Cholinesterease inhibit the ~~co~~ acetylcholine by hydrolysis.

– So, when these drugs inhibit this enzyme concentration of Ach increases.

• By ↑ ach release → can replace antibodies.

eg – Pyridostigmine, Neostigmine etc.

② Immunosuppresant –

use these drug to suppress the immune system to decrease the formation of antibodies.

eg Cyclosporine A, Methotrexate, Azathioprine, cyclophosphamide etc.

③ Carticosteroids –

→ Decrease Antibodies

– Increase synthesis of nicotinic receptor

④ Plasmapheresis - (plasma exchange) -

- It is a technique used to treat myasthenia gravis.

⇒ the plasma of the blood is exchange with substitute plasma, so Antibodies 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 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 (inter ocular pressure).

⇒ Intra-ocular pressure is more than 21 mmHg

two main reason -

① ↑ production of aqueous humorous

② ↓ drainage of aqueous humorous

↓

Loss of vision (blindness) ← optic nerve damage ← eye pain

mid dilated pupil

Symptoms -

- Eye pain. → redness of the eye
- mid dilated pupil ⇒ 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 (eg alcohal)
- excessive use of steroids.

⇒ Diagnosis:

- Dilated eye examination.

Type-

① Open angle glaucoma
② Closed angle glaucoma

① Open angle glaucoma -

- Also known as chronic wide angle glaucoma

Symptoms -

- gradual vision less
- optic nerve damage
- most common type of glaucoma

② Closed Angle Glaucoma -

- Also known as acute and narrow angle glaucoma
- flow of Aqueous humour blocked
- It is an emergency condition.

Symptoms- severe pain, Nausea, blurred vision.

Treatment -

By ↓ I.O.P (Intraocular pressure)

↓ production of aq. humour ↑ drainage of aq. humour.

① α - Adrenergic agonists - ↓ IOP by increasing the uveoscleral outflow

↑ drainage of aq. humor.

eg. Brimonidine, Apraclonidine, Dipivefrine etc

② β - blocker - ↓ I.O.P. by decreasing the formation of aq. humor.

eg. Timolol, Betaxolol, Levobumolol etc

③ Prostaglandin analogues - Same as α - agonist

↓ I.O.P. by ↑ uveoscleral outflow.

eg - Latanoprost, Travoprost, Bimatoprost etc,

④ Cardiac anhydrase inhibitors.

used orally. ↓ aqueous formation by ↓ bicarbonate ion in ciliary epithelium.

eg - Acetazolamide, Dorzolamide etc.

⑤ Miotics Agent – ↓ I.O.P. by increasing ciliary muscle tone (used rarely for glaucoma).

eg - Pilocarpine etc.