PHARMACY INDIA

PHARMACOLOGY

A COMPETITIVE EXAMINATION BOOK

MODULE- 4

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Chapter 1

GENERAL PHARMACOLOGY

Pharmacodynamics: -What drug does to body.

Pharmacokinetics: - What body does to the drug.

Pharmacotherapeutics: -Use of drugs in prevention & treatment of disease.

Clinical pharmacology: - Scientific study of drugs in man.

Toxicology: - Aspect of pharmacology deals with adverse effects of Drugs.

Pharmacodynamic agents: - Designed to have pharmacodynamic effects in the recipient.

Chemotherapeutic agents: - Designed to inhibit/kill parasites/malignant cells & does not have or with minimal pharmacodynamic effects in recipient.

Clinical pharmacology: - it is the systemic study of a drug in man both in kinetic healthy volunteers and in patients .it includes the evaluation of pharmacokinetics and pharmacodynamics data, safety, efficacy and adverse effect of a drug by comparative clinical trials.

Essential medicines: -

- E → Effective and economical
- S → Safe
- S → Single drug formulation mostly
- E → Environmental factor are also considered in making the choice
- N → Needed by the majority of population
- T → They must be available at all times
- I → In proper dosage form
- A → Aim is to optimally use the limited financial resources
- L → List of essential drugs is made locally with the help of WHO model list

ORPHAN DRUGS	OVER THE COUNTER DRUGS	PRESCRIPTION DRUGS
Drugs that are used for the diagnosis, treatment or prevention of rare diseases	These drugs can be sold to a patient without the need for a doctor's prescription	These are drugs which can be obtained only upon producing the prescription of a RPM.
"SR DDLG Ka FAN Hai" S → Sumatriptan R → Rifabutin D → Digoxin immune fab L → Liyothyronine (T3) F → Fomipizole A → Amphoteracin B N → Nitrates	e.g., Paracetamol, antacids etc.	e.g., antibiotics, antipsychotics etc.

Active Drug	Active Metabolite
Amitriptyline	Nortriptyline
Codeine	Morphine
Diazepam	Oxazepam
Digitoxin	Digoxin
Imipramine	Desipramine
Phenacetin	Paracetamol
Primidone	Phenobarbitone
Sipronolactone	Canrenone
Allopurinol	Alloxanthine
Morphine	Morphine-6-glucuronide

Inactive Drug (Prodrug)	Active Metabolite
Proguanil	Cycloguanil
Levodopa	Dopamine
Enalapril	Enalaprilat
Dipivefrine	Epinephrine
Sulindac	Sulfide metabolite
Prednisone	Prednisolone
Bacampicillin	Ampicillin
Sulfasalazine	5-Amino salicylic acid
Acyclovir	Acyclovir triphosphate
Cyclophosphamide	Aldophosphamide, Acrolein

Prodrugs

- It is an inactive form of a drug which is converted to an active form after metabolism.

Uses of prodrugs (advantages)

- To improve bioavailability: - parkinsonism is due to deficiency of dopamine. Dopamine itself cannot be used since it does not cross the blood brain barrier (BBB). So, it is given in the form of a prodrug, levodopa. Levodopa crosses the BBB and it is then converted into dopamine.
- To prolong the duration of action: - phenothiazines have a short duration of action whereas esters of phenothiazines (fluphenazine) have a longer of duration of action
- To improve the taste: - clindamycin has a bitter taste so clindamycin palmitate suspension has been developed for pediatric use to improve the taste.
    1. Inactive form
    2. Active metabolite, e.g.: - Codeine to morphine
    3. Prodrug to active drug, e.g.: - L-Dopa to Dopamine

Pathways of Drug Metabolism

PHASE I METABOLISM: - NON-SYNTHETIC

Reaction	Definition	Examples
Oxidation	Addition of oxygen /removal of hydrogen	Phenytoin ,phenobarbitone, pentobarbitone ,propranolol
Reduction	Removal of oxygen/addition of hydrogen	Chloramphenicol ,methadone
Hydrolysis	Break down of compound by addition of water	Esters – procaine ,succinylcholine Amides – lignocaine, procainamide
Cyclization	Conversion of straight chain compound into ring structure.	Proguanil to cycloguanil
Decyclization	Breaking up of the ring structure of the drug.	Phenobarbitone & Phenytoin

COMPETITIVE (EQUILIBRIUM TYPE) ENZYME INHIBITORS

Enzyme	Endogenous substrate	Competitive inhibitor
Cholinesterase	Acetylcholine	Physostigmine, Neostigmine
Monoamine-oxidase A (MAO-A)	Catecholamines	Moclobemide
Dopa decarboxylase	Levodopa	Carbidopa, Benserazide
Xanthine oxidase	Hypoxanthine	Allopurinol
Angiotensin converting enzyme (ACE)	Angiotensin-1	Captopril
5α-Reductase	Testosterone	Finasteride
Aromatase	Testosterone, Androstenedione	Letrozole, Anastrozole
Bacterial folate synthase	Para-amino benzoic acid (PABA)	Sulfadiazine

NON-COMPETITIVE ENZYME INHIBITORS

Non-competitive inhibitor	Enzyme
Acetazolamide	Carbonic anhydrase
Aspirin, indomethacin	Cyclooxygenase
Disulfiram	Aldehyde dehydrogenase
Omeprazole	H+ K+ ATPase
Digoxin	Na+ K+ ATPase
Theophylline	Phosphodiesterase
Propylthiouracil	Peroxidase in thyroid
Lovastatin	HMG-CoA reductase
Sildenafil	Phosphodiesterase-5

IV. **Through ion channel**: -some drugs directly bind to ion channels and alter the flow of ions e.g., local anesthetic blocks sodium channel in neuronal membrane to produce local anesthesia.

V. **Through antibody production**: -vaccines produce their effect by stimulating the formation of antibodies e.g., Vaccine against tuberculosis (BCG), oral polio vaccine, etc.

VI. **Transporters**: - some drugs produce their effect by binding to transporters. Selective serotonin reuptake inhibitors (SSRIs)→bind to 5HT transporter → block 5-HT reuptake into neurons → antidepressant effect.

VII. **Others: -** anticancer drugs like cyclophosphamide produce their effect by binding to nucleic acid drugs like colchicine's bind to tubulin and prevent mitigation neutrophils.

SOME IMPORTANT TERMINOLOGY IN PHARMACODYNAMICS

TERMINOLOGY	DESCRIPTION
Receptor	• It is defined as a macromolecule or binding site located on the surface or inside the effector cell that serves to recognize the signal molecule/drug and initiate the response to it, but itself has no other function. **Drug (D)+ Receptor (R) ⇌ Drug-receptor complex → Response**

TYPES OF HYPERSENSITIVITY REACTION

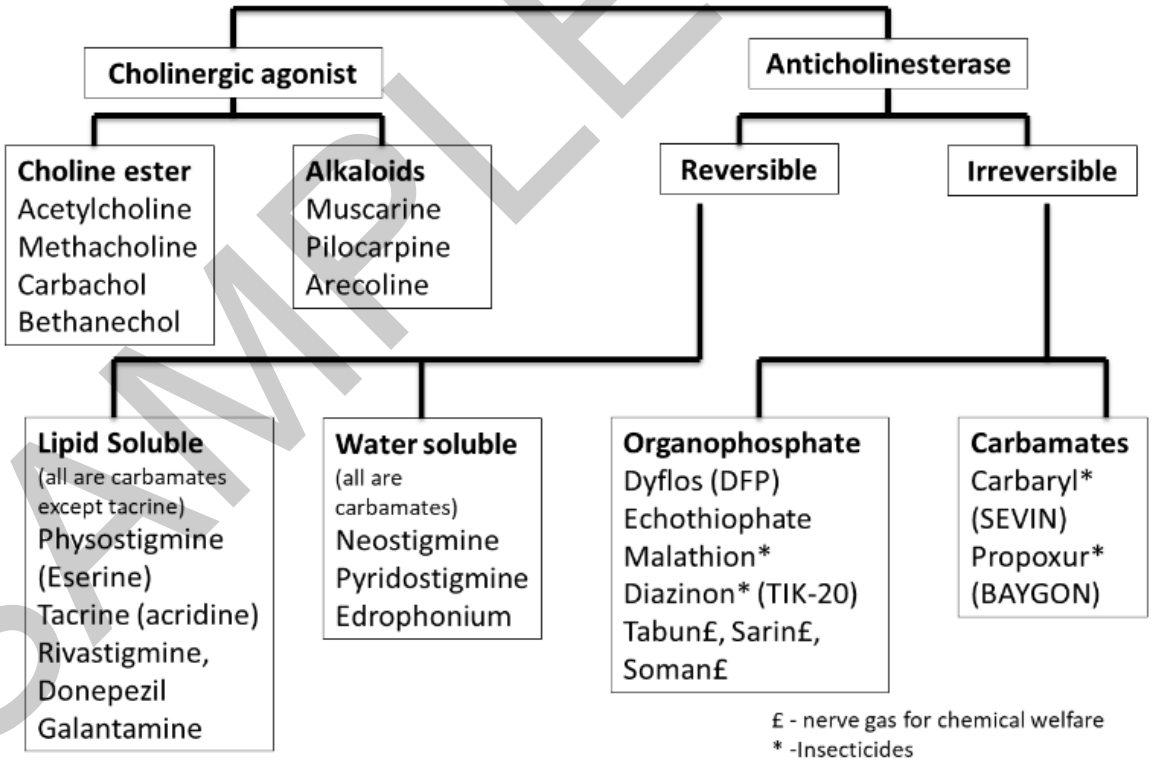
CHARACTERISTIC	TYPE I (Immediate & anaphylactic)	TYPE II (Cytotoxic)	TYPE III (Arthus, serum sickness)	TYPE IV (cell-mediated)
Immune response alters	Humoral	Humoral	Humoral	Mediated
Immediate or delay	Immediate & anaphylactic	Immediate	Immediate	Delay
Antigen	Soluble	Cell surface	Soluble	Soluble or bound
Mediator	IgE	IgG, lgM	Ag-Ab complex, and lgG	T- cell
Effector mechanism	Mast cell	1, ADCC 2. Complement mediated cytolysis	Complement activation and inflammatory response	Macrophages activation lead to phagocytosis or cell cytotoxicity
Typical manifestation	1. Anaphylaxis 2. Asthma 3. Atopic Dermatitis	1. Transfusion reaction 2. Rh incompatibility 3. Hemolytic anemia	1. Arthus reaction 2. Serum sickness 3. Rheumatiod arthritis 4. Steven johnson syndrome	1.Tuberculin test 2. Leposy 3. dermatitis

ANTIDOTES OF POISONING

S.NO.	POISONS	ANTIDOTES
1.	Acetaminophen	N - Acetylcysteine
2.	Aspirin	Sodium bicarbonate
3.	Anticoagulant	Vitamin K
4.	Atropine	Physostigmine
5.	Arsenic	EDTA
6.	Benzodiazepines	Flumazenil
7.	Beta blocker	Glucagon and calcium
8.	Cyanide	Hydroxy cobalamin, amyl nitrate, sodium thiosulfate
9.	Calcium channel blocker	Calcium
10.	Carbon monoxide	Hyperbaric acid
11.	Carbamates	Atropine

		smooth muscle: Contraction	cause → vasodilation
NATURE	**Gq – Protein coupled**	**Gq/Go-protein coupled**	**Gq –protein coupled**
TRANSDUCER	**IP3/DAG**— ↑se cytosolic Ca2+, **PLA2 ↑ses** —PG synthesis.	**K* channel opening:** ↓se cAMP	**IP3/DAG**— ↑se cytosolic Ca2+, **PLA2 ↑ses** — PG synthesis.
AGONIST	Oxotremorine	Methacholine	Bethanechol
ANTAGONIST	Pirenzepine, Talenzepine	Methoctramine, Tripitramine	Solifenacin, Dariferacin

CHOLINERGIC AGENTS



	• **trigone and sphincter muscle contracts – urinary retention**
• **Bronchi**	Atropine causes - • Bronchial muscles relax **(bronchodilation)**
Glands	Atropine causes - • **Decreased secretion of exocrine glands.** • Decreases sweat, salivary, tracheobronchial and lacrimal secretion (M3 blockade). • Skin and eyes become dry, talking and swallowing may be difficult.
Body temperature	• Rise in body temperature occurs at higher doses. • It is due to both inhibition of sweating as well as stimulation of temperature regulating centre in the hypothalamus.
Local anaesthetic	• Atropine has a mild anaesthetic action on the cornea.

USES AND ADVERSE EFFECTS

THERAPEUTIC USES OF ATROPINE	ADVERSE EFFECTS OF ATROPINE
Trick (ATROPA)	**Trick (DHATURA)**
A – As mydriatics and cycloplegic **T –** Traveller's diarrhoea **R –** Rapid onset of mushroom poisoning **O –** Organophosphate poisoning **P –** Preanaesthetic medication **A –** Arrhythmia	**D –** Dry mouth **H –** Hot dry skin **A –** Accommodation paralysis **T –** Tachycardia **U –** Urinary retention **R –** Respiratory depression **A –** Ataxia (lack of voluntary constriction)

DRUGS ACTING ON AUTONOMIC GANGLIA

Class	Sub-class	Drugs
Ganglionic stimulants	**Selective nicotinic agonist**	Nicotine, Lobeline, Dimethyl phenyl piperazinium(DMPP), Tetramethyl ammonium (TMA),Varenicline
	Nonselective/ muscarinic agonist	Acetylcholine, Carbachol, Pilocarpine, Anticholinesterase, MCN 343-A.
Ganglionic blocking agents	**Competitive blockers**	
	Quaternary ammonium compounds	Hexamethonium, Pentolinium
	Amines (secondary/tertiary)	Mecamylamine, Pempidine
	Monosulfonium compound	Trimethaphan camforsulfonate
Persistent depolarizing blocker	Nicotine, Anticholinesterases	

SYMPATHETIC NERVOUS SYSTEM

Adrenergic Transmission: -

- Adrenergic (more precisely 'Noradrenergic') transmission is restricted to the sympathetic division of the ANS.
- **Catecholamines** (CAs).

Endogenous catecholamines	Exogenous catecholamines	Non-catecholamines

5-HT$_2$	**α – methyl 5-HT**	**5-HT$_{2A}$**	**Cyproheptadine, Ketanserine (additional α–blocking property), Ritanserine**
5-HT$_3$	**2 – methyl 5-HT**	**5-HT$_{2A/2C}$**	**Clozapine, methysergide** (Used in migraine)
5-HT$_4$	**Metoclopramide, Cisapride, Renzapride** (Used in GERD)	**5-HT$_3$**	**Ondensetron, Granisetron** (Used in chemotherapy induced therapy)

Pharmacological Actions of 5-HT

Organs/ Parts of body	Pharmacological actions
CVS	• Arteries are **constricted** (by direct action on vascular smooth muscle) **as well as dilated** (through EDRF release) by 5-HT. • BP: a **triphasic response** is classically seen on i.v. injection of 5-HT in animals. ▪ Early sharp fall in BP—due to coronary chemoreflex. ▪ Brief rise in BP—due to vasoconstriction and increased cardiac output. ▪ Prolonged fall in BP—due to arteriolar dilatation and extravasation of fluid.
Visceral smooth muscles	• 5-HT is **a potent stimulator of g.i.t.** • **Peristalsis is increased** and **diarrhoea can occur** (also due to increased secretion).
Glands	• 5-HT **inhibits gastric secretion** (both acid and pepsin), but **increases mucus production.**
Nerve endings and adrenal medulla	• Afferent nerve endings are activated **causing tingling and pricking sensation, as well as pain.** • Depolarization of visceral afferents elicits respiratory and cardiovascular reflexes, **nausea and vomiting**.
Respiration	• Stimulation of respiration (mostly reflex from bronchial afferents) and • hyperventilation are the usual response
Platelets	• 5-HT causes **changes in shape of platelets**, but is a **weak aggregator**.
CNS	• Injected i.v., 5-HT **does not produce central effects** because of poor entry across blood brain barrier.

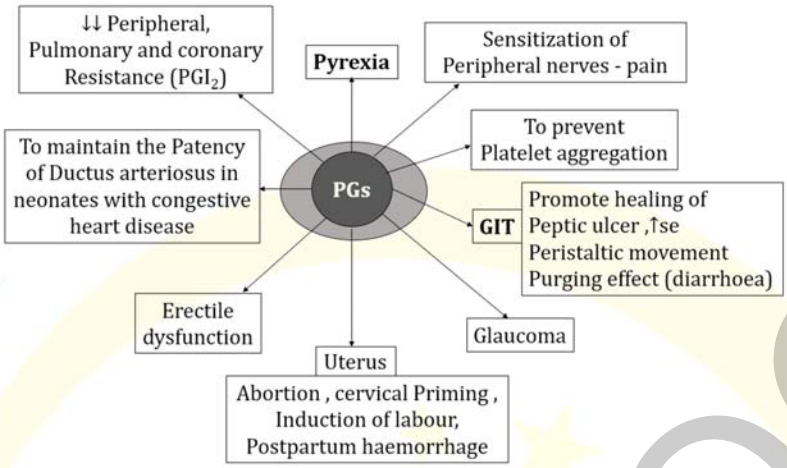
Drugs Affecting 5-HT System

5-HT SYSTEM	DRUGS
5-HT precursor	Tryptophan hydroxylase
Synthesis	P- chlorophenyl alanine
Uptake inhibitor	Tricyclic antidepressants (TCAS)
Storage inhibitor	Reserpine
Degradation inhibitor	Tranlcypromine, chlorhyline
Neuronal degeneration	5,6- dihydroxytryptamine

Ergot Alkaloids

Class	Drugs	Uses
Natural ergot alkaloids		
Amine alkaloids	Ergometrine (Ergonovine)	oxytocic
Amino acid alkaloids	Ergotamine,	Migrane

Uses of PGs



LEUKOTRIENES

- The straight chain lipoxygenase products of arachidonic acid are produced by a more limited number of tissues –
  - **LTB4 mainly by neutrophils**
  - **LTC4 and LTD4**—the cysteinyl LTs—mainly by **macrophages**)
- They are pathophysiologically as important as PGs.
- Important mediator of inflammations.

Pharmacological actions

Organs/ Part of body	Actions
CVS and blood	• LTC4 and LTD4 injected i.v. evoke a brief rise in BP followed by a more prolonged fall because of no relaxant action in blood vessels. • Coronary constriction induced decrease in cardiac output and reduction in circulating volume due to increased capillary permeability.
Smooth muscles	• LTC4 and D4 contract most smooth muscles. • Potent bronchoconstrictors • Induce spastic contraction of g.i.t. at low concentrations. • Also increase mucus secretion in the airways.
Afferent nerves	• LTB4 also sensitizes afferents carrying pain impulses → contributes to pain and tenderness of inflammation.

Leukotriene Receptors

- **All LT receptors couple with Gq protein** and function through the **IP3/DAG transducer mechanism.**
- The ***cys*LT1 receptor** is mainly expressed in **bronchial and intestinal muscle** and has higher affinity for LTD4 than for LTC4.
- The primary location of ***cys*LT2 receptor** is **leucocytes and spleen**.
- The ***cys*LT1 receptor antagonists**, *viz.* **Montelukast, Zafirlukast**.

Actions of LTs can be inhibited by: -

1. Corticosteroids
2. Lipoxygenase inhinitor **(Zileuton)**
3. LT receptor antagonist **(Montelukast, Zafirlukast)**

THROMBOXANES A_2

	• Effect on platelet TXA2 (COX-1 generated) predominates → therapeutic doses of most NSAIDs inhibit platelet aggregation: bleeding time is prolonged.
Ductus arteriosus closure	• NSAIDs in late pregnancy has been found to promote premature closure of ductus in some cases. • Risk of post-partum haemorrhage is increased. • Prescribing of NSAIDs near term should be avoided.
Parturition	• potential to delay and retard labour
Gastric mucosal damage	• enhance aggressive factors and contain defensive factors in gastric mucosa—are ulcerogenic.
Renal effects	• NSAIDs produce renal effects by at least 3 mechanisms: - COX-1 dependent impairment of renal blood flow and reduction of g.f.r. → can worsen renal insufficiency. - Juxtaglomerular COX-2 (probably COX-1 also) dependent Na+ and water retention. - Ability to cause papillary necrosis on habitual intake.
Anaphylactoid reactions	• Aspirin precipitates asthma, angioneurotic swellings, urticaria or rhinitis in certain susceptible individuals.

Non selective COX inhibitors

Aspirin –

- Acetylsalicylic acid
- It is rapidly converted in the body to its active metabolite → **salicylic acid**

Pharmacokinetics	• rapidly deacetylated in gut wall liver, plasma and other tissue to release salicylic acid • Both aspirin and salicylic acid are conjugated in liver with glycine → Salicyluric acid (major pathway) and with glucoronic acid. • $t_{1/2}$ - 15 to 20 minute.
Adverse effects	A → Asthma S → Salicylism P → Peptic ulcer I → Ion uncoupling / platelet disaggeration R → Reye's syndrome I → Idiosyncracy N → Noise (tinnitus)
Contraindication	• peptic ulcer patients and asthmatic patients • children suffering from chicken pox and influenza • Prohibited to paediatric formulation. • Pregnancy, hepatic necrosis • Possibility of premature closure of ductus arteriosus. • Avoids high dose in G-6-PD deficient individual haemolysis
Interactions	• Aspirin displaces → **warfarin, naproxen, sulfonylureas, phenytoin and methotrexate from binding sites on plasma protein** → toxicity of these drugs may occur. • **Inhibits tubular secretion of uric acid** • Antagonizes uricosuric action of probenecid

INDIVIDUAL DRUGS

Drugs	Description
Ibuprofen	• It has been rated as the safest traditional NSAID by the spontaneous adverse drug reaction reporting system in U.K.

	➢ Lidocaine is depressant, i.e., drowsiness, mental clouding, dysphoria, altered taste and tinnitus.
Tetracaine (Amethocaine)	➢ A highly lipid soluble PABA ester, more potent and more toxic due to slow hydrolysis by plasma pseudocholinesterase.
Bupivacaine	➢ It is used in obstetrics (mother can actively cooperate in vaginal delivery) and for postoperative pain relief) ➢ Epidural anaesthesia with 0.75% bupivacaine during labour has caused few fatalities due to cardiac arrest. So it is contraindicated. ➢ It also prolongs QT interval and cause tachycardia.
Benoxinate	➢ It is a good surface anaesthetic for the eye.
Benzocaine and Butylamino-benzoate (Butamben)	➢ Used as lozenges for stomatitis, sore throat; as dusting powder/ointment. ➢ Both are PABA derivative—can antagonize sulphonamides

IMPORTANT POINTS WITH TRICKS

LA causing methemoglobinemia	
B	Benzocaine
P	Prilocaine (Max)
L	Lignocaine

Chapter 5

CENTRAL NERVOUS SYSTEM

SEDATIVE HYPNOTICS

POINTS TO BE REMEMBER

$GABA_A$-BZD-Cl^- channel complex	Opening increases the conductance of chloride ions resulting in CNS depression
Barbiturates	Bind to another site on this channel to exert GABA mimetic (direct activation of $GABA_A$ receptors) as well as GABA facilitatory (increase the binding of GABA to $GABA_A$ receptors) actions.
Benzodiazepines	Bind to a different site (BZD receptor) and increase the binding of GABA to $GABA_A$ receptor (GABA facilitatory action). These drugs increase the frequency of Cl– channel opening.
Bicuculline	Binds to GABAA receptor and acts as a competitive inhibitor of GABA and non-competitive inhibitor of benzodiazepines.
β-carboline	Acts as an inverse agonist at benzodiazepine site and thus produces convulsions due to stimulation of the brain.
Flumazenil	Acts as a competitive antagonist at BZD site and therefore inhibits the action of benzodiazepines as well as Beta-carboline.

CLASSIFICATION OF NEUROTRANSMITTER ON THE BASIS OF ACTION

Types of neurotransmitters	Comments
	Inhibitory neurotransmitter
GABA	• GABA plays an important role in anxiety and sedative and hypnotics Types: GABA, GABA
Glycine	• Glycine especially in the spinal cord, Brainstem, and retina • When glycine receptors are activated, chloride enters the neuron via ionotropic receptors, causing an inhibitory potential (IPSP).
Dopamine	• Dopamine system plays a central role in several significant medical conditions including Parkinson's disease, attention deficit hyperactivity disorder, Tourette syndrome, schizophrenia, bipolar disorder, and addiction.
	Excitatory neurotransmitter
Glutamate	• Excitotoxicity due to excessive glutamate release and impaired uptake part of the ischemic cascade and is associated with stroke, autism, some forms of intellectual disability, and diseases such as amyotrophic lateral sclerosis, lathyrism, and Alzheimer's disease. • In decreased glutamate release is observed under conditions of classical phenylketonuria leading to developmental disruption of glutamate receptor expression,

T	**Triazolam, Temazepam**
O	**Oxazepam**
L	**Lorazepam**
E	**Estazolam**

BENZODIAZAPINES: ONES NOT METABOLIZED BY THE LIVER (SAFE TO USE IN LIVER FAILURE)

TRICK - "Outside The Liver"	
Outside	O Xszepam
The	T emazapam
Liver	L orazepam

BENZODLAZEPINES: ANTIDOTE

TRICK - "Ben is off with the flu" - Benzodiatzpine effects off with **Flumazenit**

BENZODIAZEPINES: ACTIONS

TRICK - "Ben SCAMs Pam into redaction not by brain but by muscle"	
S	Sedation
C	Anti-Convulsant
A	anti-Anxiety
Ms	Muscle relaxant
Not by brain	No antipsychotic activity

ANTI-PARKINSON DRUGS AND THEIR MECHANISM OF ACTION

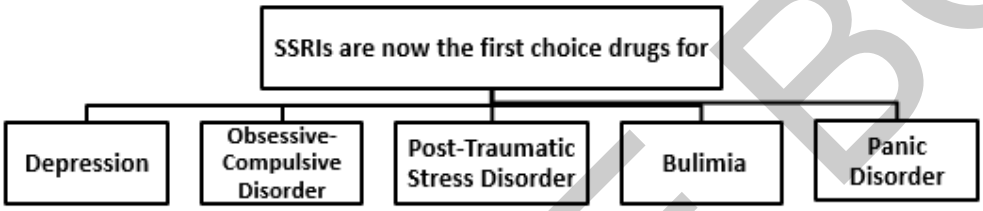
Category	Drug	Mechanism of action
Dopamine precursor	Levodopa	Levodopa crosses the blood brain barrier and decarboxylate to dopamine there by increasing the level of dopamine in brain.
Peripheral decarboxylase inhibitor	Carbidopa, benserazide	Inhibit dopamine decarboxylase for prevent the conversion of levodopa peripherally into dopamine but do not cross the blood brain barrier.
Monoamine oxidase (MAO-B) inhibitor	Selegiline	It inhibits MAO-B irreversible and delay the degradation of dopamine centrally thereby increase the duration action of levodopa.
COMT inhibitor	Tolcapone, entacapone	When peripherally decarboxylation of levodopa is blocked by carbidopa and benserazide, it is mainly metabolized by COMT to 3-0 methyldopa and COMT blocked by act peripherally Entacapone and centrally tolcapone.
Ergot alkaloids	Tolcapone entacapone	It acts as potent agonist on D2 receptor but as partial agonist or antagonist on D1 receptor.
	Ropinirole, pramipexole	It is selective D2 receptor agonist and has no affinity to D1 and increase the dopamine level.

TRICYCLIC ANTIDEPRESSANTS (TCA) SIDE EFFECTS

TRICK – "TCA'S"	
T	Thrombocytopenia
C	Cardiac (arrythmia, MI, stroke)
A	Anticholinergic (tachycardia, urinary retention, etc)
S	Seizures

MECHANISM OF ADVERSE EFFECTS OF TCA

Inhibition of	Adverse Effects
Presynaptic NT reuptake	Tremors, Insomnia
Cardiac fast Na+ channels	Conduction defects, arrhythmias, hypotension
Muscarinic ACh receptors	Hyperthermia, flushing, mydriasis, Paralytic ileus, urinary retention, sinus tachycardia
Alpha-1 Adrenergic receptors	Postural hypotension
H1 histamine receptors	Sedation



USES OF ANTIDEPRESSANTS

TRICK= DEPRESSION	
D	Depression
E	Enuresis (Imipramine)
P	Phobia
R	Recurrent panic attacks
E	Eating disorders (Bulimia)
S	Smoking cessation (Bupropion)
S	Stress disorder (Post-traumatic)
I	Impulse disorder (Kleptomania)
O	Obsessive compulsive disorder
N	Neuropathic pain

POINTS TO REMEMBER

It is a prototype SSRI and is longest acting SSRI	Fluoxetine
Shortest acting SSRI	Fluvoxamine
Most teratogenic among SSRIs	Paroxetine
Most potent blocker of 5-HT reuptake	Paroxetine
Least potent blocker of 5-HT reuptake	Bupropion
Most potent blocker of NA reuptake	Desipramine
Least potent blocker of NA reuptake	Mirtazapine
Most selective inhibitor of 5-HT reuptake	Escitalopram
Most selective inhibitor of DA reuptake	Bupropion
Most selective inhibitor of NA reuptake	Oxaprotiline
Maximum antimuscarinic activity	Amitriptyline
Maximum antihistaminic activity	Nefazodone

Chapter 6

DIURETICS

Class	Sub-class	Drugs
High efficacy diuretics (Inhibitors of Na^+-K^+-$2Cl^-$ cotransport)	**Sulphamoyl derivatives**	Furosemide, Bumetanide, Torasemide
	Phenoxyacetic acid derivatives	Ethacrynic acid
	Organomercurials	Mersalyl
Medium efficacy diuretics (Inhibitors of Na^+-Cl^- symport)	**Benzothiadiazines (thiazides)**	Hydrochlorothiazide, Benzthiazide, Hydroflumethiazide, Bendroflumethiazide
	Thiazide like (related heterocyclics)	Chlorthalidone, Metolazone, Xipamide, Indapamide, Clopamide
Weak or adjunctive diuretics	**Carbonic anhydrase inhibitors**	Acetazolamide
	Potassium sparing diuretics (TRICK → SEAT)	**Aldosterone antagonist:** Spironolactone, Eplerenone
		Inhibitors of renal epithelial Na+ channel: Triamterene, Amiloride
	Osmotic diuretics (TRICK → MUG I)	Mannitol, Urea, Isosorbide, Glycerol
	Xanthine's	Theophylline

MECHANISM OF ACTION OF DIURETICS

Classification	Site of action	Mechanism of action
Carbonic anhydrase inhibitor	Proximal Convoluted tubules (PCT)	Carbonic anhydrase enzyme convoluted catalyzes the reaction $H_2O + CO_2 \leftrightarrow H_2CO3$ and further it to H2-CO3↔ H+ + HCO3. CA inhibitors inhibits this process and prevent the formation of H+ ion. Thus Na+ H+ exchange is prevented and leads to diuretic action
Loop diuretics	Act on thick ascending loop of Henle	Inhibit Na-K-2Cl cotransporter so increase excretion of Na+ and in urine

Chapter 8

CARDIOVASCULAR SYSTEM

CARDIAC GLYCOSIDES

Drugs	Mechanism of action
Digoxin, digitoxin, strophanthin and ouabain	Act by inhibiting Na^+-K^+ ATPase of myocardial fibres by binding to its extracellular face

CONTRAINDICATIONS OF DIGITALIS

TRICK = Contraindicated in WEAK HEART	
Contraindicated	Cardiatits (myocarditits)
In	Increases Ca (Hypercalcimeia)
Weak	WPW syndrome
H	Hypokalemia and hypomagnesimia
E	Elderly
A	AV Block (partial)
R	Renal failure (digoxin)
T	Thyroid (hyper or hypothyroidism)

TYPES OF ANGINAS

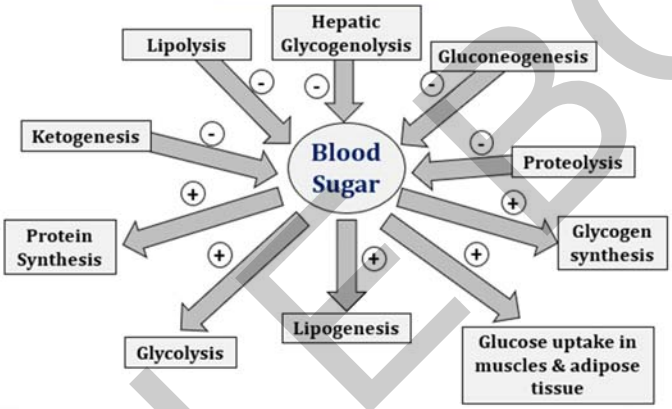
Classical angina /stable angina	Variant /prinzmetal/ vasopastic angina	Unstable angina
Provoked by exercise, emotion, eating or coitus	occurs at rest during sleep and are unpredictable (due to vasospasm)	mostly due to the rupture of an artheromateous plaque attracting platelet disposition & progressive occlusion of the coronary artery

CLASSIFICATION OF ANTI-ANGINALS

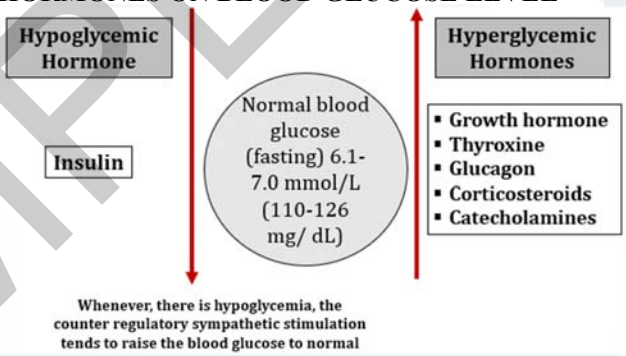
Classes	Sub classes	Drugs
Nitrates	Short acting	Glyceryl trinitrate (Nitroglycerine)
	Long acting	Isosorbide dinitrate, Isosorbide mononitrate, erythrityl tetranitrate, pentaerythrityl tetranitrate
β – Blocker	Propranolol, metoprolol, atenolol (other)	
Calcium channel blocker	Phenyl alkylamine	Verapamil
	Benzothiazepine	Diltiazem

PANCREAS CELLS	HORMONES	FUNCTION	DISORDER
α cells	Glucagon	Stimulates the conversion of stored glycogen (stored in the to glucose and Stimulates glycogenesis.	Hyposecretion cause hypoglycaemia.
β cells	Insulin	Control blood glucose levels by signalling the liver, muscle and fat cells to take in glucose from the blood.	Hyposecretion cause diabetes mellitus
δ cells	Somatostatin	Suppresses the release of insulin and glucagon	-
F-cells	Pancreatic polypeptide	Inhibits the release of digestive secretion of the pancreas.	-

ACTIONS OF INSULIN



EFFECT OF VARIOUS HORMONES ON BLOOD GLUCOSE LEVEL



INSULIN PREPARATIONS

Type of action	Insulins
Rapid acting	Insulin lispro, Insulin aspart, Insulin glulisine
Short acting	Regular (soluble, crystalline zinc) insulin, Humulin
Intermediate acting	Insulin zinc suspension or Lente, Neutral protamine hagedorn or isophane insulin
Long acting	Insulin glargine, Insulin detemir, Insulin degludeg
Recombinant	

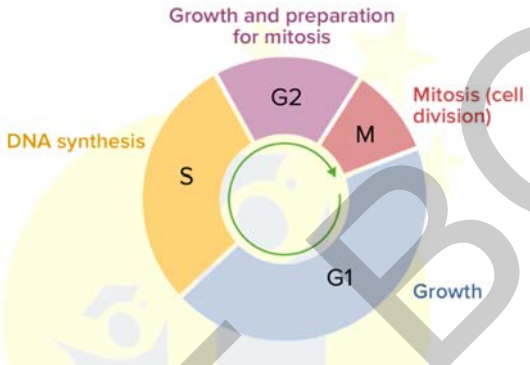
ORAL HYPOGLYCAEMIC DRUGS

Class	Sub class	Drugs

Chapter 13

ANTICANCER DRUGS

- The anticancer drugs either kill cancer cells or modify their growth.
- Cancer is a disease of cells characterized by Progressive. Persistent. Purposeless and uncontrolled Proliferation of tissues.
- Both normal as well as cancerous cells must pass through the following phases of cell cycle:



CELL CYCLE SPECIFIC AND NON-SPECIFIC DRUGS

Phase	Cell cycle specific drugs	Cell cycle non-specific drugs
G1	**Etoposide**	**Platinum compound**
S	**ANTIMETABOLITE** • Methotrexate • 6- mercaptopurine • 5-Flurouracil • Capecitabine • Cytarabine • Cytosine arabinoside (G-05)	**ALKYLATING AGENT** • Melphalan • Cyclophosphamide • Nitrosoureas
G	**TOPOISOMERASE INHIBITOR** • Irinotecan • Topotecan • Etoposide • Bloemycin	**ANTHRACYCLINS** • Doxorubicin • Daunorubicin • Epirubicin • Mitoxantrone
M	**VINCA ALKALOIDS** • Vincristine • Vinblastine • Vinorelbine **TAXENES** • Paclitaxel • Docetaxel	**MITOMYCINS C** • Dactinomycin • Camptothecin

AIIMS

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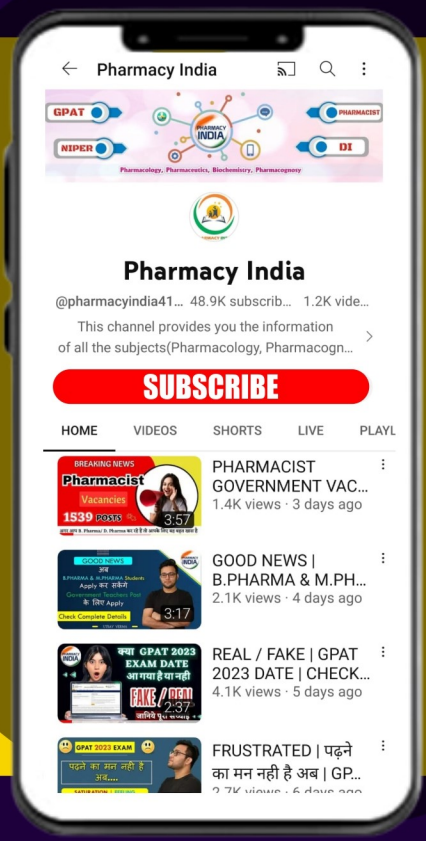
HPPSC, BTSC, ISRO,
KERALA PSC,
MP PHARMACIST,
SECL, DSSSB,
BCCL

ESIC

UP NHM

ABOUT PHARMACY INDIA

Our classes set up with an aim to provide coaching to the aspiring students who are dedicated and want to achieve excellence in their career. we nurture aspirants and facilitated achievement and we specialized in providing correct and relevant information related to Pharma institute admission for higher education.



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