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PHARMACOLOGY - I

UNIT 3

TOPIC :

- **Pharmacology of drugs acting on peripheral nervous system**
  a. Organization and function of ANS.
  b. Neurohumoral transmission, co-transmission and classification of neurotransmitters.
  c. Parasympathomimetics, Parasympatholytics, Sympathomimetics, sympatholytics.
  d. Neuromuscular blocking agents and skeletal muscle relaxants (peripheral).
  e. Local anesthetic agents.
  f. Drugs used in myasthenia gravis and glaucoma

Drugs Acting on the Autonomic Nervous System (ANS)

Drugs acting on the ANS influence the sympathetic and parasympathetic branches, which regulate involuntary functions like:

- Heart rate
- Blood pressure
- Respiratory rate
- Digestion
- Pupil size
- Glandular activity

Nervous System Overview

1. Central Nervous System (CNS)

- Brain and spinal cord

2. Peripheral Nervous System (PNS)

- Nerves outside CNS
- Divided into:
  - **Somatic Nervous System** – voluntary control (skeletal muscles)
  - **Autonomic Nervous System (ANS)** – involuntary control
    - **Sympathetic Nervous System (SNS)** – "fight or flight"
    - **Parasympathetic Nervous System (PNS)** – "rest and digest"

Functional Differences Between SNS And PNS

Organ / Tissue	Sympathetic (SNS)	Parasympathetic (PNS)
Heart	↑ Heart rate	↓ Heart rate
Bronchi / Lungs	Bronchodilation	Bronchoconstriction
GIT	Inhibits digestion	Stimulates digestion
Bladder	Relaxes bladder	Contracts bladder
Pupil	Dilation (Mydriasis)	Constriction (Miosis)
Glands	↑ Secretion (sweat, adrenal)	↑ Secretion (saliva, lacrimal)

SYMPATHETIC NERVOUS SYSTEM (SNS)

Also called: "Fight or Flight" system

Function: Prepares the body to respond to stress, danger, or emergency situations by mobilizing energy and increasing alertness.

Key Functions of SNS

Organ / System	Effect of Sympathetic Activation
Heart	↑ Heart rate & ↑ contractility
Bronchi	Dilation (↑ airflow)
Pupil	Dilation (Mydriasis)
GIT	Inhibition of digestion & ↓ saliva secretion
Liver	Stimulates glucose release (glycogenolysis)
Bladder	Relaxation → urine retention
Blood Vessels	Vasoconstriction → ↑ blood pressure
Sweat Glands	↑ sweating

Neurotransmitters in SNS

Nerve Type	Neurotransmitter
Preganglionic	Acetylcholine (ACh)
Postganglionic	Norepinephrine (NE) / Epinephrine (E)

Parasympathetic Nervous System (PNS)

Also called: "Rest and Digest" system

Function: Maintains normal body functions at rest, conserves energy, and facilitates digestion and excretion.

Key Functions Of PNS

Organ / System	Effect of Parasympathetic Activation
Heart	↓ Heart rate & ↓ contractility
Bronchi	Constriction (↓ airflow)
Pupil	Constriction (Miosis)
GIT	Stimulation of digestion & ↑ saliva secretion
Liver	↑ glycogen synthesis (storage of glucose)
Bladder	Contraction → facilitates urination
Blood Vessels	Mostly no direct effect
Glands	↑ secretion (lacrimal, salivary, gastric, pancreatic)

Neurotransmitters In PNS

Nerve Type	Neurotransmitter
Preganglionic	Acetylcholine (ACh)
Postganglionic	Acetylcholine (ACh)

Difference Between Sympathetic Nervous System (SNS) And Parasympathetic Nervous System (PNS)

Feature	Sympathetic Nervous System (SNS)	Parasympathetic Nervous System (PNS)
Also called	"Fight or Flight" system	"Rest and Digest" system
Main Function	Prepares body for stress, danger, or emergency	Conserves energy, promotes relaxation and routine body maintenance
Heart	↑ Heart rate & ↑ contractility	↓ Heart rate & ↓ contractility
Bronchi / Lungs	Dilation (↑ airflow)	Constriction (↓ airflow)
Pupil	Dilation (Mydriasis)	Constriction (Miosis)
Gastrointestinal Tract	Inhibits digestion	Stimulates digestion & motility
Saliva	↓ Secretion	↑ Secretion
Liver / Glucose	Stimulates glucose release	Promotes glycogen storage
Bladder	Relaxes bladder (urine retention)	Contracts bladder (facilitates urination)
Blood Vessels	Vasoconstriction (↑ BP)	Mostly no direct effect
Glands (Lacrimal, Sweat, etc.)	↑ Sweat secretion	↑ Secretions (lacrimal, salivary, gastric)
Preganglionic Neurotransmitter	Acetylcholine (ACh)	Acetylcholine (ACh)
Postganglionic Neurotransmitter	Norepinephrine / Epinephrine (except sweat glands use ACh)	Acetylcholine (ACh)
Response Speed	Fast, short-term action	Slow, long-term action
Dominant During	Stress, exercise, emergencies	Rest, sleep, digestion

Neurohumoral Transmission

Neurohumoral transmission is the process by which nerve cells (neurons) communicate with each other or with effector organs (muscles, glands, etc.) using chemical messengers called neurotransmitters.

Steps of Neurohumoral Transmission

1. Synthesis of Neurotransmitter

- Neurotransmitters are synthesized either in the cell body or nerve terminal.
- Example: Acetylcholine (ACh) is synthesized from choline + acetyl CoA by choline acetyltransferase.

2. Storage in Vesicles

- Neurotransmitters are stored in synaptic vesicles at the nerve terminal.
- Ready for release upon stimulation.

3. Nerve Impulse (Action Potential)

- Arrival of a nerve impulse at the terminal causes depolarization of the membrane.

4. Influx & Release of Neurotransmitters

- Depolarization opens voltage-gated calcium channels (Ca^{2+}).
- Calcium influx triggers fusion of synaptic vesicles with the membrane.
- Neurotransmitters are released into the synaptic cleft.

5. Receptor Binding

- Neurotransmitters bind to specific receptors on the postsynaptic membrane (next neuron, muscle, or gland).

6. Postsynaptic Response

- Receptor activation initiates a response in the effector cell:
  - Muscle contraction
  - Gland secretion
  - Generation of new nerve impulses

7. Termination of Action

- Neurotransmitter action is terminated by:
  1. **Enzymatic degradation**
     - Example: Acetylcholinesterase breaks down ACh → choline + acetate
  2. **Reuptake into presynaptic neuron**

3. **Diffusion away from synaptic cleft**

CO-TRANSMISSION

Co-transmission refers to the phenomenon where a single neuron releases more than one type of neurotransmitter (classical neurotransmitter + modulatory molecule) at the same synapse or target site. These neurotransmitters can act together to produce a more complex or finely tuned physiological response.

Key Points:

- Not all neurons release a single neurotransmitter; many release **combinations** of neurotransmitters.
- Co-transmitters can include small molecule neurotransmitters (e.g., acetylcholine, norepinephrine) and neuropeptides (e.g., substance P, vasoactive intestinal peptide).
- The released neurotransmitters may act on different receptors on the post-synaptic cell.
- Co-transmission allows modulation of the main neurotransmitter's effect or produces additional effects.

Classification of Neurotransmitters

- Neurotransmitters are chemical messengers released by neurons at synapses to transmit signals to other neurons, muscles, or glands. They bind to specific receptors on the target cell to produce a physiological response.
- Neurotransmitters are classified based on chemical structure, function, or receptor type.

1. Based on Chemical Structure

A. Acetylcholine (ACh)

- Class: Cholinergic neurotransmitter
- Found in: CNS & PNS (Parasympathetic postganglionic neurons)
- Function: Muscle contraction, autonomic regulation, memory

B. Amino Acids

- **Excitatory:** Glutamate, Aspartate → increase neuronal firing
- **Inhibitory:** GABA (gamma-aminobutyric acid), Glycine → decrease neuronal firing

C. Biogenic Amines

- Derived from amino acids like tyrosine or tryptophan

1. **Catecholamines (from Tyrosine):** Dopamine, Norepinephrine, Epinephrine
2. **Indoleamines (from Tryptophan):** Serotonin (5-HT), Melatonin
3. **Histamine:** from Histidine

D. Purines

- ATP, Adenosine → act as neurotransmitters in CNS and autonomic nervous system

E. Peptides (Neuropeptides)

- Substance P, Vasoactive Intestinal Peptide (VIP), Neuropeptide Y, Endorphins, Enkephalins

F. Gases & Lipid-derived Molecules

- Nitric Oxide (NO), Carbon Monoxide (CO), Anandamide (endocannabinoids)

2. Based on Function

A. Excitatory Neurotransmitters

- Promote depolarization of post-synaptic membrane → Action Potential
- Examples: Glutamate, Aspartate, Acetylcholine (at NMJ)

B. Inhibitory Neurotransmitters

- Promote hyperpolarization → decrease neuronal firing
- Examples: GABA, Glycine

3. Based on Site of Action

A. Central Nervous System (CNS) Neurotransmitters

- Glutamate, GABA, Dopamine, Serotonin, Acetylcholine

B. Peripheral Nervous System (PNS) Neurotransmitters

- Acetylcholine (Parasympathetic)
- Norepinephrine/Epinephrine (Sympathetic)

4. Based on Receptor Type

A. Ionotropic Receptors (Ligand-gated channels)

- Fast response, direct ion flow
- Examples: Glutamate (NMDA, AMPA receptors), GABA-A, Nicotinic ACh receptors

B. Metabotropic Receptors (G-protein coupled)

- Slow, modulatory response
- Examples: Dopamine receptors, Muscarinic ACh receptors, Adrenergic receptors

Parasympathomimetics (Cholinomimetics)

Drugs that mimic the action of acetylcholine (ACh) at parasympathetic nerve endings, stimulating muscarinic or nicotinic receptors.

Mechanism of Action:

- Direct-acting: Bind directly to muscarinic or nicotinic receptors.
- Indirect-acting: Inhibit acetylcholinesterase → increase ACh levels.

Examples:

- **Direct-acting:** Pilocarpine, Bethanechol, Carbachol
- **Indirect-acting (AChE inhibitors):** Neostigmine, Physostigmine, Donepezil

Effects:

Organ/System	Effect
Heart	↓ Heart rate (bradycardia)
Eye	Miosis (pupil constriction), ↓ IOP
GIT	↑ Motility and secretion
Urinary Bladder	↑ Contraction → facilitates urination
Respiratory	↑ Bronchial secretions

Therapeutic Uses:

- Glaucoma (Pilocarpine)
- Myasthenia gravis (Neostigmine)
- Postoperative urinary retention (Bethanechol)
- Alzheimer's disease (Donepezil)

Parasympatholytics (Anticholinergics / Muscarinic antagonists)

Drugs that block the action of acetylcholine at parasympathetic receptors.

Mechanism of Action:

- Competitive antagonists at muscarinic receptors (M1–M5)
- Nicotinic antagonists at neuromuscular junction (less common)

Examples:

- Atropine, Scopolamine, Ipratropium, Tropicamide

Effects:

Organ/System	Effect
Heart	↑ Heart rate (tachycardia)
Eye	Mydriasis (pupil dilation), cycloplegia
GIT	↓ Motility and secretions
Urinary Bladder	↓ Contraction → urinary retention
Respiratory	Bronchodilation

Therapeutic Uses:

- Pre-anesthetic medication (Atropine)
- Motion sickness (Scopolamine)
- Asthma/COPD (Ipratropium)
- Ophthalmic examination (Tropicamide)

Sympathomimetics (Adrenergic agonists)

Drugs that mimic the effects of norepinephrine (NE) or epinephrine by activating adrenergic receptors (α and β).

Mechanism of Action:

- **Direct-acting:** Bind directly to α or β receptors (e.g., Epinephrine, Phenylephrine)
- **Indirect-acting:** Increase release or inhibit reuptake of NE (e.g., Amphetamine)

Examples:

- Epinephrine, Norepinephrine, Dopamine, Dobutamine, Phenylephrine, Salbutamol

Effects:

Receptor	Effect
α1	Vasoconstriction → ↑ BP, pupil dilation
α2	↓ NE release (feedback inhibition)
β1	↑ Heart rate, ↑ contractility
β2	Bronchodilation, ↑ glycogenolysis
Dopamine	↑ Renal perfusion (low dose), ↑ cardiac output (moderate dose)

Therapeutic Uses:

- Shock and hypotension (Epinephrine, Norepinephrine)
- Asthma (Salbutamol)
- Heart failure (Dobutamine)
- Nasal decongestion (Phenylephrine)

Sympatholytics (Adrenergic antagonists / blockers)

Drugs that block adrenergic receptors, inhibiting sympathetic activity.

Mechanism of Action:

- **α-blockers:** Block α1 or α2 receptors
- **β-blockers:** Block β1 or β2 receptors

Examples:

- α-blockers: Prazosin, Doxazosin
- β-blockers: Propranolol, Metoprolol, Atenolol

Effects:

Receptor	Effect
α1	↓ Vasoconstriction → ↓ BP
α2	↑ NE release (rare effect)
β1	↓ Heart rate and contractility
β2	Bronchoconstriction, ↓ glycogenolysis

Therapeutic Uses:

- Hypertension (Prazosin, Metoprolol)
- Angina pectoris (β-blockers)
- Heart failure (Carvedilol)
- Benign prostatic hyperplasia (α1-blockers)

Neuromuscular Blocking Agents (NMBAs)

Drugs that interfere with transmission at the NMJ, preventing acetylcholine (ACh) from activating skeletal muscle contraction.

Mechanism of Action:

- **Non-depolarizing (Competitive) blockers:**
  - Bind to nicotinic ACh receptors at NMJ, blocking ACh without activating the receptor.
  - Muscle cannot depolarize → flaccid paralysis.
  - Reversed by AChE inhibitors (e.g., Neostigmine).
- **Depolarizing blockers:**
  - Bind to ACh receptor and depolarize the muscle (like ACh).
  - Initial fasciculations → sustained depolarization → paralysis.
  - Not reversed by AChE inhibitors; duration limited by plasma metabolism (e.g., Succinylcholine).

Examples:

Type	Drugs	Duration/Notes
Non-depolarizing	Tubocurarine, Atracurium, Vecuronium, Rocuronium	Reversible by Neostigmine
Depolarizing	Succinylcholine	Rapid onset, short duration

Effects:

- Skeletal muscle relaxation: face, limbs, abdomen, respiratory muscles.
- **No effect** on cardiac muscle or consciousness.

Therapeutic Uses:

- Muscle relaxation during **surgery** or **intubation**
- Facilitate **mechanical ventilation**

Adverse Effects:

- Hypotension, tachycardia (esp. Tubocurarine)
- Histamine release → bronchospasm
- Hyperkalemia (Succinylcholine)
- Malignant hyperthermia (rare, with Succinylcholine)

Peripheral (Direct-Acting) Skeletal Muscle Relaxants

Drugs that act directly on skeletal muscle or indirectly at NMJ to reduce muscle tone and relieve spasticity.

Mechanism of Action:

- **Direct-acting:** Act on skeletal muscle fibers, interfering with excitation-contraction coupling.
- **Indirect-acting:** Reduce acetylcholine release or block nicotinic receptor activity.

Examples:

Drug	Mechanism	Use
Dantrolene	Inhibits Ca^{2+} release from sarcoplasmic reticulum	Spasticity, Malignant hyperthermia
Baclofen	GABA_B agonist → inhibits spinal reflexes	Spasticity from spinal cord injury
Diazepam	GABA_A agonist → CNS mediated muscle relaxation	Spasticity, anxiety
Tizanidine	α2-adrenergic agonist → inhibits spinal motor neurons	Spasticity

Effects:

- ↓ Muscle tone and spasticity
- ↓ reflex activity (for CNS-acting agents)
- Preserve consciousness

Therapeutic Uses:

- Spasticity in **cerebral palsy, multiple sclerosis, stroke**
- Malignant hyperthermia (Dantrolene)
- Muscle spasms from **injury**

Adverse Effects:

- Muscle weakness
- Sedation (esp. Baclofen, Diazepam)
- Hypotension (Tizanidine)
- Hepatotoxicity (Dantrolene)

Comparison Table: NMBAs vs Peripheral Muscle Relaxants

Feature	Neuromuscular Blocking Agents	Peripheral/Skeletal Muscle Relaxants
Site of Action	NMJ (pre/post-synaptic nicotinic receptors)	Skeletal muscle fibers or spinal neurons
Mechanism	Non-depolarizing: block ACh; Depolarizing: depolarize & desensitize	Interfere with Ca^{2+} release, inhibit spinal reflexes
Effect	Complete paralysis (surgery, intubation)	Reduce muscle tone/spasticity
Onset	Rapid (min)	Slower (hrs for oral agents)
Reversal	Non-depolarizing: AChE inhibitors	Usually not reversible
Examples	Succinylcholine, Vecuronium, Atracurium	Dantrolene, Baclofen, Tizanidine, Diazepam

LOCAL ANESTHETIC AGENTS (LAs)

Local anesthetics are drugs that **temporarily block sensation of pain** in a specific part of the body **without affecting consciousness**.

- They act by **blocking nerve conduction**, preventing pain signals from reaching the brain.
- Commonly used in **minor surgical procedures, dental treatments, and localized pain management**.

Mechanism of Action

- Local anesthetics **block voltage-gated sodium channels** in neuronal membranes.
- This prevents **depolarization** of the nerve fiber → **action potential cannot propagate → pain sensation is blocked**.
- Effect is **reversible** after the drug is metabolized or diffuses away.

Classification of Local Anesthetics

1. Based on Route of Administration

Type	Description	Examples
Injectable	Administered directly into tissue, nerve, or epidural space	Procaine, Lidocaine, Bupivacaine, Ropivacaine
Surface (Topical)	Applied to mucous membranes or skin	Cocaine, Lidocaine, Benzocaine, Tetracaine

2. Based on Chemical Structure

1. **Ester-type LAs** – Hydrolyzed by plasma esterases
    - Examples: Procaine, Chloroprocaine, Tetracaine, Cocaine
2. **Amide-type LAs** – Metabolized in the liver
    - Examples: Lidocaine, Bupivacaine, Ropivacaine, Prilocaine

3. Based on Potency, Duration & Solubility

Drug	Potency	Duration	Notes
Procaine	Low	Short	Ester type
Lidocaine	Medium	Medium	Amide type, widely used
Bupivacaine	High	Long	Amide type, cardiac toxicity risk
Ropivacaine	High	Long	Less cardiotoxic than Bupivacaine
Tetracaine	High	Long	Ester type, topical or spinal
Cocaine	Medium	Medium	Topical use, vasoconstrictor
Prilocaine	Medium	Medium	Amide type
Benzocaine	Low	Short	Topical use, ester type

Properties of Local Anesthetics

- **Lipophilicity:** ↑ Lipid solubility → ↑ potency & duration
- **Protein Binding:** ↑ Protein binding → ↑ duration of action
- **pKa:** Determines **onset**; closer to physiological pH → faster onset
- **Vasodilation/Vasoconstriction:** Most LAs cause vasodilation (shortens duration); epinephrine can be added to prolong action

Clinical Uses

1. Minor surgical procedures (suturing, excision)
2. Dental anesthesia
3. Epidural and spinal anesthesia
4. Topical pain relief (burns, mucosal lesions)

Adverse Effects

- CNS: Dizziness, tinnitus, seizures (especially with Lidocaine overdose)
- Cardiovascular: Hypotension, bradycardia, arrhythmias (esp. Bupivacaine)
- Allergic reactions: Rare, more with ester-type LAs
- Methemoglobinemia: Rare, seen with Prilocaine and Benzocaine

1. Injectable (Parenteral) Anesthetics

- Drugs administered via injection to induce loss of sensation or unconsciousness, used for surgery, procedural sedation, or pain management.

Classification by Potency & Duration

Potency	Examples	Notes
Low potency, short duration	Procaine, Chloroprocaine	Used for short-term induction, rapid onset
Intermediate potency & duration	Lidocaine, Mepivacaine	Moderate strength & duration; commonly used IV
High potency, long duration	Bupivacaine, Ropivacaine, Dibucaine	Prolonged anesthesia or sedation; slow metabolism

Mechanism of Action

- Block voltage-gated Na^+ channels in nerve membranes near the site of injection.
- Prevents action potential propagation, temporarily stopping pain signals from reaching CNS.

Pharmacokinetics

- **Absorption:** Rapid via IV; slower with IM.
- **Distribution:** Rapid to highly perfused organs (brain, heart); redistributed to muscle and fat.
- **Metabolism:** Primarily in liver (amide-type); ester-type by plasma esterases.
- **Excretion:** Renal excretion of metabolites.
- **Duration:** Short (15–60 min) to long (several hours), depending on drug and dose.

Adverse Effects

- Hypotension, bradycardia
- Respiratory depression, apnea
- CNS toxicity: dizziness, seizures, nystagmus

- Arrhythmias (esp. Bupivacaine)

Therapeutic Uses

- Induction & maintenance of anesthesia
- Procedural sedation
- Analgesia (burns, minor surgeries)

2. Surface (Topical) Anesthetics

Drugs applied to skin or mucous membranes to produce localized numbness without systemic anesthesia.

Types

Type	Examples	Notes
Soluble	Cocaine, Lidocaine, Tetracaine, Proparacaine	Rapid action on mucosa; limited systemic absorption
Insoluble	Benzocaine, Butylaminobenzoate	Poorly water-soluble; used only topically; prolonged local effect

Mechanism of Action

- Block sodium channels in nerve endings at the site of application.
- Prevents nerve signal transmission, causing temporary local numbness.

Pharmacokinetics

- **Absorption:** Rapid via mucous membranes; slow via intact skin (insoluble type).
- **Distribution:** Mainly at site of application; limited systemic exposure.
- **Metabolism:** Mostly in liver (for absorbed portion); insoluble types minimally metabolized.
- **Excretion:** Renal excretion of metabolites.
- **Duration:** Short for soluble agents; prolonged for insoluble agents due to slow release from site.

Adverse Effects

- Irritation, burning, tingling at application site
- Allergic reactions, dermatitis
- Rare systemic toxicity with large absorption

Therapeutic Uses

- Topical anesthesia for dentistry, ENT procedures, ophthalmology, burns, teething pain

Drugs Used In Myasthenia Gravis (MG)

Myasthenia Gravis (MG):

A chronic autoimmune neuromuscular disorder causing skeletal muscle weakness and fatigue.

- Caused by autoantibodies that block or destroy acetylcholine (ACh) receptors at the neuromuscular junction.
- Result: Impaired transmission of nerve impulses to muscles → weakness.

Common Symptoms

- Muscle weakness (especially ocular and facial muscles)
- Ptosis (drooping eyelids)
- Diplopia (double vision)
- Dysphagia (difficulty swallowing)
- Neck weakness, head drop
- Fatigue, chewing difficulty

Causes / Risk Factors

- Autoimmunity: Abnormal antibody production
- Genetic factors
- Infections
- Medications (can exacerbate symptoms)
- Thyroid gland abnormalities

Anticholinesterases (Cholinesterase Inhibitors)

Mechanism of Action:

- Inhibit acetylcholinesterase, increasing acetylcholine levels at nerve terminals.
- Enhances neuromuscular transmission, improving muscle contraction.

Examples:

- Pyridostigmine
- Neostigmine
- Physostigmine (less common)

Pharmacokinetics:

- Absorption: Moderate oral bioavailability
- Distribution: Limited CNS penetration (poor blood-brain barrier crossing)
- Metabolism: Partially in liver
- Excretion: Mainly via kidneys

Adverse Effects:

- Muscle cramps
- Diarrhea
- Sweating, salivation
- Bradycardia
- Blurred vision

Therapeutic Uses:

- Myasthenia Gravis
- Urinary retention
- Reversal of neuromuscular blockade
- Ileus
- Prophylaxis against nerve agent poisoning

Immunosuppressants

Mechanism of Action:

- Reduce immune system activity
- Decrease production of autoantibodies targeting ACh receptors

Examples:

- Cyclosporine
- Cyclophosphamide
- Azathioprine

Pharmacokinetics:

- **Absorption:** Well absorbed orally (variable)
- **Distribution:** Widely distributed; some cross BBB
- **Metabolism:** Liver
- **Excretion:** Urine and bile

Adverse Effects:

- Leukopenia, anemia
- Increased infection risk
- Hepatotoxicity, nephrotoxicity
- Hair loss, nausea

Therapeutic Uses:

- Myasthenia Gravis
- Rheumatoid arthritis
- Lupus nephritis
- Multiple sclerosis

Corticosteroids

Mechanism of Action:

- Anti-inflammatory and immunosuppressive
- Reduce autoantibody production and inflammation at neuromuscular junction

Examples:

- Prednisolone
- Hydrocortisone
- Methylprednisolone

Pharmacokinetics:

- **Absorption:** Rapid oral absorption
- **Distribution:** Widely distributed; crosses plasma & BBB
- **Metabolism:** Liver
- **Excretion:** Urine

Adverse Effects:

- Infection risk
- Hyperglycemia
- Hypertension
- Weight gain
- Glaucoma, acne

Therapeutic Uses:

- Myasthenia Gravis
- Asthma
- Allergic conditions
- Autoimmune disorders

Drugs Used in Glaucoma

- **Glaucoma:**
  A group of eye disorders characterized by optic nerve damage, often due to increased intraocular pressure (IOP). Untreated glaucoma can lead to irreversible blindness.

Pathophysiology

- Obstruction of **aqueous humor outflow** → accumulation of fluid
- Increased **intraocular pressure (IOP)**
- Compression of **optic nerve fibers**
- Reduced blood flow to optic nerve → **vision loss**

Causes

- Genetics
- Trauma
- Steroid use
- Surgery
- Myopia
- Tumors

Symptoms

- Blurred vision
- Headache, eye pain
- Tearing, redness
- Vomiting (in acute cases)
- Gradual vision loss

β-Adrenergic Blockers

Mechanism of Action:

- Block β-receptors in ciliary body
- Reduce aqueous humor production → lowers IOP

Examples:

- Timolol
- Betaxolol

Pharmacokinetics:

- Absorption: Rapid via ocular drops
- Distribution: Local ocular distribution
- Metabolism: Partially metabolized in liver
- Excretion: Mainly renal

Adverse Effects:

- Bronchospasm
- Bradycardia, hypotension
- Fatigue, depression
- Dryness

Therapeutic Uses:

- Glaucoma
- Hypertension
- Angina
- Arrhythmias
- Migraine prophylaxis

α-Adrenergic Agonists

Mechanism of Action:

- Activate α-receptors
- Reduce aqueous humor production and increase outflow

Examples:

- Brimonidine
- Apraclonidine

Pharmacokinetics:

- **Absorption:** Well absorbed via ocular drops
- **Distribution:** Widely distributed locally
- **Metabolism:** Mostly liver
- **Excretion:** Renal

Adverse Effects:

- Dizziness, headache
- Hypotension
- Palpitations
- Blurred vision

Therapeutic Uses:

- Glaucoma
- Ocular hypertension
- Refractory cases

Prostaglandin Analogues

Mechanism of Action:

- Mimic prostaglandins
- Increase aqueous humor outflow → reduce IOP

Examples:

- Latanoprost
- Travoprost
- Bimatoprost

Pharmacokinetics:

- **Absorption:** Rapid ocular absorption
- **Distribution:** Local in eye
- **Metabolism:** Converted to active form in eye and liver
- **Excretion:** Renal

Adverse Effects:

- Eyelash growth
- Iris pigmentation
- Blurred vision
- Itching

Therapeutic Uses:

- Glaucoma
- Ocular hypertension
- Cosmetic eyelash growth (hypertrichosis)

Carbonic Anhydrase Inhibitors (CAIs)

Mechanism of Action:

- Inhibit carbonic anhydrase enzyme in ciliary body
- Reduce aqueous humor formation → lower IOP

Examples:

- **Acetazolamide**
- **Dorzolamide**

Pharmacokinetics:

- **Absorption:** Oral or topical
- **Distribution:** Eye and systemic fluids
- **Metabolism:** Minimal
- **Excretion:** Mainly unchanged in urine

Adverse Effects:

- Fatigue
- Kidney stones
- Metallic taste
- Diarrhea, tingling

Therapeutic Uses:

- Glaucoma
- Epilepsy
- Altitude sickness

Miotics / Cholinergic Agonists

Mechanism of Action:

- Constrict pupils (miosis)
- Opens trabecular meshwork → increases aqueous humor outflow

Examples:

- Pilocarpine
- Carbachol

Pharmacokinetics:

- **Absorption:** Well absorbed via ocular drops
- **Distribution:** Mainly ocular; minimal systemic
- **Metabolism:** Local ocular metabolism
- **Excretion:** Renal

Adverse Effects:

- Blurred vision
- Headache
- Tearing
- Myopia, night vision difficulty

Therapeutic Uses:

- Glaucoma
- Myasthenia Gravis (diagnostic use)
- Eye surgery preparation