

MBBS Curriculum

Course Features

- 3D & 2D Animated Videos
- India's Only CBME Program for Pre-Clinical & Para - Clinical Subjects
- Videos Covering CBME Based NMC Approved Syllabus
- Theory & Practical Exams Preparation
- University PYQs for Professional Exam
- Qbank for Formative Assessments
- Mentorship & Guidance by India's Best Faculty Team
- Special Videos for AETCOM
- Hard Copy Notes

India's Best Faculty Team



Dr Tejaswi
Anatomy



Dr Amit Jain
Biochemistry



Dr Naveen Porwal
Physiology



Dr Nileshraj
Pharmacology



Dr Vandana Puri
Pathology



Dr Sonu Panwar
Microbiology



Dr Rajasi
PSM



Dr Jayaprakash K
FMT

TABLE OF CONTENTS

S. NO	Table of contents	Page No
1	The Two Pillars: Pharmacokinetics vs. Pharmacodynamics	1
2	Pharmacokinetics: The Journey of a Drug (ADME)	2
3	Pharmacodynamics: Receptor Interactions and Cellular Response	3
4	Autonomic Nervous System: Cholinergic & Adrenergic Pathways	4
5	Hormones & Related Drugs	5
6	Anti-Microbial Agents: Mechanisms of Selective Toxicity	6
7	Geriatric Pharmacology & Central Nervous System (CNS) Drugs	7
8	Gastrointestinal & Peripheral Nervous System Agents	8
9	Cardiovascular System: Angina & Hypertension Management Pathways	9
10	Heart Failure: Acute & Chronic Management	10
11	Blood, Anticoagulation & Diuretics	11
12	Respiratory System & Autacoids/NSAIDs	13
13	Chemotherapy of Neoplastic Disease	14
14	Synthesis & High-Yield Principles	15

The Two Pillars: Pharmacokinetics vs. Pharmacodynamics



Pharmacokinetics (PK):

What the body does to the drug.

- Governs Absorption, Distribution, Metabolism, and Excretion (ADME).
- Determines the drug concentration at its site of action.





Pharmacodynamics (PD):

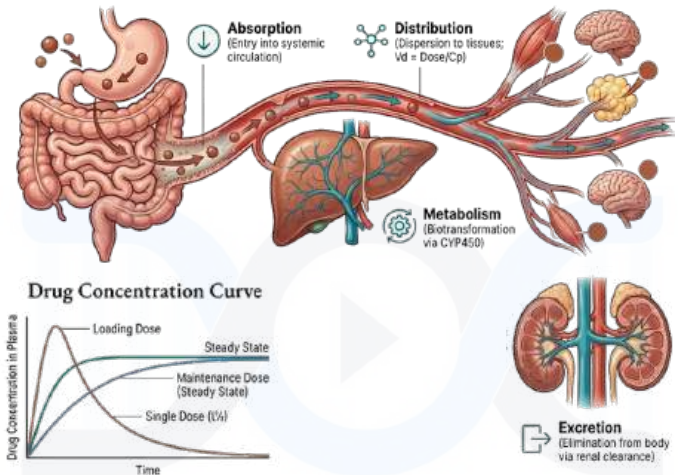
What the drug does to the body.

- Describes drug-receptor interaction and the subsequent cellular response.
- Links receptor binding to the clinical effect.

Foundational Concepts:

Category	Definition	Key Concepts	Example (Paracetamol)
PK	What the body does to the drug	ADME, Half-life, Clearance	Body absorbs, metabolizes, and excretes the drug.
PD	What the drug does to the body	Receptors, Agonists, Antagonists	Drug binds to COX enzymes to reduce pain.
Nomenclature	 Chemical: N-(4-hydroxyphenyl)acetamide	 Generic (INN): Paracetamol	TM Brand™: Tylenol®

Pharmacokinetics: The Journey of a Drug (ADME)



Key Metrics



Steady State: Achieved after approximately 4-5 half-lives ($t_{1/2}$).



Loading Dose: Used for rapid onset of effect.

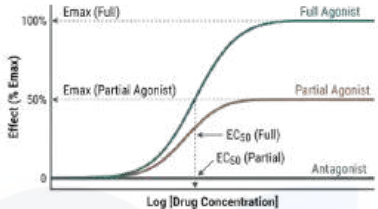


Maintenance Dose: Used to maintain steady-state concentrations.

Phase	Key Factors & Considerations
Absorption	Bioavailability (F), First-pass effect, pH/pKa, Passive vs. active transport
Distribution	Volume of distribution (V_d). Plasma protein binding, Blood-brain barrier
Metabolism	Phase I (CYP450) & Phase II (Conjugation) reactions, Active metabolites
Excretion	Half-life ($t_{1/2}$), Clearance (Cl). First-order vs. Zero-order kinetics

Pharmacodynamics: Receptor Interactions and Cellular Response





- Drug effects are mediated through binding to specific receptor superfamilies.
- The relationship between drug concentration and effect is defined by dose-response curves, measuring potency (EC_{50}) and efficacy (E_{max}).



Drug-Receptor Actions

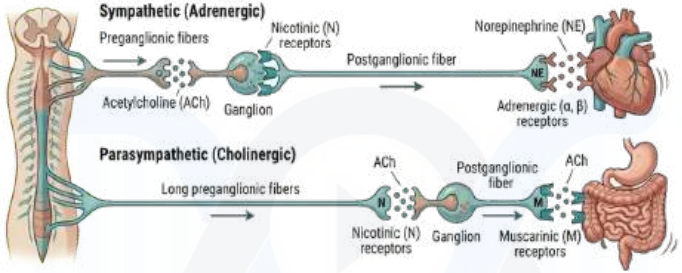
Action	Description	Efficacy
Full Agonist	Binds and elicits a maximum response.	High Efficacy
Partial Agonist	Binds but elicits a submaximal response.	Submaximal Efficacy
Antagonist	Binds and blocks agonist; no intrinsic activity.	No Efficacy
Inverse Agonist	Binds and elicits an opposite effect to the agonist.	Opposite Effect

Major Receptor Superfamilies



Receptor Type	Mechanism & Response Speed	Examples
 GPCR (G-protein)	Gs, Gi, Gq subtypes mediate second messenger cascades; Slower.	Most common drug targets.
 Ion Channels	Ligand-gated or voltage-gated ion flux; Very fast responses.	Benzodiazepines, Local Anesthetics.
 Enzyme-Linked	Receptor Tyrosine Kinase activity upon binding; Slower.	Insulin, Growth Factors.
 Nuclear Receptors	Intracellular; Regulate gene transcription; Slowest.	Steroids, Thyroid Hormone.

Autonomic Nervous System: Cholinergic & Adrenergic Pathways




- The ANS is divided into the parasympathetic (cholinergic) and sympathetic (adrenergic) systems, which are modulated by drugs targeting their specific receptors.



Comparison of Autonomic Systems

System	Cholinergic 	Adrenergic 
Receptors	Muscarinic (M1-M5), Nicotinic (NN, NM)	Alpha (α1, α2), Beta (β1, β2, β3)
Key Functions	"Rest and Digest;" Neuromuscular transmission	"Fight or Flight" response
Example Agonists (↑)	Bethanechol, Pilocarpine	Epinephrine, Albuterol
Example Antagonists (↓)	Atropine, Pancuronium	Propranolol, Prazosin






Hormones & Related Drugs

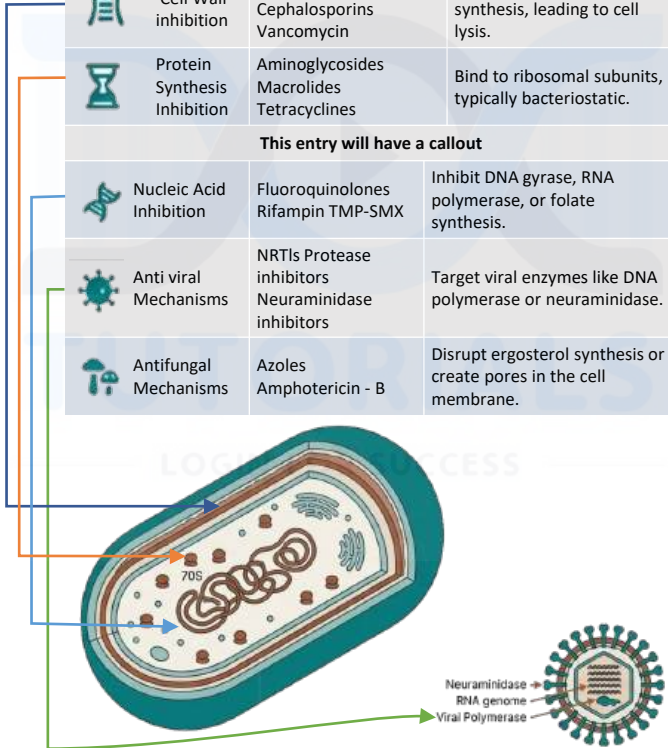
Key Endocrine Drug Classes		
 Glucocorticoids	 Thyroid Drugs	 Diabetes Drugs
Examples Prednisolone, Dexamethasone	Examples Levothyroxine, Methimazole	Examples Insulin, Metformin, SGLT2i (Empagliflozin)
Primary Effects Potent anti-inflammatory, immunosuppressive; Risk of hyperglycemia, osteoporosis, adrenal suppression.	Primary Effects T4 replacement (Levothyroxine); Inhibit hormone synthesis (Methimazole).	Primary Effects Lower blood glucose via diverse mechanisms (e.g., AMPK activation, urinary glucose excretion, ↑insulin secretion).
Key Monitoring Parameters (📄) Blood glucose, Blood pressure, Bone density, Signs of infection.	Key Monitoring Parameters (❤️) TSH, free T 4 levels, Heart rate, Weight changes.	Key Monitoring Parameters (🩸) HbA1c, Blood glucose, Renal function (eGFR), Signs of hypoglycemia.

LOGIN FOR SUCCESS

Anti-Microbial Agents: Mechanisms of Selective Toxicity

- Combination therapy is standard for organisms like Mycobacterium tuberculosis (HRZE regimen: Isoniazid, Rifampin, Pyrazinamide, Ethambutol).

Major Anti-Microbial Classes by Mechanism		
Mechanism of Action	Drug Classes	Examples
 Cell Wall inhibition	Penicillins Cephalosporins Vancomycin	Inhibit peptidoglycan synthesis, leading to cell lysis.
 Protein Synthesis Inhibition	Aminoglycosides Macrolides Tetracyclines	Bind to ribosomal subunits, typically bacteriostatic.
This entry will have a callout		
 Nucleic Acid Inhibition	Fluoroquinolones Rifampin TMP-SMX	Inhibit DNA gyrase, RNA polymerase, or folate synthesis.
 Anti viral Mechanisms	NRTIs Protease inhibitors Neuraminidase inhibitors	Target viral enzymes like DNA polymerase or neuraminidase.
 Antifungal Mechanisms	Azoles Amphotericin - B	Disrupt ergosterol synthesis or create pores in the cell membrane.



Geriatric Pharmacology & Central Nervous System (CNS) Drugs

Geriatric PK/PD Changes

PK:





- ↑ Body fat (lipophilic drug V_d)
- ↓ Hepatic metabolism (CYP450)
- ↓ Renal elimination (GFR)
- ↓ Albumin (↑ free drug)



PD:





- ↑ Sensitivity to CNS depressants, increasing the risk of confusion and falls.

Key CNS Drug Classes & Considerations

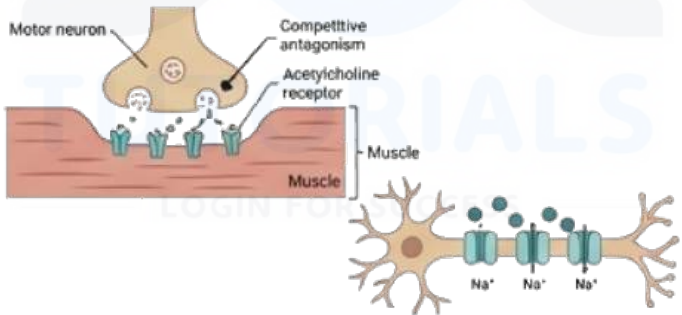
Class	Mechanism	Key Clinical Points
 Anti-psychotics	D2 and/or 5-HT _{2A} antagonism	↓ Clozapine requires weekly WBC monitoring for agranulocytosis.
 Mood Stabilizers	Multiple (ion pumps, channels)	↓ Lithium has a narrow therapeutic index (0.6-1.2 mEq/L); monitor renal/thyroid function.
 Anti-convulsants	Na ⁺ /Ca ²⁺ channel blockade, GABA enhancement	↓ Phenytoin follows zero-order kinetics. Levetiracetam has minimal drug interactions. Lamotrigine requires slow titration (SJS risk).
 Sedatives	GABA-A modulation (Benzos), 5-HT _{1A} agonism	↓ Risk of tolerance, dependence, and withdrawal seizures with benzodiazepines.

Gastrointestinal & Peripheral Nervous System Agents

GIT Disorder Therapies

	Condition	Drug Class	Mechanism	Critical Note
	Acid-Peptic Disease	PPIs (Omeprazole)	Irreversible H ⁺ /K ⁺ ATPase blockade.	A Increased risk of C. diff; take 30-60 min before meals.
	Motility Disorders	Opioid Agonists (Loperamide)	μ-opioid agonism (anti-diarrheal).	A Contraindicated in bacterial enteritis (risk of toxic megacolon).
	Nausea/Vomiting	5-HT ₃ Antagonists (Ondansetron)	Block serotonin receptors in CTZ.	A Risk of QT prolongation; monitor ECG.
	Inflammatory Bowel	Biologics (Infliximab)	Anti-TNF-α therapy.	A Increased infection risk; screen for TB before starting.

PNS Drug Comparison



Agent Type	Mechanism	Primary Use	Key Risk
Neuromuscular Blockers	Competitive antagonism at NMJ	Surgical paralysis	Provide no analgesia; require mechanical ventil.
Local Anesthetics	Na ⁺ channel blockade in nerves	Regional anesthesia	Systemic toxicity (LAST); Bupivacaine has high cardiotoxicity risk.

Cardiovascular System: Angina & Hypertension Management Pathways

Angina Management Pathway

**Acute
Attack**

Step 1: Immediate Relief

Sublingual Nitroglycerin

Step 2: Background Therapy

13-blockers (1st Line).
Add CCB if needed/contraindicated

Step 3: ASCVD Risk Reduction

High-intensity Statin + Antiplatelet + ACE Inhibitor

Hypertension Therapy Pathway

Diagnosis

Step 1: Initial Monotherapy

Thiazide, ACEi, ARB, or CCB

Step 2: Dual Therapy

e.g., ACEi + CCB or ARB + Thiazide

If BP >20/10 mmHg above target →

Step 3: Compelling Indications

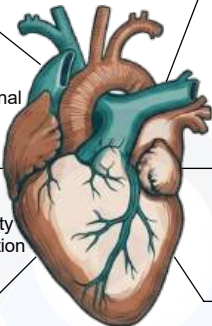
CKD: ACEi/ ARB

CAD: 13-blocker

Heart Failure: Acute & Chronic Management

Heart Failure Therapeutic Strategies




HF Type:	Management Focus	Key Drug Classes
♥ HFrEF (Chronic)	Foundational 4-Drug Therapy	<ol style="list-style-type: none">1. ARNI (Sacubitril / Valsartan) or ACEi2. Evidence-Based β - blocker (Metoprolol Succinate, Carvedilol)3. MRA (Spironolactone)4. SGLT2i
♥ HFpEF (Chronic)	Comorbidity & Congestion Control	Diuretics (for volume), Treat HTN/AF/DM.
📄 Acute Decompensation	Emergency Management	<ol style="list-style-type: none">1. IV Loop Diuretics (Furosemide)2. Vasodilators (Nitroglycerin)3. Inotropes if needed (Dobutamine)



TUTORIALS

LOGIN FOR SUCCESS

Blood, Anticoagulation & Diuretics

Blood Modifying Agents			
Class	Mechanism	Monitoring	Reversal Agent
 Anticoagulants	Inhibit coagulation cascade (Factor Xa, IIa)	INR (Warfarin). aPTT (Heparin)	Vitamin K, Protamine, Andexanet alfa
 Antiplatelets	Inhibit COX-1 or P2Y12 receptor	Platelet function assays	N/A (platelet transfusion)
 Thrombolytics	Activate plasminogen to dissolve fibrin clots	Bleeding, fibrinogen levels	Aminocaproic acid (limited use)

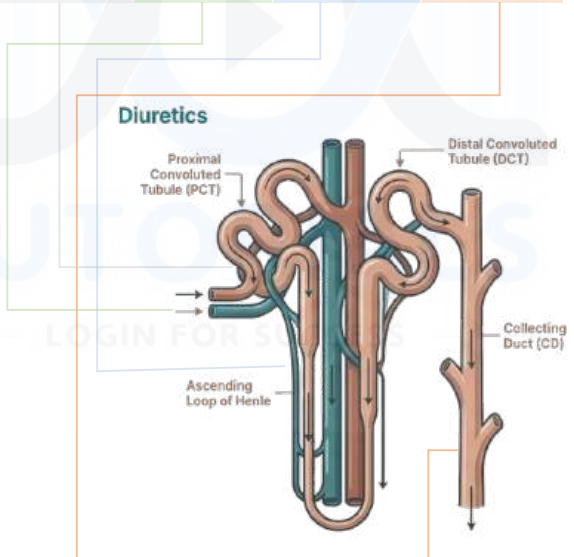
TUTORIALS

LOGIN FOR SUCCESS

Blood, Anticoagulation & Diuretics

Major Diuretic Classes by Site of Action

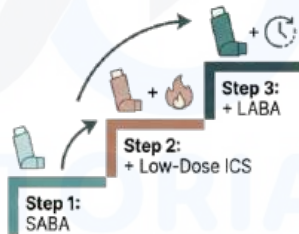
Class	Loop Diuretics	Thiazide Diuretics	K-Sparing Diuretics	CA Inhibitors
Site of Action	Ascending Loop of Henle	Distal Convoluted Tubule (OCT)	Collecting Duct (CD)	Proximal Convoluted Tubule (PCT)
Efficacy	High	Moderate	Weak	Weak
Key Indication	Acute pulmonary edema	Hypertension (first-line)	HF (Spironolactone as MRA)	Glaucoma



Respiratory System & Autacoids/NSAIDs

Respiratory

Asthma & COPD Management			
Category	Drug Class	Mechanism	Role in Therapy
Relievers	SABA (Albuterol)	B2 agonist bronchodilation	Quick relief of symptoms
Controllers	ICS (Fluticasone)	Topical anti- inflammatory	Daily maintenance therapy
COPD	LABA/LAMA Combo	Sustained bronchodilation/a nticholinergic	First-line maintenance








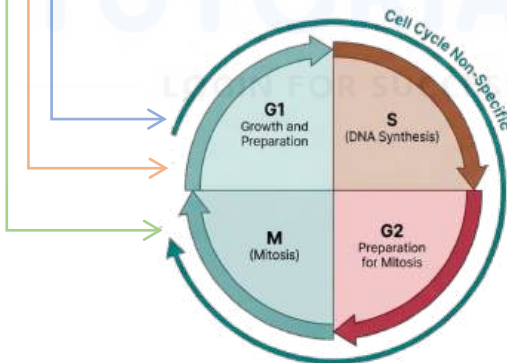
Autacoids & NSAIDs

Class	Examples	Mechanism	Key Clinical Point
Antihistamines	Loratadine, Cetirizine	H1 receptor antagonists	2nd generation agents are less sedating.
E Gout Meds	Allopurinol, Colchicine	Xanthine oxidase inhibition/ Anti- inflammatory	Differentiate acute flare vs. chronic urate- lowering therapy.
NSAIDs	Ibuprofen, Celecoxib	COX-1 and/or COX-2 inhibition	Risk of Gland Cardiovascular events with long-term use.

Chemotherapy of Neoplastic Disease:

- Combination therapy is standard for organisms like Mycobacterium tuberculosis (HRZE regimen: Isoniazid, Rifampin, Pyrazinamide, Ethambutol).

Major Chemotherapy Classes by Mechanism		
Class	Mechanism of Action	Examples
 Alkylating Agents	Induce DNA cross-linking; cell cycle non-specific.	Cyclophosphamide, Cisplatin
 Anti-metabolites	Act as purine/pyrimidine analogs to inhibit DNA synthesis (S -phase).	Methotrexate, 5-Fluorouracil
 Anti-micro Tubule Agents	Interfere with mitotic spindle function (M-phase).	Paclitaxel (Taxane), Vincristine (Vinca)
 Topoisomerase Inhibitors	Prevent DNA re-ligation after strand breakage.	Doxorubicin, Etoposide
 Targeted/Biologics	Target specific molecules (e.g., receptors, kinases).	Trastuzumab, Imatinib



Synthesis & High-Yield Principles



Core Frameworks are Essential

- ADME Framework: Master the principles of Absorption, Distribution, Metabolism, and Excretion.
 - D Receptor Theory: Understand agonist/antagonist concepts, potency vs. efficacy.
 - 0 Dose-Response: Grasp the relationship between concentration and effect.
-



High-Yield Clinical Concepts

- Link MOA to Effects: Connect the Mechanism of Action (MOA) directly to both therapeutic effects and hallmark adverse events (AEs).
-



Safety First

- Safety First: Prioritize understanding of therapeutic index, contraindications, and required monitoring for high-risk drugs.
-



Context Matters

- Context Matters: Recognize that drug choice and dosing are modified by patient factors like age, renal function, and comorbidities.

DocTutorials

The Jarvis to 'Your' Iron Man



Hardcopy Notes



High-quality videos

India's top faculty



Scan QR Code to Get the App

Contact

+91 79951 11755, 70976 34567

help@doctutorials.com

www.doctutorials.com



**PHARMACOLOGY:
A COMPREHENSIVE GUIDE**
A Visual Companion to Principles, Systems, and Therapies
