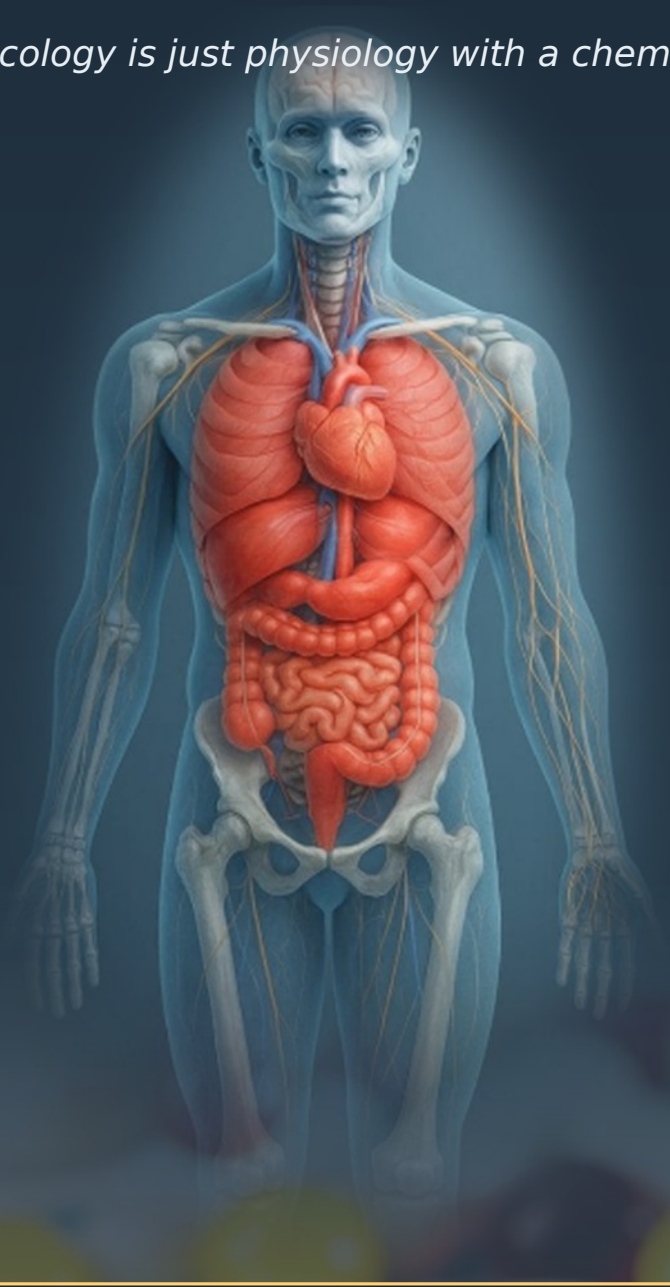
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PHYSIOLOGY
— meets —
PHARMACOLOGY

“Pharmacology is just physiology with a chemical key”

1. Physiology
2. Pathology
3. Disease
4. Drug action
5. Adverse effects



ONE DRUG CLASS
ONE PAGE
58 CLASSES
ALLIED HEALTH,
NURSING & DENTAL

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ACADEMY FOR EMERGENCY, TRAUMA & CRITICAL CARE MEDICINE

By

Dr. Gireesh Kumar K P

www.aetcm.com

How to use this book

Pharmacology is just physiology with a chemical key. A drug is easiest to remember when you know **why** it works. Each page of this book follows one drug class through the same five steps: the **normal physiology** of the system the drug acts on, the **pathology** that disturbs it, the **disease** that results, how the **drug acts** to correct the problem — and whether that brings cure, control or remission — and the important **adverse effects**, which usually follow from the same physiology. Read the five boxes from top to bottom as a story; then cover the lower boxes and try to predict the drug action and the side-effects from the physiology alone.

The drug classes follow the chapters of **Applied Pharmacology for Undergraduates** (Allied Health Sciences, Nursing & Dental). Use the two books together: this one explains the reasoning; the textbook gives classifications, **doses**, contraindications, interactions, antidotes and self-assessment questions. Always check a current formulary and local protocol before prescribing.

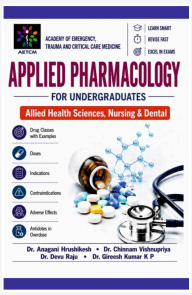
1 Physiology

2 Pathology

3 Disease caused by this problem

4 Drug action — cure, control or remission

5 Adverse effects



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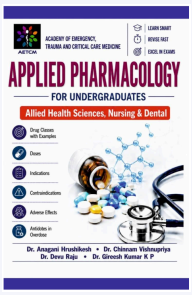
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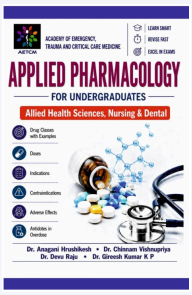
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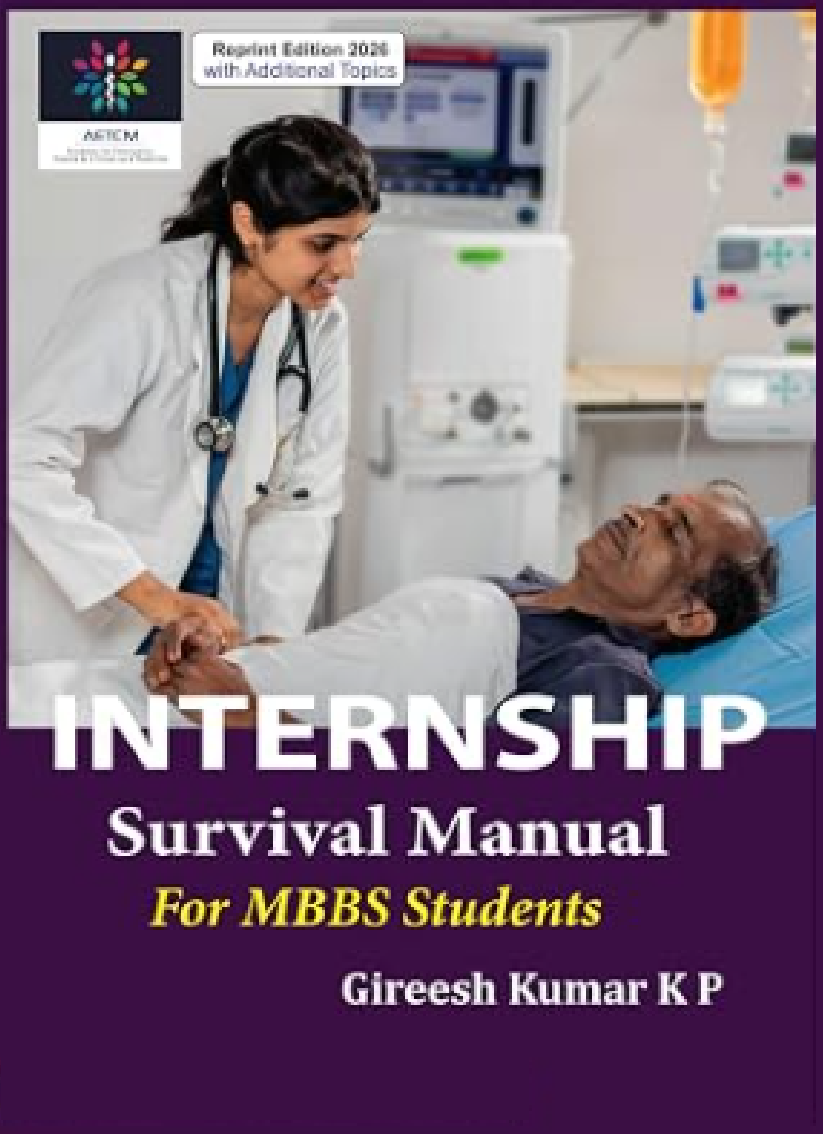
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1. Cholinergic Agonists (Parasympathomimetics)

Key drugs: Pilocarpine, bethanechol, neostigmine, pyridostigmine

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Acetylcholine (ACh), released from parasympathetic nerve endings, acts on muscarinic receptors (M1–M3) to slow the heart, constrict pupils, increase secretions, contract the bladder and gut; at the neuromuscular junction it acts on nicotinic receptors to contract skeletal muscle. Acetylcholinesterase rapidly breaks down ACh to end its action.

2 PATHOLOGY

Too little cholinergic transmission: autoantibodies against nicotinic receptors (myasthenia gravis), atonic bladder after surgery, raised intraocular pressure from blocked aqueous outflow, post-operative ileus, or residual neuromuscular blockade after anaesthesia.

3 DISEASE CAUSED BY THIS PROBLEM

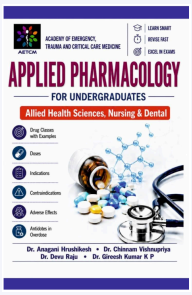
Myasthenia gravis (fatigable ptosis, diplopia, weakness), glaucoma, urinary retention, paralytic ileus, xerostomia (Sjögren's), reversal of non-depolarising muscle relaxants.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Direct agonists (pilocarpine, bethanechol) stimulate muscarinic receptors. Anticholinesterases (neostigmine, pyridostigmine) block breakdown of ACh, raising its level at the synapse — improving muscle strength in myasthenia (symptomatic control; remission needs immunotherapy/thymectomy), contracting bladder and gut, and lowering intraocular pressure.

5 ADVERSE EFFECTS

SLUDGE effects — salivation, lacrimation, urination, diarrhoea, GI cramps, emesis; bradycardia, bronchospasm, sweating, miosis/blurred vision. Overdose → cholinergic crisis (weakness, respiratory failure). Avoid in asthma, bowel/bladder obstruction.



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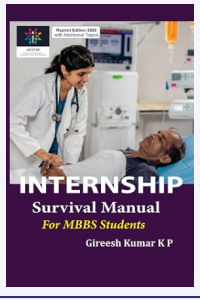
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Drug Table — 1. Cholinergic Agonists (Parasympathomimetics)

Drugs in this class: Pilocarpine, bethanechol, neostigmine, pyridostigmine

Drug	Indications	Dose (adult)	Caution
Pilocarpine	Open-angle and acute angle-closure glaucoma; xerostomia (Sjögren's, post-radiotherapy)	Eye: 1–2% drops 1 drop 3–4 times daily; oral 5 mg tds	Brow ache, blurred vision; sweating; avoid in asthma, iritis
Bethanechol	Post-operative / post-partum non-obstructive urinary retention; atonic bladder	10–50 mg orally 3–4 times daily	Contraindicated in mechanical obstruction, asthma, peptic ulcer, bradycardia
Neostigmine	Reversal of non-depolarising neuromuscular block; myasthenia gravis; paralytic ileus	Reversal 0.05 mg/kg IV (max 5 mg) with glycopyrrolate; MG 15–30 mg orally 4-hourly	Give with antimuscarinic to prevent bradycardia; avoid in bowel obstruction
Pyridostigmine	Myasthenia gravis (maintenance)	30–120 mg orally 3–6 times daily (max 1.2 g/day)	Cholinergic crisis on overdose — weakness worsens; GI upset
Edrophonium (historic)	Tensilon test for myasthenia gravis	2 mg IV test, then 8 mg	Resuscitation equipment and atropine ready; largely replaced by antibody tests

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol.
Facing page: physiology → pathology → disease → drug action → adverse effects.



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2. Anticholinergics (Muscarinic Antagonists)

Key drugs: Atropine, hyoscine, ipratropium, oxybutynin, tropicamide

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Muscarinic receptors mediate parasympathetic "rest and digest" effects: M2 slows the SA node and AV conduction; M3 contracts smooth muscle of bronchi, bladder and gut and stimulates glands; M1 is involved in gastric secretion and cognition.

2 PATHOLOGY

Excess vagal or muscarinic activity: vagal bradycardia, bronchoconstriction with mucus, detrusor overactivity, intestinal spasm, excessive secretions, and organophosphate-induced cholinergic overload.

3 DISEASE CAUSED BY THIS PROBLEM

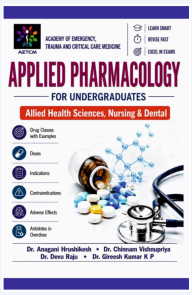
Symptomatic bradycardia, organophosphate poisoning, overactive bladder, COPD/asthma, intestinal colic/IBS spasm, motion sickness, terminal respiratory secretions; also used to dilate the pupil for eye examination.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Competitive blockade of muscarinic receptors removes vagal brake on the heart (↑ heart rate, ↑ AV conduction), relaxes bronchial, bladder and gut smooth muscle, dries secretions and dilates the pupil. Atropine reverses muscarinic toxicity in organophosphate poisoning (titrate to clear chest and HR >80).

5 ADVERSE EFFECTS

Dry mouth, blurred vision, photophobia, tachycardia, constipation, urinary retention, flushing, hyperthermia; confusion and delirium (especially elderly). Contraindicated in angle-closure glaucoma, BPH with retention, myasthenia gravis.



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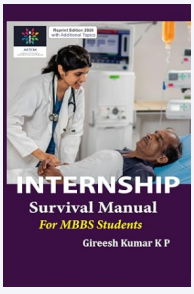
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Drug Table — 2. Anticholinergics (Muscarinic Antagonists)

Drugs in this class: Atropine, hyoscine, ipratropium, oxybutynin, tropicamide

Drug	Indications	Dose (adult)	Caution
Atropine	Symptomatic bradycardia; organophosphate poisoning; pre-anaesthetic antisialagogue	Bradycardia 0.5–1 mg IV every 3–5 min (max 3 mg); OP poisoning 1.8–3 mg IV, double every 5 min until atropinised	Tachycardia, urinary retention, delirium, hyperthermia; glaucoma
Hyoscine butylbromide	Intestinal / renal colic; death rattle; malignant bowel obstruction	20 mg IM/IV/SC, repeat; 60–120 mg/24 h by syringe driver	Tachycardia, dry mouth; does not cross blood–brain barrier
Hyoscine hydrobromide	Motion sickness; hypersalivation	300 µg orally 30 min before travel; 1.5 mg transdermal patch every 72 h	Sedation and confusion in elderly
Ipratropium	COPD and acute asthma (bronchodilator)	Nebulised 250–500 µg 4–6-hourly; MDI 20–40 µg 3–4 times daily	Dry mouth; angle-closure glaucoma if nebulised into eyes
Oxybutynin	Overactive bladder, detrusor instability, urge incontinence	2.5–5 mg orally 2–3 times daily	Dry mouth, constipation, confusion in elderly; urinary retention



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Facing page: physiology → pathology → disease → drug action → adverse effects.

3. Adrenergic Agonists (Sympathomimetics)

Key drugs: Adrenaline, noradrenaline, dobutamine, salbutamol, phenylephrine

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

The sympathetic system ("fight or flight") releases noradrenaline at nerve endings and adrenaline from the adrenal medulla. α1 receptors constrict vessels; β1 increase heart rate and contractility; β2 relax bronchi and vessels in muscle and shift potassium into cells.

2 PATHOLOGY

Failure of circulatory or airway tone: profound vasodilation and capillary leak (anaphylaxis, sepsis), pump failure (cardiogenic shock), cardiac arrest, or bronchoconstriction.

3 DISEASE CAUSED BY THIS PROBLEM

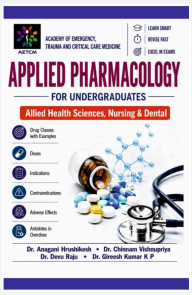
Anaphylaxis, cardiac arrest, septic and cardiogenic shock, acute asthma, hypotension during anaesthesia, nasal congestion.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Adrenaline (α + β) restores blood pressure, reverses airway oedema and bronchospasm, and stabilises mast cells — life-saving in anaphylaxis. Noradrenaline (mainly α1) restores vascular tone in septic shock. Dobutamine (β1) increases cardiac output. Salbutamol (β2) relaxes bronchial smooth muscle.

5 ADVERSE EFFECTS

Tachycardia, arrhythmias, hypertension, myocardial ischaemia, anxiety, tremor, hyperglycaemia, hypokalaemia, lactic acidosis; extravasation causes tissue necrosis (noradrenaline — treat with phentolamine).



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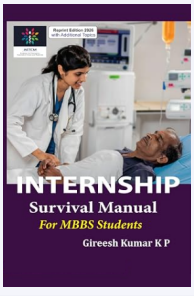
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Drug Table — 3. Adrenergic Agonists (Sympathomimetics)

Drugs in this class: Adrenaline, noradrenaline, dobutamine, salbutamol, phenylephrine

Drug	Indications	Dose (adult)	Caution
Adrenaline	Anaphylaxis; cardiac arrest; severe asthma; with local anaesthetic	Anaphylaxis 0.5 mg IM (1:1000); cardiac arrest 1 mg IV (1:10 000) every 3–5 min	Arrhythmias, hypertension; check strength of ampoule carefully
Noradrenaline	Septic and vasodilatory shock	0.05–0.5 µg/kg/min IV infusion (central line)	Extravasation necrosis; peripheral ischaemia; reflex bradycardia
Dobutamine	Cardiogenic shock; acute heart failure; stress echocardiography	2.5–20 µg/kg/min IV infusion	Tachyarrhythmias, hypotension (vasodilatation), myocardial ischaemia
Salbutamol	Acute asthma, COPD; hyperkalaemia; preterm labour (historic)	Inhaled 100–200 µg PRN; nebulised 2.5–5 mg; hyperkalaemia 10–20 mg nebulised	Tremor, tachycardia, hypokalaemia
Phenylephrine	Hypotension during spinal anaesthesia; nasal decongestant; mydriasis	50–100 µg IV bolus; 2.5–10% eye drops	Reflex bradycardia, hypertension



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4. Alpha-Adrenergic Blockers

Key drugs: Prazosin, doxazosin, tamsulosin, phenoxybenzamine

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

α1 receptors on vascular smooth muscle maintain arteriolar tone, and α1A receptors in the prostate and bladder neck keep the outlet closed. Noradrenaline acting on these receptors raises blood pressure and resistance to urine flow.

2 PATHOLOGY

Excess α-mediated tone: enlarged prostate with increased smooth-muscle tone obstructs urine flow; catecholamine-secreting tumours (phaeochromocytoma) cause severe paroxysmal hypertension.

3 DISEASE CAUSED BY THIS PROBLEM

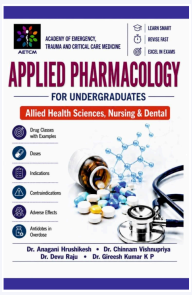
Benign prostatic hyperplasia (lower urinary tract symptoms, retention), hypertension (add-on), phaeochromocytoma, ureteric stones (expulsion), Raynaud's phenomenon.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Blocking α1 receptors relaxes prostate and bladder-neck smooth muscle (improves urine flow within days) and dilates arterioles (lowers BP). Phenoxybenzamine (irreversible, non-selective) is given before surgery for phaeochromocytoma — alpha blockade must precede beta blockade.

5 ADVERSE EFFECTS

First-dose postural hypotension and syncope (take at bedtime), dizziness, falls in elderly, reflex tachycardia, nasal congestion, retrograde ejaculation, intra-operative floppy iris syndrome (tamsulosin).



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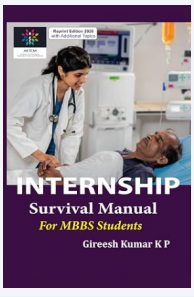
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Drug Table — 4. Alpha-Adrenergic Blockers

Drugs in this class: Prazosin, doxazosin, tamsulosin, phenoxybenzamine

Drug	Indications	Dose (adult)	Caution
Prazosin	Hypertension (add-on); scorpion sting envenomation; PTSD nightmares	Start 0.5 mg at night, then 1–5 mg bd; scorpion sting 0.5 mg orally 3-hourly (adult)	First-dose postural hypotension — give at bedtime; dizziness
Doxazosin	Hypertension; benign prostatic hyperplasia	1 mg daily, increase to 4–8 mg (max 16 mg)	Postural hypotension, dizziness, oedema
Tamsulosin	BPH lower urinary tract symptoms; ureteric stone expulsion	400 µg once daily after food	Floppy-iris syndrome in cataract surgery; retrograde ejaculation
Phenoxybenzamine	Pre-operative preparation of phaeochromocytoma	10 mg bd, increase to 1–2 mg/kg/day in divided doses	Irreversible blockade; marked postural hypotension, tachycardia — add beta-blocker after alpha-blockade
Phentolamine	Hypertensive crisis in phaeochromocytoma; noradrenaline extravasation	2–5 mg IV repeated; 5–10 mg in 10 mL saline infiltrated locally	Tachycardia, arrhythmias, hypotension

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.



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5. ACE Inhibitors

Key drugs: Enalapril, ramipril, lisinopril, perindopril, captopril

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

The renin–angiotensin–aldosterone system (RAAS) maintains blood pressure and volume: renin converts angiotensinogen to angiotensin I; angiotensin-converting enzyme (ACE) forms angiotensin II, which constricts arterioles, releases aldosterone (salt and water retention) and stimulates cardiac and vascular remodelling. ACE also breaks down bradykinin.

2 PATHOLOGY

Chronic RAAS overactivity raises peripheral resistance and volume, causes left ventricular hypertrophy, fibrosis of heart and kidney, and raised glomerular pressure leading to proteinuria.

3 DISEASE CAUSED BY THIS PROBLEM

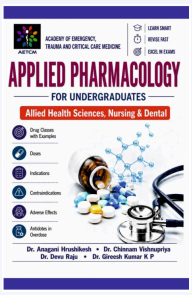
Hypertension, heart failure with reduced ejection fraction, post-myocardial infarction remodelling, diabetic nephropathy and proteinuric chronic kidney disease.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Inhibiting ACE lowers angiotensin II and aldosterone, dilates arterioles, reduces preload and afterload, reduces intraglomerular pressure and proteinuria, and limits remodelling — prolonging survival in heart failure and slowing kidney disease progression (control, not cure).

5 ADVERSE EFFECTS

Dry cough (bradykinin), first-dose hypotension, hyperkalaemia, acute kidney injury (especially with renal artery stenosis, NSAIDs, diuretics), angioedema (rare, life-threatening), taste disturbance. Contraindicated in pregnancy.



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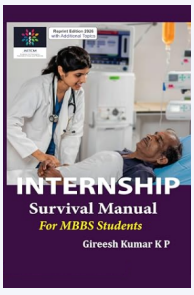
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Drug Table — 5. ACE Inhibitors

Drugs in this class: Enalapril, ramipril, lisinopril, perindopril, captopril

Drug	Indications	Dose (adult)	Caution
Enalapril	Hypertension; heart failure; post-MI; diabetic nephropathy	2.5–5 mg daily, up to 20 mg bd	Dry cough, hyperkalaemia, first-dose hypotension; contraindicated in pregnancy
Ramipril	Hypertension; heart failure; cardiovascular prevention (HOPE)	1.25–2.5 mg daily, up to 10 mg daily	Check creatinine and potassium at 1–2 weeks
Lisinopril	Hypertension; heart failure; post-MI	2.5–10 mg daily (max 40 mg)	Angioedema (more in Black patients); AKI with NSAIDs and diuretics
Perindopril	Hypertension; stable coronary disease; secondary stroke prevention	2–8 mg daily	Avoid in bilateral renal artery stenosis
Captopril	Hypertensive urgency (historic); heart failure; renography	6.25–25 mg 2–3 times daily	Short-acting; taste disturbance, rash, neutropenia

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.



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6. Angiotensin Receptor Blockers (ARBs)

Key drugs: Losartan, valsartan, candesartan, telmisartan

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Angiotensin II acts on AT1 receptors to constrict vessels, release aldosterone, increase sympathetic activity and promote cardiac and renal fibrosis; AT2 receptors have opposing, protective effects.

2 PATHOLOGY

Sustained AT1 stimulation causes hypertension, left ventricular hypertrophy, heart failure progression and glomerular hyperfiltration with proteinuria.

3 DISEASE CAUSED BY THIS PROBLEM

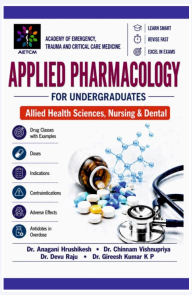
Hypertension, heart failure (when ACE inhibitor not tolerated), diabetic and proteinuric kidney disease, post-MI; combined with sacubitril (ARNI) in heart failure.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Selective AT1 blockade produces the same haemodynamic and renal benefits as ACE inhibitors, without raising bradykinin — hence no cough. Controls blood pressure and slows organ damage; no cure.

5 ADVERSE EFFECTS

Hypotension, dizziness, hyperkalaemia, acute kidney injury, rarely angioedema. Contraindicated in pregnancy. Do not combine with ACE inhibitors (no benefit, more harm).



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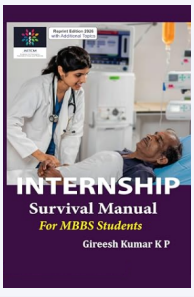
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Drug Table — 6. Angiotensin Receptor Blockers (ARBs)

Drugs in this class: Losartan, valsartan, candesartan, telmisartan

Drug	Indications	Dose (adult)	Caution
Losartan	Hypertension; diabetic nephropathy; ACE-inhibitor cough	50 mg daily (25–100 mg)	Hyperkalaemia; contraindicated in pregnancy; mild uricosuric effect
Telmisartan	Hypertension; cardiovascular risk reduction	40–80 mg daily	Long half-life; check potassium and creatinine
Valsartan	Hypertension; heart failure; post-MI	80–320 mg daily (40–160 mg bd in HF)	Hypotension; with sacubitril as ARNI in HFrEF
Candesartan	Hypertension; heart failure	8–32 mg daily	Renal impairment with volume depletion
Sacubitril–valsartan (ARNI)	Heart failure with reduced ejection fraction	24/26 mg bd → 97/103 mg bd	Stop ACE inhibitor 36 h before; angioedema, hypotension

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.



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7. Calcium Channel Blockers

Key drugs: Amlodipine, nifedipine, verapamil, diltiazem

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

L-type calcium channels let calcium enter vascular smooth muscle and cardiac cells. In vessels, calcium entry causes contraction (vascular tone); in the heart it drives contractility, SA node pacemaker activity and AV node conduction.

2 PATHOLOGY

Increased vascular tone and stiffness raise blood pressure; coronary vasospasm and increased myocardial oxygen demand cause angina; rapid AV conduction allows fast ventricular rates in atrial fibrillation.

3 DISEASE CAUSED BY THIS PROBLEM

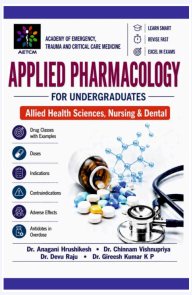
Hypertension (especially isolated systolic in elderly), stable and vasospastic angina, supraventricular tachycardia, rate control in atrial fibrillation, Raynaud's phenomenon.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Dihydropyridines (amlodipine, nifedipine) mainly dilate arterioles — lower BP and relieve angina. Non-dihydropyridines (verapamil, diltiazem) slow the heart and AV conduction — control heart rate and terminate SVT.

5 ADVERSE EFFECTS

Dihydropyridines — ankle oedema, flushing, headache, reflex tachycardia, gum hypertrophy. Verapamil/diltiazem — bradycardia, heart block, worsening heart failure, constipation. Never combine verapamil with beta-blockers (asystole risk).



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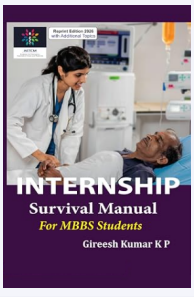
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Drug Table — 7. Calcium Channel Blockers

Drugs in this class: Amlodipine, nifedipine, verapamil, diltiazem

Drug	Indications	Dose (adult)	Caution
Amlodipine	Hypertension; chronic stable angina	5–10 mg once daily	Ankle oedema, flushing, headache, gum hypertrophy
Nifedipine (modified release)	Hypertension; angina; preterm labour (tocolysis); Raynaud's	MR 20–60 mg daily; tocolysis 20 mg orally then 10–20 mg 6–8-hourly	Avoid short-acting capsules — reflex tachycardia, stroke/MI risk
Verapamil	SVT; rate control in AF; angina; migraine and cluster headache prophylaxis	SVT 5–10 mg IV over 2 min; oral 40–120 mg tds	Never with IV beta-blocker (asystole); constipation; heart failure
Diltiazem	Angina; AF rate control; hypertension	60–120 mg tds or MR 120–360 mg daily	Bradycardia, AV block; CYP3A4 interactions
Nimodipine	Prevention of vasospasm after subarachnoid haemorrhage	60 mg orally 4-hourly for 21 days	Hypotension

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8. Beta-Blockers

Key drugs: Atenolol, bisoprolol, metoprolol, propranolol, carvedilol

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

β1 receptors in the heart increase heart rate, contractility and AV conduction and release renin from the kidney; β2 receptors relax bronchi and blood vessels. Sympathetic drive matches cardiac output to demand.

2 PATHOLOGY

Chronic sympathetic overactivity raises myocardial oxygen demand (angina), drives arrhythmias, promotes remodelling and apoptosis in heart failure, and maintains high blood pressure; thyrotoxicosis and anxiety amplify adrenergic symptoms.

3 DISEASE CAUSED BY THIS PROBLEM

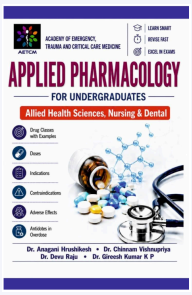
Angina, post-myocardial infarction, heart failure with reduced EF, atrial fibrillation rate control, hypertension, thyrotoxicosis symptoms, essential tremor, migraine prophylaxis, portal hypertension (propranolol), glaucoma (timolol).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Blocking β1 slows heart rate, reduces contractility and oxygen demand, prolongs diastolic filling, suppresses arrhythmias and renin — reduces mortality after MI and in heart failure (long-term control).

5 ADVERSE EFFECTS

Bradycardia, heart block, fatigue, cold extremities, bronchospasm (avoid in asthma), masked hypoglycaemia, sleep disturbance, erectile dysfunction; abrupt withdrawal can precipitate angina/MI. Avoid in acute decompensated heart failure.



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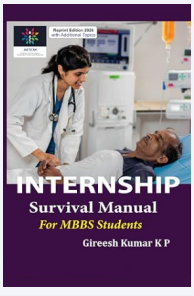
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Drug Table — 8. Beta-Blockers

Drugs in this class: Atenolol, bisoprolol, metoprolol, propranolol, carvedilol

Drug	Indications	Dose (adult)	Caution
Atenolol	Hypertension; angina; rate control	25–100 mg daily	Renally excreted — reduce in CKD; bradycardia, cold peripheries
Bisoprolol	Heart failure (HFrEF); angina; AF; hypertension	1.25 mg daily titrated to 10 mg	Start low and slow in heart failure; avoid in asthma
Metoprolol	Hypertension; angina; acute MI; arrhythmias; HF (succinate)	25–100 mg bd; IV 5 mg up to 3 doses in MI/SVT	Bronchospasm, bradycardia, fatigue
Propranolol	Thyrotoxicosis; portal hypertension (variceal prophylaxis); migraine; anxiety; essential tremor	10–40 mg 2–3 times daily	Non-selective — contraindicated in asthma; masks hypoglycaemia
Carvedilol	Heart failure; portal hypertension; hypertension	3.125 mg bd titrated to 25 mg bd	Alpha-blocking — postural hypotension
Esmolol	Perioperative tachycardia and hypertension; aortic dissection	500 µg/kg IV over 1 min, then 50–200 µg/kg/min	Ultra-short acting — needs infusion monitoring

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9. Nitrates

Key drugs: Glyceryl trinitrate (GTN), isosorbide mononitrate, isosorbide dinitrate

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Nitric oxide (NO) produced by endothelium activates guanylate cyclase in smooth muscle, raising cyclic GMP and causing relaxation. Venous dilation reduces return of blood to the heart (preload); arterial dilation reduces afterload; coronary dilation improves blood flow.

2 PATHOLOGY

Atherosclerotic coronary narrowing limits oxygen supply; when demand rises (exercise), ischaemia causes angina. Acute left ventricular failure raises filling pressure, causing pulmonary oedema.

3 DISEASE CAUSED BY THIS PROBLEM

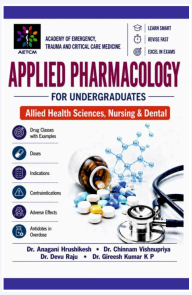
Stable angina, unstable angina/acute coronary syndrome (symptom relief), acute pulmonary oedema, hypertensive emergency with heart failure; oesophageal spasm.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Nitrates release NO, mainly dilating veins: lower preload and wall tension, reducing myocardial oxygen demand; also dilate coronary arteries. They relieve angina rapidly and reduce pulmonary congestion — symptomatic relief, not disease modification.

5 ADVERSE EFFECTS

Throbbing headache, flushing, postural hypotension, dizziness, reflex tachycardia, tolerance with continuous use. Never give with PDE-5 inhibitors (sildenafil) — severe hypotension; caution in right ventricular infarction and severe aortic stenosis.



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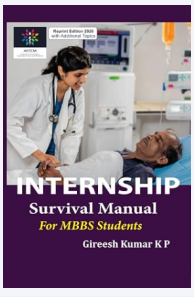
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Drug Table — 9. Nitrates

Drugs in this class: Glyceryl trinitrate (GTN), isosorbide mononitrate, isosorbide dinitrate

Drug	Indications	Dose (adult)	Caution
Glyceryl trinitrate (GTN) sublingual	Acute angina; prophylaxis before exertion	300–600 µg SL tablet or 400 µg spray, repeat every 5 min (max 3)	Headache, hypotension; contraindicated with sildenafil/tadalafil
GTN infusion	Unstable angina; acute LVF; hypertensive emergency	10–200 µg/min IV, titrated to BP	Tolerance after 24 h; methaemoglobinaemia (rare)
Isosorbide mononitrate	Angina prophylaxis	20 mg bd (asymmetric 8 am and 2 pm) or MR 30–60 mg daily	Nitrate-free interval to prevent tolerance
Isosorbide dinitrate	Angina; with hydralazine in heart failure	10–40 mg 2–3 times daily	Postural hypotension, headache
GTN transdermal patch	Angina prophylaxis	5–15 mg/24 h patch, remove at night	Remove patch for 8–12 h daily (tolerance)

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.



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10. Antiarrhythmic Drugs (Vaughan-Williams)

Key drugs: Lidocaine, flecainide, amiodarone, adenosine, sotalol

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

The cardiac action potential has phases: phase 0 depolarisation (Na+ entry), plateau (Ca2+ entry), repolarisation (K+ exit). The SA node fires automatically; the AV node delays conduction so atria and ventricles contract in sequence.

2 PATHOLOGY

Arrhythmias arise from abnormal automaticity (ectopic foci), triggered activity (after-depolarisations — e.g. long QT) or re-entry circuits (scar after MI, accessory pathways); electrolyte disturbance and ischaemia promote them.

3 DISEASE CAUSED BY THIS PROBLEM

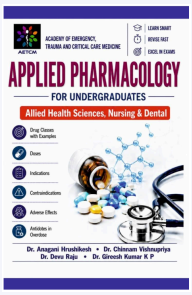
Atrial fibrillation/flutter, supraventricular tachycardia (AVNRT, WPW), ventricular tachycardia, ventricular fibrillation, ectopic beats.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Class I block Na+ channels (slow conduction); Class II beta-blockers; Class III (amiodarone, sotalol) block K+ channels — prolong repolarisation and refractory period; Class IV calcium blockers slow the AV node. Adenosine transiently blocks the AV node to terminate SVT (cure for that episode; ablation can cure re-entry).

5 ADVERSE EFFECTS

All can be pro-arrhythmic. Amiodarone — thyroid dysfunction, pulmonary fibrosis, hepatitis, photosensitivity, corneal deposits, slate-grey skin, many interactions. Flecainide — dangerous in structural heart disease. Adenosine — flushing, chest tightness, brief asystole, bronchospasm.



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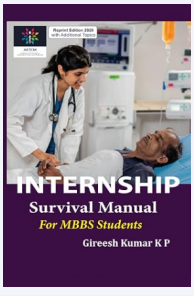
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Drug Table — 10. Antiarrhythmic Drugs (Vaughan-Williams)

Drugs in this class: Lidocaine, flecainide, amiodarone, adenosine, sotalol

Drug	Indications	Dose (adult)	Caution
Adenosine	Termination of narrow-complex SVT (AVNRT/AVRT)	6 mg rapid IV bolus, then 12 mg, then 12 mg, with saline flush	Transient asystole, chest discomfort, bronchospasm — avoid in severe asthma
Amiodarone	VF/pulseless VT (after 3rd shock); AF; VT	Arrest 300 mg IV, then 150 mg; loading 5 mg/kg over 20–60 min then 900 mg/24 h	Thyroid, lung, liver toxicity; photosensitivity; QT prolongation; many interactions
Lidocaine	Ventricular arrhythmias (alternative to amiodarone)	1–1.5 mg/kg IV bolus, then 1–4 mg/min	CNS toxicity: perioral tingling, seizures
Flecainide	Paroxysmal AF in structurally normal heart ('pill in the pocket'); SVT	50–150 mg bd; pill-in-pocket 200–300 mg	Contraindicated after MI / structural heart disease (CAST trial)
Sotalol	Ventricular arrhythmias; AF maintenance	40–160 mg bd	Torsades de pointes — monitor QTc and potassium
Digoxin (see class 11)	AF rate control in heart failure	Loading 0.5–1 mg; maintenance 62.5–250 µg daily	Toxicity with hypokalaemia, renal failure

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.



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11. Digoxin and Positive Inotropes

Key drugs: Digoxin, dobutamine, milrinone

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Cardiac contraction depends on intracellular calcium. The Na+/K+-ATPase pump keeps sodium low inside cells; the Na+/Ca2+ exchanger uses this gradient to remove calcium. Vagal tone slows conduction through the AV node.

2 PATHOLOGY

In heart failure the weakened ventricle cannot maintain cardiac output; in atrial fibrillation rapid irregular impulses reach the ventricles, causing fast ventricular rates and worsening failure.

3 DISEASE CAUSED BY THIS PROBLEM

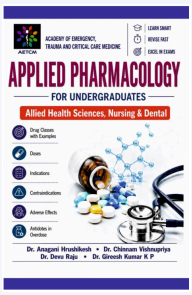
Atrial fibrillation with fast ventricular rate (especially with heart failure), symptomatic heart failure with reduced EF; cardiogenic shock (dobutamine, milrinone).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Digoxin inhibits Na+/K+-ATPase → more intracellular Na+ → less Ca2+ removal → stronger contraction; it also increases vagal tone, slowing AV conduction and ventricular rate. Reduces hospital admissions in HF but not mortality. Dobutamine (β1) and milrinone (PDE3 inhibitor) increase contractility acutely.

5 ADVERSE EFFECTS

Narrow therapeutic index. Toxicity: nausea, vomiting, anorexia, visual disturbance (yellow-green vision), confusion, bradycardia, heart block, ventricular arrhythmias — worsened by hypokalaemia, renal failure, amiodarone, verapamil. Antidote: digoxin-specific antibody fragments (DigiFab).



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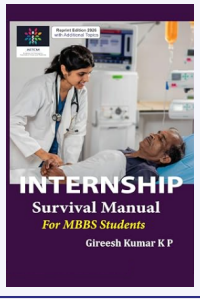
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Drug Table — 11. Digoxin and Positive Inotropes

Drugs in this class: Digoxin, dobutamine, milrinone

Drug	Indications	Dose (adult)	Caution
Digoxin	AF with rapid ventricular rate (especially with heart failure); HFrEF symptom control	Load 500 µg then 250 µg after 6 h; maintenance 62.5–250 µg daily	Narrow therapeutic index; toxicity with hypokalaemia, renal failure, elderly — nausea, xanthopsia, arrhythmias; antidote Digibind
Dobutamine	Cardiogenic shock; low-output heart failure	2.5–10 µg/kg/min IV	Tachyarrhythmias; ischaemia
Milrinone	Acute decompensated heart failure; post-cardiac surgery low output	Load 50 µg/kg over 10 min, then 0.375–0.75 µg/kg/min	Hypotension, arrhythmias; reduce in renal impairment
Levosimendan	Acute decompensated heart failure	0.05–0.2 µg/kg/min for 24 h	Hypotension, hypokalaemia
Ivabradine	HFrEF with sinus rhythm HR ≥ 70 despite beta-blocker; angina	5 mg bd → 7.5 mg bd	Phosphenes (visual brightness), bradycardia; not in AF

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol.
Facing page: physiology → pathology → disease → drug action → adverse effects.



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12. Lipid-Lowering Drugs (Statins and Others)

Key drugs: Atorvastatin, rosuvastatin, ezetimibe, fenofibrate, PCSK9 inhibitors

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

The liver makes cholesterol via HMG-CoA reductase and clears LDL from blood through LDL receptors. Cholesterol is needed for cell membranes, steroid hormones and bile acids; LDL carries cholesterol to tissues and HDL returns it to the liver.

2 PATHOLOGY

Excess LDL enters the arterial wall, becomes oxidised and is engulfed by macrophages (foam cells), forming atherosclerotic plaques that narrow arteries and may rupture, causing thrombosis.

3 DISEASE CAUSED BY THIS PROBLEM

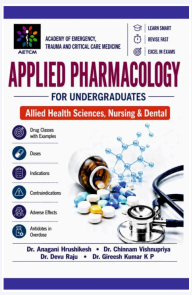
Coronary artery disease, myocardial infarction, ischaemic stroke, peripheral arterial disease, familial hypercholesterolaemia; severe hypertriglyceridaemia causes pancreatitis.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Statins inhibit HMG-CoA reductase → less liver cholesterol → more LDL receptors → LDL falls 30–60%; they also stabilise plaques. Ezetimibe blocks intestinal absorption; PCSK9 inhibitors increase LDL receptors; fibrates lower triglycerides. Reduce heart attacks and strokes (prevention, not cure).

5 ADVERSE EFFECTS

Myalgia, myopathy, rarely rhabdomyolysis (risk ↑ with macrolides, azoles, fibrates, hypothyroidism); raised liver enzymes; small rise in diabetes risk. Avoid in pregnancy. Fibrates — gallstones, myopathy with statins.



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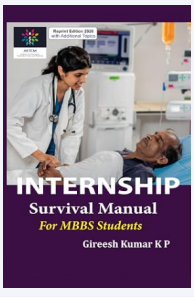
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Drug Table — 12. Lipid-Lowering Drugs (Statins and Others)

Drugs in this class: Atorvastatin, rosuvastatin, ezetimibe, fenofibrate, PCSK9 inhibitors

Drug	Indications	Dose (adult)	Caution
Atorvastatin	Primary and secondary prevention of ASCVD; hypercholesterolaemia	10–80 mg once daily (80 mg after ACS)	Myalgia, raised transaminases; rhabdomyolysis with macrolides, azoles
Rosuvastatin	High-intensity lipid lowering	5–40 mg once daily	Lower doses in Asian patients and renal impairment
Simvastatin	Hypercholesterolaemia	20–40 mg at night	Many CYP3A4 interactions (amlodipine, diltiazem, clarithromycin)
Ezetimibe	Add-on to statin; statin intolerance	10 mg daily	Well tolerated; diarrhoea
Fenofibrate	Severe hypertriglyceridaemia; mixed dyslipidaemia	145–160 mg daily	Myopathy with statins; gallstones; raised creatinine
PCSK9 inhibitors (evolocumab)	Familial hypercholesterolaemia; very high risk not at target	140 mg SC every 2 weeks or 420 mg monthly	Injection-site reactions; cost

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol.
Facing page: physiology → pathology → disease → drug action → adverse effects.



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13. Centrally-Acting and Other Antihypertensives

Key drugs: Methyldopa, clonidine, moxonidine, hydralazine, minoxidil

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

The brainstem vasomotor centre controls sympathetic outflow to heart and blood vessels; α2 receptors in the brain reduce this outflow. Arteriolar smooth muscle tone determines peripheral resistance.

2 PATHOLOGY

Increased sympathetic outflow and arteriolar tone sustain high blood pressure; in pregnancy, abnormal placental vessels cause pre-eclampsia with hypertension and organ damage.

3 DISEASE CAUSED BY THIS PROBLEM

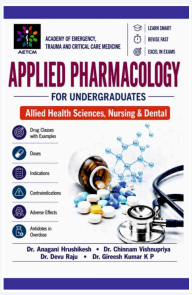
Resistant hypertension, hypertension in pregnancy and pre-eclampsia (methyldopa, labetalol, nifedipine, hydralazine), hypertensive emergencies, heart failure (hydralazine + nitrate).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Methyldopa and clonidine stimulate central α2 receptors, reducing sympathetic outflow and blood pressure. Hydralazine and minoxidil directly relax arteriolar smooth muscle. These control blood pressure; they do not cure the underlying disease.

5 ADVERSE EFFECTS

Methyldopa — sedation, depression, haemolytic anaemia (positive Coombs), hepatitis. Clonidine — dry mouth, sedation, rebound hypertension on abrupt stop. Hydralazine — reflex tachycardia, headache, lupus-like syndrome. Minoxidil — fluid retention, hirsutism.



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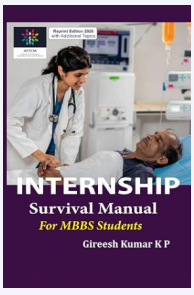
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Drug Table — 13. Centrally-Acting and Other Antihypertensives

Drugs in this class: Methyldopa, clonidine, moxonidine, hydralazine, minoxidil

Drug	Indications	Dose (adult)	Caution
Methyldopa	Hypertension in pregnancy	250 mg bd–tds, up to 3 g/day	Sedation, depression, positive Coombs test, haemolytic anaemia, hepatitis
Clonidine	Resistant hypertension; opioid/ alcohol withdrawal; ADHD	50–300 µg bd–tds	Rebound hypertension on abrupt stopping; sedation, dry mouth
Moxonidine	Essential hypertension	200–600 µg daily	Dry mouth; avoid in severe heart failure
Hydralazine	Severe hypertension in pregnancy; heart failure (with nitrate)	5–10 mg IV slowly, repeat 20 min; oral 25–50 mg tds	Reflex tachycardia, headache; drug-induced lupus
Labetalol	Hypertension in pregnancy; hypertensive emergency	Oral 100–400 mg bd; IV 20 mg then 40–80 mg every 10 min (max 300 mg)	Avoid in asthma; postural hypotension
Minoxidil	Resistant hypertension; topical for alopecia	2.5–40 mg daily (with beta-blocker + diuretic)	Fluid retention, hirsutism, pericardial effusion

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.



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14. Loop Diuretics

Key drugs: Furosemide, torsemide, bumetanide

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

In the thick ascending limb of the loop of Henle, the Na+-K+-2Cl− cotransporter reabsorbs about 25% of filtered sodium, creating the concentrated medulla needed for water reabsorption.

2 PATHOLOGY

In heart failure, cirrhosis and nephrotic syndrome, the kidney retains sodium and water (RAAS activation, low effective circulating volume), leading to oedema and pulmonary congestion; impaired excretion of potassium or calcium may also occur.

3 DISEASE CAUSED BY THIS PROBLEM

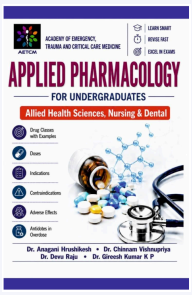
Acute pulmonary oedema, chronic heart failure with congestion, oedema of renal or hepatic disease, hypercalcaemia (with saline), hyperkalaemia, resistant hypertension with CKD.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Blocking the Na-K-2Cl cotransporter causes powerful natriuresis and diuresis ("high-ceiling"); IV furosemide also dilates veins, reducing preload within minutes. Relieves congestion and breathlessness — symptom control, not cure.

5 ADVERSE EFFECTS

Hypokalaemia, hyponatraemia, hypomagnesaemia, hypocalcaemia, dehydration and hypotension, pre-renal AKI, metabolic alkalosis, hyperuricaemia/gout, hyperglycaemia, ototoxicity (rapid high IV doses), urinary incontinence in elderly.



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Drug Table — 14. Loop Diuretics

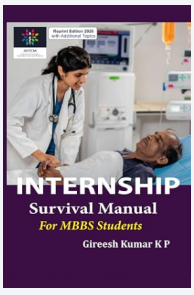
Drugs in this class: Furosemide, torsemide, bumetanide

Drug	Indications	Dose (adult)	Caution
Furosemide	Acute pulmonary oedema; oedema (heart, liver, renal failure); hypercalcaemia (after rehydration)	20–40 mg IV/oral, up to 80–250 mg; infusion 5–20 mg/h	Hypokalaemia, hyponatraemia, dehydration, ototoxicity (rapid IV), gout
Torsemide	Oedema; hypertension; heart failure	5–20 mg daily (up to 200 mg)	Better oral bioavailability than furosemide; electrolytes
Bumetanide	Oedema resistant to furosemide (gut oedema)	1 mg daily, up to 5 mg	1 mg ≈ 40 mg furosemide; myalgia
Ethacrynic acid	Loop diuretic in sulfonamide allergy	50–100 mg daily	Most ototoxic loop diuretic
Acetazolamide (carbonic anhydrase inhibitor)	Glaucoma; altitude sickness; idiopathic intracranial hypertension; metabolic alkalosis	250 mg 1–4 times daily	Metabolic acidosis, hypokalaemia, paraesthesia; sulfonamide allergy

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.

★ REMEMBER — EXAM POINTS

- Loop diuretics act on the Na^+–K^+–$2Cl^-$ cotransporter in the thick ascending limb — the most powerful diuretics (up to 25% of filtered sodium).
- IV furosemide also causes early venodilatation — relief in pulmonary oedema starts before diuresis.
- Monitor weight, urine output, sodium, potassium and creatinine daily in hospital.
- Diuretic resistance: increase dose, give twice daily or by infusion, or add metolazone (sequential nephron blockade).
- Ototoxicity rises with rapid IV injection (> 4 mg/min) and with aminoglycosides.



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15. Thiazide and Thiazide-Like Diuretics

Key drugs: Hydrochlorothiazide, chlorthalidone, indapamide, metolazone

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

The distal convoluted tubule reabsorbs about 5–10% of filtered sodium via the Na+-Cl− cotransporter; this segment also reabsorbs calcium and contributes to urine dilution.

2 PATHOLOGY

Salt and water retention and increased vascular tone sustain essential hypertension; excessive calcium in urine forms kidney stones; in heart failure, diuretic resistance develops from distal sodium reabsorption.

3 DISEASE CAUSED BY THIS PROBLEM

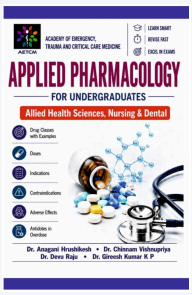
Hypertension (first-line, especially elderly), mild oedema, calcium kidney stones (hypercalciuria), nephrogenic diabetes insipidus, resistant oedema (metolazone with loop diuretic).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Blocking the Na-Cl cotransporter produces modest natriuresis and, long term, vasodilation — lowering blood pressure and reducing stroke and heart failure. Increases calcium reabsorption (reduces stones). Long-term control of BP.

5 ADVERSE EFFECTS

Hyponatraemia (especially elderly women), hypokalaemia, hypomagnesaemia, hypercalcaemia, hyperuricaemia/gout, hyperglycaemia, dyslipidaemia, erectile dysfunction, photosensitivity. Less effective when eGFR <30 (except metolazone).



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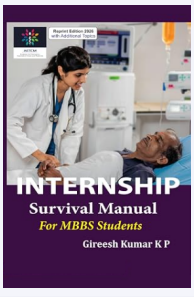
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Drug Table — 15. Thiazide and Thiazide-Like Diuretics

Drugs in this class: Hydrochlorothiazide, chlorthalidone, indapamide, metolazone

Drug	Indications	Dose (adult)	Caution
Hydrochlorothiazide	Hypertension; oedema; nephrogenic diabetes insipidus; calcium stones	12.5–25 mg daily	Hyponatraemia, hypokalaemia, hyperuricaemia, hyperglycaemia, photosensitivity
Chlorthalidone	Hypertension (long-acting, outcome evidence)	12.5–25 mg daily	Electrolyte disturbance — check sodium and potassium
Indapamide	Hypertension; stroke prevention (PROGRESS)	1.5 mg MR or 2.5 mg daily	Less metabolic effect; hyponatraemia in elderly women
Metolazone	Resistant oedema (with loop diuretic — sequential nephron blockade)	2.5–5 mg daily or alternate days	Profound diuresis, hypokalaemia — monitor closely
Bendroflumethiazide	Hypertension; oedema	2.5 mg daily	Ineffective when eGFR < 30 (except metolazone)

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol.
Facing page: physiology → pathology → disease → drug action → adverse effects.



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16. Potassium-Sparing Diuretics and Aldosterone Antagonists

Key drugs: Spironolactone, eplerenone, amiloride, triamterene

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

In the collecting duct, aldosterone binds mineralocorticoid receptors to increase epithelial sodium channels (ENaC) and Na+/K+-ATPase, reabsorbing sodium and secreting potassium and hydrogen ions.

2 PATHOLOGY

Excess aldosterone (primary hyperaldosteronism or secondary in heart failure and cirrhosis) causes salt retention, hypokalaemia, hypertension and myocardial and vascular fibrosis.

3 DISEASE CAUSED BY THIS PROBLEM

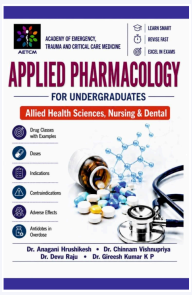
Heart failure with reduced EF, ascites in cirrhosis, primary hyperaldosteronism (Conn's syndrome), resistant hypertension, hypokalaemia from other diuretics, nephrotic oedema.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Spironolactone/eplerenone block aldosterone receptors — natriuresis with potassium retention, and anti-fibrotic effects that reduce mortality in heart failure. Amiloride/triamterene directly block ENaC. Control, not cure (Conn's adenoma may be cured surgically).

5 ADVERSE EFFECTS

Hyperkalaemia (especially with ACEi/ARB, CKD, potassium supplements), hyponatraemia, AKI, gynaecomastia and breast tenderness (spironolactone), menstrual irregularity, impotence.



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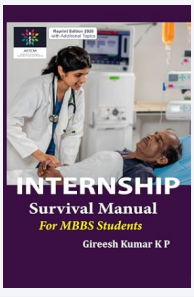
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Drug Table — 16. Potassium-Sparing Diuretics and Aldosterone Antagonists

Drugs in this class: Spironolactone, eplerenone, amiloride, triamterene

Drug	Indications	Dose (adult)	Caution
Spironolactone	Heart failure (HFrEF); ascites in cirrhosis; primary hyperaldosteronism; resistant hypertension	HF 25–50 mg daily; ascites 100–400 mg daily	Hyperkalaemia (with ACEi/ARB), gynaecomastia, menstrual disturbance
Eplerenone	Heart failure post-MI; spironolactone gynaecomastia	25–50 mg daily	Hyperkalaemia; CYP3A4 interactions
Amiloride	With thiazide/loop to prevent hypokalaemia; Liddle syndrome; lithium-induced DI	5–10 mg daily	Hyperkalaemia, especially in renal impairment
Triamterene	With thiazide to conserve potassium	50–100 mg daily	Hyperkalaemia; renal stones
Finerenone	CKD with type 2 diabetes	10–20 mg daily	Hyperkalaemia — check potassium

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.



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17. Anticoagulants — Heparins

Key drugs: Unfractionated heparin, enoxaparin, dalteparin, fondaparinux

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Blood clotting is a cascade of clotting factors ending in thrombin, which converts fibrinogen to fibrin. Antithrombin is a natural inhibitor that neutralises thrombin (IIa) and factor Xa, keeping blood fluid inside intact vessels.

2 PATHOLOGY

Virchow's triad — stasis of blood (immobility, surgery), endothelial injury and hypercoagulability (cancer, pregnancy, inherited thrombophilia) — causes inappropriate clot formation inside veins or arteries.

3 DISEASE CAUSED BY THIS PROBLEM

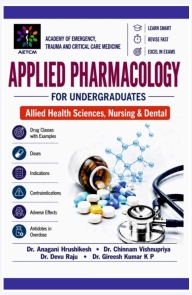
Deep-vein thrombosis, pulmonary embolism, acute coronary syndrome, prevention of clotting in dialysis and bypass circuits, VTE prophylaxis in hospitalised and post-operative patients.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Heparins bind antithrombin and greatly accelerate its inactivation of thrombin and factor Xa (LMWH mainly Xa). They prevent clot extension and new clots while the body's fibrinolytic system dissolves existing clot — allowing resolution, not direct dissolution.

5 ADVERSE EFFECTS

Bleeding, heparin-induced thrombocytopenia (HIT — fall in platelets day 5–10 with thrombosis), osteoporosis with long-term use, hyperkalaemia. Protamine sulphate reverses UFH (partially LMWH). LMWH accumulates in renal failure.



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Drug Table — 17. Anticoagulants — Heparins

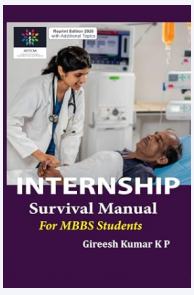
Drugs in this class: Unfractionated heparin, enoxaparin, dalteparin, fondaparinux

Drug	Indications	Dose (adult)	Caution
Unfractionated heparin	Acute VTE; ACS; dialysis; when rapid reversal needed; severe renal failure	Bolus 5000 units (80 U/kg) IV, then 18 U/kg/h; prophylaxis 5000 U SC 8–12-hourly	Monitor aPTT; HIT (check platelets); antidote protamine
Enoxaparin	VTE treatment and prophylaxis; ACS	Treatment 1 mg/kg SC 12-hourly; prophylaxis 40 mg SC daily	Reduce in CrCl < 30; bleeding; anti-Xa monitoring in pregnancy/obesity
Dalteparin	VTE (including cancer-associated); prophylaxis	200 U/kg SC daily (cancer: month 1), prophylaxis 5000 U daily	Bleeding; HIT less common than UFH
Fondaparinux	VTE prophylaxis and treatment; ACS; HIT history	2.5 mg SC daily (prophylaxis/ACS); 7.5 mg treatment	Contraindicated if CrCl < 20–30; no reversal agent

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol.
Facing page: physiology → pathology → disease → drug action → adverse effects.

★ REMEMBER — EXAM POINTS

- Heparin activates antithrombin → inhibits thrombin (IIa) and Xa; LMWH acts mainly on Xa.
- HIT: platelets fall > 50% at 5–10 days with thrombosis — stop all heparin, use a non-heparin anticoagulant.
- Protamine fully reverses UFH, only partially LMWH.



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18. Anticoagulants — Warfarin (Vitamin K Antagonist)

Key drugs: Warfarin, acenocoumarol

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

The liver makes clotting factors II, VII, IX and X (and proteins C and S) in an inactive form; vitamin K is required to carboxylate them so they can bind calcium and work. Vitamin K is recycled by the enzyme vitamin K epoxide reductase (VKORC1).

2 PATHOLOGY

Clots form in the heart and veins when blood stagnates or flows abnormally — left atrial appendage in atrial fibrillation, on mechanical heart valves, or in leg veins — and can embolise.

3 DISEASE CAUSED BY THIS PROBLEM

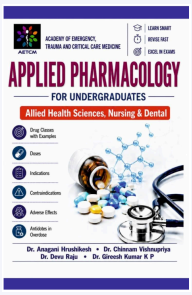
Stroke prevention in atrial fibrillation (especially with mitral stenosis), mechanical heart valves, DVT/PE treatment and prevention, antiphospholipid syndrome.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Warfarin blocks VKORC1, so new clotting factors are non-functional; effect takes 3–5 days (existing factors must be cleared), so heparin cover is needed at the start. It prevents embolic complications — long-term prevention, not cure.

5 ADVERSE EFFECTS

Bleeding (major risk), skin necrosis (early protein C deficiency), teratogenic (avoid in first trimester), purple toe syndrome. Many interactions — antibiotics, amiodarone, azoles raise INR; rifampicin, carbamazepine lower it. Reversal: vitamin K, prothrombin complex concentrate, FFP.



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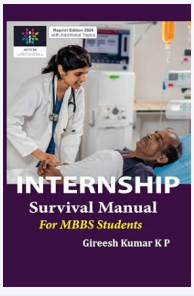
Drugs in this class: Warfarin, acenocoumarol

Drug	Indications	Dose (adult)	Caution
Warfarin	AF (valvular, mechanical valves); VTE treatment; mechanical heart valves	5–10 mg loading (by protocol), then 2–10 mg daily to target INR 2–3 (2.5–3.5 mechanical mitral)	Many drug/food interactions; teratogenic; bleeding — reversal with vitamin K + PCC/FFP
Acenocoumarol	As for warfarin (commonly used in India)	1–8 mg daily, INR guided	Shorter half-life; same interactions
Vitamin K (phytomenadione)	Reversal of warfarin over-anticoagulation	INR > 8 no bleeding 1–5 mg oral; major bleed 5–10 mg IV slowly	Anaphylactoid reaction with rapid IV
Prothrombin complex concentrate	Life-threatening warfarin-associated bleeding	25–50 IU/kg IV	Thrombosis risk
Fresh frozen plasma	Warfarin reversal when PCC unavailable; coagulopathy with bleeding	15 mL/kg IV	Volume overload, transfusion reactions; slower than PCC

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.

★ REMEMBER — EXAM POINTS

- Warfarin inhibits VKORC1 → factors II, VII, IX, X and proteins C and S are not carboxylated.
- Early prothrombotic phase (protein C falls first) — overlap with heparin for ≥ 5 days and until INR ≥ 2 for 24 h.
- INR targets: 2–3 for AF and VTE; 2.5–3.5 for mechanical mitral valves.
- Interactions: INR ↑ with amiodarone, macrolides, fluconazole, metronidazole; INR ↓ with rifampicin, carbamazepine, vitamin K-rich foods.
- Teratogenic (warfarin embryopathy, weeks 6–12) — switch to LMWH in pregnancy.



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19. Direct Oral Anticoagulants (DOACs)

Key drugs: Apixaban, rivaroxaban, edoxaban, dabigatran

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Factor Xa sits at the junction of the clotting cascade, converting prothrombin into thrombin; thrombin then forms fibrin and activates platelets. Inhibiting either step stops clot formation.

2 PATHOLOGY

Stasis in a fibrillating left atrium or in deep veins allows thrombus to form; fragments embolise to the brain (stroke) or lungs (PE).

3 DISEASE CAUSED BY THIS PROBLEM

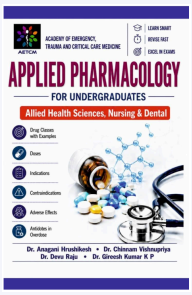
Non-valvular atrial fibrillation (stroke prevention), DVT and PE treatment and secondary prevention, VTE prophylaxis after hip/knee replacement.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Apixaban, rivaroxaban and edoxaban directly inhibit factor Xa; dabigatran directly inhibits thrombin. They have rapid onset and offset, fixed dosing and no routine INR monitoring, and prevent stroke and recurrent VTE as well as or better than warfarin, with less intracranial bleeding.

5 ADVERSE EFFECTS

Bleeding (GI bleeding more with rivaroxaban/dabigatran), dyspepsia (dabigatran). Avoid in mechanical valves, moderate–severe mitral stenosis, pregnancy, severe renal impairment. Reversal: idarucizumab (dabigatran), andexanet alfa or PCC (Xa inhibitors).



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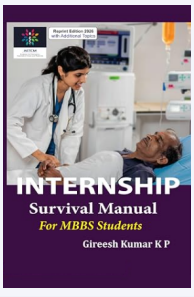
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Drug Table — 19. Direct Oral Anticoagulants (DOACs)

Drugs in this class: Apixaban, rivaroxaban, edoxaban, dabigatran

Drug	Indications	Dose (adult)	Caution
Apixaban	Non-valvular AF; VTE treatment and prevention	AF 5 mg bd (2.5 mg if ≥ 2 of: age ≥ 80, weight ≤ 60 kg, creatinine ≥ 1.5 mg/dL); VTE 10 mg bd × 7 d then 5 mg bd	Not for mechanical valves; interactions with azoles, rifampicin
Rivaroxaban	Non-valvular AF; VTE; CAD (low dose with aspirin)	AF 20 mg daily with food (15 mg if CrCl 15–49); VTE 15 mg bd × 21 d then 20 mg	Take with food; GI bleeding
Edoxaban	Non-valvular AF; VTE (after 5 days of heparin)	60 mg daily (30 mg if CrCl 15–50 or weight ≤ 60 kg)	Less effective if CrCl > 95 in AF
Dabigatran	Non-valvular AF; VTE	150 mg bd (110 mg bd if age ≥ 80 or high bleeding risk)	Renal excretion 80% — avoid CrCl < 30; dyspepsia; antidote idarucizumab
Andexanet alfa / idarucizumab	Reversal of factor Xa inhibitors / dabigatran	Idarucizumab 5 g IV; andexanet per protocol	Thrombotic events

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20. Antiplatelet Drugs

Key drugs: Aspirin, clopidogrel, ticagrelor, prasugrel, abciximab

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

When a vessel is injured, platelets adhere to exposed collagen, are activated (thromboxane A2 and ADP released), change shape and aggregate via glycoprotein IIb/IIIa receptors, forming a platelet plug.

2 PATHOLOGY

Rupture of an atherosclerotic plaque exposes a highly thrombogenic core; platelets aggregate rapidly to form an arterial "white thrombus" that blocks coronary or cerebral arteries.

3 DISEASE CAUSED BY THIS PROBLEM

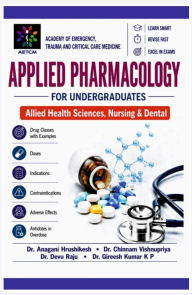
Acute coronary syndrome and myocardial infarction, after coronary stenting, ischaemic stroke and TIA, peripheral arterial disease; secondary prevention of atherothrombotic events.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Aspirin irreversibly inhibits COX-1, blocking thromboxane A2 for the platelet's lifespan (7–10 days). Clopidogrel/prasugrel (irreversible) and ticagrelor (reversible) block the P2Y12 ADP receptor. GP IIb/IIIa blockers prevent final aggregation. Prevent re-infarction and stroke (secondary prevention).

5 ADVERSE EFFECTS

Bleeding (GI, intracranial), dyspepsia and peptic ulcer (aspirin), aspirin-exacerbated asthma, Reye's syndrome in children; dyspnoea and bradycardia (ticagrelor); TTP (rare, clopidogrel). Clopidogrel efficacy reduced by CYP2C19 poor metabolism.



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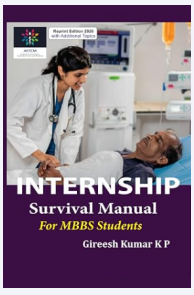
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Drug Table — 20. Antiplatelet Drugs

Drugs in this class: Aspirin, clopidogrel, ticagrelor, prasugrel, abciximab

Drug	Indications	Dose (adult)	Caution
Aspirin	ACS; secondary prevention of MI/ stroke; after PCI; acute ischaemic stroke	ACS 300 mg chewed, then 75–150 mg daily	GI bleeding, peptic ulcer; asthma exacerbation; avoid in children (Reye's)
Clopidogrel	ACS and PCI (with aspirin); stroke prevention; aspirin intolerance	Load 300–600 mg, then 75 mg daily	Reduced effect with CYP2C19 poor metabolisers / omeprazole; bleeding
Ticagrelor	ACS (more potent DAPT)	Load 180 mg, then 90 mg bd	Dyspnoea, bleeding, bradyarrhythmia; aspirin dose ≤ 100 mg
Prasugrel	ACS undergoing PCI	Load 60 mg, then 10 mg daily (5 mg if < 60 kg or ≥ 75 y)	Contraindicated after stroke/TIA; bleeding
Abciximab / tirofiban (GP IIb/IIIa)	High-risk PCI, bail-out in thrombus	Tirofiban 25 µg/kg IV bolus, then 0.15 µg/kg/min	Bleeding, thrombocytopenia
Cilostazol	Intermittent claudication	100 mg bd	Contraindicated in heart failure

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.



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21. Thrombolytics (Fibrinolytics)

Key drugs: Alteplase, tenecteplase, streptokinase, reteplase

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

The fibrinolytic system dissolves clots once healing begins: tissue plasminogen activator (tPA) released from endothelium converts plasminogen bound to fibrin into plasmin, which breaks fibrin into degradation products (D-dimers).

2 PATHOLOGY

A fresh occlusive thrombus blocks blood flow to the heart, brain or lungs; natural fibrinolysis is too slow to prevent cell death, which progresses minute by minute ("time is muscle/brain").

3 DISEASE CAUSED BY THIS PROBLEM

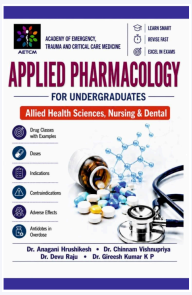
ST-elevation myocardial infarction (when primary PCI unavailable), acute ischaemic stroke within 4.5 hours, massive (high-risk) pulmonary embolism with shock, blocked catheters.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Thrombolytics convert plasminogen to plasmin, rapidly dissolving the fibrin clot and restoring blood flow (reperfusion), saving myocardium or brain if given early — potentially curative for that event.

5 ADVERSE EFFECTS

Bleeding — intracranial haemorrhage (most feared), GI bleeding; reperfusion arrhythmias; hypotension and allergy (streptokinase — antibodies, do not repeat). Contraindications: recent haemorrhagic stroke, active bleeding, recent surgery/trauma, uncontrolled hypertension.



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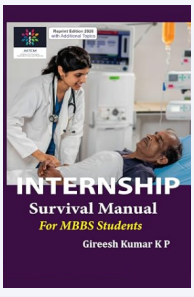
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Drug Table — 21. Thrombolytics (Fibrinolytics)

Drugs in this class: Alteplase, tenecteplase, streptokinase, reteplase

Drug	Indications	Dose (adult)	Caution
Alteplase (tPA)	Acute ischaemic stroke (≤ 4.5 h); massive PE; STEMI	Stroke 0.9 mg/kg (max 90 mg): 10% bolus, rest over 60 min; PE 100 mg over 2 h	Intracranial haemorrhage; strict exclusion checklist; BP < 185/110 before stroke lysis
Tenecteplase	STEMI when primary PCI not available within 120 min; stroke (newer)	Single weight-based IV bolus 30–50 mg (STEMI); 0.25 mg/kg stroke	Bleeding; give with antiplatelets and anticoagulant in STEMI
Streptokinase	STEMI (low-cost option in India)	1.5 million units IV over 60 min	Antigenic — allergy, hypotension; do not repeat after 5 days–12 months
Reteplase	STEMI	10 units IV bolus, repeat after 30 min	Bleeding
Tranexamic acid (reversal adjunct)	Fibrinolytic-associated bleeding; trauma haemorrhage (CRASH-2)	1 g IV over 10 min, then 1 g over 8 h	Thrombosis risk; seizures at high doses

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol.
Facing page: physiology → pathology → disease → drug action → adverse effects.



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22. Haematinics (Iron, Vitamin B12, Folate)

Key drugs: Ferrous sulphate, ferric carboxymaltose, hydroxocobalamin, folic acid

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Red cells are made in the bone marrow under erythropoietin. Haemoglobin needs iron (absorbed in the duodenum, stored as ferritin); DNA synthesis in dividing cells needs vitamin B12 (absorbed in the terminal ileum with intrinsic factor) and folate.

2 PATHOLOGY

Iron deficiency (blood loss, poor intake, pregnancy, malabsorption) produces small pale cells; B12 deficiency (pernicious anaemia, gastrectomy, vegan diet, metformin) or folate deficiency (poor diet, pregnancy, alcohol) causes impaired DNA synthesis — large immature megaloblasts.

3 DISEASE CAUSED BY THIS PROBLEM

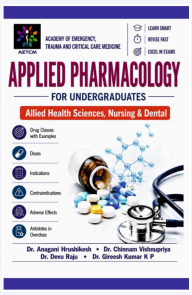
Iron-deficiency anaemia (microcytic), megaloblastic anaemia (macrocytic); B12 deficiency also causes neuropathy and subacute combined degeneration of the cord; folate deficiency in pregnancy causes neural tube defects.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Replacing the missing nutrient restores red-cell production — reticulocytes rise within 5–10 days and Hb by ~1–2 g/dL in 2–4 weeks: cure, provided the cause (e.g. bleeding) is treated. Folic acid before conception prevents neural tube defects.

5 ADVERSE EFFECTS

Oral iron — constipation, black stools, nausea; IV iron — hypersensitivity, hypophosphataemia. Iron overdose is dangerous in children (antidote desferrioxamine). Never give folic acid alone in B12 deficiency — may worsen neurological damage.



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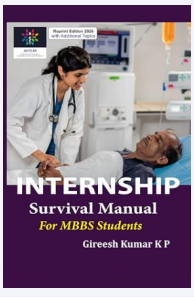
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Drug Table — 22. Haematinics (Iron, Vitamin B12, Folate)

Drugs in this class: Ferrous sulphate, ferric carboxymaltose, hydroxocobalamin, folic acid

Drug	Indications	Dose (adult)	Caution
Ferrous sulphate	Iron-deficiency anaemia; pregnancy supplementation	200 mg (65 mg elemental iron) once daily or alternate days	GI upset, black stools, constipation; separate from antacids, tetracyclines
Ferric carboxymaltose	IDA with oral intolerance/failure, CKD, pregnancy (2nd–3rd trimester), heart failure	Up to 1000 mg IV over 15 min (max 20 mg/kg) per week	Hypophosphataemia; hypersensitivity — observe 30 min
Iron sucrose	IV iron replacement (CKD, dialysis)	100–200 mg IV, repeated to total deficit	Hypotension with rapid infusion
Hydroxocobalamin (vitamin B12)	Pernicious anaemia; B12 deficiency; neuropathy	1 mg IM alternate days × 2 weeks (or until improvement), then every 3 months	Hypokalaemia when treating severe anaemia
Folic acid	Folate deficiency; pregnancy (neural-tube prevention); haemolytic anaemia	5 mg daily (treatment); 400 µg preconception (5 mg high risk)	Never give alone in B12 deficiency — risk of subacute combined degeneration

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.



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23. Beta-2 Agonists (Bronchodilators)

Key drugs: Salbutamol, terbutaline, salmeterol, formoterol, indacaterol

Normal physiology | Pathology | Disease | Drug action

1 PHYSIOLOGY

Airway smooth muscle tone is controlled by the autonomic system: β2-adrenergic stimulation raises cyclic AMP and relaxes the bronchi; parasympathetic (vagal) activity constricts them. Mucociliary clearance keeps airways clean.

2 PATHOLOGY

In asthma, allergic (type 2) inflammation makes airways hyper-responsive, causing reversible bronchospasm, mucosal oedema and mucus plugging; in COPD, chronic inflammation and loss of elastic recoil cause fixed airflow limitation.

3 DISEASE CAUSED BY THIS PROBLEM

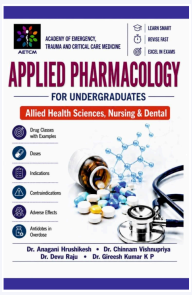
Acute asthma, chronic asthma (with inhaled steroid), COPD, exercise-induced bronchospasm; IV/nebulised salbutamol also lowers potassium in hyperkalaemia.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

β2 agonists raise cAMP in bronchial smooth muscle, causing rapid bronchodilation (SABA within minutes, for relief) or sustained bronchodilation (LABA, 12–24 h, for control). They relieve symptoms but do not treat inflammation — in asthma LABA must always be combined with an ICS.

5 ADVERSE EFFECTS

Fine tremor, palpitations, tachycardia, headache, muscle cramps, hypokalaemia, hyperglycaemia, lactic acidosis with high doses. LABA monotherapy in asthma increases asthma deaths — always with ICS.



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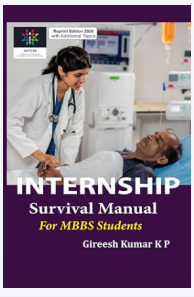
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Drug Table — 23. Beta-2 Agonists (Bronchodilators)

Drugs in this class: Salbutamol, terbutaline, salmeterol, formoterol, indacaterol

Drug	Indications	Dose (adult)	Caution
Salbutamol	Acute asthma and COPD reliever; hyperkalaemia	MDI 100–200 µg PRN; nebulised 2.5–5 mg; IV 5 µg/min in severe asthma	Tremor, tachycardia, hypokalaemia, lactic acidosis
Terbutaline	Asthma reliever; tocolysis (short term)	Inhaled 500 µg; SC 250 µg	As for salbutamol
Salmeterol (LABA)	Maintenance asthma (always with ICS); COPD	50 µg bd inhaled	Never as monotherapy in asthma — increased asthma deaths
Formoterol (LABA)	Maintenance; MART regimen with budesonide	6–12 µg bd (budesonide/ formoterol)	Fast onset allows reliever use in MART
Indacaterol / olodaterol (ultra-LABA)	COPD maintenance	Indacaterol 150–300 µg once daily	Cough after inhalation

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.



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24. Inhaled Corticosteroids

Key drugs: Budesonide, beclomethasone, fluticasone, ciclesonide

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Healthy airways have a thin epithelium and few inflammatory cells. Glucocorticoids bind intracellular receptors and switch off inflammatory genes (cytokines, chemokines) while switching on anti-inflammatory proteins.

2 PATHOLOGY

In asthma, allergens activate Th2 lymphocytes, mast cells and eosinophils, releasing IL-4, IL-5, IL-13, histamine and leukotrienes — causing chronic airway inflammation, hyper-responsiveness, mucus and remodelling.

3 DISEASE CAUSED BY THIS PROBLEM

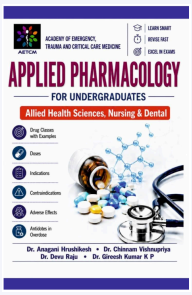
Persistent asthma (cornerstone of treatment), COPD with frequent exacerbations and high blood eosinophils, allergic rhinitis (nasal steroids).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

ICS suppress airway inflammation, reduce eosinophils and hyper-responsiveness, and prevent exacerbations and asthma deaths. They control disease (remission of symptoms) rather than cure; effects build over 1–2 weeks. Combined ICS-formoterol can be used as both reliever and maintenance (MART).

5 ADVERSE EFFECTS

Oral thrush, hoarseness (dysphonia), cough; at high doses — adrenal suppression, osteoporosis, cataract, glaucoma, skin bruising, growth slowing in children; increased pneumonia risk in COPD.



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Drug Table — 24. Inhaled Corticosteroids

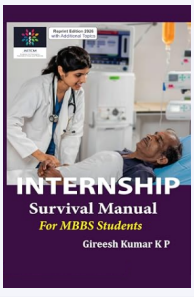
Drugs in this class: Budesonide, beclomethasone, fluticasone, ciclesonide

Drug	Indications	Dose (adult)	Caution
Budesonide	Asthma maintenance (step 2 onwards); COPD with exacerbations; croup (nebulised)	200–800 µg/day inhaled; croup 2 mg nebulised	Oral candidiasis, dysphonia — rinse mouth; pneumonia risk in COPD
Beclomethasone	Asthma prevention	100–400 µg bd (dose depends on device)	Use spacer; growth monitoring in children
Fluticasone propionate / furoate	Asthma; COPD (with LABA ± LAMA)	Propionate 100–500 µg bd; furoate 100–200 µg once daily	Adrenal suppression at high doses
Ciclesonide	Asthma	80–160 µg once daily	Prodrug — fewer local side-effects
Prednisolone (oral, for exacerbations)	Acute asthma / COPD exacerbation	40–50 mg daily for 5 days	Hyperglycaemia; no taper needed for ≤ 2 weeks

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.

★ REMEMBER — EXAM POINTS

- Inhaled steroids reduce airway inflammation and exacerbations — they are the cornerstone of asthma control.
- Rinse mouth after use; spacer reduces oral deposition and improves lung delivery.
- In COPD, ICS increase pneumonia risk — reserve for frequent exacerbators with eosinophilia.



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25. Inhaled Antimuscarinics

Key drugs: Ipratropium, tiotropium, glycopyrronium, umeclidinium

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Vagal nerves release acetylcholine onto M3 receptors in airway smooth muscle and submucosal glands, maintaining bronchial tone and mucus secretion; this cholinergic tone is the main reversible component in COPD.

2 PATHOLOGY

In COPD, cigarette smoke and pollutants cause chronic inflammation, mucus gland hyperplasia and emphysema; increased vagal tone narrows already damaged airways.

3 DISEASE CAUSED BY THIS PROBLEM

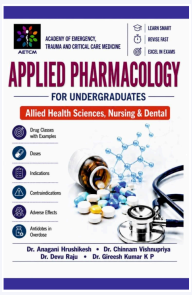
COPD (maintenance — first-line long-acting bronchodilator), acute exacerbations of COPD and severe asthma (ipratropium nebulised), asthma add-on (tiotropium).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Blocking M3 receptors relaxes airway smooth muscle and reduces mucus — improving airflow, symptoms and exercise tolerance and reducing COPD exacerbations. Symptom control; does not reverse lung damage.

5 ADVERSE EFFECTS

Dry mouth, metallic taste, cough, urinary retention (BPH), constipation; may precipitate acute angle-closure glaucoma if nebulised mist enters the eye (use mouthpiece).



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Drug Table — 25. Inhaled Antimuscarinics

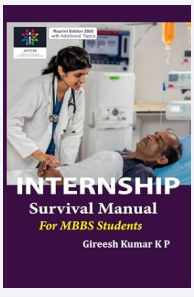
Drugs in this class: Ipratropium, tiotropium, glycopyrronium, umeclidinium

Drug	Indications	Dose (adult)	Caution
Ipratropium (SAMA)	Acute asthma (added to salbutamol); COPD	Nebulised 500 µg 4–6-hourly	Dry mouth; glaucoma with nebuliser mask
Tiotropium (LAMA)	COPD maintenance; asthma add-on	18 µg once daily (HandiHaler) / 2.5 µg × 2 puffs (Respimat)	Dry mouth, urinary retention, constipation
Glycopyrronium (LAMA)	COPD maintenance	44–50 µg once daily	Urinary retention in BPH
Umeclidinium (LAMA)	COPD maintenance (often with vilanterol)	55 µg once daily	Paradoxical bronchospasm (rare)
Aclidinium (LAMA)	COPD maintenance	322 µg bd	Anticholinergic effects

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.

★ REMEMBER — EXAM POINTS

- LAMAs block M3 receptors on airway smooth muscle → bronchodilatation and less mucus.
- COPD step-up: LAMA or LABA → LAMA + LABA → add ICS if eosinophils ≥ 300/µL or frequent exacerbations.
- Check inhaler technique at every visit — most 'treatment failure' is technique failure.



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26. Methylxanthines and Leukotriene Antagonists

Key drugs: Theophylline, aminophylline, doxofylline, montelukast

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Bronchial smooth muscle relaxes when intracellular cyclic AMP rises; phosphodiesterase enzymes break cAMP down. Leukotrienes (LTC4, LTD4) made from arachidonic acid by mast cells and eosinophils are potent airway constrictors.

2 PATHOLOGY

In asthma and allergic rhinitis, leukotrienes released during allergic inflammation cause bronchospasm, mucosal oedema and mucus; aspirin-exacerbated respiratory disease involves leukotriene overproduction.

3 DISEASE CAUSED BY THIS PROBLEM

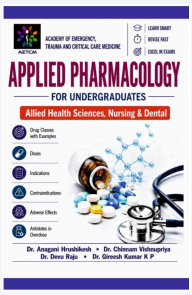
Asthma (add-on), exercise-induced and aspirin-sensitive asthma (montelukast), allergic rhinitis; COPD (theophylline add-on); apnoea of prematurity (caffeine).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Theophylline inhibits phosphodiesterase and blocks adenosine receptors, causing bronchodilation and improved diaphragm contractility. Montelukast blocks the CysLT1 receptor, reducing bronchospasm and inflammation. Both are add-on control therapies.

5 ADVERSE EFFECTS

Theophylline — narrow therapeutic index: nausea, vomiting, tachycardia, arrhythmias, seizures; levels raised by ciprofloxacin, erythromycin, cimetidine, liver disease; lowered by smoking. Montelukast — headache, abdominal pain, neuropsychiatric effects (sleep disturbance, depression).



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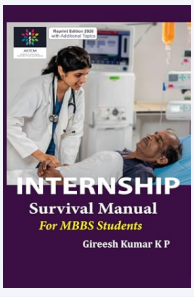
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Drug Table — 26. Methylxanthines and Leukotriene Antagonists

Drugs in this class: Theophylline, aminophylline, doxofylline, montelukast

Drug	Indications	Dose (adult)	Caution
Theophylline (SR)	Add-on in COPD and difficult asthma	200–300 mg bd; target level 10–20 mg/L	Narrow therapeutic index — arrhythmias, seizures; levels rise with ciprofloxacin, erythromycin
Aminophylline	Severe acute asthma (rarely now)	Load 5 mg/kg IV over 20 min (omit if on theophylline), then 0.5 mg/kg/h	Monitor levels; nausea, tachyarrhythmias
Doxofylline	Asthma and COPD (widely used in India)	400 mg bd–tds	Fewer cardiac side-effects than theophylline
Montelukast	Asthma (exercise-induced, aspirin-sensitive); allergic rhinitis	10 mg at night (4–5 mg in children)	Neuropsychiatric effects — mood changes, nightmares
Roflumilast (PDE-4 inhibitor)	Severe COPD with chronic bronchitis and exacerbations	500 µg daily	Weight loss, diarrhoea, psychiatric effects

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27. Antihistamines

Key drugs: Chlorpheniramine, promethazine, cetirizine, loratadine, fexofenadine

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Histamine stored in mast cells and basophils acts on H1 receptors to dilate and leak capillaries, contract bronchial and gut smooth muscle and stimulate sensory nerves (itch); in the brain it maintains wakefulness.

2 PATHOLOGY

In type I hypersensitivity, allergen cross-links IgE on mast cells, releasing histamine and other mediators — causing sneezing, rhinorrhoea, itching, urticaria, angioedema and, systemically, anaphylaxis.

3 DISEASE CAUSED BY THIS PROBLEM

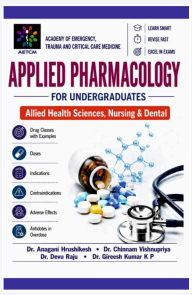
Allergic rhinitis and conjunctivitis, urticaria, insect bites, drug rashes, pruritus; adjunct in anaphylaxis (after adrenaline); motion sickness and vertigo (sedating agents).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

H1 antagonists (inverse agonists) block histamine effects — relieving itch, sneezing, rhinorrhoea and wheals. Second-generation agents do not cross the blood–brain barrier, so cause little sedation. Symptom control; avoidance of allergen and immunotherapy target the cause.

5 ADVERSE EFFECTS

First-generation — sedation, impaired driving, anticholinergic effects (dry mouth, retention, confusion in elderly), paradoxical excitation in children. Second-generation — minimal; occasional headache. Not a substitute for adrenaline in anaphylaxis.



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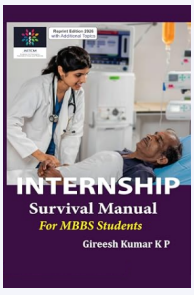
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Drug Table — 27. Antihistamines

Drugs in this class: Chlorpheniramine, promethazine, cetirizine, loratadine, fexofenadine

Drug	Indications	Dose (adult)	Caution
Chlorpheniramine	Allergic reactions; anaphylaxis (adjunct); urticaria	4 mg orally 4–6-hourly; 10 mg IM/IV	Sedation, anticholinergic effects; not first-line in anaphylaxis (adrenaline is)
Promethazine	Allergy; nausea; sedation	25 mg orally/IM	Marked sedation; respiratory depression in children < 2 y; tissue injury with IV
Cetirizine	Allergic rhinitis; urticaria	10 mg once daily	Mild sedation in some; reduce in renal impairment
Loratadine	Allergic rhinitis; urticaria	10 mg once daily	Non-sedating
Fexofenadine	Allergic rhinitis; chronic urticaria	120–180 mg once daily	Avoid with fruit juice (reduced absorption)
Hydroxyzine	Pruritus; anxiety	10–25 mg at night (max 100 mg)	Sedation; QT prolongation

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.



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28. Benzodiazepines and Related Sedative-Hypnotics

Key drugs: Diazepam, lorazepam, midazolam, alprazolam, zolpidem

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

GABA is the main inhibitory neurotransmitter of the brain; it opens chloride channels on GABA-A receptors, hyperpolarising neurons and damping excitability — important for calmness, sleep and preventing seizures.

2 PATHOLOGY

Reduced inhibitory tone or neuronal hyperexcitability underlies anxiety disorders, insomnia, seizures and status epilepticus, and alcohol withdrawal (where chronic alcohol has down-regulated GABA receptors).

3 DISEASE CAUSED BY THIS PROBLEM

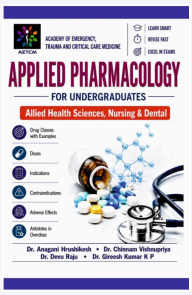
Status epilepticus and acute seizures, alcohol withdrawal and delirium tremens, acute severe anxiety/agitation, short-term insomnia, procedural sedation, muscle spasm.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Benzodiazepines bind the GABA-A receptor and increase the frequency of chloride channel opening, enhancing GABA's effect — causing anxiolysis, sedation, anticonvulsant and muscle-relaxant actions. Z-drugs act similarly at the same receptor. Symptom control; for anxiety and insomnia only short term.

5 ADVERSE EFFECTS

Drowsiness, ataxia, falls and fractures (elderly), amnesia, respiratory depression (especially with opioids/alcohol), dependence and withdrawal (seizures if stopped abruptly), paradoxical agitation. Antidote: flumazenil (caution — seizures).



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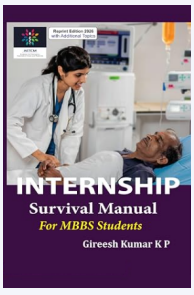
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Drug Table — 28. Benzodiazepines and Related Sedative-Hypnotics

Drugs in this class: Diazepam, lorazepam, midazolam, alprazolam, zolpidem

Drug	Indications	Dose (adult)	Caution
Diazepam	Status epilepticus (rectal/IV); alcohol withdrawal; muscle spasm; anxiety	Status 10 mg IV/PR; withdrawal 10–20 mg 6-hourly reducing	Long half-life — accumulation in elderly and liver disease; respiratory depression
Lorazepam	Status epilepticus; acute agitation; anxiety	Status 4 mg IV (0.1 mg/kg), repeat once; anxiety 0.5–1 mg	Respiratory depression; dependence
Midazolam	Procedural sedation; status epilepticus (buccal/IM); terminal agitation	Sedation 1–2.5 mg IV titrated; seizures 10 mg buccal/IM	Short-acting; apnoea — monitor SpO2; antidote flumazenil
Alprazolam	Panic disorder; anxiety (short term)	0.25–0.5 mg tds	High dependence potential; withdrawal seizures
Zolpidem (Z-drug)	Insomnia (short term ≤ 2–4 weeks)	5–10 mg at bedtime	Complex sleep behaviours, falls in elderly
Flumazenil (antidote)	Benzodiazepine overdose (selected)	200 µg IV, then 100 µg/min (max 1 mg)	May precipitate seizures in mixed overdoses / chronic users

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29. Antiepileptic Drugs

Key drugs: Sodium valproate, levetiracetam, carbamazepine, phenytoin, lamotrigine

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Normal brain function depends on a balance between excitation (glutamate, sodium and calcium channels) and inhibition (GABA). Neurons fire in controlled, synchronised patterns.

2 PATHOLOGY

In epilepsy, a group of neurons becomes hyperexcitable (genetic channel defects, scar from stroke/trauma, tumour, infection) and fires in abnormal, excessive synchrony that spreads through the brain.

3 DISEASE CAUSED BY THIS PROBLEM

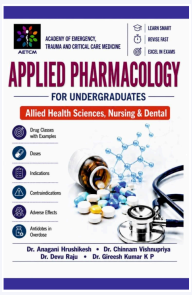
Epilepsy — focal seizures, generalised tonic-clonic, absence and myoclonic seizures; status epilepticus; also trigeminal neuralgia (carbamazepine), bipolar disorder (valproate, lamotrigine), neuropathic pain.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Phenytoin, carbamazepine and lamotrigine block voltage-gated sodium channels; valproate has multiple actions (Na+ channels, ↑ GABA, T-type Ca2+); levetiracetam binds synaptic vesicle protein SV2A. They suppress seizures — about 70% achieve seizure freedom (remission); epilepsy may remit after 2+ years seizure-free.

5 ADVERSE EFFECTS

Valproate — teratogenic (neural tube defects; avoid in women of childbearing potential), weight gain, tremor, hepatotoxicity, pancreatitis. Carbamazepine/phenytoin — enzyme induction, hyponatraemia, SJS (HLA-B*1502), gum hypertrophy (phenytoin). Lamotrigine — rash. Levetiracetam — irritability, mood change.



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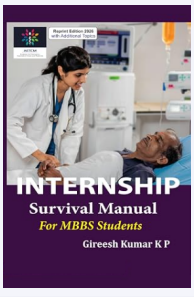
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Drug Table — 29. Antiepileptic Drugs

Drugs in this class: Sodium valproate, levetiracetam, carbamazepine, phenytoin, lamotrigine

Drug	Indications	Dose (adult)	Caution
Sodium valproate	Generalised and focal epilepsy; status; bipolar disorder; migraine	500 mg bd → 1–2 g/day; IV 20–40 mg/kg loading	Teratogenic (neural tube defects) — avoid in women of child-bearing age; hepatotoxicity, pancreatitis, weight gain
Levetiracetam	Focal and generalised epilepsy; status (second line); pregnancy-safe option	250–500 mg bd → 1.5 g bd; status 60 mg/kg IV (max 4.5 g)	Irritability, depression; reduce in renal impairment
Carbamazepine	Focal epilepsy; trigeminal neuralgia; bipolar disorder	100–200 mg bd → 400 mg bd	SJS/TEN (HLA-B*1502 in Asians), hyponatraemia, enzyme induction
Phenytoin	Status epilepticus; focal epilepsy	Status 20 mg/kg IV at ≤ 50 mg/min; maintenance 300 mg/day	Zero-order kinetics — monitor levels; gum hypertrophy, ataxia; cardiac monitoring with IV
Lamotrigine	Focal and generalised epilepsy; bipolar depression; women of child-bearing age	25 mg daily, slow titration to 100–200 mg bd	Rash/SJS with rapid titration or with valproate
Phenobarbitone	Neonatal seizures; refractory status; low-cost epilepsy treatment	Status 15–20 mg/kg IV; maintenance 60–180 mg at night	Sedation, respiratory depression, enzyme induction

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30. Antipsychotics

Key drugs: Haloperidol, chlorpromazine, risperidone, olanzapine, quetiapine, clozapine

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Dopamine pathways in the brain regulate reward and motivation (mesolimbic), cognition (mesocortical), movement (nigrostriatal) and prolactin release (tuberoinfundibular). Serotonin 5-HT2A receptors modulate dopamine release.

2 PATHOLOGY

In schizophrenia and mania, excess dopamine activity in the mesolimbic pathway produces positive symptoms (delusions, hallucinations); reduced mesocortical activity contributes to negative and cognitive symptoms.

3 DISEASE CAUSED BY THIS PROBLEM

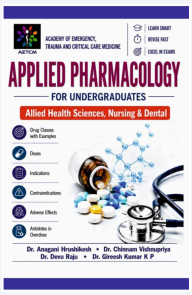
Schizophrenia, acute mania and bipolar disorder, acute agitation and delirium (short term), psychotic depression; nausea and vomiting (prochlorperazine), intractable hiccups (chlorpromazine).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Antipsychotics block dopamine D2 receptors (typicals strongly; atypicals also block 5-HT2A), reducing positive psychotic symptoms within days to weeks. Long-term treatment maintains remission and prevents relapse; clozapine is used for treatment-resistant schizophrenia.

5 ADVERSE EFFECTS

Extrapyramidal effects (acute dystonia, parkinsonism, akathisia, tardive dyskinesia), neuroleptic malignant syndrome, hyperprolactinaemia, QT prolongation, sedation, metabolic syndrome (olanzapine, clozapine), agranulocytosis and myocarditis (clozapine); increased stroke and death in elderly with dementia.



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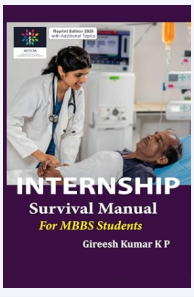
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Drug Table — 30. Antipsychotics

Drugs in this class: Haloperidol, chlorpromazine, risperidone, olanzapine, quetiapine, clozapine

Drug	Indications	Dose (adult)	Caution
Haloperidol	Acute psychosis; agitation and delirium; nausea (palliative); Tourette's	Agitation 2.5–5 mg IM; delirium 0.5–1 mg; schizophrenia 5–10 mg/day	Extrapyramidal effects, QT prolongation, NMS
Chlorpromazine	Schizophrenia; intractable hiccups; agitation	25–100 mg tds	Sedation, hypotension, photosensitivity, cholestatic jaundice
Risperidone	Schizophrenia; bipolar mania; behavioural problems in autism	1–6 mg daily	Hyperprolactinaemia, EPS at higher doses, weight gain
Olanzapine	Schizophrenia; mania; agitation; chemotherapy nausea	5–20 mg daily; IM 10 mg for agitation	Weight gain, diabetes, dyslipidaemia (metabolic syndrome)
Quetiapine	Schizophrenia; bipolar depression; psychosis in Parkinson's disease	50 mg → 300–800 mg/day	Sedation, postural hypotension, metabolic effects
Clozapine	Treatment-resistant schizophrenia	12.5 mg → 200–450 mg/day (slow titration)	Agranulocytosis — mandatory blood monitoring; myocarditis, seizures, constipation

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31. Antidepressants

Key drugs: Sertraline, fluoxetine, escitalopram, amitriptyline, venlafaxine, mirtazapine

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Serotonin (5-HT) and noradrenaline released by brainstem neurons regulate mood, sleep, appetite, motivation and pain modulation; transporters (SERT, NET) recycle them back into nerve endings.

2 PATHOLOGY

Depression involves reduced monoamine signalling, stress-hormone (HPA axis) overactivity, reduced neuroplasticity and BDNF in the hippocampus and prefrontal cortex, often triggered by genetic vulnerability and life stress.

3 DISEASE CAUSED BY THIS PROBLEM

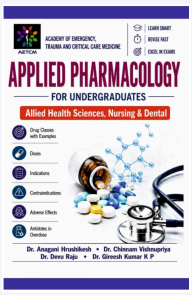
Major depressive disorder, generalised anxiety, panic disorder, obsessive-compulsive disorder, PTSD; neuropathic pain and migraine prophylaxis (amitriptyline, duloxetine).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

SSRIs block serotonin reuptake; SNRIs block serotonin and noradrenaline reuptake; TCAs block both plus other receptors; mirtazapine blocks α2 autoreceptors. Monoamines rise immediately but mood improves over 2–6 weeks (neuroadaptation). Continued for ≥6–12 months to maintain remission.

5 ADVERSE EFFECTS

SSRIs — nausea, headache, insomnia, sexual dysfunction, hyponatraemia (elderly), GI bleeding with NSAIDs, serotonin syndrome, increased suicidal thoughts in young people early on. TCAs — anticholinergic effects, postural hypotension, cardiotoxic and lethal in overdose. Discontinuation symptoms if stopped abruptly.



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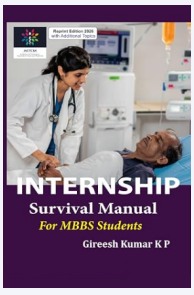
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Drug Table — 31. Antidepressants

Drugs in this class: Sertraline, fluoxetine, escitalopram, amitriptyline, venlafaxine, mirtazapine

Drug	Indications	Dose (adult)	Caution
Sertraline (SSRI)	Depression; anxiety disorders; OCD; PTSD; depression after MI	50–200 mg daily	GI upset, sexual dysfunction, hyponatraemia; bleeding with NSAIDs
Fluoxetine (SSRI)	Depression (including adolescents); OCD; bulimia	20–60 mg daily	Long half-life; CYP2D6 inhibition; agitation early
Escitalopram (SSRI)	Depression; generalised anxiety; panic	10–20 mg daily	QT prolongation at high doses
Amitriptyline (TCA)	Depression (rarely now); neuropathic pain; migraine prophylaxis	Pain 10–75 mg at night; depression 75–150 mg	Anticholinergic, sedation, arrhythmia; lethal in overdose
Venlafaxine (SNRI)	Depression; generalised anxiety	75–225 mg daily	Hypertension at higher doses; discontinuation syndrome
Mirtazapine	Depression with insomnia/poor appetite	15–45 mg at night	Weight gain, sedation

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol.
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32. Mood Stabiliser — Lithium

Key drugs: Lithium carbonate, lithium citrate

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Mood is regulated by neurotransmitter systems and intracellular second messengers (inositol phosphate, protein kinase C, GSK-3) that control neuronal excitability and plasticity.

2 PATHOLOGY

In bipolar disorder, dysregulated intracellular signalling and circadian rhythms produce recurrent episodes of mania (elevated mood, reduced sleep, grandiosity) and depression.

3 DISEASE CAUSED BY THIS PROBLEM

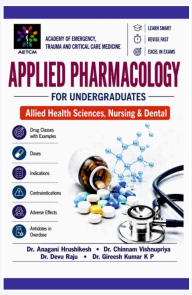
Bipolar affective disorder (acute mania and long-term prophylaxis), treatment-resistant depression (augmentation), reduction of suicide risk in mood disorders.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Lithium inhibits inositol monophosphatase and GSK-3, stabilising neuronal signalling; it treats mania, prevents relapses of both mania and depression and uniquely reduces suicide — long-term maintenance of remission.

5 ADVERSE EFFECTS

Narrow therapeutic index. Fine tremor, polyuria and nephrogenic diabetes insipidus, hypothyroidism, weight gain, hyperparathyroidism, teratogenic (Ebstein anomaly). Toxicity (>1.5 mmol/L): coarse tremor, vomiting, ataxia, confusion, seizures — precipitated by dehydration, NSAIDs, ACE inhibitors, diuretics. Haemodialysis for severe toxicity.



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Drug Table — 32. Mood Stabiliser — Lithium

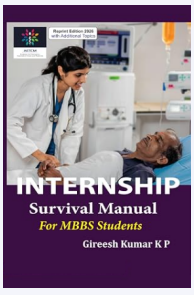
Drugs in this class: Lithium carbonate, lithium citrate

Drug	Indications	Dose (adult)	Caution
Lithium carbonate	Bipolar disorder (acute mania and prophylaxis); augmentation in depression	400–1200 mg/day; target 0.4–1.0 mmol/L (12 h post-dose)	Narrow therapeutic index — toxicity with dehydration, NSAIDs, ACEi, thiazides; hypothyroidism, nephrogenic DI, teratogenic
Lithium citrate (liquid)	As above when liquid needed	Doses differ from carbonate — prescribe by brand	Same toxicity; tremor, polyuria
Sodium valproate (alternative)	Acute mania; bipolar prophylaxis	750 mg–2 g/day	Avoid in women of child-bearing potential
Quetiapine / olanzapine (alternatives)	Mania; bipolar depression (quetiapine)	Quetiapine 300–600 mg; olanzapine 10–15 mg	Metabolic syndrome
Lamotrigine	Bipolar depression prophylaxis	25 mg daily, slow titration to 200 mg	Rash/SJS — titrate slowly; halve dose with valproate

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.

★ REMEMBER — EXAM POINTS

- Lithium is handled by the kidney like sodium — dehydration, low-salt diet, NSAIDs, ACE inhibitors and thiazides raise levels.
- Therapeutic level 0.4–1.0 mmol/L (12 h after the last dose); toxicity > 1.5 mmol/L — coarse tremor, ataxia, vomiting, confusion, seizures.
- Check TSH, calcium, creatinine before starting and every 6 months; levels every 3 months.
- Severe toxicity: stop lithium, IV saline; haemodialysis for level > 4 mmol/L or neurological features.
- Counsel: maintain fluid intake, avoid OTC NSAIDs, carry a lithium card.



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33. Antiparkinsonian Drugs

Key drugs: Levodopa–carbidopa, pramipexole, ropinirole, rasagiline, entacapone, trihexyphenidyl

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Dopaminergic neurons of the substantia nigra project to the striatum (nigrostriatal pathway), balancing cholinergic activity to allow smooth, initiated voluntary movement.

2 PATHOLOGY

In Parkinson's disease, progressive loss of nigral dopaminergic neurons (with α-synuclein Lewy bodies) causes dopamine deficiency and relative cholinergic excess in the striatum; symptoms appear after ~60–80% of neurons are lost.

3 DISEASE CAUSED BY THIS PROBLEM

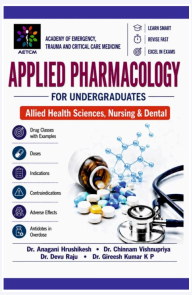
Parkinson's disease — resting tremor, rigidity, bradykinesia, postural instability; drug-induced parkinsonism (anticholinergics only); restless legs syndrome (dopamine agonists).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Levodopa is converted to dopamine in the brain (carbidopa prevents peripheral breakdown, reducing nausea); agonists stimulate dopamine receptors; MAO-B and COMT inhibitors prolong dopamine action; anticholinergics restore balance. They control symptoms — no drug halts neuronal loss.

5 ADVERSE EFFECTS

Levodopa — nausea, postural hypotension, dyskinesias and on-off fluctuations (long term), hallucinations, confusion. Agonists — impulse-control disorders, sleep attacks, oedema, hallucinations. Anticholinergics — confusion in elderly. Never stop abruptly (neuroleptic-malignant-like syndrome).



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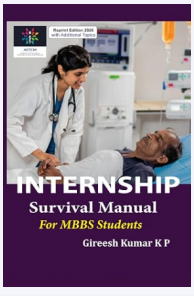
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Drug Table — 33. Antiparkinsonian Drugs

Drugs in this class: Levodopa–carbidopa, pramipexole, ropinirole, rasagiline, entacapone, trihexyphenidyl

Drug	Indications	Dose (adult)	Caution
Levodopa–carbidopa	Parkinson's disease (most effective symptomatic drug)	Start 100/25 mg tds, titrate (300–800 mg levodopa/day)	Nausea, postural hypotension, dyskinesia, 'on–off'; hallucinations
Pramipexole	Parkinson's disease; restless legs syndrome	0.125 mg tds → 1.5 mg/day; RLS 0.088 mg at night	Impulse-control disorders, sudden sleep attacks
Ropinirole	Parkinson's disease; restless legs	0.25 mg tds titrated (max 24 mg/day)	As pramipexole; oedema
Rasagiline (MAO-B inhibitor)	Early Parkinson's; adjunct for wearing-off	1 mg daily	Serotonin syndrome with SSRIs/tramadol/pethidine
Entacapone (COMT inhibitor)	Wearing-off with levodopa	200 mg with each levodopa dose	Orange urine, diarrhoea, increased dyskinesia
Trihexyphenidyl	Tremor-dominant PD; drug-induced parkinsonism	1 mg → 2 mg tds	Confusion in elderly, urinary retention, glaucoma

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34. Opioid Analgesics

Key drugs: Morphine, fentanyl, tramadol, codeine, oxycodone, buprenorphine

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Pain signals travel from nociceptors via the spinal dorsal horn to the brain; endogenous opioids (endorphins, enkephalins) act on μ, δ and κ receptors to inhibit transmission and modulate the emotional response to pain.

2 PATHOLOGY

Tissue injury (trauma, surgery, myocardial infarction, cancer infiltration) releases mediators that activate and sensitise nociceptors, producing severe acute or chronic pain; breathlessness in terminal illness also responds to opioids.

3 DISEASE CAUSED BY THIS PROBLEM

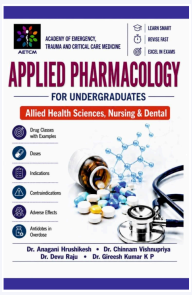
Severe acute pain (fractures, post-operative, MI), cancer pain, palliative breathlessness, acute pulmonary oedema (historic), opioid dependence (buprenorphine, methadone), cough (codeine).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Opioids activate μ receptors, reducing neurotransmitter release in the spinal cord and brain and altering pain perception — powerful analgesia; also reduce air hunger. Symptom relief, not cure of the cause.

5 ADVERSE EFFECTS

Respiratory depression, sedation, nausea and vomiting, constipation (no tolerance), pruritus, miosis, urinary retention, hypotension, tolerance and dependence. Tramadol — seizures, serotonin syndrome. Antidote: naloxone 0.4 mg IV (titrate 0.1 mg in dependence).



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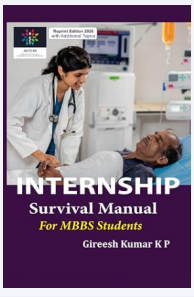
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Drug Table — 34. Opioid Analgesics

Drugs in this class: Morphine, fentanyl, tramadol, codeine, oxycodone, buprenorphine

Drug	Indications	Dose (adult)	Caution
Morphine	Severe acute and cancer pain; acute LVF breathlessness; palliative dyspnoea	Oral 5–10 mg 4-hourly (2.5 mg in elderly); IV 2–5 mg titrated	Respiratory depression, constipation (prescribe laxative), nausea; accumulates in renal failure
Fentanyl	Procedural/ICU analgesia; stable cancer pain (patch); renal failure	IV 25–100 µg; patch 12–100 µg/h every 72 h	Patch not for opioid-naive or acute pain; chest-wall rigidity with rapid IV
Tramadol	Moderate pain	50–100 mg 4–6-hourly (max 400 mg/day)	Seizures, serotonin syndrome, hyponatraemia; abuse potential
Codeine	Mild–moderate pain; cough; diarrhoea	30–60 mg 4-hourly (max 240 mg)	Variable CYP2D6 activation; contraindicated in children < 12 and breastfeeding
Oxycodone	Moderate–severe pain	IR 5 mg 4–6-hourly; MR 10 mg bd	As morphine; abuse potential
Buprenorphine	Chronic/cancer pain (patch); opioid dependence (sublingual)	Patch 5–20 µg/h weekly; SL 2–16 mg/day (OST)	Partial agonist — ceiling for respiratory depression; naloxone reversal needs high doses
Naloxone (antidote)	Opioid overdose	400 µg IV/IM, repeat every 2–3 min; titrate 100 µg in palliative patients	Short half-life — re-sedation; acute withdrawal

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.



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35. General and Local Anaesthetics

Key drugs: Propofol, ketamine, sevoflurane, thiopentone, lidocaine, bupivacaine

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Consciousness depends on integrated activity of cortex, thalamus and reticular activating system; nerve impulses travel along axons by opening voltage-gated sodium channels in sequence.

2 PATHOLOGY

Surgery and painful procedures cause pain, stress responses (tachycardia, hypertension) and movement; nerve injury or inflammation causes local pain needing blockade.

3 DISEASE CAUSED BY THIS PROBLEM

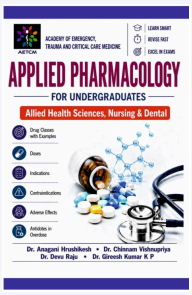
Need for general anaesthesia for surgery, sedation in ICU, rapid-sequence intubation; local and regional anaesthesia for minor procedures, dental work, spinal/epidural anaesthesia, nerve blocks, IV lidocaine for arrhythmia.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

General anaesthetics enhance GABA-A inhibition (propofol, volatile agents, thiopentone) or block NMDA receptors (ketamine) causing reversible unconsciousness. Local anaesthetics block sodium channels from inside the axon, stopping nerve conduction in a region — complete, reversible pain-free state.

5 ADVERSE EFFECTS

Propofol — hypotension, apnoea, injection pain, propofol infusion syndrome. Ketamine — emergence hallucinations, hypertension, salivation. Volatile agents — malignant hyperthermia (dantrolene). Local anaesthetic systemic toxicity — perioral tingling, seizures, arrhythmias, cardiac arrest (treat with 20% lipid emulsion).



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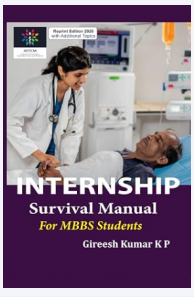
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Drug Table — 35. General and Local Anaesthetics

Drugs in this class: Propofol, ketamine, sevoflurane, thiopentone, lidocaine, bupivacaine

Drug	Indications	Dose (adult)	Caution
Propofol	Induction and maintenance of anaesthesia; ICU sedation	Induction 1.5–2.5 mg/kg IV; sedation 0.3–4 mg/kg/h	Hypotension, apnoea, pain on injection; propofol infusion syndrome
Ketamine	Induction in shock/ asthma; procedural sedation; analgesia	Induction 1–2 mg/kg IV (4–10 mg/kg IM); analgesia 0.1–0.3 mg/kg	Emergence delirium, hypertension, hypersalivation
Thiopentone	Induction; refractory status epilepticus	3–5 mg/kg IV	Hypotension; tissue necrosis if extravasated or intra-arterial; porphyria
Sevoflurane	Inhalational induction (children) and maintenance	1–8% inhaled (MAC ~2%)	Malignant hyperthermia trigger; nephrotoxicity theoretical
Lidocaine (local)	Local infiltration, nerve blocks, IV regional; antiarrhythmic	Max 3 mg/kg plain, 7 mg/kg with adrenaline	LA systemic toxicity — perioral tingling, seizures, arrhythmia (treat with lipid emulsion)
Bupivacaine	Spinal and epidural anaesthesia; nerve blocks	Max 2 mg/kg; spinal 0.5% heavy 2–3 mL	Cardiotoxic — never IV (Bier block)

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36. Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)

Key drugs: Ibuprofen, diclofenac, naproxen, aspirin, etoricoxib, celecoxib

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Cyclo-oxygenase enzymes make prostaglandins from arachidonic acid. COX-1 (constitutive) protects gastric mucosa, maintains renal blood flow and makes platelet thromboxane; COX-2 (inducible) makes prostaglandins at sites of inflammation that cause pain, swelling and fever.

2 PATHOLOGY

Tissue injury and inflammation induce COX-2; prostaglandins sensitise nociceptors (pain), dilate vessels (redness, swelling) and reset the hypothalamic thermostat (fever).

3 DISEASE CAUSED BY THIS PROBLEM

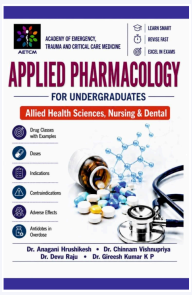
Musculoskeletal pain, osteoarthritis and rheumatoid arthritis (symptoms), acute gout, dysmenorrhoea, renal colic, dental and post-operative pain, fever; aspirin for antiplatelet effect.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

NSAIDs inhibit COX, reducing prostaglandin synthesis — analgesic, anti-inflammatory and antipyretic actions. Selective COX-2 inhibitors spare the stomach. They relieve symptoms but do not alter disease progression in arthritis.

5 ADVERSE EFFECTS

Dyspepsia, peptic ulcer and GI bleeding, acute kidney injury and fluid retention, hypertension and heart failure worsening, increased MI/stroke risk (COX-2 selective, diclofenac), asthma exacerbation, rashes. Avoid in third trimester, CKD, heart failure, active ulcer.



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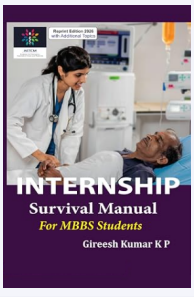
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Drug Table — 36. Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)

Drugs in this class: Ibuprofen, diclofenac, naproxen, aspirin, etoricoxib, celecoxib

Drug	Indications	Dose (adult)	Caution
Ibuprofen	Mild–moderate pain; fever; inflammatory arthritis; dysmenorrhoea	200–400 mg tds (max 2.4 g/day)	GI ulceration, AKI, asthma; avoid with anticoagulants
Diclofenac	Musculoskeletal pain; renal colic; gout	50 mg tds; 75 mg IM (renal colic)	Cardiovascular risk; GI and renal toxicity
Naproxen	Arthritis; gout; dysmenorrhoea	250–500 mg bd	Lower cardiovascular risk among NSAIDs; GI bleeding
Aspirin (analgesic dose)	Pain, fever, inflammation (rheumatic fever historic)	300–900 mg 4–6-hourly	Reye's syndrome in children; salicylism at high doses
Etoricoxib (COX-2)	Osteoarthritis; ankylosing spondylitis; acute gout	60–120 mg daily	Hypertension, cardiovascular risk; less GI ulceration
Celecoxib (COX-2)	Arthritis with GI risk	100–200 mg bd	Cardiovascular risk; sulfonamide allergy

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Facing page: physiology → pathology → disease → drug action → adverse effects.



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37. Paracetamol (Acetaminophen)

Key drugs: Paracetamol (oral, IV, rectal)

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

The hypothalamic thermoregulatory centre sets body temperature; pyrogens act through prostaglandin E2 to raise the set point. Pain perception involves central prostaglandin and serotonergic pathways.

2 PATHOLOGY

Infection and inflammation release cytokines (IL-1, IL-6, TNF) that increase central PGE2, causing fever; tissue injury produces nociceptive pain.

3 DISEASE CAUSED BY THIS PROBLEM

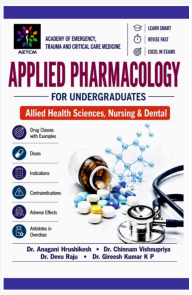
Fever, mild-to-moderate pain (headache, musculoskeletal, dental, post-operative), osteoarthritis pain (modest), as part of multimodal analgesia.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Paracetamol weakly inhibits COX in the central nervous system (where peroxide levels are low) and acts on serotonergic and cannabinoid pathways — reducing fever and pain with almost no anti-inflammatory or antiplatelet effect and little gastric toxicity. Symptom relief.

5 ADVERSE EFFECTS

Very safe in therapeutic doses; rash rarely. Overdose causes hepatotoxicity (toxic metabolite NAPQI depletes glutathione) — acute liver failure 2–4 days later. Antidote: N-acetylcysteine. Watch for hidden paracetamol in combination products.



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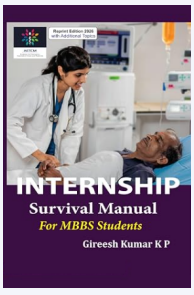
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Drug Table — 37. Paracetamol (Acetaminophen)

Drugs in this class: Paracetamol (oral, IV, rectal)

Drug	Indications	Dose (adult)	Caution
Paracetamol oral	Mild–moderate pain; fever; first step of WHO ladder	500 mg–1 g 4–6-hourly (max 4 g/day; 2–3 g in liver disease, alcoholism, < 50 kg)	Hepatotoxicity in overdose — NAPQI; check combination products
Paracetamol IV	Post-operative pain; fever when oral not possible	1 g IV over 15 min 6-hourly (15 mg/kg if < 50 kg)	Dosing errors in children and low weight adults
Paracetamol rectal	Children; vomiting patients	15–20 mg/kg PR (adult 1 g)	Variable absorption
N-acetylcysteine (antidote)	Paracetamol overdose above treatment line	SNAP or 3-bag regimen: 150 mg/kg over 1 h, then 50 mg/kg over 4 h, 100 mg/kg over 16 h	Anaphylactoid reactions (flushing, bronchospasm) — usually first bag
Paracetamol combinations	Moderate pain (with codeine / tramadol)	Count total paracetamol — max 4 g/day from all sources	Hidden overdose from multiple combination products

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol.
Facing page: physiology → pathology → disease → drug action → adverse effects.



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38. Drugs for Gout

Key drugs: Colchicine, allopurinol, febuxostat, probenecid

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Purines from DNA breakdown and diet are converted by xanthine oxidase into uric acid, which is excreted by the kidney. Normal serum urate is kept below its saturation point (~6.8 mg/dL).

2 PATHOLOGY

Over-production or under-excretion of urate (diuretics, CKD, alcohol, purine-rich diet, genetics) leads to hyperuricaemia; monosodium urate crystals deposit in joints and are engulfed by neutrophils, activating the NLRP3 inflammasome and triggering intense inflammation.

3 DISEASE CAUSED BY THIS PROBLEM

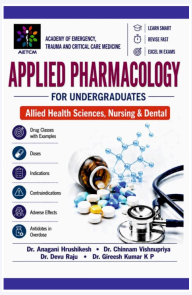
Acute gouty arthritis (classically first MTP joint — podagra), chronic tophaceous gout, urate kidney stones and nephropathy, tumour lysis syndrome.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Colchicine blocks microtubule assembly in neutrophils, stopping their migration and inflammasome activation — settles acute attacks. Allopurinol and febuxostat inhibit xanthine oxidase, lowering urate (target <6 mg/dL) and dissolving crystals over months — long-term remission of attacks. Probenecid increases urate excretion.

5 ADVERSE EFFECTS

Colchicine — diarrhoea, vomiting, myopathy, toxicity with clarithromycin/statins and in renal failure. Allopurinol — rash, severe hypersensitivity/SJS (HLA-B*5801), interaction with azathioprine. Febuxostat — liver enzyme rise, cardiovascular concern.



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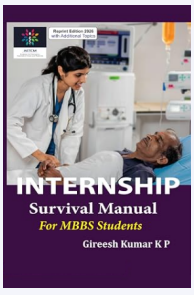
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Drug Table — 38. Drugs for Gout

Drugs in this class: Colchicine, allopurinol, febuxostat, probenecid

Drug	Indications	Dose (adult)	Caution
Colchicine	Acute gout; prophylaxis when starting urate-lowering therapy; pericarditis; FMF	500 µg 2–3 times daily (acute); 500 µg daily–bd prophylaxis	Diarrhoea; toxicity with clarithromycin, statins, renal impairment
Allopurinol	Chronic gout; tumour lysis prophylaxis; urate stones	100 mg daily, titrate by 100 mg every 2–4 weeks to urate < 6 mg/dL (max 900 mg)	Do not start in acute attack without cover; SJS (HLA-B*5801); azathioprine interaction
Febuxostat	Gout with allopurinol intolerance	40–80 mg daily (max 120 mg)	Cardiovascular caution; flares on initiation
Probenecid	Gout in under-excretors with normal renal function	250 mg bd → 500 mg bd	Uric acid stones — high fluid intake; ineffective if eGFR < 30
Prednisolone / intra-articular steroid	Acute gout when NSAIDs/colchicine unsuitable	Prednisolone 30–35 mg daily for 5 days	Hyperglycaemia

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39. Corticosteroids (Systemic)

Key drugs: Prednisolone, hydrocortisone, dexamethasone, methylprednisolone

Normal physiology | Pathology | Disease | Drug action

1 PHYSIOLOGY

The adrenal cortex secretes cortisol (under ACTH control) which regulates glucose metabolism, blood pressure, the stress response and suppresses inflammation and immunity; aldosterone regulates sodium and potassium.

2 PATHOLOGY

Uncontrolled inflammation or autoimmunity damages tissues (asthma, rheumatoid arthritis, vasculitis, SLE, IBD); adrenal destruction (Addison's) or pituitary failure causes cortisol deficiency; oedema around tumours raises intracranial pressure.

3 DISEASE CAUSED BY THIS PROBLEM

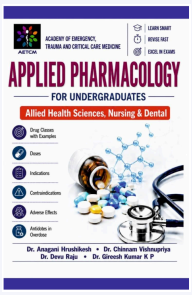
Asthma/COPD exacerbations, anaphylaxis adjunct, autoimmune and inflammatory diseases, adrenal insufficiency (replacement), cerebral oedema, spinal cord compression, severe pneumonia, COVID-19 with hypoxia, transplant rejection, antiemetic.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Corticosteroids bind intracellular glucocorticoid receptors and switch off inflammatory genes (cytokines, COX-2) and immune cell activity — rapid control of inflammation, inducing remission in many autoimmune diseases; physiological replacement in adrenal insufficiency.

5 ADVERSE EFFECTS

Hyperglycaemia, hypertension, fluid retention, weight gain, Cushingoid features, osteoporosis, myopathy, peptic ulcer (with NSAIDs), infections, mood changes and psychosis, cataract, glaucoma, adrenal suppression — taper after >3 weeks; carry steroid card.



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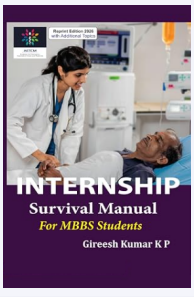
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Drug Table — 39. Corticosteroids (Systemic)

Drugs in this class: Prednisolone, hydrocortisone, dexamethasone, methylprednisolone

Drug	Indications	Dose (adult)	Caution
Prednisolone	Asthma/COPD exacerbation; autoimmune disease; nephrotic syndrome; rheumatological conditions	5–60 mg daily (1 mg/kg in many inflammatory diseases)	Hyperglycaemia, osteoporosis, infection, adrenal suppression — taper after > 3 weeks
Hydrocortisone	Adrenal crisis; anaphylaxis/asthma (adjunct); replacement therapy	Crisis 100 mg IV then 50 mg 6-hourly; replacement 15–25 mg/day split	Mineralocorticoid effects; sick-day rules
Dexamethasone	Cerebral oedema; spinal cord compression; croup; antiemetic; preterm lung maturation	4–16 mg/day; croup 0.15 mg/kg; antenatal 6 mg IM 12-hourly × 4	No mineralocorticoid effect; insomnia, psychosis, hyperglycaemia
Methylprednisolone	Pulse therapy: MS relapse, lupus nephritis, transplant rejection, vasculitis	500 mg–1 g IV daily for 3–5 days	Arrhythmias with rapid infusion; infection
Fludrocortisone	Addison's disease; postural hypotension	50–200 µg daily	Hypertension, hypokalaemia, oedema

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Facing page: physiology → pathology → disease → drug action → adverse effects.



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40. Penicillins (Beta-Lactams)

Key drugs: Benzylpenicillin, amoxicillin, co-amoxiclav, flucloxacillin, piperacillin-tazobactam

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Bacteria are surrounded by a peptidoglycan cell wall that resists osmotic pressure; penicillin-binding proteins (transpeptidases) cross-link peptidoglycan during growth. Human cells have no cell wall.

2 PATHOLOGY

Invading bacteria (streptococci, staphylococci, many Gram-negatives) multiply in tissues, triggering inflammation and tissue damage; some produce β-lactamases that destroy penicillins.

3 DISEASE CAUSED BY THIS PROBLEM

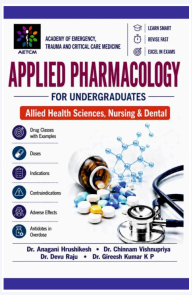
Pharyngitis, cellulitis, pneumonia, otitis media, sinusitis, urinary tract infection, meningitis, syphilis, endocarditis, rheumatic fever prophylaxis, febrile neutropenia (piperacillin-tazobactam).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Penicillins bind penicillin-binding proteins and block cross-linking of the cell wall, causing bacterial lysis (bactericidal). β-lactamase inhibitors (clavulanate, tazobactam) protect them from destruction. Eradicate susceptible infection — cure.

5 ADVERSE EFFECTS

Hypersensitivity — rash, urticaria, anaphylaxis (ask allergy history); diarrhoea, C. difficile infection; amoxicillin rash in glandular fever; cholestatic jaundice (co-amoxiclav, flucloxacillin); seizures with very high doses in renal failure.



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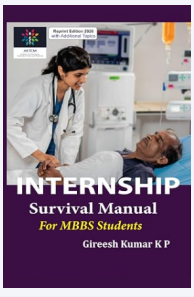
Drug Table — 40. Penicillins (Beta-Lactams)

Drugs in this class: Benzylpenicillin, amoxicillin, co-amoxiclav, flucloxacillin, piperacillin-tazobactam

Drug | Indications | Dose (adult) | Caution

Drug	Indications	Dose (adult)	Caution
Benzylpenicillin	Streptococcal and pneumococcal infections; meningococcal disease; syphilis	1.2–2.4 g IV 4–6-hourly; meningitis 2.4 g 4-hourly	Anaphylaxis (ask allergy history); seizures in high dose with renal failure
Amoxicillin	Otitis, sinusitis, community pneumonia, UTI, H. pylori, endocarditis prophylaxis	500 mg–1 g tds orally	Rash with EBV (glandular fever); diarrhoea
Co-amoxiclav (amoxicillin–clavulanate)	Bites, aspiration pneumonia, intra-abdominal, skin and soft tissue infections	625 mg tds orally; 1.2 g IV 8-hourly	Cholestatic jaundice; C. difficile
Flucloxacillin / cloxacillin	Staphylococcal skin infection, cellulitis, osteomyelitis	500 mg–1 g qds orally; 1–2 g IV 6-hourly	Cholestatic jaundice; take on empty stomach
Piperacillin–tazobactam	Hospital-acquired sepsis; febrile neutropenia; Pseudomonas	4.5 g IV 6–8-hourly	Renal adjustment; C. difficile
Benzathine penicillin	Syphilis; rheumatic fever prophylaxis	2.4 MU IM (syphilis); 1.2 MU every 3–4 weeks (RF)	Painful IM injection; never IV

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41. Cephalosporins and Carbapenems

Key drugs: Cefalexin, cefuroxime, ceftriaxone, ceftazidime, cefepime, meropenem

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Like penicillins, these β-lactams target bacterial cell-wall synthesis, which is essential for bacterial survival and absent in human cells.

2 PATHOLOGY

Serious Gram-negative and mixed infections, often hospital-acquired or with resistance mechanisms such as ESBLs (extended-spectrum β-lactamases), cause sepsis, meningitis and pneumonia.

3 DISEASE CAUSED BY THIS PROBLEM

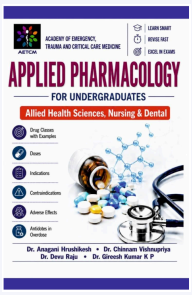
Meningitis (ceftriaxone), community-acquired pneumonia, pyelonephritis, gonorrhoea, enteric fever, intra-abdominal sepsis; hospital-acquired and ESBL infections, febrile neutropenia (carbapenems, cefepime), Pseudomonas (ceftazidime).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

They bind penicillin-binding proteins and inhibit cell-wall cross-linking — bactericidal. Each generation has broader Gram-negative activity; carbapenems resist most β-lactamases including ESBLs — reserved for resistant infections (stewardship).

5 ADVERSE EFFECTS

Hypersensitivity (small cross-reactivity with penicillin allergy), diarrhoea and C. difficile, biliary sludge (ceftriaxone), raised INR; carbapenems — seizures (especially imipenem), reduce valproate levels. Overuse drives carbapenem resistance.



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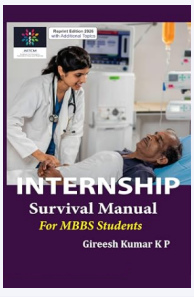
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Drug Table — 41. Cephalosporins and Carbapenems

Drugs in this class: Cefalexin, cefuroxime, ceftriaxone, ceftazidime, cefepime, meropenem

Drug	Indications	Dose (adult)	Caution
Cefalexin (1st gen)	Skin infections, UTI (including pregnancy)	500 mg bd–qds orally	~1–2% cross-reactivity with penicillin allergy
Cefuroxime (2nd gen)	Respiratory infections; surgical prophylaxis	250–500 mg bd orally; 750 mg–1.5 g IV 8-hourly	C. difficile
Ceftriaxone (3rd gen)	Meningitis, pneumonia, enteric fever, gonorrhoea, sepsis	1–2 g IV daily (2 g 12-hourly in meningitis)	Biliary sludge; never with calcium IV in neonates
Ceftazidime	Pseudomonas infections; melioidosis	1–2 g IV 8-hourly	Poor gram-positive cover
Cefepime (4th gen)	Febrile neutropenia; hospital-acquired infections	1–2 g IV 8–12-hourly	Neurotoxicity in renal failure
Meropenem (carbapenem)	ESBL infections; severe hospital sepsis; meningitis	1 g IV 8-hourly (2 g in meningitis)	Reserve drug — stewardship; lowers valproate levels

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.



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42. Macrolides

Key drugs: Azithromycin, clarithromycin, erythromycin

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Bacterial protein synthesis occurs on 70S ribosomes (30S and 50S subunits), different from human 80S ribosomes — a selective drug target.

2 PATHOLOGY

Intracellular and "atypical" organisms (Mycoplasma, Chlamydia, Legionella), and common respiratory bacteria, cause pneumonia, sexually transmitted and skin infections, particularly in penicillin-allergic patients.

3 DISEASE CAUSED BY THIS PROBLEM

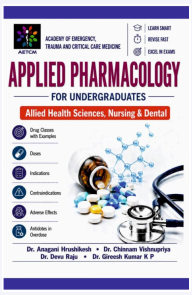
Atypical pneumonia, community-acquired pneumonia (with β-lactam), pertussis, chlamydia, H. pylori eradication (clarithromycin), skin infections in penicillin allergy, enteric fever (azithromycin), trachoma.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Macrolides bind the 50S ribosomal subunit and block translocation, stopping bacterial protein synthesis (bacteriostatic; bactericidal at high levels). Good tissue and intracellular penetration. Cure of susceptible infection.

5 ADVERSE EFFECTS

GI upset (erythromycin acts on motilin receptors), QT prolongation and arrhythmias, cholestatic hepatitis, ototoxicity at high doses. Clarithromycin and erythromycin inhibit CYP3A4 — raise levels of statins (myopathy), warfarin, colchicine, theophylline, carbamazepine.



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Drug Table — 42. Macrolides

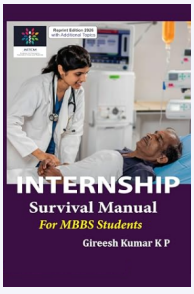
Drugs in this class: Azithromycin, clarithromycin, erythromycin

Drug	Indications	Dose (adult)	Caution
Azithromycin	Atypical pneumonia; chlamydia; enteric fever; trachoma; scrub typhus (alternative)	500 mg daily × 3 days; chlamydia 1 g single dose	QT prolongation; diarrhoea
Clarithromycin	Community pneumonia; H. pylori eradication; atypical mycobacteria	250–500 mg bd	Strong CYP3A4 inhibitor (statins, colchicine, warfarin); QT
Erythromycin	Penicillin allergy; diabetic gastroparesis (prokinetic); pertussis	250–500 mg qds	GI upset, QT prolongation, CYP3A4 inhibition
Roxithromycin	Respiratory and skin infections	150 mg bd	GI upset
Fidaxomicin (macrocyclic)	Clostridioides difficile infection (recurrence risk)	200 mg bd × 10 days	Expensive; minimal systemic absorption

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.

★ REMEMBER — EXAM POINTS

- Macrolides bind the 50S ribosomal subunit — bacteriostatic; good intracellular (atypical) cover.
- Erythromycin and clarithromycin inhibit CYP3A4 — avoid with simvastatin, colchicine, warfarin.
- Azithromycin has a long tissue half-life — 3-day courses are adequate.



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43. Aminoglycosides

Key drugs: Gentamicin, amikacin, tobramycin, streptomycin

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Aerobic bacteria take up aminoglycosides by an oxygen-dependent transport process; inside, the 30S ribosomal subunit reads messenger RNA to build bacterial proteins.

2 PATHOLOGY

Severe Gram-negative infections (E. coli, Klebsiella, Pseudomonas) cause urosepsis, intra-abdominal sepsis and bacteraemia; staphylococci and enterococci cause endocarditis.

3 DISEASE CAUSED BY THIS PROBLEM

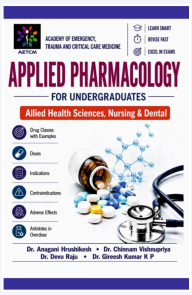
Gram-negative sepsis and pyelonephritis, febrile neutropenia, endocarditis (synergy with β-lactams), tuberculosis (streptomycin, amikacin), Pseudomonas in cystic fibrosis (tobramycin), plague, tularaemia.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Aminoglycosides bind the 30S subunit, causing misreading of mRNA and faulty proteins — rapidly bactericidal with concentration-dependent killing and a post-antibiotic effect, allowing once-daily dosing. Cure; usually short courses.

5 ADVERSE EFFECTS

Nephrotoxicity (acute tubular necrosis — usually reversible), ototoxicity (vestibular and cochlear, often irreversible; risk with loop diuretics), neuromuscular blockade (avoid in myasthenia gravis). Avoid in pregnancy (fetal deafness). Ineffective against anaerobes.



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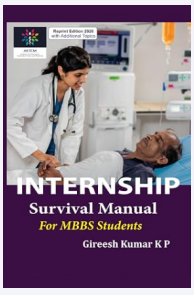
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Drug Table — 43. Aminoglycosides

Drugs in this class: Gentamicin, amikacin, tobramycin, streptomycin

Drug	Indications	Dose (adult)	Caution
Gentamicin	Severe gram-negative sepsis; pyelonephritis; endocarditis (synergy)	5–7 mg/kg IV once daily (level-guided)	Nephrotoxicity and ototoxicity — monitor levels and creatinine; avoid with loop diuretics
Amikacin	Resistant gram-negatives; MDR-TB (historic)	15 mg/kg IV once daily	Ototoxicity (irreversible), nephrotoxicity
Tobramycin	Pseudomonas (cystic fibrosis — inhaled)	5–7 mg/kg IV daily; inhaled 300 mg bd	As gentamicin
Streptomycin	TB (re-treatment); plague; tularaemia	15 mg/kg IM daily (max 1 g)	Vestibular toxicity; avoid in pregnancy
Neomycin	Bowel preparation; hepatic encephalopathy (historic)	1 g orally 4–6-hourly	Not for systemic use

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44. Fluoroquinolones

Key drugs: Ciprofloxacin, levofloxacin, moxifloxacin, ofloxacin

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Bacterial DNA must be supercoiled and untangled during replication by topoisomerase enzymes — DNA gyrase (topoisomerase II) in Gram-negatives and topoisomerase IV in Gram-positives.

2 PATHOLOGY

Gram-negative organisms (E. coli, Salmonella, Pseudomonas) and some respiratory pathogens cause urinary, enteric and respiratory infections; widespread use has led to high resistance in India.

3 DISEASE CAUSED BY THIS PROBLEM

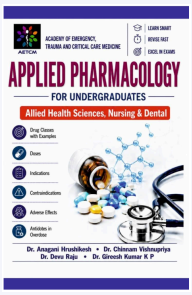
Complicated urinary tract infection and prostatitis, Pseudomonas infections (ciprofloxacin), community-acquired pneumonia (levofloxacin, moxifloxacin), drug-resistant tuberculosis, bone and joint infection, traveller's diarrhoea.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Fluoroquinolones inhibit DNA gyrase and topoisomerase IV, causing DNA breaks — rapidly bactericidal with excellent oral bioavailability and tissue penetration. Cure, but reserve for appropriate indications to limit resistance.

5 ADVERSE EFFECTS

Tendinitis and tendon rupture (elderly, steroids), QT prolongation, seizures and confusion, peripheral neuropathy, aortic aneurysm/dissection, dysglycaemia, photosensitivity, C. difficile, arthropathy concern in children. Raise theophylline and warfarin levels.



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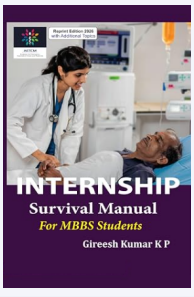
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Drug Table — 44. Fluoroquinolones

Drugs in this class: Ciprofloxacin, levofloxacin, moxifloxacin, ofloxacin

Drug	Indications	Dose (adult)	Caution
Drug	Indications	Dose (adult)	Caution
Ciprofloxacin	UTI, prostatitis, enteric fever (resistance common), gram-negative infections, anthrax	250–750 mg bd orally; 400 mg IV 12-hourly	Tendon rupture, QT, seizures; chelated by antacids/iron; raises theophylline
Levofloxacin	Community pneumonia; MDR-TB; UTI	500–750 mg daily	As ciprofloxacin; aortic aneurysm risk
Moxifloxacin	Pneumonia; MDR-TB; skin infections	400 mg daily	Greatest QT prolongation; hepatotoxicity
Ofloxacin	UTI; leprosy (alternative); eye/ear drops	200–400 mg bd	As class; avoid in children and pregnancy where possible
Norfloxacin	UTI; SBP prophylaxis	400 mg bd (prophylaxis 400 mg daily)	Mainly urinary/GI action

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45. Tetracyclines

Key drugs: Doxycycline, minocycline, tetracycline, tigecycline

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Bacterial 70S ribosomes accept aminoacyl-tRNA at the A site of the 30S subunit to add amino acids to growing protein chains.

2 PATHOLOGY

Intracellular and atypical organisms — rickettsiae, chlamydiae, mycoplasma, spirochaetes — and Propionibacterium (Cutibacterium) acnes cause infections that β-lactams cannot reach.

3 DISEASE CAUSED BY THIS PROBLEM

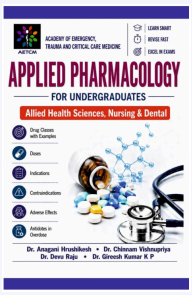
Scrub typhus and other rickettsial fevers, leptospirosis, atypical pneumonia, chlamydia, Lyme disease, brucellosis, cholera, acne and rosacea, malaria prophylaxis, MRSA skin infection.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Tetracyclines bind the 30S subunit and block tRNA attachment, stopping protein synthesis (bacteriostatic). Doxycycline concentrates well intracellularly — rapid defervescence in scrub typhus (within 48 h) — cure.

5 ADVERSE EFFECTS

Oesophageal ulceration (pill oesophagitis), photosensitivity, GI upset, tooth discolouration and bone deposition (avoid under 8 years and in pregnancy), benign intracranial hypertension, vestibular effects (minocycline). Absorption reduced by milk, antacids, iron, calcium.



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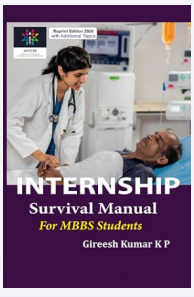
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Drug Table — 45. Tetracyclines

Drugs in this class: Doxycycline, minocycline, tetracycline, tigecycline

Drug	Indications	Dose (adult)	Caution
Doxycycline	Scrub typhus, leptospirosis, rickettsia, chlamydia, acne, malaria prophylaxis, cholera	100 mg bd (200 mg loading)	Oesophageal ulcer — take upright with water; photosensitivity; teeth staining (< 8 y, pregnancy)
Minocycline	Acne; MRSA skin infections	100 mg daily–bd	Vestibular effects, pigmentation, drug-induced lupus
Tetracycline	H. pylori quadruple therapy; acne	500 mg qds	Chelation by milk, antacids, iron; Fanconi syndrome with expired drug
Tigecycline	MDR complicated skin and intra-abdominal infections (reserve)	100 mg IV, then 50 mg 12-hourly	Nausea; increased mortality — last resort
Demeclocycline	SIADH (induces nephrogenic diabetes insipidus)	150–300 mg bd–qds	Photosensitivity, nephrotoxicity in cirrhosis

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol.
Facing page: physiology → pathology → disease → drug action → adverse effects.



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46. Other Antibacterials

Key drugs: Vancomycin, linezolid, metronidazole, nitrofurantoin, co-trimoxazole, clindamycin

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Bacteria have unique structures and metabolic pathways — Gram-positive cell walls, anaerobic metabolism, folate synthesis from PABA — that drugs can target selectively.

2 PATHOLOGY

Resistant Gram-positive organisms (MRSA, enterococci), anaerobes (Bacteroides, C. difficile), urinary pathogens and opportunistic organisms (Pneumocystis) cause specific infections that need targeted drugs.

3 DISEASE CAUSED BY THIS PROBLEM

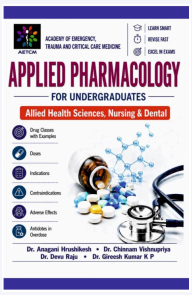
MRSA infections and C. difficile (vancomycin), VRE and MRSA pneumonia (linezolid), anaerobic and protozoal infections, amoebiasis (metronidazole), cystitis (nitrofurantoin), Pneumocystis pneumonia and UTI (co-trimoxazole), skin/bone infection (clindamycin).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Vancomycin blocks cell-wall precursors; linezolid inhibits 50S initiation complex; metronidazole forms toxic radicals in anaerobes; nitrofurantoin damages bacterial DNA in urine; co-trimoxazole blocks two steps of folate synthesis. Cure of susceptible infection.

5 ADVERSE EFFECTS

Vancomycin — nephrotoxicity, red-man syndrome. Linezolid — thrombocytopenia, serotonin syndrome, optic neuropathy. Metronidazole — metallic taste, disulfiram reaction with alcohol, neuropathy. Nitrofurantoin — pulmonary and hepatic toxicity, avoid in CKD. Co-trimoxazole — hyperkalaemia, rash/SJS, marrow suppression.



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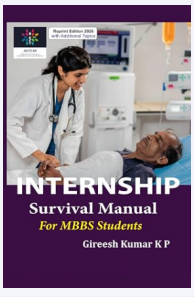
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Drug Table — 46. Other Antibacterials

Drugs in this class: Vancomycin, linezolid, metronidazole, nitrofurantoin, co-trimoxazole, clindamycin

Drug	Indications	Dose (adult)	Caution
Vancomycin	MRSA infections; C. difficile (oral); febrile neutropenia with line infection	15–20 mg/kg IV 8–12-hourly (trough/ AUC guided); C. diff 125 mg qds oral	Nephrotoxicity; red-man syndrome with fast infusion (give over ≥ 1 h)
Linezolid	MRSA and VRE; MDR-TB	600 mg bd orally/IV	Thrombocytopenia (> 2 weeks), serotonin syndrome, optic neuropathy
Metronidazole	Anaerobic and protozoal infections; C. difficile; bacterial vaginosis; amoebiasis	400–500 mg tds orally; 500 mg IV 8-hourly	Disulfiram-like reaction with alcohol; metallic taste; neuropathy
Nitrofurantoin	Uncomplicated lower UTI	100 mg MR bd × 5 days	Avoid if eGFR < 45; pulmonary fibrosis and neuropathy with long-term use
Co-trimoxazole	UTI; PJP treatment and prophylaxis; toxoplasmosis; nocardia	960 mg bd; PJP 120 mg/kg/day in divided doses	Hyperkalaemia, marrow suppression, SJS; folate antagonism
Clindamycin	Skin/soft tissue; bone infection; toxin suppression in necrotising fasciitis	300–450 mg qds orally; 600–900 mg IV 8-hourly	C. difficile colitis

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47. Antitubercular Drugs

Key drugs: Isoniazid, rifampicin, pyrazinamide, ethambutol, bedaquiline

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Mycobacterium tuberculosis has a thick, waxy, mycolic-acid-rich cell wall, grows very slowly and can survive inside macrophages and in dormant states within granulomas.

2 PATHOLOGY

Inhaled bacilli are contained in granulomas (latent TB); when immunity falls (HIV, diabetes, malnutrition, steroids, ageing), they reactivate, causing caseous necrosis, cavitation and spread to other organs.

3 DISEASE CAUSED BY THIS PROBLEM

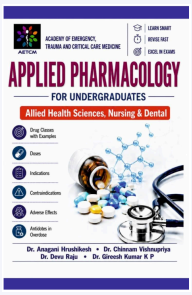
Pulmonary tuberculosis, extrapulmonary TB (lymph node, pleural, spinal, meningeal, abdominal), latent TB infection (preventive therapy), drug-resistant TB.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Isoniazid blocks mycolic acid synthesis; rifampicin blocks bacterial RNA polymerase (sterilising); pyrazinamide kills semi-dormant bacilli in acidic environments; ethambutol inhibits cell-wall arabinogalactan. Combination therapy prevents resistance — 6 months of treatment cures >85% of drug-sensitive TB.

5 ADVERSE EFFECTS

Hepatotoxicity (isoniazid, rifampicin, pyrazinamide — monitor LFT), peripheral neuropathy (isoniazid — give pyridoxine), orange-red body fluids and enzyme induction (rifampicin — reduces OCP, warfarin, ART levels), gout/arthralgia (pyrazinamide), optic neuritis (ethambutol — check vision). Bedaquiline — QT prolongation.



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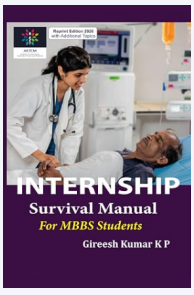
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Drug Table — 47. Antitubercular Drugs

Drugs in this class: Isoniazid, rifampicin, pyrazinamide, ethambutol, bedaquiline

Drug	Indications	Dose (adult)	Caution
Isoniazid (H)	All forms of TB; TB preventive therapy	5 mg/kg daily (max 300 mg) with pyridoxine	Hepatotoxicity, peripheral neuropathy (give pyridoxine); slow acetylators
Rifampicin (R)	TB; leprosy; prophylaxis of meningococcal contacts	10 mg/kg daily (max 600 mg)	Orange body fluids; hepatotoxicity; potent enzyme inducer — OCP, warfarin, ART
Pyrazinamide (Z)	TB intensive phase (2 months)	25 mg/kg daily	Hepatotoxicity, hyperuricaemia/ gout, arthralgia
Ethambutol (E)	TB intensive phase	15 mg/kg daily	Optic neuritis — check visual acuity and colour vision; renal adjustment
Bedaquiline	MDR/RR-TB (all-oral regimens)	400 mg daily × 2 weeks, then 200 mg thrice weekly (per NTEP)	QT prolongation — ECG monitoring
Fixed-dose combination (HRZE)	Drug-sensitive TB (NTEP daily regimen)	Weight-band FDC tablets daily × 2 months, then HRE × 4 months	Monitor LFTs; adherence (99DOTS/Nikshay)

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48. Antifungal, Antiviral and Antiparasitic Drugs

Key drugs: Fluconazole, amphotericin B, aciclovir, oseltamivir, artesunate, albendazole

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Fungi have ergosterol (not cholesterol) in their membranes; viruses replicate inside host cells using their own enzymes (DNA polymerase, neuraminidase); malaria parasites digest haemoglobin in red cells; worms rely on microtubules.

2 PATHOLOGY

Opportunistic fungi (Candida, Aspergillus, Mucor) invade immunocompromised hosts; herpes viruses and influenza replicate and destroy cells; Plasmodium multiplies in red cells causing haemolysis and microvascular obstruction; intestinal worms cause malnutrition and anaemia.

3 DISEASE CAUSED BY THIS PROBLEM

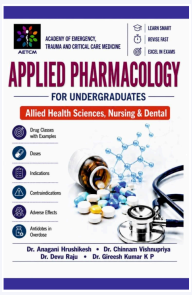
Candidiasis, cryptococcal meningitis, mucormycosis; herpes simplex and zoster, influenza; malaria (P. falciparum, vivax); helminthiasis (roundworm, hookworm, neurocysticercosis).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Azoles block ergosterol synthesis; amphotericin binds ergosterol and punches holes; aciclovir is activated by viral thymidine kinase to stop viral DNA synthesis; oseltamivir blocks viral release; artemisinins generate free radicals that kill parasites rapidly; albendazole binds worm tubulin. Cure or suppression.

5 ADVERSE EFFECTS

Azoles — hepatotoxicity, QT, many CYP interactions. Amphotericin — nephrotoxicity, hypokalaemia, infusion reactions. Aciclovir — crystal nephropathy (hydrate), neurotoxicity in renal failure. Artesunate — delayed haemolysis. Albendazole — teratogenic, seizures from dying cysts.



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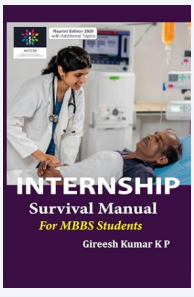
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Drug Table — 48. Antifungal, Antiviral and Antiparasitic Drugs

Drugs in this class: Fluconazole, amphotericin B, aciclovir, oseltamivir, artesunate, albendazole

Drug	Indications	Dose (adult)	Caution
Fluconazole	Oral/oesophageal/ vaginal candidiasis; cryptococcal maintenance	150 mg single (vaginal); 50–200 mg daily	CYP inhibition (warfarin, statins); QT; hepatotoxicity
Amphotericin B (liposomal)	Mucormycosis; cryptococcal meningitis; invasive fungal infection; kala-azar	3–5 mg/kg/day IV (mucor 5–10 mg/kg)	Nephrotoxicity, hypokalaemia, hypomagnesaemia, infusion reactions
Aciclovir	Herpes simplex, zoster, HSV encephalitis, chickenpox	Zoster 800 mg 5×/ day; encephalitis 10 mg/kg IV 8-hourly	Crystal nephropathy — hydrate; renal adjustment
Oseltamivir	Influenza treatment and prophylaxis	75 mg bd × 5 days	Nausea; start within 48 h for best effect
Artesunate	Severe and P. falciparum malaria (with partner drug — ACT)	Severe: 2.4 mg/kg IV at 0, 12, 24 h then daily; ACT per NVBDCP	Post-artesunate delayed haemolysis
Albendazole	Roundworm, hookworm, neurocysticercosis, hydatid disease; deworming	400 mg single dose; NCC 15 mg/kg/day × 8–28 days	Teratogenic in first trimester; seizures in NCC (give steroids)

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.



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49. Proton Pump Inhibitors (PPIs)

Key drugs: Omeprazole, pantoprazole, esomeprazole, rabeprazole, lansoprazole

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Gastric parietal cells secrete hydrochloric acid through the H+/K+-ATPase (proton pump), stimulated by histamine (H2), gastrin and acetylcholine. The mucosa is protected by mucus, bicarbonate and prostaglandins.

2 PATHOLOGY

Acid injures mucosa when defences fail — H. pylori infection and NSAIDs cause peptic ulcers; incompetent lower oesophageal sphincter causes reflux oesophagitis; gastrinoma causes massive acid output.

3 DISEASE CAUSED BY THIS PROBLEM

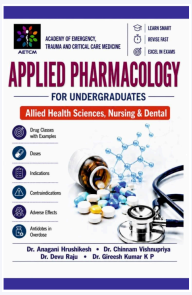
Peptic ulcer disease and bleeding ulcers, GERD and oesophagitis, H. pylori eradication (with antibiotics), NSAID gastroprotection, Zollinger-Ellison syndrome, stress-ulcer prophylaxis in ICU.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

PPIs are activated in the acidic parietal canaliculus and irreversibly block the proton pump — the final step of acid secretion — reducing acid by >90%. Ulcers heal in 4–8 weeks; with H. pylori eradication, ulcers are cured.

5 ADVERSE EFFECTS

Headache, diarrhoea, C. difficile infection, pneumonia, hypomagnesaemia, B12 deficiency, fractures, interstitial nephritis with long-term use; rebound acid hypersecretion on stopping. Omeprazole reduces clopidogrel activation (prefer pantoprazole).



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Drug Table — 49. Proton Pump Inhibitors (PPIs)

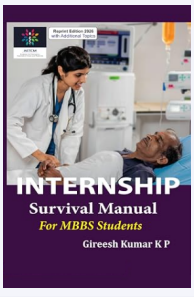
Drugs in this class: Omeprazole, pantoprazole, esomeprazole, rabeprazole, lansoprazole

Drug	Indications	Dose (adult)	Caution
Omeprazole	GERD; peptic ulcer; H. pylori eradication; NSAID gastroprotection	20–40 mg daily before breakfast	Long-term: hypomagnesaemia, B12 deficiency, fractures, C. difficile; reduces clopidogrel activation
Pantoprazole	As omeprazole; upper GI bleeding (IV); stress-ulcer prophylaxis	40 mg daily; GI bleed 80 mg IV then 8 mg/h (or 40 mg bd)	Fewer interactions — preferred with clopidogrel
Esomeprazole	Erosive oesophagitis; GERD	20–40 mg daily	As class
Rabeprazole	GERD; H. pylori	20 mg daily	As class
Lansoprazole	GERD; peptic ulcer	15–30 mg daily	As class; orodispersible form available

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.

★ REMEMBER — EXAM POINTS

- PPIs irreversibly block the H^+/K^+-ATPase — take 30–60 min before breakfast for best effect.
- Review long-term use: deprescribe when the indication has ended.
- H. pylori triple therapy: PPI + amoxicillin + clarithromycin for 14 days.



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50. H2-Receptor Antagonists and Antacids

Key drugs: Famotidine, ranitidine (withdrawn), aluminium hydroxide, magnesium hydroxide, alginates

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Histamine released from enterochromaffin-like cells acts on H2 receptors of parietal cells, raising cyclic AMP and stimulating acid secretion, especially at night.

2 PATHOLOGY

Excess acid or reflux of acid into the oesophagus causes heartburn, dyspepsia and erosions; nocturnal acid breakthrough persists despite PPIs in some patients.

3 DISEASE CAUSED BY THIS PROBLEM

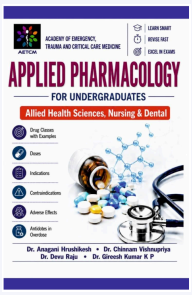
Dyspepsia, mild GERD and heartburn, peptic ulcer (less used now), nocturnal acid symptoms, aspiration prophylaxis before anaesthesia (with sodium citrate), hyperphosphataemia (aluminium salts, historic).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

H2 antagonists competitively block parietal-cell H2 receptors, reducing basal and nocturnal acid by ~60–70%. Antacids neutralise existing acid within minutes (short relief); alginates form a raft that blocks reflux. Symptom relief.

5 ADVERSE EFFECTS

H2 antagonists — headache, confusion in elderly/renal failure; cimetidine inhibits CYP enzymes and causes gynaecomastia. Antacids — constipation (aluminium), diarrhoea (magnesium), reduced absorption of tetracyclines, quinolones, iron, levothyroxine; sodium load.



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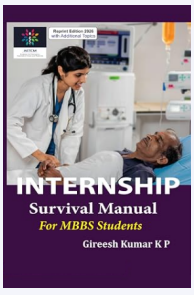
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Drug Table — 50. H2-Receptor Antagonists and Antacids

Drugs in this class: Famotidine, ranitidine (withdrawn), aluminium hydroxide, magnesium hydroxide, alginates

Drug	Indications	Dose (adult)	Caution
Famotidine	Dyspepsia; GERD (mild); stress-ulcer prophylaxis; urticaria (adjunct)	20–40 mg bd	Reduce in renal impairment; confusion in elderly
Ranitidine (withdrawn)	Formerly GERD and peptic ulcer	— (withdrawn 2020 due to NDMA contamination)	Replace with famotidine or PPI
Aluminium hydroxide	Dyspepsia; hyperphosphataemia (binder)	10–20 mL after meals	Constipation; aluminium toxicity in renal failure; chelates tetracyclines, quinolones
Magnesium hydroxide	Dyspepsia; constipation	10–20 mL	Diarrhoea; hypermagnesaemia in renal failure
Sodium alginate (Gaviscon)	GERD and reflux in pregnancy	10–20 mL after meals and at bedtime	Sodium content — caution in heart failure

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol.
Facing page: physiology → pathology → disease → drug action → adverse effects.



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51. Antiemetics

Key drugs: Ondansetron, metoclopramide, domperidone, prochlorperazine, cyclizine, aprepitant

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Vomiting is coordinated by the vomiting centre in the medulla, which receives input from the chemoreceptor trigger zone (dopamine D2, 5-HT3, NK1), the gut (5-HT3 via vagus), the vestibular system (H1, muscarinic) and the cortex.

2 PATHOLOGY

Chemotherapy releases serotonin from gut cells; drugs, uraemia and toxins stimulate the CTZ; motion and vestibular disease stimulate vestibular input; gastric stasis and raised intracranial pressure also trigger vomiting.

3 DISEASE CAUSED BY THIS PROBLEM

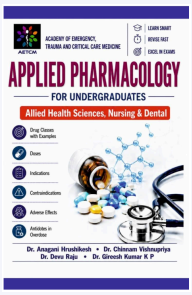
Chemotherapy- and radiotherapy-induced nausea, post-operative nausea, gastroenteritis, gastroparesis, motion sickness and vertigo, nausea of pregnancy (hyperemesis), opioid-induced nausea, palliative care.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Ondansetron blocks 5-HT3; metoclopramide and domperidone block D2 and promote gastric emptying; prochlorperazine blocks D2; cyclizine blocks H1/muscarinic; aprepitant blocks NK1. Choosing by cause gives effective symptom control.

5 ADVERSE EFFECTS

Ondansetron — constipation, headache, QT prolongation. Metoclopramide — acute dystonia (young women), tardive dyskinesia, avoid in Parkinson's and bowel obstruction. Domperidone — QT prolongation. Prochlorperazine — extrapyramidal effects, sedation. Cyclizine — anticholinergic effects.



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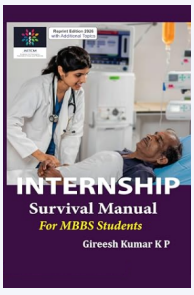
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Drug Table — 51. Antiemetics

Drugs in this class: Ondansetron, metoclopramide, domperidone, prochlorperazine, cyclizine, aprepitant

Drug	Indications	Dose (adult)	Caution
Ondansetron (5-HT3)	Chemotherapy, radiotherapy and post-operative nausea and vomiting; gastroenteritis vomiting	4–8 mg orally/IV 8–12-hourly (max 16 mg single IV)	Constipation, headache, QT prolongation
Metoclopramide (D2 + prokinetic)	Gastric stasis, migraine-associated nausea, post-operative nausea	10 mg tds (max 30 mg/day, ≤ 5 days)	Acute dystonia (young women), tardive dyskinesia; avoid in bowel obstruction and Parkinson's
Domperidone	Nausea, gastroparesis (does not cross BBB)	10 mg up to tds (max 30 mg/day)	QT prolongation — avoid in cardiac disease
Prochlorperazine	Vertigo; nausea	5–10 mg tds; 12.5 mg IM	Extrapyramidal effects, sedation
Cyclizine (H1)	Vestibular nausea, raised ICP, motion sickness, bowel obstruction	50 mg tds orally/ IM/SC	Anticholinergic; antagonises metoclopramide
Aprepitant (NK1)	Highly emetogenic chemotherapy (delayed nausea)	125 mg day 1, 80 mg days 2–3	CYP3A4 interactions — reduce dexamethasone dose

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol.
Facing page: physiology → pathology → disease → drug action → adverse effects.



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52. Laxatives and Antidiarrhoeals

Key drugs: Lactulose, polyethylene glycol, senna, bisacodyl, ispaghula, loperamide, ORS

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

The colon absorbs water and electrolytes and propels stool by coordinated peristalsis; the enteric nervous system controls motility and secretion. Normal stool needs fibre and fluid.

2 PATHOLOGY

Constipation results from low fibre/fluid, immobility, drugs (opioids, anticholinergics, iron), hypothyroidism, hypercalcaemia or neurological disease. Diarrhoea results from infection, toxins (cholera, E. coli) causing secretion, or rapid transit.

3 DISEASE CAUSED BY THIS PROBLEM

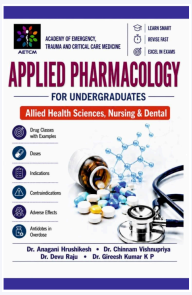
Chronic and opioid-induced constipation, bowel preparation, hepatic encephalopathy (lactulose), faecal impaction; acute infectious diarrhoea, traveller's diarrhoea, IBS-diarrhoea, high-output stomas.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Osmotic laxatives (lactulose, PEG) retain water in the bowel; stimulants (senna, bisacodyl) increase peristalsis; bulk-formers add fibre. Loperamide acts on gut μ-opioid receptors to slow transit. ORS uses sodium–glucose co-transport to replace losses — life-saving in diarrhoea.

5 ADVERSE EFFECTS

Lactulose — bloating, flatulence. Stimulants — cramps; chronic abuse causes hypokalaemia. Bulk-forming — obstruction if inadequate fluid. Loperamide — avoid in bloody diarrhoea/C. difficile (risk of toxic megacolon); cardiotoxic in overdose.



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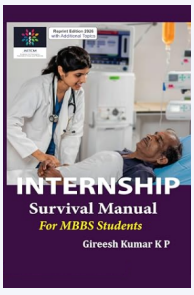
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Drug Table — 52. Laxatives and Antidiarrhoeals

Drugs in this class: Lactulose, polyethylene glycol, senna, bisacodyl, ispaghula, loperamide, ORS

Drug	Indications	Dose (adult)	Caution
Lactulose (osmotic)	Constipation; hepatic encephalopathy	15–30 mL bd; encephalopathy 30 mL titrated to 2–3 soft stools	Bloating, flatulence, takes 48 h
Polyethylene glycol (macrogol)	Chronic constipation; faecal impaction; bowel preparation	1–3 sachets daily; impaction up to 8 sachets/day	Fluid/electrolyte shifts in bowel prep
Senna (stimulant)	Constipation including opioid-induced	7.5–15 mg at night	Abdominal cramps; avoid in obstruction
Bisacodyl (stimulant)	Constipation; bowel preparation	5–10 mg at night; 10 mg suppository	Cramps; hypokalaemia with overuse
Ispaghula husk (bulk-forming)	Chronic constipation; IBS; haemorrhoids	3.5 g sachet bd with plenty of water	Avoid in faecal impaction, dysphagia, immobile patients
Loperamide (antidiarrhoeal)	Acute non-infective diarrhoea; ileostomy output	4 mg then 2 mg after each loose stool (max 16 mg/day)	Avoid in bloody diarrhoea, C. difficile, children < 4 y; cardiac toxicity in overdose
ORS (reduced osmolarity) + zinc	Acute diarrhoea and dehydration	ORS 50–100 mL/kg over 4 h; zinc 20 mg/day × 14 days (children)	Correct preparation — 1 sachet in 1 litre clean water

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53. Insulin

Key drugs: Regular (soluble) insulin, NPH, glargine, detemir, aspart, lispro, premixed

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Beta cells of the pancreatic islets secrete insulin in response to rising glucose; insulin promotes glucose uptake into muscle and fat (GLUT4), glycogen and fat storage, and suppresses liver glucose output and ketone production.

2 PATHOLOGY

Type 1 diabetes — autoimmune destruction of beta cells causes absolute insulin deficiency (ketosis). Type 2 diabetes — insulin resistance with progressive beta-cell failure. Without insulin, glucose and ketones accumulate.

3 DISEASE CAUSED BY THIS PROBLEM

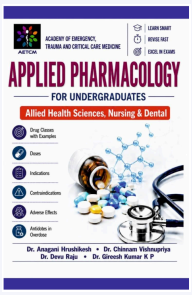
Type 1 diabetes (essential), type 2 diabetes when oral agents fail, gestational diabetes, diabetic ketoacidosis and hyperosmolar state, hyperglycaemia in critical illness/surgery, hyperkalaemia (with glucose).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Exogenous insulin replaces the missing hormone, lowering glucose and stopping ketogenesis — life-saving in DKA. Basal insulin controls fasting glucose; rapid-acting insulin covers meals. Control (remission rarely in early type 2 with intensive therapy).

5 ADVERSE EFFECTS

Hypoglycaemia (sweating, confusion, seizures — treat with glucose/glucagon), weight gain, lipohypertrophy at injection sites (rotate sites), hypokalaemia with IV insulin. Insulin is a "high-alert" drug — never abbreviate "U".



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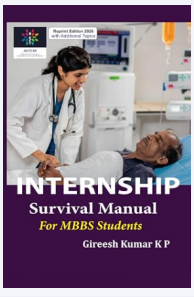
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Drug Table — 53. Insulin

Drugs in this class: Regular (soluble) insulin, NPH, glargine, detemir, aspart, lispro, premixed

Drug	Indications	Dose (adult)	Caution
Regular (soluble) insulin	DKA, hyperkalaemia, perioperative sliding scale, basal–bolus mealtime	DKA 0.1 U/kg/h IV infusion; SC 4–10 U before meals	Hypoglycaemia; hypokalaemia with IV infusion
NPH (isophane) insulin	Basal insulin; steroid-induced hyperglycaemia	0.1–0.2 U/kg/day (bd)	Nocturnal hypoglycaemia (peak 4–8 h)
Insulin glargine / detemir (long-acting)	Basal insulin in type 1 and type 2 diabetes	Start 10 U or 0.1–0.2 U/kg once daily; titrate by fasting glucose	Do not mix with other insulins
Insulin aspart / lispro (rapid-acting)	Mealtime bolus; insulin pumps	Before meals (0–15 min), dose by carbohydrate	Hypoglycaemia if meal delayed
Premixed insulin (30/70)	Type 2 diabetes — twice-daily regimens	Start 0.2–0.3 U/kg/day in 2 divided doses before breakfast and dinner	Fixed ratio — less flexible; hypoglycaemia
Glucagon / IV dextrose (rescue)	Severe hypoglycaemia	Glucagon 1 mg IM/SC; 25% dextrose 100 mL or 50 mL of 50% IV	Glucagon ineffective in liver disease/alcohol excess

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54. Oral and Non-Insulin Antidiabetic Drugs

Key drugs: Metformin, gliclazide, sitagliptin, empagliflozin, dapagliflozin, pioglitazone, semaglutide

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Blood glucose is kept between ~70 and 140 mg/dL by insulin (from beta cells), glucagon, incretin hormones from the gut (GLP-1, GIP) and renal glucose reabsorption via SGLT2 in the proximal tubule.

2 PATHOLOGY

In type 2 diabetes, insulin resistance (obesity, inactivity, genetics) and progressive beta-cell failure, plus increased hepatic glucose output, reduced incretin effect and increased renal glucose reabsorption, cause chronic hyperglycaemia.

3 DISEASE CAUSED BY THIS PROBLEM

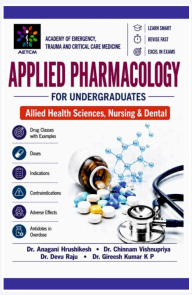
Type 2 diabetes mellitus and its complications (CVD, nephropathy, retinopathy, neuropathy); prediabetes, PCOS (metformin); obesity, heart failure and CKD (SGLT2i, GLP-1 RA).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Metformin reduces liver glucose output; sulphonylureas close beta-cell KATP channels to release insulin; DPP-4 inhibitors raise incretins; SGLT2 inhibitors excrete glucose in urine and protect heart and kidneys; GLP-1 agonists increase insulin, reduce appetite and weight. Control; remission possible with major weight loss.

5 ADVERSE EFFECTS

Metformin — GI upset, B12 deficiency, lactic acidosis (avoid eGFR <30). Sulphonylureas — hypoglycaemia, weight gain. SGLT2i — genital infections, dehydration, euglycaemic DKA. Pioglitazone — oedema, heart failure, fractures. GLP-1 RA — nausea, pancreatitis, gallstones.



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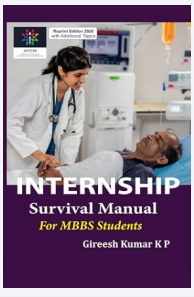
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Drug Table — 54. Oral and Non-Insulin Antidiabetic Drugs

Drugs in this class: Metformin, gliclazide, sitagliptin, empagliflozin, dapagliflozin, pioglitazone, semaglutide

Drug	Indications	Dose (adult)	Caution
Metformin	First-line type 2 diabetes; PCOS; prediabetes	500 mg with meals → 1 g bd (max 2.5 g)	GI upset, B12 deficiency, lactic acidosis — stop if eGFR < 30, before contrast/surgery
Gliclazide (sulfonylurea)	Type 2 diabetes	40–160 mg bd (MR 30–120 mg daily)	Hypoglycaemia (elderly, renal failure), weight gain
Sitagliptin (DPP-4i)	Type 2 diabetes (add-on)	100 mg daily (50 mg eGFR 30–45; 25 mg < 30)	Pancreatitis (rare); weight neutral
Empagliflozin / dapagliflozin (SGLT2i)	Type 2 diabetes; heart failure; CKD	Empagliflozin 10–25 mg; dapagliflozin 10 mg daily	Genital mycotic infections, euglycaemic DKA, volume depletion — sick-day rules
Pioglitazone	Type 2 diabetes with insulin resistance; NASH	15–45 mg daily	Fluid retention, heart failure, fractures, bladder cancer
Semaglutide (GLP-1 RA)	Type 2 diabetes; obesity; cardiovascular risk reduction	0.25 mg SC weekly → 1–2 mg; oral 3–14 mg daily	Nausea, vomiting; pancreatitis; not in MTC/MEN2 history

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.



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55. Thyroid and Antithyroid Drugs

Key drugs: Levothyroxine, carbimazole, methimazole, propylthiouracil, radioactive iodine, propranolol

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

The thyroid uses iodine and thyroid peroxidase to make T4 and T3 under TSH control from the pituitary; thyroid hormones set metabolic rate, heart rate, temperature, growth and brain development.

2 PATHOLOGY

Hypothyroidism — autoimmune destruction (Hashimoto's), iodine deficiency, post-surgery/radioiodine. Hyperthyroidism — TSH-receptor stimulating antibodies (Graves'), toxic nodules, thyroiditis.

3 DISEASE CAUSED BY THIS PROBLEM

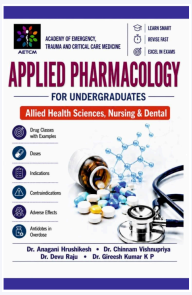
Hypothyroidism (fatigue, weight gain, cold intolerance, myxoedema), congenital hypothyroidism; hyperthyroidism (weight loss, tremor, AF, eye disease), thyroid storm.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Levothyroxine replaces T4 (converted to active T3) — lifelong replacement restores normal state. Carbimazole/methimazole and PTU inhibit thyroid peroxidase, blocking hormone synthesis (remission in ~50% of Graves' after 12–18 months); radioiodine destroys thyroid tissue (definitive cure); β-blockers control adrenergic symptoms.

5 ADVERSE EFFECTS

Levothyroxine over-replacement — AF, angina, osteoporosis. Carbimazole/PTU — agranulocytosis (stop and check blood count if sore throat or fever), rash, hepatotoxicity (PTU), teratogenicity (carbimazole in first trimester). Radioiodine — hypothyroidism, contraindicated in pregnancy.



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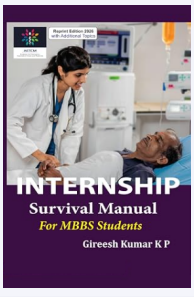
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Drug Table — 55. Thyroid and Antithyroid Drugs

Drugs in this class: Levothyroxine, carbimazole, methimazole, propylthiouracil, radioactive iodine, propranolol

Drug	Indications	Dose (adult)	Caution
Levothyroxine	Hypothyroidism; post-thyroidectomy; TSH suppression in thyroid cancer	1.6 µg/kg/day; start 25–50 µg in elderly/cardiac disease	Take on empty stomach; separate from iron/calcium; angina, AF if overdosed
Carbimazole	Graves' disease and toxic nodules	15–40 mg daily, then 5–15 mg maintenance (or block-replace)	Agranulocytosis — stop if sore throat/ fever; teratogenic in 1st trimester
Methimazole	Hyperthyroidism (active metabolite of carbimazole)	10–30 mg daily	As carbimazole
Propylthiouracil	Hyperthyroidism in first trimester; thyroid storm	100–150 mg tds; storm 200–250 mg 4-hourly	Severe hepatotoxicity — second line otherwise
Radioactive iodine (I-131)	Graves', toxic nodule, thyroid cancer ablation	Single oral dose (MBq per indication)	Contraindicated in pregnancy/ breastfeeding; hypothyroidism later
Propranolol	Symptom control in thyrotoxicosis; thyroid storm	20–40 mg 6–8-hourly	Avoid in asthma — use diltiazem

Doses are typical adult doses for orientation — always check the current formulary, renal/hepatic function, age and local protocol. Facing page: physiology → pathology → disease → drug action → adverse effects.



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56. Immunosuppressants

Key drugs: Tacrolimus, cyclosporine, mycophenolate, azathioprine, methotrexate, biologics

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

The immune system distinguishes self from non-self; T cells activated via the T-cell receptor and calcineurin–IL-2 signalling proliferate and coordinate B-cell antibody responses and inflammation.

2 PATHOLOGY

Loss of tolerance causes autoimmune diseases (rheumatoid arthritis, SLE, IBD, vasculitis); a transplanted organ is recognised as foreign and rejected by T cells and antibodies.

3 DISEASE CAUSED BY THIS PROBLEM

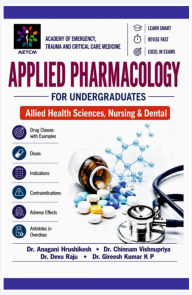
Organ transplantation (prevention of rejection), rheumatoid arthritis, psoriasis, SLE and lupus nephritis, inflammatory bowel disease, vasculitis, autoimmune hepatitis.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Calcineurin inhibitors (tacrolimus, cyclosporine) block IL-2 production; mycophenolate and azathioprine block lymphocyte purine synthesis; methotrexate inhibits folate metabolism; biologics block specific cytokines (TNF, IL-6) or cells (CD20). They induce and maintain remission of autoimmune disease and prevent rejection.

5 ADVERSE EFFECTS

Infections (including TB, opportunistic), malignancy (skin cancer, lymphoma), bone marrow suppression; tacrolimus/cyclosporine — nephrotoxicity, hypertension, diabetes, tremor, many CYP3A4 interactions; methotrexate — hepatotoxicity, pneumonitis, mucositis, teratogenic; azathioprine — toxicity with allopurinol.



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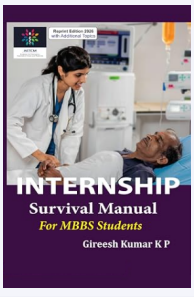
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Drug Table — 56. Immunosuppressants

Drugs in this class: Tacrolimus, cyclosporine, mycophenolate, azathioprine, methotrexate, biologics

Drug	Indications	Dose (adult)	Caution
Tacrolimus	Prevention of transplant rejection; atopic dermatitis (topical)	0.1–0.2 mg/kg/day in 2 doses, trough-level guided	Nephrotoxicity, diabetes, tremor, hypertension; CYP3A4 interactions
Cyclosporine	Transplant rejection; psoriasis; rheumatoid arthritis; nephrotic syndrome	2.5–5 mg/kg/day, level-guided	Nephrotoxicity, gum hypertrophy, hirsutism, hypertension
Mycophenolate mofetil	Transplant; lupus nephritis; autoimmune disease	1–1.5 g bd	Leucopenia, GI upset; teratogenic
Azathioprine	IBD; autoimmune hepatitis; transplant; vasculitis	1–2.5 mg/kg/day (check TPMT first)	Myelosuppression; allopurinol interaction (reduce dose by 75%)
Methotrexate	Rheumatoid arthritis, psoriasis (weekly); ectopic pregnancy; cancer	7.5–25 mg ONCE WEEKLY with folic acid 5 mg weekly	Fatal errors with daily dosing; marrow suppression, hepatotoxicity, pneumonitis
Biologics (e.g. infliximab, rituximab)	RA, IBD, psoriasis, vasculitis, lymphoma	Infliximab 5 mg/kg IV at 0, 2, 6 weeks then 8-weekly; rituximab per protocol	Screen TB and hepatitis B before starting; infusion reactions

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Facing page: physiology → pathology → disease → drug action → adverse effects.



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57. Drugs Acting on the Uterus and in Obstetrics

Key drugs: Oxytocin, misoprostol, carboprost, methylergometrine, magnesium sulphate, nifedipine

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Uterine smooth muscle contracts in response to oxytocin (receptors increase near term) and prostaglandins; after delivery, contraction of the myometrium compresses spiral arteries ("living ligatures") to stop bleeding.

2 PATHOLOGY

Uterine atony (prolonged labour, overdistension, infection) causes postpartum haemorrhage; failure of labour to progress needs induction; abnormal placentation causes pre-eclampsia with seizures (eclampsia); premature contractions cause preterm labour.

3 DISEASE CAUSED BY THIS PROBLEM

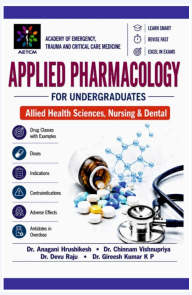
Induction and augmentation of labour, postpartum haemorrhage (leading cause of maternal death), medical termination and miscarriage management, eclampsia prevention and treatment, preterm labour (tocolysis).

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Oxytocin and prostaglandins (misoprostol, carboprost) and ergometrine contract the uterus — prevent and treat PPH. Magnesium sulphate stabilises neuronal membranes, preventing eclamptic seizures. Nifedipine and atosiban relax the uterus to delay preterm delivery for steroids to work.

5 ADVERSE EFFECTS

Oxytocin — uterine hyperstimulation, fetal distress, water intoxication/hyponatraemia. Ergometrine — hypertension (avoid in pre-eclampsia). Carboprost — bronchospasm (avoid in asthma). Misoprostol — fever, shivering, diarrhoea. MgSO4 toxicity — loss of patellar reflex, respiratory depression (antidote calcium gluconate).



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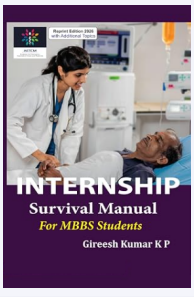
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Drug Table — 57. Drugs Acting on the Uterus and in Obstetrics

Drugs in this class: Oxytocin, misoprostol, carboprost, methylergometrine, magnesium sulphate, nifedipine

Drug	Indications	Dose (adult)	Caution
Oxytocin	Induction/ augmentation of labour; prevention and treatment of PPH (AMTSL)	PPH prevention 10 U IM; infusion 40 U in 500 mL; induction 1–2 mU/ min titrated	Uterine hyperstimulation, water intoxication (hyponatraemia), hypotension with rapid IV bolus
Misoprostol	PPH (where oxytocin unavailable); medical abortion; cervical ripening	PPH 600–800 µg SL/oral; induction 25 µg vaginal 4–6-hourly	Fever, shivering, diarrhoea; uterine rupture in scarred uterus
Carboprost (15-methyl PGF2α)	Refractory PPH due to atony	250 µg IM every 15–90 min (max 8 doses)	Bronchospasm — avoid in asthma; diarrhoea
Methylergometrine	PPH due to atony	200 µg IM/IV, repeat every 2–4 h	Contraindicated in hypertension and pre-eclampsia
Magnesium sulphate	Eclampsia and severe pre-eclampsia (seizure prophylaxis); fetal neuroprotection	Pritchard: 4 g IV + 10 g IM loading, then 5 g IM 4-hourly; Zuspan: 4 g IV then 1 g/h	Check reflexes, RR > 16, urine > 30 mL/h; antidote calcium gluconate 1 g IV
Nifedipine (tocolytic)	Preterm labour (to allow steroids); severe hypertension in pregnancy	Tocolysis 20 mg orally, then 10–20 mg 6–8-hourly × 48 h	Hypotension, headache

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58. Vaccines and Immunoglobulins

Key drugs: BCG, OPV/IPV, MMR, hepatitis B, tetanus toxoid, rabies vaccine and immunoglobulin, anti-snake venom

Normal physiology → Pathology → Disease → Drug action

1 PHYSIOLOGY

Adaptive immunity creates memory B and T cells after exposure to an antigen, allowing a faster, stronger response on re-exposure. Antibodies (immunoglobulins) neutralise toxins and microbes.

2 PATHOLOGY

Infectious diseases and toxins cause illness when the body lacks prior immunity; some (tetanus, rabies, diphtheria, snake venom) act faster than an immune response can develop.

3 DISEASE CAUSED BY THIS PROBLEM

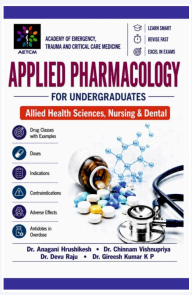
Vaccine-preventable infections (tuberculosis, polio, measles, hepatitis B, tetanus, diphtheria, pertussis, HPV, influenza, pneumococcus, COVID-19); post-exposure needs — rabies, tetanus-prone wounds, hepatitis B exposure, snakebite envenomation.

4 DRUG ACTION — CURE, CONTROL OR REMISSION

Vaccines (live attenuated, inactivated, toxoid, subunit, mRNA) induce active immunity and memory — long-term prevention. Immunoglobulins and antivenoms give immediate passive immunity, neutralising virus or toxin — life-saving post-exposure.

5 ADVERSE EFFECTS

Local pain, fever, malaise; rare anaphylaxis (keep adrenaline ready). Live vaccines — avoid in pregnancy and immunosuppression. Antivenom — early anaphylactoid reactions (adrenaline 0.5 mg IM), serum sickness later.



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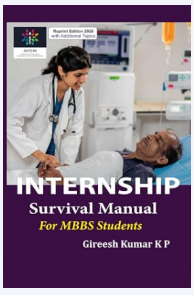
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Drug Table — 58. Vaccines and Immunoglobulins

Drugs in this class: BCG, OPV/IPV, MMR, hepatitis B, tetanus toxoid, rabies vaccine and immunoglobulin, anti-snake venom

Drug	Indications	Dose (adult)	Caution
BCG vaccine	Prevention of severe childhood TB (UIP); bladder cancer (intravesical)	0.05 mL intradermal at birth (0.1 mL > 1 month)	Live — contraindicated in immunodeficiency; local abscess/BCG-itis
OPV / IPV	Poliomyelitis prevention (UIP)	OPV 2 drops at birth, 6, 10, 14 weeks; fIPV 0.1 mL ID at 6 & 14 weeks (+9 months)	OPV live — VAPP rare; IPV for immunocompromised
MMR / MR	Measles, mumps, rubella	0.5 mL SC at 9–12 months and 16–24 months	Live — avoid in pregnancy and immunosuppression
Hepatitis B vaccine	Birth dose; health-care workers; at-risk adults	0.5 mL (child) / 1 mL (adult) IM at 0, 1, 6 months	Check anti-HBs in health-care workers
Tetanus toxoid (Td) / TIG	Wound management; antenatal (2 doses)	Td 0.5 mL IM; TIG 250–500 IU IM for tetanus-prone wounds	Do not over-boost (Arthus reaction)
Rabies vaccine + RIG	Post-exposure prophylaxis (category II and III bites)	Vaccine ID (Thai Red Cross 0, 3, 7, 28) or IM (0, 3, 7, 14, 28); RIG 20 IU/kg (human) infiltrated into wound for category III	Never delay; wound washing 15 min with soap and water
Anti-snake venom (polyvalent)	Envenomation by big four Indian snakes (cobra, krait, Russell's viper, saw-scaled viper)	10 vials IV (100 mL) over 1 h; repeat per 20WBCT/ neuroparalysis	Anaphylaxis — adrenaline 0.5 mg IM ready; no test dose

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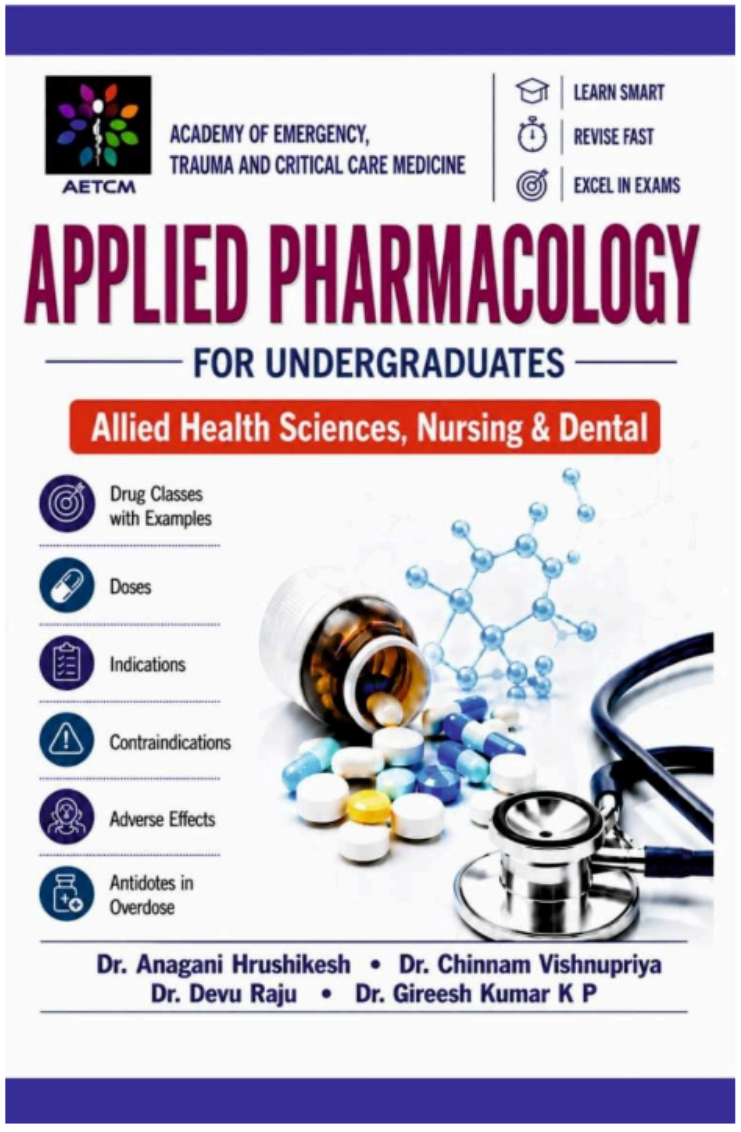
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