

LMR Notes 2.0 Medicine General Index

One index for all 12 systems of the NEET-SS Medicine notes. Use it to find the right system and section, then open that system's detailed index for topic-level pages. Page numbers match the circle at the top right of each page.

12 systems

478 pages

166 indexed topics

Find a system

Use Quick Jump below. Click any card to jump to its detailed table.

Find a topic

Bold rows are main headings; ↳ rows are sub-topics continuing on the next pages.

Go to the page

Open the notes at the coloured page number shown on the right.

Quick Jump

All 12 systems of LMR Notes 2.0 Medicine, in page order (pages 1–478). Click a system to jump to its section list.

1	Neurology	1–53	2	Oncology	54–78
3	Gastroenterology & Hepatology	79–116	4	Endocrinology	117–158
5	Hematology	159–186	6	Cardiology	187–221
7	Nephrology	222–261	8	Pulmonology & Critical Care Medicine	262–280
9	Rheumatology, Immunology & Infectious Diseases	281–338	10	Genetics, Environmental Disorders & Toxicology	339–364
11	High Yield Pointers	365–429	12	Recent Advances in Medicine	431–478

Each system has its own detailed index (separate PDFs) with topic-level page numbers. Environmental & Toxicology page numbers (346–364) are estimated – those pages all show "1" in the top-right circle. Pages 355–357 (missing Toxicology sections 1–3), 397, 413, 418 and 430 are not in the files received.

Detailed Index

Topic-wise contents of every system, with page numbers.

1 Neurology		Pages 1–53
TOPIC	WHAT YOU'LL FIND	PAGE
I. Cerebrovascular accidents	Ischemic stroke · Thrombolysis & thrombectomy · TIA & secondary prevention · NIHSS, anticoagulation, BP · Stroke syndromes – overview	1–12
II. Epilepsy	Status epilepticus · Drug choices for seizures · Epilepsy surgery · EEG & major epilepsy syndromes · Antiepileptic drugs	13–18
III. Movement disorders	Essential vs parkinsonian tremor · Genetics of Parkinson's disease · PD treatment – levodopa · Huntington's disease · Inherited ataxias – AD	18–30
IV. Demyelinating disorders	Multiple sclerosis · NMOSD (Devic's) · ADEM · Guillain–Barré syndrome	31–38
V. Neurodegenerative disorders	Alzheimer disease · Dementia with Lewy bodies · Frontotemporal dementia · ARIA	38–43
VI. Neuromuscular junction disorders	MG vs LEMS vs botulism	43–44
VII. Peripheral neuropathy	Neuropathy comparison · Patterns of diabetic neuropathy	45
VII*. CNS infections	CSF findings · Bacterial meningitis · Tuberculous meningitis · Viral meningitis / encephalitis · Neurocysticercosis · Cryptococcal meningitis	46–48
VIII. Paraneoplastic syndromes in CNS	Paraneoplastic syndromes	48
IX. Spinal cord lesions	Spinal cord lesions	49
X. Headache disorders	Migraine · Trigeminal autonomic cephalalgias · Idiopathic intracranial hypertension	50–53
2 Oncology		Pages 54–78
TOPIC	WHAT YOU'LL FIND	PAGE
1. Epidemiology	Cancer epidemiology · ECOG performance scale · Tumour markers	54
2. Symptom control in cancer	Cancer pain · GI & oral mucositis · Chemotherapy-induced nausea & vomiting	54–56
3. Smoking cessation & chemoprevention	Smoking cessation · Chemoprevention	56–57
4. Molecular basis of cancer	Oncogenes · Tumour suppressor genes · Familial cancer syndromes · Viral oncogenes	58–59
5. Cancer cell biology	Hallmarks of cancer cells · Cell-cycle checkpoints · Synthetic lethality & Warburg effect	59–60
6. Gompertzian growth & radiation oncology	Gompertzian tumour growth · Radiation oncology · Molecularly targeted agents	60–61
7. Neutropenic fever & enterocolitis	Neutropenic fever · Neutropenic enterocolitis (typhlitis)	62

TOPIC	WHAT YOU'LL FIND	PAGE
8. Oncologic emergencies	Superior vena cava syndrome · Spinal cord compression · Hypercalcaemia of malignancy · SIADH · Tumour lysis syndrome · Haemorrhagic cystitis	62–64
1. Breast cancer	Screening & diagnosis · Pathology, staging & management	64–66
2. Lung cancer (NSCLC & SCLC)	Presentation & staging · NSCLC – tests & management · SCLC	66–68
3. Testicular cancers	Essentials	68–70
4. Stomach (gastric) cancer	Essentials & diagnosis	70–71
5. Colon cancer	Essentials, diagnosis & staging	71–73
6. Hepatocellular carcinoma	HCC	74
7. Pancreatic cancer	Pancreatic cancer	75
8. GIST	Gastrointestinal stromal tumour	75
9. Ovarian cancer	Ovarian cancer	76
10. Cervical cancer	Cervical cancer	76
11. Osteosarcoma	Osteosarcoma	77
12. Ewing sarcoma	Ewing sarcoma	77
13. Renal cell carcinoma	Renal cell carcinoma	77
14. Prostate cancer	Prostate cancer	78

3 Gastroenterology & Hepatology

Pages 79–116

TOPIC	WHAT YOU'LL FIND	PAGE
1. Esophagus	Achalasia · Triple A (Allgrove) syndrome · Oesophageal motility disorders · Barrett's oesophagus · Eosinophilic oesophagitis · Infectious & pill oesophagitis	79–82
2. Stomach & duodenum	H. pylori – treatment regimens · Zollinger–Ellison syndrome · Chronic gastritis · Ménétrier disease	83–87
3. Small intestine	Disorders of absorption · Celiac disease · Whipple disease · SIBO · Inflammatory bowel disease – UC	88–97
3*. Pancreas	Acute pancreatitis · Autoimmune pancreatitis · Chronic pancreatitis	98–99
4. Liver & biliary	Primary sclerosing cholangitis · Primary biliary cholangitis	100
5. Vascular liver disorders	Budd–Chiari syndrome	100
6. Cirrhosis & portal hypertension	Portal hypertension · (i) Ascites · (ii) Spontaneous bacterial peritonitis · (iii) Variceal bleeding · (iv) Hepatic encephalopathy · (v) Hepatorenal syndrome	100–105
7. Hepatitis	Hepatitis B – high yield · Chronic hepatitis C · Autoimmune hepatitis · Drug-induced liver injury	105–111
8. Metabolic liver diseases	Wilson disease · Haemochromatosis · Alpha-1 antitrypsin deficiency	112–113
9. Trials in GI & liver	Landmark trials	113

TOPIC	WHAT YOU'LL FIND	PAGE
10. ALD & MASLD	MASLD · Alcohol-related liver disease	114–116

4 Endocrinology Pages 117–158

TOPIC	WHAT YOU'LL FIND	PAGE
1. Pituitary disorders	Pituitary disorders – summary table · Hyperprolactinaemia · Cushing disease vs ectopic ACTH · Diabetes insipidus vs SIADH	117–120
2. Thyroid disorders	Thyroid disorders – summary table · Sick euthyroid syndrome · Amiodarone & the thyroid	121–123
3. Adrenal disorders	Adrenal disorders – summary table · Cushing syndrome – stepwise diagnosis · Aldosterone–renin ratio · Thyroid in pregnancy † · Graves disease & thyroid storm † · Subacute & silent thyroiditis †	123–132
4. Parathyroid, calcium & osteoporosis	Calcium disorders – summary table · Primary hyperparathyroidism · Hypercalcaemia · Pseudohypoparathyroidism & PPHP · Hypoparathyroidism · Defective vitamin D metabolism	133–143
5. Diabetes mellitus & metabolic disorders	Diagnostic criteria (ADA 2025) · Classification of diabetes · Glycaemic targets (ADA 2025) · Pharmacologic management (ADA 2025) · Hypoglycaemia · Obesity	143–151
6. Genetic & syndromic disorders	Endocrine genetic syndromes · Autoimmune polyglandular syndromes	152–153
8. Disorders of lipoprotein metabolism	Primary hyperlipoproteinaemias · Secondary dyslipidaemia · Novel lipid drugs (2024–25)	154–155
9. Porphyrrias	Porphyrias	156
10. Storage diseases (lysosomal)	Lysosomal storage diseases	156–157
11. Sex & gender-based disorders	Disorders of sex development	157
12. Trials in endocrinology	Landmark trials	158

5 Hematology Pages 159–186

TOPIC	WHAT YOU'LL FIND	PAGE
A. Iron metabolism & microcytic anaemias	Iron metabolism & regulation · Microcytic hypochromic anaemias	159–161
B. Macrocytic anaemias & B12	Macrocytosis without megaloblastic change · Megaloblastic anaemia – other causes · Neurology of B12 deficiency	160–163
C. Marrow failure & MDS	Congenital bone marrow failure syndromes · Myelodysplastic syndromes	164–165
D. Myeloproliferative neoplasms	Polycythaemia vera · Essential thrombocythaemia	165–167
E. Haemoglobinopathies & PNH	Sickle cell anaemia · Thalassaemia · Paroxysmal nocturnal haemoglobinuria	167–170
F. Acute leukaemias	Acute myeloid leukaemia · Acute lymphoblastic leukaemia	171

TOPIC	WHAT YOU'LL FIND	PAGE
G. Chronic leukaemias	Chronic myeloid leukaemia · Chronic lymphocytic leukaemia	172
H. Plasma cell disorders	Multiple myeloma · MGUS · Waldenström macroglobulinaemia	172–174
I. Lymphomas	Hodgkin lymphoma · Non-Hodgkin lymphoma	174–178
J. DIC & thrombotic microangiopathies	Disseminated intravascular coagulation · TTP & HUS	179–180
K. Antiphospholipid & HIT	Antiphospholipid antibody syndrome · Heparin-induced thrombocytopenia	180–181
L. Bleeding disorders	von Willebrand disease · Haemophilia A & B · Immune thrombocytopenia · Trials in haematology	182–184
M. Transfusion medicine	Complications of blood transfusion	185–186

6 Cardiology

Pages 187–221

TOPIC	WHAT YOU'LL FIND	PAGE
1. Ischemic heart disease	Stable angina · Unstable angina / NSTEMI · STEMI · Acute coronary syndrome · Silent ischaemia & microvascular angina	187–192
2. Heart failure	Heart failure – causes & types · Cardiomyopathies · Mechanical circulatory support · Prosthetic valves · ICD & CRT	193–200
3. Valvular heart disease	Valve lesion severity · Mitral stenosis · Mitral regurgitation	201–203
4. Congenital heart disease in adults	Atrial septal defect · PAPVR, Ebstein anomaly, tetralogy of Fallot	204–205
5. Infective endocarditis	Endocarditis prophylaxis · Infective endocarditis – 2023 Duke-ISCVID criteria	206–208
6. Arrhythmias	Narrow-complex tachycardias · Broad-complex tachycardia · Atrial fibrillation · Heart blocks · Antiarrhythmic drugs · Brugada syndrome & POTS	209–213
7. Hypertension	Hypertension	214–215
8. Pericardial disease & pulmonary hypertension	Pericardial diseases · Pulmonary hypertension & cor pulmonale	216
9. PE, aorta & peripheral arteries	Pulmonary embolism · Aorta & peripheral artery disease	217
10. Miscellaneous & new concepts	Miscellaneous / new concepts · Cardiorenal syndrome · Thyrotoxic heart disease · Rheumatic heart disease & cardiac amyloid	218–220
11. Trials in cardiology	Landmark trials	221

7 Nephrology

Pages 222–261

TOPIC	WHAT YOU'LL FIND	PAGE
1. Glomerular diseases	Glomerular diseases – rapid summary · Nephrotic syndrome · Minimal change disease · Focal segmental glomerulosclerosis · Membranous nephropathy · IgA nephropathy – newer therapies	222–237
2. Tubulointerstitial diseases	Acute interstitial nephritis · Chronic tubulointerstitial nephritis	238–240

TOPIC	WHAT YOU'LL FIND	PAGE
3. Acute kidney injury	Acute kidney injury	240–241
4. Chronic kidney disease	CKD – classification & management · Anaemia of CKD · CKD–mineral bone disorder · Dialysis indications & bone phenotypes · Haemodialysis complications	242–252
5. Cystic kidney diseases	ADPKD – diagnosis	250–254
6. Renovascular disease & RRT	Hypertension & renovascular disorders · Renal replacement therapy	255
7. Electrolytes & tubular disorders	Sodium & potassium disorders · Renal tubular disorders · Nephrolithiasis	256–258
8. Acid–base balance	Acid–base disorders	259–260
9. Trials in nephrology	Trials in nephrology	261

8 Pulmonology & Critical Care Medicine

Pages 262–280

TOPIC	WHAT YOU'LL FIND	PAGE
1. Pulmonary physiology & PFT	Pulmonary physiology & PFT	262
2. COPD	COPD – GOLD 2025 update	263–264
3. Bronchiectasis	Bronchiectasis	265
4. Asthma	Asthma – GINA 2024 update	266–267
5. Pneumonia & lung abscess	Pneumonia – IDSA/ATS 2023 · Lung abscess	267–269
6. Interstitial & occupational lung disease	ILD – ATS/ERS comparison · Eosinophilic pneumonias · Occupational lung diseases · Idiopathic interstitial pneumonias · Aspergillosis	269–272
7. Pleura, sleep & ABPA criteria	Obstructive sleep apnoea · Pleural effusion · Sleep medicine (AASM 2023–24) · ABPA – revised ISHAM 2021 criteria	272–274
8. ARDS	ARDS – rapid review	275
9. Shock & haemodynamic assessment	Fluid responsiveness · POCUS in shock – RUSH protocol · Sepsis trials · Cardiogenic shock · Poisoning	276–277
10. Environmental emergencies	Heat-related illness	278
11. Genetics, ageing & emerging topics	Genetic diseases · Ageing · Emerging topics in medicine	279–280

9 Rheumatology, Immunology & Infectious Diseases

Pages 281–338

TOPIC	WHAT YOU'LL FIND	PAGE
1. SLE, APS & connective tissue diseases	Connective tissue diseases – autoantibodies · Systemic lupus erythematosus · Antiphospholipid syndrome · Systemic sclerosis · Sjögren syndrome · Myositis & MCTD	281–288
2. Rheumatoid arthritis & JIA	RA – classification & remission · Juvenile idiopathic arthritis · RA – extra-articular & treatment	289–297
3. Spondyloarthritis	SpA comparison table · Spondyloarthritis · Psoriatic & reactive arthritis	292–300

TOPIC	WHAT YOU'LL FIND	PAGE
4. Fibromyalgia & autoinflammatory syndromes	Fibromyalgia · Autoinflammatory syndromes · Crystal arthropathies	293–304
5. Sarcoidosis, IgG4 & trials	Sarcoidosis † · IgG4-related disease † · Trials in rheumatology · Rheumatology rapid recall	305–310
6. General principles, sepsis & HAIs	General principles in infectious diseases · Sepsis & ICU infections · Clostridioides difficile	311–313
7. Tuberculosis & mycobacteria	NTEP anti-TB regimens · Atypical mycobacteria	313–317
8. HIV & viral infections	HIV – system-wise manifestations	318–320
9. Fungal, parasitic & tropical infections	Fungal infections · Malaria · Tropical & zoonotic infections · Transplant & immunocompromised host infections	321–325
10. Bioterrorism, endocarditis & diarrhoea	Bioterrorism-related infections · Infective endocarditis · Infectious diarrhoea	326–331
11. Rapid revision	Most commons · Investigation of choice & gold standards · Named signs · First-line treatment · Poor prognostic factors · Important trials	332–338

10 Genetics, Environmental Disorders & Toxicology

Pages 339–364

TOPIC	WHAT YOU'LL FIND	PAGE
1. Core concepts & inheritance	Core genetic concepts · Mendelian inheritance · Non-Mendelian patterns	339–341
2. Chromosomal & microdeletion syndromes	Cytogenetics – chromosome disorders · Microdeletion / microduplication syndromes · Molecular diagnostic tests	342–343
3. Epigenetics, cancer & pharmacogenetics	Epigenetics · Cancer genetics · Pharmacogenetics · Inherited metabolic disorders	344–345
4. Health effects of climate change	Climate change & health	346–347
5. Altitude illness	Altitude illness	347–348
6. Hyperbaric oxygen & diving medicine	Hyperbaric oxygen therapy	349–350
7. Hypothermia & cold injuries	Hypothermia	351–352
8. Heat-related illness	Heat-related illness	353–354
9. Toxidromes & decontamination	Toxidromes · GI decontamination & enhanced elimination	358–359
10. Antidotes & heavy metals	Antidote list · Heavy metal poisoning	360–362
11. Drug overdoses & toxic alcohols	High-yield drug overdoses · Toxic alcohols & metabolic poisons	363–364

11 High Yield Pointers

Pages 365–429

TOPIC	WHAT YOU'LL FIND	PAGE
1.1. Cardiology classifications	ACC/AHA HF stages · AF patterns · Killip–Kimball (acute MI) · Aortic stenosis	365–366 · 379–381

TOPIC	WHAT YOU'LL FIND	PAGE
1.2. Respiratory & critical care	COPD GOLD grades · Practical ILD patterns · COPD symptom groups (mMRC/CAT) · PSI/PORT	367–368 · 382–384
1.3. Gastroenterology & hepatology	Child–Pugh · Forrest classification · Maddrey DF · West Haven grading	368–370 · 385–387
1.4. Renal & electrolytes	Etiologic classification · KDIGO GFR & albuminuria categories · GN immunopathology · Hyponatraemia by tonicity & volume	370–372 · 388–389
1.5. Neurology	Epilepsy · ICH severity score components · TOAST · Hoehn & Yahr	372–374 · 390–391
1.6. Endocrinology	Diabetes types · Bethesda thyroid cytology · MEN expanded · DKA severity	374–375 · 392–393
1.7. Rheumatology & immunology	2019 EULAR/ACR SLE · APS domains · AAV	376–377 · 394–395
1.8. Haematology, oncology & toxicology	Anaemia by MCV & reticulocytes · Toxidrome classification · Myeloma spectrum	378–379 · 395–396
2.1. Cardiology signs	Pulsus paradoxus, Beck triad, Kussmaul sign · HCM, MR, AS, TR, MS, AR – dynamic auscultation · IE (Janeway, Osler, Roth, splinter)	398–400
2.2. Pulmonology signs	Hoover sign, silent chest, stridor, egophony, crackles, tracheal deviation · Hamman, Hampton hump, Westermarck, Palla	401–402
2.3. GI & hepatology signs	Murphy, Courvoisier, Charcot, Reynolds, Cullen, Grey–Turner, Rovsing, psoas, obturator, Virchow, Sister Mary Joseph · Caput medusae, Cruveilhier–Baumgarten, asterixis, spider angiomas	403–404
2.4. Nephrology signs	Oedema · RBC, WBC, muddy brown, waxy, fatty & broad casts · uraemic frost · renal bruit	405
2.5. Neurology signs & localisation	Babinski, Hoffmann, clonus, fasciculations, Romberg, dysidiadochokinesia, nystagmus, INO · Argyll Robertson, Adie, Horner · Festinating gait, PSP, DLB, MSA clues	406–408
2.6. Endocrinology signs	Graves eye signs (Dalrymple, von Graefe) · Striae, buffalo hump	409–410
2.7. Rheumatology signs	Gotttron, heliotrope, shawl, mechanic's hands · Z-thumb	411–412
3. Triads, tetrads & pentads	Beck, Virchow, Charcot, Reynolds, Whipple · Ménière · Gout · Atrial myxoma	414–417
4.1. Core diagnostic thresholds	Diabetes & prediabetes · Pre- vs post-capillary PH	419–420
4.2. GI & hepatology criteria	Acute liver failure · Rome IV	421–422
4.3. Cardiology criteria	LVEF definitions (HFrEF / HFmrEF / HFpEF) · low-flow low-gradient AS · myocardial injury · long-QT risk	423
4.4. Pulmonology criteria	Asthma · ABPA · PE risk · CURB-65 · PSI/PORT	424
4.5. Nephrology criteria	Nephrotic vs nephritic · RTA types 1, 2, 4 · SIADH · Fanconi · central vs nephrogenic DI	425

TOPIC	WHAT YOU'LL FIND	PAGE
4.6. Neurology criteria	McDonald (MS) · GBS · myasthenia · NPH · WFNS & modified Fisher (SAH) · Parkinson disease	426
4.7. Endocrinology & metabolism criteria	DKA & euglycaemic DKA · HHS · Burch-Wartofsky · primary aldosteronism · pheochromocytoma · PHPT · osteoporosis · MEN2	427
4.8. Rheumatology & immunology criteria	EULAR/ACR SLE · systemic sclerosis · APS · Sjögren · EGPA · GPA	428
4.9. Haematology & oncology criteria	Myeloma SLiM-CRAB & smouldering · DIC (ISTH) · HLH-2004 · anaemia	429

12 Recent Advances in Medicine

Pages 431–478

TOPIC	WHAT YOU'LL FIND	PAGE
1. Cardiology	2026 ESC heart failure – reclassification · HFpEF + obesity · Cardiovascular–kidney disease · Fifth universal definition of MI (2026) · Transthyretin cardiomyopathy · Sotatercept	431–433
2. Pulmonology	GINA 2026 · GOLD 2026	434–436
3. Diabetes, obesity & metabolic medicine	ADA 2026 – type 2 diabetes · Type 1 diabetes & monitoring · Obesity pharmacotherapy · MASH	437–439
4. Nephrology	Cardiorenal therapies · Glomerular disease	440–441
5. Gastroenterology & hepatology	MASLD / MASH · Portal hypertension · Inflammatory bowel disease · Hepatitis C	442–444
6. Neurology	Alzheimer disease · Migraine & myasthenia gravis · CIDP & NMOSD	444–446
7. Rheumatology & immunology	Rheumatoid arthritis · Lupus nephritis · Systemic sclerosis ILD & gout	447–448
8. Infectious disease	HIV · Tuberculosis · Antimicrobial resistance & C. auris · Malaria vaccines	449–451
9. Hematology	Cell therapy & bispecifics · Bleeding & thrombotic disorders · Sickle cell disease	452–454
10. Oncology	Antibody–drug conjugates · HER2–low breast cancer · bispecifics · checkpoint inhibitors · Precision oncology · PARP inhibition	454–457
11. Critical care & emergency medicine	Sepsis & shock · ARDS · Anticoagulant reversal	458–460
12. Rare diseases & other new therapies	New targeted therapies · CIDP – FcRn blockade	461–462
B1. Sepsis, ARDS & critical care (1–10)	Sepsis – SSC 2026 · Sepsis (contd.) · ARDS	462–465
B2. Lipids, hypertension & heart (11–15)	Lipid medicine · Hypertension & HCM · Heart failure	465–467
B3. Kidney (16–19)	CKD · IgA nephropathy	467–468
B4. Neurology (20–25)	ALS · Parkinson disease · NMOSD & MOGAD · Migraine	468–470
B5. Infection & AMR (26–32)	HIV · Antimicrobial resistance · C. auris & malaria	470–472

TOPIC	WHAT YOU'LL FIND	PAGE
B6. Haematology & oncology (33–40)	Haematology · Multiple myeloma · Oncology	473–475
B7. Other specialties (41–47)	Toxicology · Digital medicine & transplantation · Geriatrics & prevention	475–477
B8. Obesity & diabetes (48–50)	Obesity & diabetes	478

LMR Notes 2.0 · LMR Notes 2.0 Medicine Index

3.TIA and secondary prevention:

^a Definition: transient neurologic deficit resolving within 24 h without imaging (modern practice emphasizes tissue-based definition: TIA = transient symptoms without diffusion MRI lesion). Rapid evaluation is crucial because early recurrence risk is high.

•Components of the ABCD3I Score

Parameter	Criteria	Points
A – Age	≥ 60 years	1
B – Blood Pressure	≥ 140/90 mmHg at presentation	1
C – Clinical Features	Unilateral weakness = 2 points Speech impairment without weakness = 1 point	2
D – Duration of Symptoms	≥ 60 min = 2 points 10–59 min = 1 point	2
D – Diabetes Mellitus	Present	1
D ₂ – Dual TIA	≥ 2 TIAs within 7 days	2
I – Imaging (MRI/CT)	Acute diffusion-weighted imaging (DWI) lesion = 2 points	2

•Total Score Range: 0 – 13

• Interpretation:

Score Range	Stroke Risk	Interpretation
0–3	Low	Minimal early stroke risk
4–7	Moderate	Requires prompt outpatient evaluation
≥8	High	High short-term stroke risk; hospital admission recommended for urgent evaluation and management

(II) EPILEPSY:

1. STATUS EPILEPTICUS(SE)

Definition (ILAE 2015–2022 Consensus)

- Time point t1: Continuous seizure activity ≥ 5 min (convulsive SE) — treatment should start.
- Time point t2: Beyond 30 min $\rightarrow$ risk of long-term neuronal injury.

Types

- Convulsive SE (generalized tonic–clonic)
- Nonconvulsive SE (complex partial, absence, subtle motor)
- Refractory SE: Seizures persist despite first-line (benzodiazepine) and second-line AEDs.
- Super-refractory SE: Continues >24 h after anesthetic therapy initiation.

2. DRUG CHOICES FOR SEIZURES

Seizure Type	First-Line Drugs	Alternatives	Avoid
Focal (Partial) seizures	Carbamazepine, Oxcarbazepine,	Lacosamide, Topiramate,	Valproate (esp. in women)
Generalized tonic–clonic (GTCS)	Valproate, Lamotrigine, Levetiracetam	Topiramate	Carbamazepine (may worsen)
Absence seizures	Ethosuximide, Valproate	Lamotrigine	Carbamazepine, Phenytoin
Myoclonic seizures	Valproate	Levetiracetam, Clonazepam	Carbamazepine, Phenytoin
Atonic seizures	Valproate	Lamotrigine, Rufinamide	—
Status epilepticus	Benzodiazepine $\rightarrow$ Valproate /	Phenobarbital	—

Childhood Absence Epilepsy (CAE)

- Childhood onset; frequent brief staring spells with abrupt onset/offset.
- No significant postictal confusion.
- Hyperventilation commonly provokes an event.
- EEG: classic 3-Hz generalized spike-and-wave.

Important: Ethosuximide is the classic first-line drug when absence seizures occur without other seizure types.

Lennox-Gastaut Syndrome (LGS)

- Childhood-onset developmental epileptic encephalopathy with multiple seizure types.
- Classic seizures: tonic, atonic/drop attacks and atypical absence; other types may coexist.
- EEG: slow <3-Hz spike-and-wave + generalized paroxysmal fast activity, especially in sleep.
- Developmental/cognitive impairment is common; seizures are often drug-resistant.

Important: MULTIPLE seizure types + SLOW spike-wave + developmental impairment.

West Syndrome / Infantile Epileptic Spasms Syndrome

- Infancy; epileptic spasms often occur in clusters, commonly around awakening.
- Developmental arrest/regression may occur.
- EEG: hypsarrhythmia is classic.
- Important etiologies include structural brain injury, genetic and metabolic disorders.

Treatment: ACTH/high-dose corticosteroid therapy or vigabatrin are key therapies; vigabatrin is particularly important for tuberous sclerosis–associated spasms.

Mesial Temporal Lobe Epilepsy (MTLE)

- Most common focal epilepsy syndrome in many adult series.
- Typical aura: rising epigastric sensation, fear, déjà vu or experiential symptoms.
- Impaired awareness with automatisms is classic.
- Postictal confusion is common.
- MRI may show hippocampal sclerosis.
- Drug-resistant MTLE is a major surgically remediable epilepsy; epilepsy surgery should be considered after appropriate presurgical evaluation.

PARKINSON DISEASE

Treatment

LEVODOPA — GOLD STANDARD

Levodopa adverse effects

- Early: nausea, vomiting, orthostatic hypotension.
- Long-term motor: wearing-off, ON-OFF fluctuations and dyskinesias.
- Neuropsychiatric: hallucinations/confusion may occur, particularly with advanced disease or susceptibility.
- Dyskinesia is often dose-related and may limit further levodopa escalation.

Important: Do NOT withhold levodopa simply because of fear of dyskinesia. It remains the most effective symptomatic therapy; use the lowest dose that provides adequate benefit and manage complications when they emerge.

3. LEVODOPA MOTOR COMPLICATIONS

Wearing-off

- Predictable return of parkinsonian symptoms before the next levodopa dose.
- Due to shortening duration of benefit as disease progresses.
- Management: shorten dosing interval, adjust levodopa regimen, or add COMT inhibitor / MAO-B inhibitor / dopamine agonist.

Mnemonic: WEARING-OFF = 'Dose works → then wears out BEFORE next dose'.

ON-OFF phenomenon

- Unpredictable transitions between ON and severe OFF states, sometimes independent of dosing.
- OFF may be associated with severe parkinsonism and painful dystonia.
- More difficult to manage than predictable wearing-off.

AMANTADINE

- Useful symptomatic antiparkinsonian drug and particularly important for levodopa-induced dyskinesia.
- Mechanism: NMDA receptor antagonism is the key exam concept.
- Adverse effects: livedo reticularis, ankle edema, confusion/hallucinations; dose reduction is needed in renal impairment.

Single best association: LEVODOPA-INDUCED DYSKINESIA → AMANTADINE.

ANTICHOLINERGICS

- Examples: trihexyphenidyl, benztropine.
- Best suited to younger patients with prominent tremor and relatively little cognitive impairment.
- Poor choice in elderly patients because of confusion, memory impairment, urinary retention, constipation and other anticholinergic effects.

Important: Anticholinergics help TREMOR more than bradykinesia/rigidity; avoid routine use in older patients.

OTHER MEDICAL OPTIONS

- Adenosine A2A receptor antagonist: istradefylline—used as adjunct for OFF episodes in selected settings.
- Zonisamide has antiparkinsonian effects and is used in Japan.
- Several disease-modifying/neuroprotective approaches remain investigational; no established therapy has definitively been shown to stop PD progression.

CONTINUOUS DOPAMINERGIC DELIVERY — ADVANCED PD

- Rationale: intermittent short-acting levodopa produces fluctuating plasma/brain dopamine exposure → motor complications.
- Continuous delivery aims to provide more stable levodopa levels, reduce OFF time and increase ON time without troublesome dyskinesia.
- Options include continuous intestinal levodopa/carbidopa delivery and continuous subcutaneous dopaminergic infusion in appropriate settings.
- Continuous infusion can be considered in advanced PD with disabling motor fluctuations not adequately controlled with optimized oral therapy.

Concept: Advanced motor fluctuations → think DEVICE-AIDED / CONTINUOUS DOPAMINERGIC THERAPY, not simply repeated rescue levodopa tablets.

ON-DEMAND TREATMENT OF OFF PERIODS

- Used for rapid, predictable rescue of disabling OFF episodes.
- Subcutaneous apomorphine: rapid onset; useful when oral levodopa absorption is unreliable.
- Sublingual apomorphine: noninvasive rescue option where available.
- Inhaled levodopa: rapid pulmonary delivery; useful for intermittent OFF episodes where available.

BEHAVIORAL / PSYCHIATRIC COMPLICATIONS

- Dopamine dysregulation syndrome: compulsive use of dopaminergic medication despite unnecessary use/craving.
- Punding: repetitive, purposeless stereotyped activities such as sorting/collecting/assembling objects.
- Impulse-control disorders: pathological gambling, hypersexuality, compulsive shopping/eating.
- Hallucinations and cognitive impairment become more important with advanced disease and in vulnerable patients.

Drug association: Impulse-control disorders are particularly associated with DOPAMINE AGONISTS.

SURGICAL TREATMENT — DEEP BRAIN STIMULATION (DBS)

- Consider in carefully selected patients with disabling motor fluctuations, medication-refractory tremor or dyskinesia despite optimized medical therapy.
- Best candidates generally have clear levodopa responsiveness, without severe uncontrolled dementia or major psychiatric contraindication.

Alemtuzumab

- Anti-CD52 monoclonal antibody → profound lymphocyte depletion followed by immune reconstitution.
- Course: 12 mg/day IV for 5 consecutive days; second course 12 mg/day for 3 consecutive days about 1 year later (if indicated).
- Reserved for highly active disease after failure of other DMTs in many treatment strategies.
- Major adverse effects: secondary autoimmune disease—especially thyroid disease and immune thrombocytopenic purpura (ITP); infections and malignancy concerns require prolonged monitoring.

Remember: ALEMTUZUMAB = 'immune reset' + delayed AUTOIMMUNITY.

S1P RECEPTOR MODULATORS

- Mechanism: functional S1P receptor modulation/sequestration → Traps lymphocytes in lymphoid tissue and reduces CNS trafficking.
- Fingolimod: 0.5 mg orally once daily; binds S1P1, S1P3, S1P4 and S1P5 receptors.
- Fingolimod first-dose issue: bradycardia/AV conduction slowing can occur → first-dose cardiac observation/ECG monitoring is required in appropriate patients.
- Other key issues: macular edema, elevated liver enzymes, lymphopenia/infection risk and teratogenicity; VZV immunity/vaccination considerations are important.
- Ongoing cardiac and ophthalmologic precautions depend on patient risk and local guidance.
- Ozanimod: oral once-daily S1P1/S1P5 modulator; dose titration is used at initiation.
- Ponesimod: oral S1P1-selective modulator; titration at initiation; cardiac/pulmonary/hepatic considerations.
- Siponimod: S1P1/S1P5-selective modulator; dosing is CYP2C9 genotype dependent. Key exam point: genotype-guided dosing.

S1P : Fingolimod → FIRST-DOSE BRADYCARDIA + MACULAR EDEMA. Siponimod → CYP2C9 genotype-guided dosing.

FUMARATES

- Dimethyl fumarate: activates Nrf2-related cytoprotective pathways and has immunomodulatory effects.
- Typical dose: 240 mg orally twice daily (often after a short initial lower-dose period).
- Common adverse effects: flushing and gastrointestinal symptoms.
- Lymphopenia can occur → monitor CBC; rare PML has been reported in the setting of significant/prolonged lymphopenia.
- Diroximel fumarate: converted to monomethyl fumarate; similar efficacy/side-effect profile, with a major practical aim of improved GI tolerability.

FUMARATES: FLUSHING + GI symptoms + LYMPHOPENIA → think dimethyl fumarate.

CLADRIBINE

- Purine nucleoside analogue; preferentially depletes lymphocytes, producing a long-lasting immune-reconstitution effect.
- Oral weight-based dosing is given in short treatment courses over 2 years rather than continuous daily treatment.
- Major concerns: lymphopenia and infection risk, including herpes zoster; malignancy and reproductive precautions require attention.

Concept: CLADRIBINE = short-course immune reconstitution rather than continuous immunosuppression.

II) MODERATELY EFFECTIVE / LOWER-EFFICACY DMTs

Glatiramer acetate

- Synthetic random polypeptide; immunomodulatory effect with relatively favorable safety profile.
- 20 mg SC daily or 40 mg SC three times weekly.
- Common: injection-site reactions; transient post-injection systemic reaction can occur.
- No routine laboratory toxicity burden comparable to many systemic immunosuppressive DMTs.

Interferon- β

- Options include interferon- β 1a and interferon- β 1b formulations; pegylated interferon- β 1a permits less frequent dosing.
- Common adverse effects: flu-like symptoms, injection-site reactions and laboratory abnormalities.
- Important toxicity: hepatotoxicity; monitor liver function and blood counts.

Teriflunomide

- Inhibits dihydroorotate dehydrogenase $\rightarrow$ reduces proliferation of activated T and B lymphocytes.
- Dose: 14 mg orally once daily.
- Major adverse effects: hepatotoxicity, GI symptoms/diarrhea, alopecia/hair thinning and teratogenicity.

TERIFLUNOMIDE : Oral daily DMT + HEPATOTOXICITY + TERATOGENICITY. It also has a very long effective half-life; accelerated elimination can be used when rapid drug clearance is required.

PPMS / SPMS —IMPORTANT

Primary progressive MS (PPMS)

- Progressive disability from onset rather than clear relapses and remissions.
- Ocrelizumab is the key disease-modifying therapy with established benefit/approval for PPMS, particularly in appropriately selected patients with evidence of inflammatory activity/younger age in many treatment frameworks.

Secondary progressive MS (SPMS)

- Determine whether disease remains ACTIVE (relapses and/or new MRI inflammatory activity) because this influences DMT choice.
- Siponimod is a key exam drug for active SPMS; genotype-guided dosing is required because of CYP2C9 metabolism.
- Rehabilitation, symptomatic treatment and disability prevention remain central as progression becomes dominant.

CLUSTER = CLOCK + RESTLESSNESS: 15–180 min, up to 8/day, often nocturnal, ipsilateral autonomic signs; oxygen + SC sumatriptan acutely; verapamil preventively.

Paroxysmal hemicrania

- Shorter, frequent unilateral TAC: severe orbital/temporal pain lasting 2–30 min, typically >5/day.
- Absolute hallmark: COMPLETE response to indomethacin.
- Indomethacin is diagnostic and therapeutic; monitor GI/renal adverse effects.

PAROXYSMAL HEMICRANIA = Indomethacin. If a TAC disappears with indomethacin, think paroxysmal hemicrania.

SUNCT / SUNA

- Very short attacks of unilateral orbital/temporal pain lasting seconds to minutes, with prominent ipsilateral cranial autonomic symptoms.
- SUNCT: conjunctival injection + tearing are both prominent/required. SUNA: only one or neither may be present.
- Attacks may occur many times/day and can be triggered by touch, chewing or movement.
- Treatment: lamotrigine is the classic preventive/first-line drug; refractory cases may require specialist neuromodulation strategies.

SUNCT vs SUNA: SUNCT = conjunctival injection + tearing. SUNA = short-lasting unilateral neuralgiform attacks with autonomic symptoms, but not necessarily both red eye + tearing.

Hemicrania continua

- Strictly unilateral, continuous headache >3 months with exacerbations; autonomic symptoms may accompany flares.
- Absolute response to indomethacin is the key diagnostic feature.
- Think: CONTINUOUS unilateral headache + indomethacin response.

Other High-Yield Paraneoplastic Syndromes

Syndrome	Cancer	Mechanism
Cushing syndrome	Small-cell lung cancer	Ectopic ACTH
Lambert-Eaton	SCLC	Anti-VGCC antibodies
Polycythemia	RCC, HCC	↑EPO
Dermatomyositis/Polymyositis	Ovarian, lung	Autoimmune
Hyperviscosity	Waldenström	IgM excess
Hypertrophic osteoarthropathy	Lung cancer	Periostitis

SECTION 2 — SYSTEMIC ONCOLOGY

CHAPTER 1 — BREAST CANCER

Step	Key points
Screening	Mammography reduces mortality in women ≥50; many guidelines screen 40–70; MRI for lifetime risk ≥20% (BRCA carriers) or dense breasts selectively.
Diagnostic imaging	Diagnostic mammography ± ultrasound for palpable/mammographic abnormalities; MRI after diagnosis is controversial (may detect occult disease but can increase surgery).
Tissue diagnosis	Image-guided core biopsy (not FNA) — confirm invasive vs in situ, histology, grade, and perform ER / PR / HER2 IHC and HER2 FISH as needed. Biopsy suspicious nodes when present.
Additional tests	Stage-directed systemic imaging only if symptoms or signs of metastasis (CT, bone scan/PET); contralateral imaging as up to ~3% have occult disease.
Genetic testing	Germline panel testing indicated for personal/family risk features (≤65 y, TNBC, male BC, family history, Ashkenazi ancestry).

Adjuvant endocrinotherapy

Patient group	Preferred adjuvant endocrine therapy
Premenopausal ER+	Tamoxifen ± ovarian suppression (LHRH agonist) for higher risk
Postmenopausal ER+	Aromatase inhibitor (anastrozole/exemestane/letrozole) or
High recurrence risk / node-positive	Extended therapy and consider ovarian suppression in

Perioperative / fertility / pregnancy considerations

- **Fertility preservation** discussion for premenopausal women before systemic therapy; LHRH agonist during chemo may preserve ovarian function.
- **Pregnancy:** Surgery safe in 2nd/3rd trimester; radiation deferred until after delivery; some anthracycline-based chemo can be given in 2nd/3rd trimester; endocrine/targeted therapies avoided until after delivery.

Prevention / risk reduction

- **BRCA carriers:** Offer germline testing when indicated; consider prophylactic bilateral mastectomy (~90% risk reduction) and risk-reducing oophorectomy (reduces ovarian and lowers breast risk in premenopausal women). Chemoprevention (tamoxifen, raloxifene, AIs) for high-risk patients.

IMPORTANT:

Always obtain ER/PR/HER2 on diagnostic core biopsy — drives systemic therapy.

- Lumpectomy + RT = mastectomy for many early cancers (survival equivalent).
- CDK4/6 inhibitors + endocrine therapy are standard first-line for most ER+ metastatic cases.
- HER2+ first-line metastatic: taxane + trastuzumab + pertuzumab; second-line trastuzumab-deruxtecan (ADC) has high activity.
- PARP inhibitors indicated in BRCA-mutated advanced disease; sacituzumab govitecan for refractory TNBC.

CHAPTER 2 — LUNG CANCER (NSCLC & SCLC)

• Diagnosis

Step	Key points
Presentation	Cough, hemoptysis, dyspnea, weight loss, paraneoplastic syndromes. New or changing pulmonary nodule warrants workup.
Imaging	Contrast CT chest ± upper abdomen is first step; PET-CT for staging (mediastinal nodes, distant mets) when no contraindication; brain MRI for suspected/advanced disease. LDCT screening recommended for high-risk individuals (age and smoking criteria).
Tissue diagnosis	Image-guided core biopsy / bronchoscopy / EBUS — obtain histology (NSCLC vs SCLC) and enough tissue for molecular testing and PD-L1 IHC. Do nodal sampling (EBUS/TBNA) for mediastinal staging prior to definitive therapy.
Staging	AJCC TNM for NSCLC; limited vs extensive disease for SCLC (limited = confined to one hemithorax/mediastinum that can be encompassed in a radiation field).

STRUCTURAL DISORDERS

Hiatal hernia

- Large symptomatic paraesophageal hernias may require surgical repair.

Rings & webs

- Schatzki ring: thin lower-esophageal mucosal ring near the squamocolumnar junction → intermittent solid-food dysphagia / 'steakhouse syndrome'.
- Esophageal webs, especially proximal/cervical webs, may occur with iron-deficiency anemia → Plummer–Vinson syndrome.
- Symptomatic rings/webs are treated with dilation; correct associated iron deficiency.

Diverticula

- Zenker diverticulum: hypopharyngeal diverticulum near Killian triangle → dysphagia, regurgitation, halitosis and aspiration.
- Epiphrenic diverticula are distal and may be associated with motility disorders such as achalasia.

Type	Key point
Sliding / Type I	GE junction and proximal stomach move above diaphragm; most common; strongly associated with GERD.
Paraesophageal / Types II–IV	Herniation beside esophagus; large hernias may contain stomach/other viscera and can cause volvulus or obstruction.

3. SMALL INTESTINE

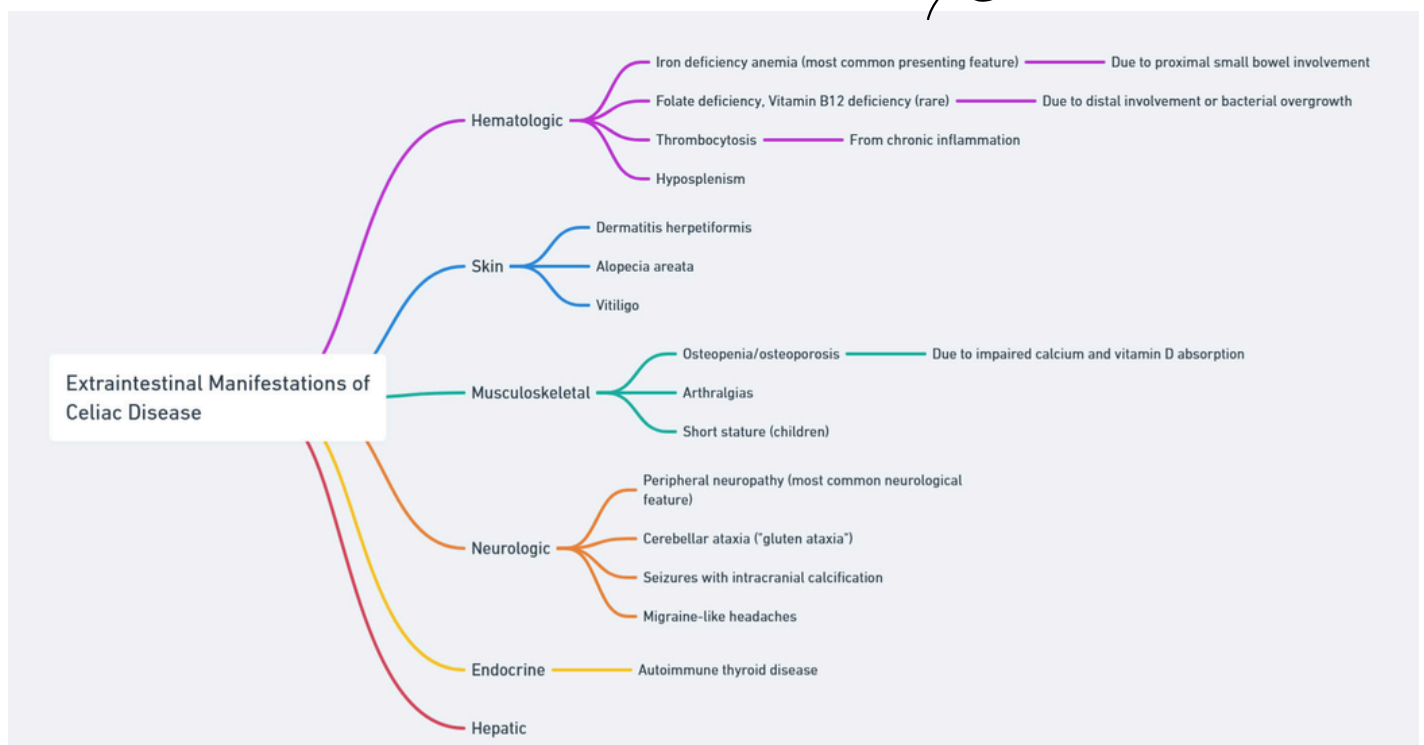
(i) Celiac Disease –

- Total IgA deficiency → order IgG DGP or IgG tTG.
- Marsh 1 is NOT diagnostic unless clinical + serology positive.
- Dermatitis herpetiformis diagnosis = skin biopsy: granular IgA in dermal papillae.
- Refractory Celiac Type 2 → high risk of EATL (T-cell lymphoma).

Tropical Sprue vs Celiac

Feature	Celiac	Tropical sprue
Location	Worldwide	South India, SE Asia
Onset	Gradual	Post-infectious
Serology	Positive	Negative
Folate/B12	Folate ↓	Both ↓

EXTRAINTESTINAL MANIFESTATIONS OF CELIAC DISEASE



Complications

Refractory celiac disease

Enteropathy-associated T-cell lymphoma (EATL)

Small-bowel adenocarcinoma

Hyposplenism

Refractory celiac disease

Type 1: intraepithelial lymphocytes have a relatively normal phenotype; generally better prognosis.

Type 2: aberrant/clonal CD3+ intraepithelial lymphocyte population; associated with T-cell clonal rearrangement.

Type 2 has a worse prognosis and is strongly associated with EATL.

(ii) WHIPPLE DISEASE — MANIFESTATIONS & TREATMENT

Caused by *Tropheryma whippiei*, a gram-positive actinomycete.

Clinical Manifestations



Diagnosis:

Duodenal biopsy:

- PAS-positive macrophages in lamina propria
- Filled with rod-shaped organisms
- **Confirmation:** PCR for *Tropheryma whippiei* in tissue, CSF, synovial fluid
- If neurologic symptoms → MUST evaluate CSF PCR

Always evaluate the CNS because even asymptomatic involvement requires prolonged therapy.

3. PANCREAS

(i) Acute Pancreatitis –

- BISAP $\geq 3 \rightarrow$ high mortality.
- Early CT (<72 hours) is not recommended.
- Fluid of choice = LR, not NS (less acidosis & inflammation).
- Infected necrosis suspicion = gas on CT or clinical deterioration.
- No role of antibiotics in early treatment; to be given only if infection supervenes in the later stage of necrotizing pancreatitis.

PANCREATITIS

BISAP — remember the 5 components

- B — BUN >25 mg/dL
- I — Impaired mental status (GCS <15)
- S — SIRS ≥ 2 criteria
- A — Age >60 years
- P — Pleural effusion

BISAP ≥ 3 is associated with substantially increased risk of in-hospital mortality.

AUTOIMMUNE PANCREATITIS

Feature	Type 1 AIP	Type 2 AIP
Association	IgG4-related disease	Usually isolated; associated with IBD
Serum IgG4	Often elevated	Usually normal
Histology	Lymphoplasmacytic sclerosing pancreatitis; storiform fibrosis; obliterative phlebitis	Granulocytic epithelial lesions
Relapse	Moderate–high	Low

Treatment

- Glucocorticoids are the main treatment;
- Relapsing/refractory disease may require steroid-sparing immunomodulation; rituximab is an option in selected recurrent cases.

- Ribavirin dose: 1000 mg/day if body weight <75 kg; 1200 mg/day if ≥ 75 kg. In CTP-C, start at 600 mg/day and increase as tolerated.
- Protease-inhibitor-containing regimens are NOT recommended in decompensated cirrhosis: avoid glecaprevir, grazoprevir, voxilaprevir-containing regimens.
- Interferon-based therapy is not recommended in decompensated cirrhosis.

RIBAVIRIN Major adverse effect = hemolytic anemia. It is teratogenic; contraindicated during pregnancy. Pregnancy prevention is required for the patient and relevant partner during therapy and after exposure according to prescribing guidance.

Pituitary Disorders

Hyperprolactinemia

Causes

- Physiologic: pregnancy, lactation, sleep, stress, nipple/chest-wall stimulation, sexual activity.
- Drugs: dopamine-receptor blockers (antipsychotics), metoclopramide, opioids, H₂ blockers, verapamil, estrogens and some antidepressants.
- Hypothyroidism, chronic renal failure, cirrhosis.
- Stalk compression → loss of hypothalamic dopamine inhibition.

Evaluation

1. Fasting morning serum prolactin.
2. Exclude pregnancy and secondary causes; review medications and check thyroid/renal status as appropriate.
3. Pituitary MRI when hyperprolactinemia remains unexplained or a pituitary lesion is suspected.

Hook effect: a very large pituitary tumor with an unexpectedly modest PRL should prompt repeat PRL after sample dilution. Very high PRL ($>1000 \mu\text{g/L}$ in the source) is particularly prone to assay interference.

Prolactinoma — complete management

First-line: dopamine agonist

Cabergoline is the preferred dopamine agonist in the source: long-acting, potent D₂-receptor agonist with high efficacy for prolactin suppression and tumor shrinkage.

- Cabergoline: start 0.5–1 mg twice weekly; titrate according to prolactin response and tolerance.
- Headache and visual symptoms often improve rapidly when tumor shrinkage occurs.

Bromocriptine

- Alternative dopamine agonist, particularly when pregnancy is desired.
- Start low: 0.625–1.25 mg at bedtime; increase gradually according to response/tolerance.
- More frequent dosing and adverse effects make it less convenient than cabergoline for many patients.

Plasma ACTH	Interpretation	Next step
Suppressed / <5 pg/mL	ACTH-independent Cushing	Adrenal CT → assess unilateral mass vs bilateral nodular/hyperplastic disease
Normal/high / >15 pg/mL	ACTH-dependent Cushing	Pituitary MRI + biochemical/localization testing; consider ectopic ACTH

ACTH-dependent: pituitary vs ectopic

- Pituitary MRI is part of localization, but a small incidental pituitary lesion does not automatically prove Cushing disease.
- High-dose dexamethasone suppression and CRH/desmopressin-based testing can support pituitary origin in selected patients.
- Inferior petrosal sinus sampling (IPSS) is the key invasive localization test when biochemical differentiation remains uncertain.
- central/peripheral ACTH ratio >3 after CRH/desmopressin supports pituitary source; basal ratio >2 is also supportive.

Treatment of Cushing syndrome

- Cushing disease: transsphenoidal resection of pituitary corticotroph adenoma is first-line when resectable.

CKD-MBD — PATHOPHYSIOLOGY

Definition

- CKD-MBD is a systemic disorder of mineral and bone metabolism due to CKD involving one or more of: abnormal calcium/phosphate/PTH/vitamin D metabolism; abnormal bone turnover/mineralization/volume/strength; vascular or soft-tissue calcification.
- Renal osteodystrophy = the bone morphology abnormality component of CKD-MBD.

The classic sequence

↓ nephron mass → phosphate retention → ↑ FGF23 + ↑ PTH → ↓ calcitriol → reduced intestinal Ca absorption + persistent phosphate burden → secondary hyperparathyroidism → altered bone turnover + vascular/soft-tissue calcification.

FGF23

- Produced by bone; rises early in CKD, often before overt hyperphosphatemia.
- Promotes phosphaturia and suppresses renal calcitriol production; its renal actions are klotho dependent.
- High FGF23 is associated with progression and mortality in observational cohorts, but routine measurement is not standard clinical monitoring.

PTH

- Hypocalcemia is a potent stimulus to PTH release through the calcium-sensing receptor system.
- Phosphate increases PTH production, including via effects on PTH mRNA stability.
- Calcitriol suppresses PTH through the vitamin D receptor.
- PTH increases renal calcium reabsorption and phosphate excretion

HEMODIALYSIS

AV fistula — “Rule of 6”

- ~6 mm diameter.
- ~6 cm of usable cannulation length.
- ≤ 6 mm deep beneath the skin.
- A mature AVF should provide robust flow; ~400–500 mL/min is a useful functional-flow benchmark.

Conventional maintenance HD

- Common regimen: thrice weekly, ~3–4 h/session .
- Shortening sessions below ~3 h increases intermittence, disequilibrium, UF rate and hypotension risk.
- Longer/more frequent HD can improve BP, phosphate and volume control but increases access burden and resource use.

Adequacy

- URR is simple but less comprehensive than Kt/V.
- Single-pool Kt/V is the standard familiar measure of intermittent HD dose.
- The notes cite a minimum PD weekly Kt/V of 1.7 and a conventional HD target framework around Kt/V 1.2–1.4 per treatment; interpret according to dialysis schedule and adequacy method.

HEMODIALYSIS — INTRADIALYTIC HYPOTENSION

Definition + mechanism

- KDOQI definition: symptomatic fall in systolic BP ≥ 20 mm Hg or fall in MAP ≥ 10 mm Hg.
- Risk increases with large interdialytic weight gain, aggressive UF, autonomic dysfunction, cardiac disease and electrolyte/temperature factors.

UF rates >10 – 13 mL/kg/h have been associated with higher cardiovascular morbidity/mortality

Acute management

Stop/reduce UF $\rightarrow$ supine/Trendelenburg $\rightarrow$ 100–250 mL normal saline boluses as needed $\rightarrow$ oxygen/reduce Qb if significant $\rightarrow$ stop dialysis if persistent/severe or dangerous cause suspected.

Acute vs chronic eosinophilic pneumonia

Feature	Acute eosinophilic pneumonia	Chronic eosinophilic pneumonia
Course	Acute, usually <1 month	Weeks to months
Blood eosinophilia	Often absent early	Common
BAL eosinophils	>25% highlighted	High
Imaging	Diffuse infiltrates; can resemble edema/ARDS	Peripheral upper-lobe predominant infiltrates
Asthma/atopy	Less typical	Common
Relapse	Uncommon after treatment withdrawal	Common
Treatment	Systemic corticosteroids; rapid response	Systemic corticosteroids; prolonged course often needed

Asbestosis

- Diffuse interstitial fibrosis, classically lower-lobe and subpleural.
- Pleural plaques are a major imaging clue; pleural thickening/effusion may occur.
- HRCT: subpleural curvilinear lines, parenchymal bands, honeycombing and rounded atelectasis.
- Restrictive PFT with reduced DLCO.
- Long latency; smoking synergistically increases lung-cancer risk.

Silicosis

- Exposure: mining, quarrying, sandblasting, construction, stone cutting, ceramics and engineered stone.
- Upper-lobe nodules ± hilar/mediastinal adenopathy; eggshell calcification is a classic clue.
- Progressive massive fibrosis can develop and may progress even after exposure ends.
- Strong association with tuberculosis.
- Caplan syndrome = pneumoconiosis + rheumatoid arthritis.
- Chronic beryllium disease: noncaseating sarcoid-like granulomas; BeLPT supports diagnosis.
- Hard-metal disease: tungsten carbide/cobalt exposure; giant-cell interstitial pneumonia with characteristic giant-cell changes.

Idiopathic pulmonary fibrosis

- Usually older adults with progressive exertional dyspnea and dry cough; bibasal fine crackles and clubbing may occur.
- HRCT UIP: subpleural/basal reticulation, traction bronchiectasis and honeycombing; absence of a strong alternative pattern is important.
- Antifibrotic therapy is central to disease management; oxygen and pulmonary rehabilitation support function and quality of life.
- Acute exacerbation: acute deterioration with new bilateral ground-glass/consolidative opacities superimposed on underlying fibrosis.

NSIP

- Often associated with connective-tissue disease.
- HRCT: diffuse/basal ground-glass and reticular abnormalities, volume loss and traction bronchiectasis; relative subpleural sparing is a useful clue.
- Cellular NSIP has more inflammation and tends to have a more favorable course than fibrotic NSIP.

Cryptogenic organizing pneumonia

- Subacute flu-like illness, cough, fever and dyspnea; may mimic infection.
- Patchy/migratory peripheral consolidation and ground-glass opacities are typical.
- Histology: organizing plugs in distal airspaces (Masson bodies) with surrounding inflammation.
- Corticosteroids are commonly used; recurrence can occur.

Acute interstitial pneumonia

- Abrupt severe respiratory illness with hypoxemia and diffuse infiltrates; clinically resembles ARDS.
- Histology: diffuse alveolar damage.
- No established disease-specific drug therapy; supportive critical care is central.

3. NON-MENDELIAN PATTERNS

Anticipation

- Progressively earlier onset and/or increased severity in successive generations due to repeat expansion.
- Huntington disease → CAG expansion; paternal transmission often shows greater anticipation.
- Myotonic dystrophy → CTG expansion; maternal transmission can produce congenital severe disease.
- Fragile X syndrome → CGG expansion in FMR1; full mutation is associated with methylation/silencing.
- Friedreich ataxia → GAA expansion, typically intronic; recessive inheritance.

⚡ **EXAM PEARL:** Repeat expansion disorders = think ANTICIPATION.

Genomic imprinting

- Expression depends on whether the pathogenic allele is inherited from the mother or father.
- Prader-Willi syndrome → loss of paternally expressed genes on chromosome 15q11-q13.
- Angelman syndrome → loss of maternally expressed UBE3A in relevant brain regions.
- Mechanisms include deletion, uniparental disomy and imprinting defects.

Uniparental disomy (UPD)

- Both homologues of a chromosome are inherited from one parent.
- Can cause disease through imprinting abnormalities or unmasking a recessive variant.
- Classic chromosome 15 association: Prader-Willi/Angelman.

Mosaicism

- Postzygotic mutation → genetically distinct cell populations within one individual.
- Somatic mosaicism → may produce segmental/localized disease.
- Germline mosaicism → unaffected parent can have multiple affected children with a pathogenic variant not detectable in blood.
- Mosaicism may make blood genetic testing falsely reassuring.

Founder effect / consanguinity

- Founder effect = high frequency of a pathogenic variant in descendants of a small ancestral population.
- Consanguinity increases homozygosity and the chance of autosomal-recessive disorders.

5. HEAT-RELATED ILLNESS

Thermoregulation — high yield

- Evaporation is the most efficient mechanism of heat loss and becomes progressively ineffective when relative humidity is high.
- Skin blood flow can increase dramatically during heat stress.
- Heat acclimatization takes roughly 1–several weeks: earlier/more efficient sweating, lower sweat sodium concentration, plasma-volume expansion, lower heart rate and improved heat dissipation.
- WBGT (wet-bulb globe temperature) is a better environmental heat-load index than the simple heat index because it incorporates radiant heat and wind.

Heat edema / heat cramps / heat exhaustion

- Heat edema: mild swelling of hands/feet/ankles early after heat exposure due to vasodilation and fluid shifts.
- Heat cramps: painful muscle cramps during heat stress; relative sodium/fluid/potassium depletion contributes.
- Heat exhaustion: thermoregulation is still functioning; CNS dysfunction is absent or limited. Treat with cooling, rest, oral/IV fluids and electrolyte correction as needed.

⚠ **REMEMBER::** CNS dysfunction should push you toward heatstroke rather than simple heat exhaustion.

Heatstroke

- Life-threatening hyperthermia with CNS dysfunction and systemic organ injury.
- Core temperature is usually >40–40.5°C, but do not delay treatment waiting for a specific number.
- Rapid cooling is the priority—diagnostic workup proceeds in parallel.

Classic heatstroke	Exertional heatstroke
Older/vulnerable patient	Younger, healthy, strenuous exercise
Heat wave / prolonged ambient heat	Sporadic intense exertion
May have anhidrosis / hot dry skin	Diaphoresis may persist
CNS dysfunction prominent	CNS dysfunction + severe muscle/organ injury
Oliguria; hepatic/renal injury	Rhabdomyolysis, AKI, DIC, marked lactic acidosis
Usually less dramatic muscle injury	Hyperkalemia/hyponatremia/hypoglycemi a more common

Arsenic — must remember

- Acute: severe GI toxicity → fluid loss/hypotension; delayed cardiomyopathy; ATN; hemolysis; neuropathy.
- Chronic: hyperkeratosis + hyperpigmentation + peripheral neuropathy + vascular disease/gangrene + malignancy.
- Classic nail clue → Mees lines.
- Seafood organic arsenic (arsenobetaine/arsenocholine) is relatively nontoxic; inorganic arsenic is the major toxic form.

Lead — must remember

- Measure lead in WHOLE BLOOD, not serum.
- Most lead is initially in RBCs; long-term storage occurs in bone.
- Chronic clues: abdominal colic + anemia + cognitive/behavioral dysfunction + motor neuropathy + nephropathy.
- Basophilic stippling and elevated erythrocyte protoporphyrin are classic laboratory clues.
- Children are particularly vulnerable to neurodevelopmental effects.

Cadmium — classic association

- Kidney + bone: proximal tubular injury, proteinuria, β 2-microglobulin $\uparrow$, calciuria → osteomalacia/fractures.
- Itai-itai disease = painful bone disease associated with cadmium exposure.

⚠ **REMEMBER::** Cadmium: chelation is NOT useful; dimercaprol may worsen nephrotoxicity.

Mercury — classic clues

- Mercurial erethism = excitability, memory loss, insomnia, timidity and delirium ('mad as a hatter').
- Acrodynia ('pink disease') in children: flushing, itching, swelling, tachycardia, hypertension, sweating/salivation, irritability, weakness, rash and desquamation of palms/soles.
- Organic mercury: recent exposure → blood; chronic exposure → hair is useful.

Other metals — rapid hits

- Aluminum → encephalopathy in severe renal disease/dialysis.
- Manganese → parkinsonian syndrome; typically poorly responsive to L-dopa.
- Hexavalent chromium → corrosive/sensitizing; occupational exposure associated with lung cancer.
- Cobalt → cardiomyopathy with substantial exposure.
- Selenium excess → GI/respiratory irritation, liver inflammation, alopecia, depigmentation, neuropathy.

ALL IMPORTANT CLASSIFICATIONS • STAGING • GRADING • PHENOTYPES

ACC/AHA Heart Failure — Stages

Stage	Definition	Recall
A	At risk for HF; no structural heart disease/symptoms	Risk stage
B	Pre-HF: structural heart disease or abnormal biomarkers, without HF symptoms/signs	Pre-HF
C	Structural heart disease with current or previous HF symptoms	Symptomatic HF
D	Advanced HF with marked symptoms/interference despite guideline-directed therapy	Advanced HF

NYHA Functional Classification

Class	Functional limitation
I	No limitation; ordinary activity does not cause symptoms
II	Slight limitation; ordinary activity causes symptoms
III	Marked limitation; less-than-ordinary activity causes symptoms
IV	Symptoms at rest; unable to carry out physical activity without discomfort

Pulmonary Hypertension — Clinical Classification

Group	Category	High-yield examples
1	Pulmonary arterial hypertension	Idiopathic; heritable; drugs/toxins; CTD; HIV; portal HTN; CHD; schistosomiasis; PVOD/PCH features
2	PH due to left-heart disease	HFpEF/HFrEF/HFmrEF; valvular disease; other left-heart conditions
3	PH due to lung disease/hypoxia	Obstructive, restrictive, mixed lung disease; hypoventilation; hypoxia; developmental disorders
4	PH due to pulmonary artery obstruction	CTEPH; other pulmonary artery obstructions
5	PH with unclear/multifactorial mechanisms	Hematologic, systemic, metabolic, CKD, tumor thrombotic microangiopathy, fibrosing mediastinitis

Hemodynamic phenotype: precapillary PH = mPAP >20 mmHg + PAWP ≤15 mmHg + elevated PVR; postcapillary PH has PAWP >15 mmHg.

Spondyloarthritis — ASAS-style Phenotypes

Phenotype	Examples
Axial SpA	Radiographic axial SpA / ankylosing spondylitis and non-radiographic axial SpA
Peripheral SpA	Peripheral arthritis, enthesitis, dactylitis
Disease-associated forms	Psoriatic, reactive and IBD-associated SpA

19. HEMATOLOGY & ONCOLOGY — ADDITIONAL CLASSIFICATIONS

Multiple Myeloma — Disease Spectrum

Entity	Core definition
MGUS	Monoclonal protein without myeloma-defining end-organ damage/biomarker criteria
Smoldering MM	Higher clonal burden but no myeloma-defining event
Multiple myeloma	Clonal plasma-cell disorder with CRAB features and/or myeloma-defining biomarkers

Hodgkin Lymphoma — Broad Classification

Group	Subtypes
Classical Hodgkin lymphoma	Nodular sclerosis, mixed cellularity, lymphocyte-rich, lymphocyte-depleted
Nodular lymphocyte-predominant HL	Distinct biologic entity from classical HL

Lymphoma — Ann Arbor / Lugano Staging

Stage	Concept
I	Single lymph-node region or single extralymphatic site
II	≥2 nodal regions on same side of diaphragm ± contiguous extranodal site
III	Nodal disease on both sides of diaphragm ± spleen/extranodal extension
IV	Diffuse/disseminated extranodal involvement such as marrow, liver or lung

4. NEPHROLOGY — IMPORTANT SIGNS

Sign / finding	What it suggests
Periorbital edema	Nephrotic syndrome/glomerular disease; also systemic edema causes.
Pitting pedal edema	Nephrotic syndrome, renal failure, heart failure, cirrhosis and other causes.
RBC casts	Glomerular bleeding; highly important clue for glomerulonephritis.
Dysmorphic RBCs	Glomerular hematuria.
Muddy brown granular casts	Acute tubular injury/necrosis.
WBC casts	Tubulointerstitial inflammation/pyelonephritis; can occur in other renal inflammatory states.
Waxy casts	Advanced/chronic kidney disease, low-flow dilated tubules.
Fatty casts / oval fat bodies	Nephrotic syndrome.
Broad casts	Advanced chronic kidney disease.
Frothy urine	Proteinuria clue; nonspecific.
Costovertebral angle tenderness	Pyelonephritis/renal inflammation or obstruction; not specific.
Pruritus + scratch marks	Advanced CKD/uremia.
Uremic frost	Severe azotemia/uremia; uncommon late sign.
Asterixis	Uremic encephalopathy or other toxic-metabolic encephalopathy.
Pericardial rub in renal failure	Uremic pericarditis.
Hypertension + retinopathy + renal disease	Chronic hypertensive target-organ damage.
Renal bruit	Renovascular disease clue in appropriate phenotype.