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PULMONARY MEDICINE

AT YOUR FINGERTIPS

CBME-Based Respiratory Medicine Exam-Preparatory Manual

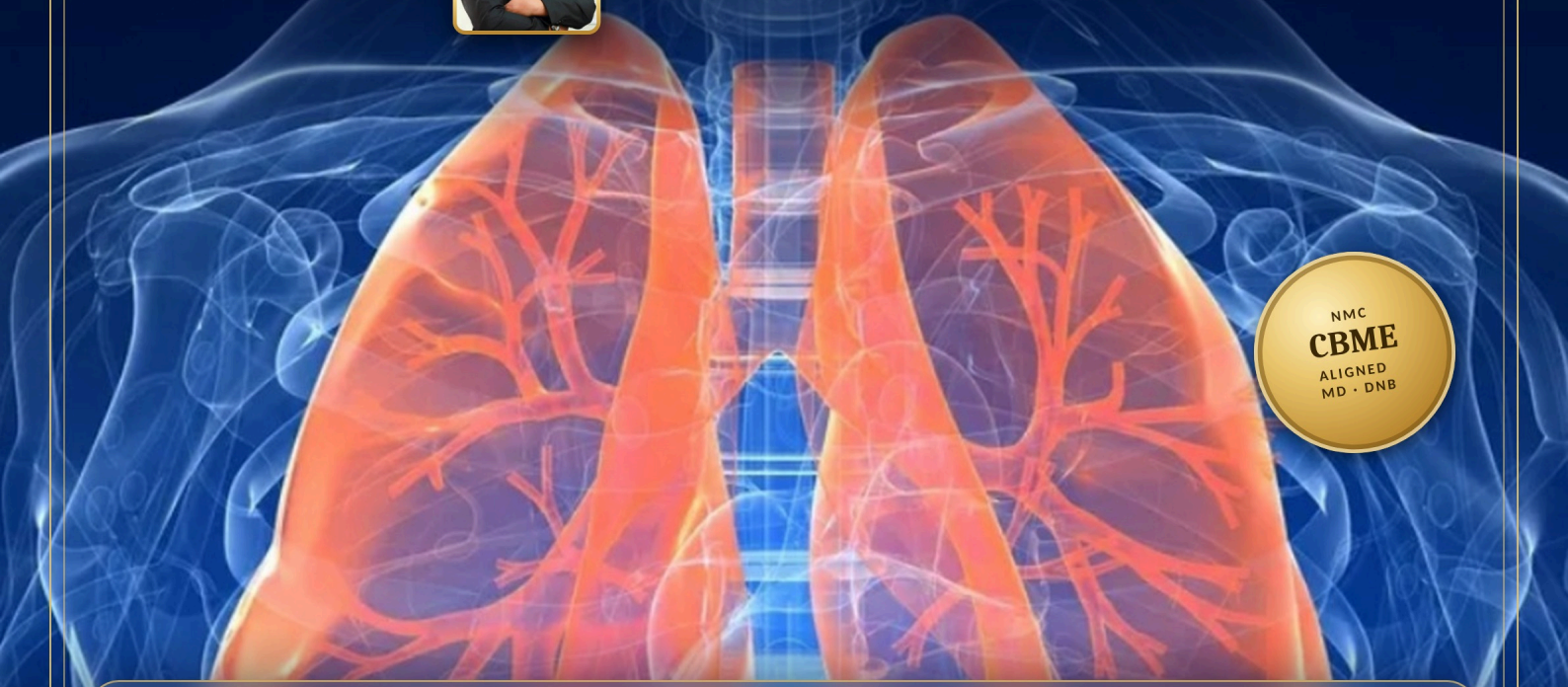
MD Pulmonary Medicine · DNB Respiratory Medicine · Previous-year questions (2014–2025)



Dr. Gireesh Kumar K P

Principal, Amrita School of Medicine

Head & Professor, Emergency Medicine · Director, AETCM



NMC CBME ALIGNED MD · DNB

EXPLORE BY SECTION — tap to jump

1 Applied Anatomy & Embryology

2 Respiratory Physiology & Function

3 Microbiology, Pathology & Pharmacology

4 Tuberculosis

5 Non-Tuberculous Lung Infections

6 Obstructive Airway Diseases

7 Interstitial & Immunological Disease

8 Occupational & Environmental Disease

9 Pulmonary Circulation & Embolism

10 Lung Cancer & Thoracic Tumours

11 Pleura, Mediastinum & Chest Wall

12 Critical Care Pulmonary Medicine

13 Sleep-Related Breathing Disorders

14 Thoracic Imaging & Procedures

15 Special Environments & Systemic Disease

16 Surgical Aspects of Pulmonary Medicine

17 Research Methodology & Biostatistics

18 Clinical Examination — Cases & Viva

19 Oxygen, NIV & Mechanical Ventilation

II Part II — Algorithms & Image Atlas

NMC MD Pulmonary Medicine curriculum ›

Question bank ›

Amrita School of Medicine · Amrita Institute of Medical Sciences, Kochi

FIRST EDITION · 2027

FOREWORD

Foreword

From Dr. Gireesh Kumar K. P. · To the postgraduates of MD Pulmonary Medicine and DNB Respiratory Medicine



Dear postgraduate,

Respiratory medicine asks more of its trainees than almost any other specialty. In a single morning you may read a spirometry tracing, decide on a drug-resistant tuberculosis regimen, look down a bronchoscope, titrate a ventilator for a patient with ARDS and counsel a family about a lung cancer diagnosis. The MD examination tests all of these — and expects you to recall the right physiology, the right guideline and the right number under pressure.

Pulmonary Medicine at Your Fingertips takes the competency-based syllabus of the National Medical Commission — the NMC *Guidelines for Competency Based Postgraduate Training Programme for MD in Pulmonary Medicine* — and maps it, section by section, to the questions that have actually been asked in the DNB Respiratory Medicine papers and in university MD examinations. Each topic opens with its CBME competencies and closes with the previous-year questions that bear on it, so that you study with the examiner's intent in view.

The answers are written in the clean, high-yield house style of our academy: physiology in bullet form, clinical-feature, investigation and treatment tables, and decision algorithms for every important emergency. Alongside the classical teaching I have placed the current guidance — the National TB Elimination Programme, GINA, GOLD, the ATS/ERS/JRS/ALAT IPF statements, the ESC/ERS pulmonary hypertension guideline, the BTS pleural guideline and the new global definition of ARDS — so that your answers remain current.

Use the algorithms to think under pressure, the tables to revise at speed, and the questions to test yourself honestly. My hope is that this volume sits beside you through the long months of preparation and earns its title — that good pulmonology, well organised, truly comes to rest at your fingertips.

With best wishes for your preparation,

Dr. Gireesh Kumar K. P.
Principal, Amrita School of Medicine
Director, Academy for Emergency, Trauma & Critical Care Medicine (AETCM), Kochi
Kochi · 2027

How to use this manual

- **Read the questions first.** Each topic closes with the actual previous-year questions — study with the examiner in view.
- **Think in tables and algorithms.** Use them to revise at speed and to answer under pressure.
- **Quote the evidence.** Close each answer with a guideline or landmark trial from the green box.
- **Navigate by tapping.** Cover, contents, section pages, topic buttons, the question bank and the curriculum are all hyperlinked.

Contents

Foreword 2

How to Use This Book 7

NMC Syllabus Map 8

Section 1 · Applied Anatomy, Embryology & Developmental Anomalies 10

1.1 Development of the Lung & Postnatal Growth 10
1.2 Airway Structure, Epithelium, Cilia & Alveolar Cells 12
1.3 Bronchopulmonary Segments, Secondary Lobule & Fissures 13
1.4 Pulmonary & Bronchial Circulation and Lymphatics 15
1.5 Mediastinum & Pleura: Anatomy and Innervation 16
1.6 Diaphragm, Respiratory Muscles & Phrenic Nerve 17
1.7 Developmental Anomalies I — Agenesis, Sequestration, CPAM, Bronchogenic Cyst 18
1.8 Developmental Anomalies II — TEF, Scimitar, PAVM, Unilateral Hyperlucent Lung 20
› Section 1 — previous-year question bank *273*

Section 2 · Respiratory Physiology & Pulmonary Function 22

2.1 Lung Volumes & Capacities and Their Measurement 22
2.2 Spirometry & Flow–Volume Loops 24
2.3 Diffusion & DLCO 26
2.4 Respiratory Mechanics — Compliance, Resistance, Surfactant & Atelectasis 28
2.5 Ventilation, Dead Space, V/Q Matching & Mechanisms of Hypoxaemia 29
2.6 Gas Transport — Oxygen and Carbon Dioxide Dissociation Curves 32
2.7 Control of Ventilation, Chemoreceptors & Reflexes 33
2.8 Acid–Base Balance & ABG Interpretation; Electrolytes 35
2.9 Non-Respiratory Functions & Pulmonary Defence Mechanisms 37
2.10 Exercise Physiology, CPET & Six-Minute Walk Test 39
2.11 Bronchoprovocation, Oscillometry, Small Airway Tests, FeNO & Respiratory Muscle Tests 40
2.12 Physiological Changes in Pregnancy, Ageing & High Altitude 41
2.13 Physiological Basis of Pulmonary Symptoms & Signs — Dyspnoea, Cough, Clubbing 43
2.14 Aerosol Kinetics & Inhaled Drug Delivery Devices 44
2.15 Pulmonary Circulation Physiology — West Zones, Starling Resistor, HPV & Fluids 45
2.16 Principles of Electrocardiography & Cardiac Changes in Lung Disease 47
› Section 2 — previous-year question bank *275*

Section 3 · Microbiology, Pathology, Immunology & Pharmacology 49

3.1 Mycobacteria — Biology, Staining, Culture & Phenotypic DST 49
3.2 Molecular Diagnosis of TB — CBNAAT, Truenat, LPA & Whole-Genome Sequencing 51
3.3 Respiratory Viruses & Their Laboratory Diagnosis 52
3.4 Respiratory Fungi — Classification & Laboratory Diagnosis 53
3.5 Bacteria, Atypical Organisms, Anaerobes & Biomarkers of Infection 54
3.6 Pathology — Inflammation, Granuloma & Emphysema 56
3.7 Hypersensitivity, Allergy Testing & Immunology of Lung Disease 57
3.8 Pharmacology of First-Line Anti-TB Drugs & Drug-Induced Liver Injury 58
3.9 Second-Line & Newer Anti-TB Drugs — Bedaquiline, Delamanid, Pretomanid, Linezolid, Fluoroquinolones 60
3.10 Bronchodilators & Inhaled Corticosteroids 61
3.11 Antimicrobials, Antifungals & Macrolides in Respiratory Medicine 63
3.12 Antineoplastic, Immunosuppressant & Biologic Drugs; Pharmacovigilance 64
› Section 3 — previous-year question bank *278*

Section 4 · Tuberculosis 66

4.1 Epidemiology of TB, End TB Strategy & the National TB Elimination Programme 66
4.2 Pathogenesis — Primary & Post-Primary TB, Wallgren Timetable 68
4.3 Diagnosis of Pulmonary TB — NTEP Algorithm, Radiology & Differential Diagnosis 70
4.4 Latent TB Infection — TST, IGRA & TB Preventive Treatment 71
4.5 Treatment of Drug-Susceptible TB under NTEP 74
4.6 Drug-Resistant TB — Definitions, Risk Factors, Surveillance & Diagnostic Algorithm 76
4.7 Treatment of MDR/RR-TB — BPaLM, Shorter & Longer Oral Regimens 78
4.8 TB in Special Situations — Pregnancy, Renal Failure, Liver Disease, Diabetes & the Elderly 79
4.9 HIV–TB Co-infection, ART Interactions & IRIS 81

4.10 Paediatric TB & Childhood Contact Management ... 82
4.11 Miliary TB & TB Meningitis ... 84
4.12 Pleural & Pericardial Tuberculosis ... 86
4.13 Extrapulmonary TB — Lymph Node, Abdominal, Spinal, Genitourinary, Endobronchial & Cutaneous ... 87
4.14 Complications & Post-TB Lung Disease; Corticosteroids and Surgery in TB ... 89
4.15 Patient Support, Infection Control, Stigma, BCG & TB Vaccines ... 91
4.16 Non-Tuberculous Mycobacteria ... 92
› Section 4 — previous-year question bank ... 280

Section 5 · Non-Tuberculous Infections of the Lung ... 95
5.1 Approach to Pulmonary Infection, Antibiotic Principles & Bronchoalveolar Lavage ... 95
5.2 Community-Acquired Pneumonia ... 97
5.3 Hospital-Acquired & Ventilator-Associated Pneumonia ... 99
5.4 Atypical & Tropical Pneumonias — Mycoplasma, Legionella, Melioidosis, Scrub Typhus, Leptospirosis ... 100
5.5 Viral Pneumonias — Influenza, COVID-19, RSV — and Vaccines ... 101
5.6 Aspiration Pneumonia, Lung Abscess & Lipoid Pneumonia ... 103
5.7 Bronchiectasis, Cystic Fibrosis & Primary Ciliary Dyskinesia ... 104
5.8 Pulmonary Aspergillosis Spectrum & Mucormycosis ... 106
5.9 Pneumocystis, Cryptococcosis, Histoplasmosis & Other Fungal Pneumonias ... 108
5.10 Parasitic Lung Disease — Hydatid, Amoebiasis, Paragonimiasis & Zoonoses ... 109
5.11 Pulmonary Infections in the Immunocompromised Host (HIV, Transplant, Immunosuppressants) ... 110
5.12 Recurrent & Non-Resolving Pneumonia; MRSA Pneumonia ... 113
› Section 5 — previous-year question bank ... 283

Section 6 · Obstructive Airway Diseases ... 115
6.1 Asthma — Pathogenesis, Phenotypes & Diagnosis ... 115
6.2 Management of Chronic Asthma — GINA Tracks, MART & Control Assessment ... 117
6.3 Acute Severe Asthma — Assessment, ICU Management & Mechanical Ventilation ... 118
6.4 Difficult-to-Treat & Severe Asthma — Phenotyping and Biologics ... 120
6.5 Allergic Bronchopulmonary Aspergillosis ... 122
6.6 Occupational Asthma, Allergen Immunotherapy & Inducible Laryngeal Obstruction ... 124
6.7 COPD — Definition, Pathogenesis, Risk Factors & Assessment ... 125
6.8 Pharmacotherapy of Stable COPD ... 127
6.9 Acute Exacerbation of COPD & Role of NIV ... 129
6.10 Systemic Manifestations, Comorbidities, Hyperinflation & Cor Pulmonale ... 131
6.11 Asthma–COPD Overlap & Small Airway Diseases (Bronchiolitis) ... 132
6.12 Oxygen Therapy, LTOT, Oxygen Toxicity & Hyperbaric Oxygen ... 133
6.13 Pulmonary Rehabilitation & Airway Clearance Techniques ... 135
6.14 Interventions for Emphysema — LVRS, Bronchoscopic Lung Volume Reduction & Bullous Disease ... 136
6.15 Alpha-1 Antitrypsin Deficiency ... 137
› Section 6 — previous-year question bank ... 285

Section 7 · Interstitial Lung Diseases & Immunological Disorders ... 139
7.1 Classification & Approach to Interstitial Lung Disease; HRCT Patterns ... 139
7.2 Idiopathic Pulmonary Fibrosis — Diagnosis, Severity, Antifibrotics & Acute Exacerbation ... 141
7.3 Other Idiopathic Interstitial Pneumonias — NSIP, COP, RB-ILD, DIP, AIP, PPFE; Progressive Pulmonary Fibrosis ... 144
7.4 Hypersensitivity Pneumonitis ... 145
7.5 Sarcoidosis ... 147
7.6 Connective Tissue Disease–Associated ILD & IPAF ... 149
7.7 Pulmonary Vasculitis & Diffuse Alveolar Haemorrhage ... 150
7.8 Eosinophilic Lung Diseases — AEP, CEP, Tropical Pulmonary Eosinophilia & HES ... 152
7.9 Drug-Induced & Transfusion-Related Lung Disease; Radiation Injury ... 153
7.10 Cystic Lung Diseases — LAM, PLCH, BHD & LIP ... 155
7.11 Pulmonary Alveolar Proteinosis, Alveolar Microlithiasis & Amyloidosis ... 156
› Section 7 — previous-year question bank ... 287

Section 8 · Occupational & Environmental Lung Disease, Smoking & Inhalational Injury ... 159
8.1 Approach to Occupational Lung Disease; Silicosis ... 159
8.2 Coal Workers' Pneumoconiosis, Asbestos-Related Diseases, Byssinosis & Other Dusts ... 161
8.3 Air Pollution, Ozone & Climate Change — Respiratory and Extrapulmonary Effects ... 162
8.4 Health Hazards of Smoking & Smoking Cessation ... 164
8.5 Toxic Inhalational Injury, Chemical & Biological Agents, Radiation Sickness & Strangulation ... 165

› *Section 8 — previous-year question bank* *289*

Section 9 · Pulmonary Circulation — Hypertension, Embolism, Oedema & Haemoptysis 168
9.1 Pulmonary Hypertension — Definition, Classification & Diagnosis 168
9.2 Pulmonary Arterial Hypertension — Risk Stratification & Treatment; CTD-PAH and Drug-Induced PAH 170
9.3 CTEPH, Portopulmonary Hypertension & Hepatopulmonary Syndrome 172
9.4 Pulmonary Embolism — Diagnosis & Risk Stratification 173
9.5 Management of PE & VTE; Prophylaxis, HIT, DOACs & Fat Embolism 175
9.6 Pulmonary Oedema; Cardiac–Pulmonary Interactions 177
9.7 Haemoptysis — Causes, Massive Haemoptysis & Bronchial Artery Embolisation 178
› *Section 9 — previous-year question bank* *290*

Section 10 · Lung Cancer & Other Thoracic Tumours 181
10.1 Epidemiology, Risk Factors, Genetic Susceptibility & Lung Cancer Screening 181
10.2 WHO Classification, Pathology, Tumour Markers & Liquid Biopsy 183
10.3 Clinical Features, Paraneoplastic Syndromes & Electrolyte Disorders 184
10.4 Diagnosis & Staging — TNM 9th Edition, EBUS & Mediastinal Staging 186
10.5 Treatment of NSCLC — Surgery, SBRT, Chemoradiation, Targeted Therapy & Immunotherapy 187
10.6 Small Cell Lung Cancer 189
10.7 Solitary Pulmonary Nodule & Incidental Nodules 190
10.8 Carcinoid Tumours, Metastatic Lung Disease & Other Tumours 192
10.9 Interventional Bronchoscopy — Central Airway Obstruction, Heat Ablation & Palliation 193
› *Section 10 — previous-year question bank* *291*

Section 11 · Pleura, Mediastinum, Diaphragm & Chest Wall 196
11.1 Pleural Physiology & Approach to Pleural Effusion 196
11.2 Parapneumonic Effusion, Empyema & Bronchopleural Fistula 198
11.3 Malignant & Recurrent Pleural Effusions, Hepatic Hydrothorax & Indwelling Pleural Catheters 200
11.4 Malignant Pleural Mesothelioma 202
11.5 Chylothorax, Pseudochylothorax, Haemothorax, Fibrothorax & Trapped Lung 203
11.6 Pneumothorax — Primary, Secondary, Recurrent, Iatrogenic; Persistent Air Leak 204
11.7 Mediastinal Masses, Mediastinitis & Pneumomediastinum 206
11.8 Diseases of the Diaphragm — Paralysis, Eventration & Elevated Hemidiaphragm 207
11.9 Chest Wall Disorders — Kyphoscoliosis, Thoracoplasty, Flail Chest, Marfan Syndrome & Neuromuscular Restriction 209
› *Section 11 — previous-year question bank* *292*

Section 12 · Critical Care Pulmonary Medicine 211
12.1 Respiratory Failure — Types, Mechanisms & Approach to Acute Hypoxaemia 211
12.2 Oxygen Delivery Devices, High-Flow Nasal Cannula & ROX Index 213
12.3 Non-Invasive Ventilation 215
12.4 Mechanical Ventilation — Modes, Waveforms, Alarms & Auto-PEEP 216
12.5 ARDS — Definition, Lung-Protective Ventilation, Prone Positioning & Refractory Hypoxaemia 219
12.6 Ventilating Obstructive Airway Disease — COPD & Dynamic Hyperinflation 221
12.7 Weaning, Tracheostomy, VILI & Complications of Mechanical Ventilation 222
12.8 Sepsis & Septic Shock; SOFA Score; Common Medical Emergencies in the Respiratory ICU 224
12.9 Monitoring in Respiratory Critical Care — Pulse Oximetry, Capnography, Haemodynamics & Central Lines 225
12.10 Airway Management & the Difficult Airway 227
12.11 Ethics, End-of-Life Care & Brain Death in Critical Care 228
› *Section 12 — previous-year question bank* *294*

Section 13 · Sleep-Related Breathing Disorders 230
13.1 Sleep Physiology, Polysomnography & Daytime Sleepiness Testing 230
13.2 Obstructive Sleep Apnoea — Epidemiology, Pathogenesis, Clinical Features & Diagnosis 232
13.3 Treatment of Obstructive Sleep Apnoea — PAP, Oral Appliances, Surgery & Pharmacotherapy 234
13.4 Central Sleep Apnoea, Obesity Hypoventilation, Overlap Syndrome & Narcolepsy 236
› *Section 13 — previous-year question bank* *296*

Section 14 · Thoracic Imaging & Pulmonary Procedures 238
14.1 Chest Radiograph Interpretation — Collapse, Silhouette & Classic Signs 238
14.2 CT & HRCT Signs in Pulmonary Medicine 240
14.3 Thoracic Ultrasound — Effusion, Pneumothorax & Trauma 241
14.4 PET-CT, MRI & Interventional Radiology in Chest Medicine 242
14.5 Flexible & Rigid Bronchoscopy, BAL, Transbronchial Lung Biopsy & Cryobiopsy 244

14.6 EBUS, Navigational & Virtual Bronchoscopy ... 245
14.7 Medical Thoracoscopy & Pleural Biopsy (Abrams, Cope, Image-Guided) ... 246
14.8 Tube Thoracostomy, Pleurodesis (incl. Autologous Blood Patch) & Image-Guided FNAC ... 248
› *Section 14 — previous-year question bank* ... *297*

Section 15 · Special Environments, Systemic Disease, Pregnancy & Preventive Pulmonology ... 250
15.1 High-Altitude Illness — AMS, HACE, HAPE & Chronic Mountain Sickness ... 250
15.2 Diving, Aviation & Aerospace Medicine; Miners ... 252
15.3 Extrapulmonary Manifestations & Pulmonary Complications of Systemic Disease ... 253
15.4 Respiratory Diseases in Pregnancy ... 254
15.5 Vaccination in Pulmonary Diseases ... 255
15.6 Chronic Cough Syndromes (UACS, GERD) & Quality-of-Life Questionnaires ... 257
15.7 Obesity-Related Pulmonary Disorders ... 259
› *Section 15 — previous-year question bank* ... *298*

Section 16 · Surgical Aspects of Pulmonary Medicine ... 261
16.1 Pre-Operative Evaluation for Lung Resection & Extrathoracic Surgery ... 261
16.2 Chest Trauma & Trauma-Related Lung Dysfunction ... 263
16.3 Lung Transplantation ... 264
16.4 Upper Airway Obstruction & Approach to Stridor ... 265
› *Section 16 — previous-year question bank* ... *299*

Section 17 · Research Methodology, Epidemiology & Biostatistics ... 267
17.1 Epidemiological Terms & Study Designs ... 267
17.2 Biostatistics — Central Tendency, Dispersion, Hypothesis Testing & Sampling ... 268
17.3 Clinical Trials, Bias, Blinding, Randomisation, Outcomes & Research Ethics ... 270
› *Section 17 — previous-year question bank* ... *300*

Previous-Year Question Bank (DNB & University papers) ... 273

NMC Curriculum — MD Pulmonary Medicine (hyperlinked) ... 301

Abbreviations ... 308

Part II · Advanced Theory, Clinical Algorithms & Image Atlas 310
Extended answers, decision algorithms, model-answer frameworks, drug charts, Radiopaedia image links and Merck Manual links — Sections 1–17
Section 18 · Clinical Examination in Respiratory Medicine — Long Case, Short Case & Viva 513
Section 19 · Oxygen Delivery, Non-Invasive Support & Mechanical Ventilation 528

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NEW IN THIS EDITION

Investigations & Treatment at a Glance — Index

Every topic in Part I now has an added page immediately after it, with (1) a table of the most important investigations — reference (normal) values, abnormal/diagnostic values and interpretation — and (2) a treatment table — drug or intervention, adult dose, indication and caution. Added pages carry a letter after the page number (e.g. **Page 24-A**), so all original page numbers, the contents, the question bank and every hyperlink remain unchanged. Tap a topic to jump to its tables; each table page links back to its topic.

No.	Topic	Page
1.1	Development of the Lung & Postnatal Growth	11-A
1.2	Airway Structure, Epithelium, Cilia & Alveolar Cells	13-A
1.3	Bronchopulmonary Segments, Secondary Lobule & Fissures	15-A
1.4	Pulmonary & Bronchial Circulation and Lymphatics	16-A
1.5	Mediastinum & Pleura: Anatomy and Innervation	17-A
1.6	Diaphragm, Respiratory Muscles & Phrenic Nerve	18-A
1.7	Developmental Anomalies I — Agenesis, Sequestration, CPAM, Bronchogenic Cyst	19-A
1.8	Developmental Anomalies II — TEF, Scimitar, PAVM, Unilateral Hyperlucent Lung	21-A
2.1	Lung Volumes & Capacities and Their Measurement	24-A
2.2	Spirometry & Flow–Volume Loops	26-A
2.3	Diffusion & DLCO	27-A
2.4	Respiratory Mechanics — Compliance, Resistance, Surfactant & Atelectasis	29-A
2.5	Ventilation, Dead Space, V/Q Matching & Mechanisms of Hypoxaemia	31-A
2.6	Gas Transport — Oxygen and Carbon Dioxide Dissociation Curves	33-A
2.7	Control of Ventilation, Chemoreceptors & Reflexes	35-A
2.8	Acid–Base Balance & ABG Interpretation; Electrolytes	37-A
2.9	Non-Respiratory Functions & Pulmonary Defence Mechanisms	39-A
2.10	Exercise Physiology, CPET & Six-Minute Walk Test	40-A
2.11	Bronchoprovocation, Oscillometry, Small Airway Tests, FeNO & Respiratory Muscle Tests	41-A
2.12	Physiological Changes in Pregnancy, Ageing & High Altitude	43-A
2.13	Physiological Basis of Pulmonary Symptoms & Signs — Dyspnoea, Cough, Clubbing	44-A
2.14	Aerosol Kinetics & Inhaled Drug Delivery Devices	45-A
2.15	Pulmonary Circulation Physiology — West Zones, Starling Resistor, HPV & Fluids	46-A
2.16	Principles of Electrocardiography & Cardiac Changes in Lung Disease	48-A
3.1	Mycobacteria — Biology, Staining, Culture & Phenotypic DST	51-A
3.2	Molecular Diagnosis of TB — CBNAAT, Truenat, LPA & Whole-Genome Sequencing	52-A
3.3	Respiratory Viruses & Their Laboratory Diagnosis	53-A
3.4	Respiratory Fungi — Classification & Laboratory Diagnosis	54-A
3.5	Bacteria, Atypical Organisms, Anaerobes & Biomarkers of Infection	55-A
3.6	Pathology — Inflammation, Granuloma & Emphysema	56-A
3.7	Hypersensitivity, Allergy Testing & Immunology of Lung Disease	58-A
3.8	Pharmacology of First-Line Anti-TB Drugs & Drug-Induced Liver Injury	60-A
3.9	Second-Line & Newer Anti-TB Drugs — Bedaquiline, Delamanid, Pretomanid, Linezolid, Fluoroquinolones	61-A

No.	Topic	Page
3.10	Bronchodilators & Inhaled Corticosteroids	62-A
3.11	Antimicrobials, Antifungals & Macrolides in Respiratory Medicine	64-A
3.12	Antineoplastic, Immunosuppressant & Biologic Drugs; Pharmacovigilance	65-A
4.1	Epidemiology of TB, End TB Strategy & the National TB Elimination Programme	68-A
4.2	Pathogenesis — Primary & Post-Primary TB, Wallgren Timetable	69-A
4.3	Diagnosis of Pulmonary TB — NTEP Algorithm, Radiology & Differential Diagnosis	71-A
4.4	Latent TB Infection — TST, IGRA & TB Preventive Treatment	74-A
4.5	Treatment of Drug-Susceptible TB under NTEP	75-A
4.6	Drug-Resistant TB — Definitions, Risk Factors, Surveillance & Diagnostic Algorithm	77-A
4.7	Treatment of MDR/RR-TB — BPaLM, Shorter & Longer Oral Regimens	79-A
4.8	TB in Special Situations — Pregnancy, Renal Failure, Liver Disease, Diabetes & the Elderly	81-A
4.9	HIV–TB Co-infection, ART Interactions & IRIS	82-A
4.10	Paediatric TB & Childhood Contact Management	83-A
4.11	Miliary TB & TB Meningitis	85-A
4.12	Pleural & Pericardial Tuberculosis	87-A
4.13	Extrapulmonary TB — Lymph Node, Abdominal, Spinal, Genitourinary, Endobronchial & Cutaneous	89-A
4.14	Complications & Post-TB Lung Disease; Corticosteroids and Surgery in TB	90-A
4.15	Patient Support, Infection Control, Stigma, BCG & TB Vaccines	92-A
4.16	Non-Tuberculous Mycobacteria	94-A
5.1	Approach to Pulmonary Infection, Antibiotic Principles & Bronchoalveolar Lavage	96-A
5.2	Community-Acquired Pneumonia	98-A
5.3	Hospital-Acquired & Ventilator-Associated Pneumonia	100-A
5.4	Atypical & Tropical Pneumonias — Mycoplasma, Legionella, Melioidosis, Scrub Typhus, Leptospirosis	101-A
5.5	Viral Pneumonias — Influenza, COVID-19, RSV — and Vaccines	102-A
5.6	Aspiration Pneumonia, Lung Abscess & Lipoid Pneumonia	104-A
5.7	Bronchiectasis, Cystic Fibrosis & Primary Ciliary Dyskinesia	106-A
5.8	Pulmonary Aspergillosis Spectrum & Mucormycosis	108-A
5.9	Pneumocystis, Cryptococcosis, Histoplasmosis & Other Fungal Pneumonias	109-A
5.10	Parasitic Lung Disease — Hydatid, Amoebiasis, Paragonimiasis & Zoonoses	110-A
5.11	Pulmonary Infections in the Immunocompromised Host (HIV, Transplant, Immunosuppressants)	112-A
5.12	Recurrent & Non-Resolving Pneumonia; MRSA Pneumonia	114-A
6.1	Asthma — Pathogenesis, Phenotypes & Diagnosis	117-A
6.2	Management of Chronic Asthma — GINA Tracks, MART & Control Assessment	118-A
6.3	Acute Severe Asthma — Assessment, ICU Management & Mechanical Ventilation	120-A
6.4	Difficult-to-Treat & Severe Asthma — Phenotyping and Biologics	122-A
6.5	Allergic Bronchopulmonary Aspergillosis	124-A
6.6	Occupational Asthma, Allergen Immunotherapy & Inducible Laryngeal Obstruction	125-A
6.7	COPD — Definition, Pathogenesis, Risk Factors & Assessment	126-A
6.8	Pharmacotherapy of Stable COPD	129-A
6.9	Acute Exacerbation of COPD & Role of NIV	131-A
6.10	Systemic Manifestations, Comorbidities, Hyperinflation & Cor Pulmonale	132-A

No.	Topic	Page
6.11	Asthma–COPD Overlap & Small Airway Diseases (Bronchiolitis)	133-A
6.12	Oxygen Therapy, LTOT, Oxygen Toxicity & Hyperbaric Oxygen	135-A
6.13	Pulmonary Rehabilitation & Airway Clearance Techniques	136-A
6.14	Interventions for Emphysema — LVRS, Bronchoscopic Lung Volume Reduction & Bullous Disease	137-A
6.15	Alpha-1 Antitrypsin Deficiency	138-A
7.1	Classification & Approach to Interstitial Lung Disease; HRCT Patterns	141-A
7.2	Idiopathic Pulmonary Fibrosis — Diagnosis, Severity, Antifibrotics & Acute Exacerbation	143-A
7.3	Other Idiopathic Interstitial Pneumonias — NSIP, COP, RB- ILD, DIP, AIP, PPFE; Progressive Pulmonary Fibrosis	145-A
7.4	Hypersensitivity Pneumonitis	146-A
7.5	Sarcoidosis	148-A
7.6	Connective Tissue Disease–Associated ILD & IPAF	150-A
7.7	Pulmonary Vasculitis & Diffuse Alveolar Haemorrhage	151-A
7.8	Eosinophilic Lung Diseases — AEP, CEP, Tropical Pulmonary Eosinophilia & HES	153-A
7.9	Drug-Induced & Transfusion-Related Lung Disease; Radiation Injury	155-A
7.10	Cystic Lung Diseases — LAM, PLCH, BHD & LIP	156-A
7.11	Pulmonary Alveolar Proteinosis, Alveolar Microlithiasis & Amyloidosis	158-A
8.1	Approach to Occupational Lung Disease; Silicosis	161-A
8.2	Coal Workers' Pneumoconiosis, Asbestos-Related Diseases, Byssinosis & Other Dusts	162-A
8.3	Air Pollution, Ozone & Climate Change — Respiratory and Extrapulmonary Effects	164-A
8.4	Health Hazards of Smoking & Smoking Cessation	165-A
8.5	Toxic Inhalational Injury, Chemical & Biological Agents, Radiation Sickness & Strangulation	167-A
9.1	Pulmonary Hypertension — Definition, Classification & Diagnosis	170-A
9.2	Pulmonary Arterial Hypertension — Risk Stratification & Treatment; CTD-PAH and Drug-Induced PAH	171-A
9.3	CTEPH, Portopulmonary Hypertension & Hepatopulmonary Syndrome	172-A
9.4	Pulmonary Embolism — Diagnosis & Risk Stratification	174-A
9.5	Management of PE & VTE; Prophylaxis, HIT, DOACs & Fat Embolism	176-A
9.6	Pulmonary Oedema; Cardiac–Pulmonary Interactions	178-A
9.7	Haemoptysis — Causes, Massive Haemoptysis & Bronchial Artery Embolisation	180-A
10.1	Epidemiology, Risk Factors, Genetic Susceptibility & Lung Cancer Screening	182-A
10.2	WHO Classification, Pathology, Tumour Markers & Liquid Biopsy	184-A
10.3	Clinical Features, Paraneoplastic Syndromes & Electrolyte Disorders	185-A
10.4	Diagnosis & Staging — TNM 9th Edition, EBUS & Mediastinal Staging	187-A
10.5	Treatment of NSCLC — Surgery, SBRT, Chemoradiation, Targeted Therapy & Immunotherapy	189-A
10.6	Small Cell Lung Cancer	190-A
10.7	Solitary Pulmonary Nodule & Incidental Nodules	192-A
10.8	Carcinoid Tumours, Metastatic Lung Disease & Other Tumours	193-A
10.9	Interventional Bronchoscopy — Central Airway Obstruction, Heat Ablation & Palliation	195-A
11.1	Pleural Physiology & Approach to Pleural Effusion	198-A
11.2	Parapneumonic Effusion, Empyema & Bronchopleural Fistula	199-A
11.3	Malignant & Recurrent Pleural Effusions, Hepatic Hydrothorax & Indwelling Pleural Catheters	201-A
11.4	Malignant Pleural Mesothelioma	202-A

No.	Topic	Page
11.5	Chylothorax, Pseudochylothorax, Haemothorax, Fibrothorax & Trapped Lung	204-A
11.6	Pneumothorax — Primary, Secondary, Recurrent, Iatrogenic; Persistent Air Leak	206-A
11.7	Mediastinal Masses, Mediastinitis & Pneumomediastinum	207-A
11.8	Diseases of the Diaphragm — Paralysis, Eventration & Elevated Hemidiaphragm	208-A
11.9	Chest Wall Disorders — Kyphoscoliosis, Thoracoplasty, Flail Chest, Marfan Syndrome & Neuromuscular Restriction	210-A
12.1	Respiratory Failure — Types, Mechanisms & Approach to Acute Hypoxaemia	213-A
12.2	Oxygen Delivery Devices, High-Flow Nasal Cannula & ROX Index	214-A
12.3	Non-Invasive Ventilation	216-A
12.4	Mechanical Ventilation — Modes, Waveforms, Alarms & Auto-PEEP	219-A
12.5	ARDS — Definition, Lung-Protective Ventilation, Prone Positioning & Refractory Hypoxaemia	221-A
12.6	Ventilating Obstructive Airway Disease — COPD & Dynamic Hyperinflation	222-A
12.7	Weaning, Tracheostomy, VILI & Complications of Mechanical Ventilation	223-A
12.8	Sepsis & Septic Shock; SOFA Score; Common Medical Emergencies in the Respiratory ICU	225-A
12.9	Monitoring in Respiratory Critical Care — Pulse Oximetry, Capnography, Haemodynamics & Central Lines	227-A
12.10	Airway Management & the Difficult Airway	228-A
12.11	Ethics, End-of-Life Care & Brain Death in Critical Care	229-A
13.1	Sleep Physiology, Polysomnography & Daytime Sleepiness Testing	232-A
13.2	Obstructive Sleep Apnoea — Epidemiology, Pathogenesis, Clinical Features & Diagnosis	234-A
13.3	Treatment of Obstructive Sleep Apnoea — PAP, Oral Appliances, Surgery & Pharmacotherapy	235-A
13.4	Central Sleep Apnoea, Obesity Hypoventilation, Overlap Syndrome & Narcolepsy	237-A
14.1	Chest Radiograph Interpretation — Collapse, Silhouette & Classic Signs	240-A
14.2	CT & HRCT Signs in Pulmonary Medicine	241-A
14.3	Thoracic Ultrasound — Effusion, Pneumothorax & Trauma	242-A
14.4	PET-CT, MRI & Interventional Radiology in Chest Medicine	243-A
14.5	Flexible & Rigid Bronchoscopy, BAL, Transbronchial Lung Biopsy & Cryobiopsy	245-A
14.6	EBUS, Navigational & Virtual Bronchoscopy	246-A
14.7	Medical Thoracoscopy & Pleural Biopsy (Abrams, Cope, Image-Guided)	247-A
14.8	Tube Thoracostomy, Pleurodesis (incl. Autologous Blood Patch) & Image-Guided FNAC	249-A
15.1	High-Altitude Illness — AMS, HACE, HAPE & Chronic Mountain Sickness	252-A
15.2	Diving, Aviation & Aerospace Medicine; Miners	253-A
15.3	Extrapulmonary Manifestations & Pulmonary Complications of Systemic Disease	254-A
15.4	Respiratory Diseases in Pregnancy	255-A
15.5	Vaccination in Pulmonary Diseases	257-A
15.6	Chronic Cough Syndromes (UACS, GERD) & Quality-of-Life Questionnaires	259-A
15.7	Obesity-Related Pulmonary Disorders	260-A
16.1	Pre-Operative Evaluation for Lung Resection & Extrathoracic Surgery	262-A
16.2	Chest Trauma & Trauma-Related Lung Dysfunction	264-A
16.3	Lung Transplantation	265-A
16.4	Upper Airway Obstruction & Approach to Stridor	266-A
17.1	Epidemiological Terms & Study Designs	268-A

No.	Topic	Page
17.2	Biostatistics — Central Tendency, Dispersion, Hypothesis Testing & Sampling	270-A
17.3	Clinical Trials, Bias, Blinding, Randomisation, Outcomes & Research Ethics	272-A

How to Use This Book

This manual follows the syllabus of the National Medical Commission (NMC) *Guidelines for Competency Based Postgraduate Training Programme for MD in Pulmonary Medicine*. The syllabus — basic sciences, clinical pulmonary medicine (infections and non-infectious disease), critical care, sleep, preventive pulmonology and the surgical aspects of pulmonary medicine — is arranged into 17 sections. Every topic follows the same blueprint so that it can be used for seminars, bedside discussion and rapid revision before the four theory papers and the clinical examination.

Chapter element	What it covers
CBME competencies	Cognitive, psychomotor and affective outcomes expected of the MD resident for the topic, with the NMC syllabus area it maps to.
In brief / definition	A two-line summary you can write as the opening of any answer.
Applied basic science	Anatomy, physiology, pathology and pharmacology needed to understand presentation and treatment (Paper I).
Clinical features & differential tables	Symptoms, signs, phenotypes and complications arranged for bedside use, with discriminating clues.
Algorithmic investigations	Step-wise tests from bedside to specialised, with expected findings and interpretation.
Flowcharts	Decision algorithms for important diseases and emergencies.
Evidence-based treatment	Rational drug therapy with doses, programme regimens (NTEP), emergency management and non-drug measures (Papers II–III).
Recent advances	Guideline changes and new therapies (Paper IV).
Clinical pearls & exam focus	High-yield points for theory, viva and clinical cases.
Landmark trials & guidelines	The evidence to quote at the end of an answer.
Previous-year questions	DNB and university questions on the topic, with source; the full topic-wise bank is at the end of the book.

Flowchart colour key

Start / end | Action | **Decision** | **Urgent step** | Information / alternative

Theory papers (NMC)

Paper	Scope
Paper I	General pulmonary medicine and basic sciences
Paper II	Clinical pulmonary medicine including medical emergencies
Paper III	Clinical pulmonary medicine including critical care medicine
Paper IV	Recent advances in pulmonary medicine and research methodology

Clinical examination: one long case and two short cases, followed by an oral/viva-voce examination on investigative procedures, therapeutic techniques and instruments (see Section 14).

Important note

Doses are for adults with normal renal and hepatic function unless stated. Programme regimens (NTEP) and international guidelines evolve rapidly — verify drug doses and protocols against the current national guidelines, institutional policy and product literature. This book supports, but does not replace, clinical judgement and supervised training.

NMC Syllabus Map

Each section of this book is mapped to the syllabus of the NMC *Guidelines for Competency Based Postgraduate Training Programme for MD in Pulmonary Medicine* and to the theory paper in which it is usually examined.

Book section	NMC syllabus areas covered	Theory paper
1. Applied Anatomy, Embryology & Developmental Anomalies	Development and anatomy of the respiratory system; applied embryology of lungs, mediastinum and diaphragm; developmental anomalies (NMC syllabus I-A 1–3). Frequently examined as 5-mark short notes in Paper I — secondary lobule, bronchopulmonary segments, cilia, sequestration, CPAM, TEF.	Paper I
2. Respiratory Physiology & Pulmonary Function	Assessment of pulmonary function; control of ventilation and mechanics; ventilation, perfusion, gas exchange and transport; non-respiratory functions; inhalation kinetics; acid–base and electrolytes; defence mechanisms; exercise physiology; pregnancy, altitude and ageing; physiological basis of symptoms (NMC syllabus I-B 1–13). The single most examined area of Paper I.	Paper I
3. Microbiology, Pathology, Immunology & Pharmacology	Mycobacteria and their laboratory diagnosis; respiratory bacteria, viruses, fungi and parasites; pathology of inflammation, granuloma and emphysema; hypersensitivity and allergy testing; pharmacology of anti-TB drugs, bronchodilators, anti-inflammatories, antimicrobials, antineoplastics and immunosuppressants; pharmacovigilance (NMC syllabus I-C, I-D, I-F, I-G).	Paper I
4. Tuberculosis	Aetiopathogenesis, diagnosis, differential diagnosis and management of pulmonary TB under the National TB Elimination Programme (NTEP) and the Standards for TB Care in India; complications; TB in children, elderly, pregnancy, diabetes, HIV and organ failure; pleural, pericardial and extrapulmonary TB; NTM; MDR/XDR-TB; prevention (NMC syllabus II-A1, I-E3). The core of Paper III in DNB and most university MD papers.	Paper II / III
5. Non-Tuberculous Infections of the Lung	Approach to pulmonary infection and principles of antibiotic use; community-acquired, hospital-acquired and ventilator-associated pneumonia; atypical, viral, fungal, parasitic, rickettsial and anaerobic infections; aspiration, lung abscess and bronchiectasis; infections in the immunocompromised host; zoonoses (NMC syllabus II-A2, I-C).	Paper II
6. Obstructive Airway Diseases	Asthma including ABPA, allergen immunotherapy and immunomodulation; COPD and small airway diseases; special aspects of management — long-term oxygen therapy, inhalation therapy and pulmonary rehabilitation; emphysema interventions and α1-antitrypsin deficiency (NMC syllabus II-B6, I-D4).	Paper II / III
7. Interstitial Lung Diseases & Immunological Disorders	Immune defence and reactions of the interstitium; idiopathic interstitial pneumonias; hypersensitivity pneumonitis; sarcoidosis; connective tissue disease, vasculitis and diffuse alveolar haemorrhage; eosinophilic pneumonias and tropical eosinophilia; drug- and radiation-induced lung disease; cystic lung diseases (PLCH, LAM); PAP and other rare diseases (NMC syllabus II-B3, II-B4, I-D6).	Paper II / IV
8. Occupational & Environmental Lung Disease, Smoking & Inhalational Injury	Occupational lung diseases and pneumoconioses; air-pollution induced disease and global warming; health hazards of smoking and smoking cessation; toxic lung, inhalational injury, chemical/biological agents and radiation (NMC syllabus II-B3, II-B4, I-E4, Preventive pulmonology).	Paper II / III
9. Pulmonary Circulation — Hypertension, Embolism, Oedema & Haemoptysis	Pulmonary hypertension and cor pulmonale; pulmonary oedema; pulmonary thromboembolic disease and infarction; cardiac problems in pulmonary patients and pulmonary complications of cardiac disease; haemoptysis and vascular malformations (NMC syllabus II-B5).	Paper II / III
10. Lung Cancer & Other Thoracic Tumours	Neoplastic and non-neoplastic lesions of the lung — epidemiology, natural history, pathology, staging and principles of medical, surgical and radiation treatment; solitary pulmonary nodule; carcinoid and metastatic tumours; interventional bronchoscopy in central airway obstruction (NMC syllabus II-B7, I-D7, I-E2).	Paper II / IV
11. Pleura, Mediastinum, Diaphragm & Chest Wall	Pleural dynamics and effusions; non-neoplastic and neoplastic pleural disease; pneumothorax; pyothorax and bronchopleural fistula; fibrothorax; non-neoplastic and neoplastic disorders of the mediastinum; diseases of the diaphragm and chest wall (NMC syllabus II-B8, II-B9, II-B13).	Paper II

Book section	NMC syllabus areas covered	Theory paper
12. Critical Care Pulmonary Medicine	Management of emergency problems in pulmonary disease; ARDS; respiratory failure in obstructive and other diseases; sepsis; respiratory and haemodynamic monitoring; non-invasive and mechanical ventilation; principles of critical care, complications and severity-of-illness scores; ethical and end-of-life issues (NMC syllabus II-B10).	Paper III
13. Sleep-Related Breathing Disorders	Physiology of sleep and its disorders; polysomnography (full-night and split-night) including CPAP titration and evaluation of daytime sleepiness; sleep apnoeas and other sleep-disordered breathing syndromes (NMC syllabus I-B8, II-B12).	Paper III
14. Thoracic Imaging & Pulmonary Procedures	Interpretation of plain radiography, ultrasound, CT, PET and MRI; fibreoptic and rigid bronchoscopy (diagnostic and therapeutic), BAL, cryobiopsy, EBUS and navigation; medical thoracoscopy, pleural biopsy, tube thoracostomy, pleurodesis and image-guided FNAC (NMC diagnostic tests and therapeutic procedures). Also the core of the clinical/viva examination (instruments and X-rays).	Paper I / IV · Clinical / Viva
15. Special Environments, Systemic Disease, Pregnancy & Preventive Pulmonology	High altitude, diving and aerospace medicine; extrapulmonary manifestations of pulmonary diseases and pulmonary manifestations of systemic diseases; respiratory disease in pregnancy; vaccination and preventive pulmonology; quality-of-life assessment and chronic cough syndromes; obesity-related pulmonary disorders (NMC syllabus II-B11, II-B13, II-B14).	Paper II / IV
16. Surgical Aspects of Pulmonary Medicine	Pre- and post-operative evaluation and management of thoracic and extrathoracic surgical patients; chest trauma and trauma-related lung dysfunction; lung transplantation; upper airway obstruction syndromes (NMC syllabus III, II-B4).	Paper II / III
17. Research Methodology, Epidemiology & Biostatistics	Epidemiological terms, definitions and methods; study designs; research methodology, statistics, clinical trials, bias and ethics (NMC syllabus I-E1, I-E5, cognitive domain 9; thesis requirements).	Paper I / IV

AETCM
Academy for Emergency, Trauma & Critical care Medicine

SECTION 1

Applied Anatomy, Embryology & Developmental Anomalies

Paper I

Development and anatomy of the respiratory system; applied embryology of lungs, mediastinum and diaphragm; developmental anomalies (NMC syllabus I-A 1–3). Frequently examined as 5-mark short notes in Paper I — secondary lobule, bronchopulmonary segments, cilia, sequestration, CPAM, TEF.

1.1 Development of the Lung & Postnatal Growth 10
1.2 Airway Structure, Epithelium, Cilia & Alveolar Cells 12
1.3 Bronchopulmonary Segments, Secondary Lobule & Fissures 13
1.4 Pulmonary & Bronchial Circulation and Lymphatics 15
1.5 Mediastinum & Pleura: Anatomy and Innervation 16
1.6 Diaphragm, Respiratory Muscles & Phrenic Nerve 17
1.7 Developmental Anomalies I — Agenesis, Sequestration, CPAM, Bronchogenic Cyst 18
1.8 Developmental Anomalies II — TEF, Scimitar, PAVM, Unilateral Hyperlucent Lung 20

1.1 Development of the Lung & Postnatal Growth

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-A1, I-A2 Development and applied embryology
- ✓ Describe the five stages of lung development with their timing and clinical correlates
- ✓ Explain the molecular regulation of branching morphogenesis and surfactant maturation
- ✓ Correlate arrested development with congenital anomalies and prematurity (BPD, hypoplasia)

IN BRIEF The lung arises as a ventral outpouching of the foregut (respiratory diverticulum) in the 4th week; airways are complete by 16 weeks, gas-exchange units develop from 16 weeks to term, and 85–90% of alveoli form after birth, continuing into adolescence.

Stages of lung development

Timeline of lung development

Stage	Period	Key events	Clinical correlate
Embryonic	4–7 weeks	Laryngotracheal groove → lung bud; tracheo-oesophageal septum; main, lobar and segmental bronchi; pulmonary arteries from 6th arch	TEF, agenesis, sequestration, bronchogenic cyst

Stage	Period	Key events	Clinical correlate
Pseudoglandular	5–17 weeks	Branching to terminal bronchioles (conducting airways complete, ~16 generations); cartilage, glands, smooth muscle	CDH-related hypoplasia, CPAM, congenital lobar emphysema
Canalicular	16–26 weeks	Respiratory bronchioles; capillaries approach epithelium; type I and II pneumocytes appear; surfactant begins ~24 weeks	Limit of viability (~22–24 weeks)
Saccular	24–38 weeks	Terminal sacs; thinning of interstitium; surfactant increases	RDS of prematurity
Alveolar	36 weeks – 8 years (to adolescence)	Secondary septation creates alveoli (≈50 million at birth → 300–480 million adult); microvascular maturation	Bronchopulmonary dysplasia = arrested alveolarisation

Molecular control

- **Branching:** FGF10 (mesenchyme) – FGFR2b (epithelium), SHH, BMP4, Wnt/β-catenin, retinoic acid; TBX4 marks lung field; NKX2-1 (TTF-1) specifies respiratory epithelium.
- **Surfactant maturation:** glucocorticoids, thyroid hormone and cAMP induce SP-A, B, C and phosphatidylcholine synthesis; antenatal steroids (betamethasone) reduce RDS.
- **Lung growth needs:** adequate thoracic space, amniotic fluid, fetal breathing movements — oligohydramnios, CDH and neuromuscular disease cause pulmonary hypoplasia.

Development of airways and blood vessels

- **Pre-acinar arteries and veins** follow airway branching and are complete by 16 weeks (Reid's laws); intra-acinar vessels develop with alveoli after birth.
- Bronchial arteries arise from the aorta at 9–12 weeks; pulmonary veins develop from the common pulmonary vein (sinus venosus) — anomalous return (scimitar) results from faulty incorporation.
- **Reid's laws:** (1) bronchial tree complete by 16 weeks; (2) alveoli develop mostly after birth; (3) pre-acinar vessels follow airways, intra-acinar vessels follow alveoli.

Postnatal lung development

- Alveolar number increases rapidly until 2 years, then mainly by enlargement; microvascular maturation (single capillary layer) completes by 2–3 years.
- Lung function rises until early adulthood (peak FEV1 ~20–25 years), plateaus, then declines ~25–30 mL/year (faster in smokers).
- Low birth weight, maternal smoking, early infections and air pollution reduce peak lung function — the "lung development trajectory" origin of COPD (GOLD "TETRA" aetiotypes).

✦ RECENT ADVANCES & GUIDELINE UPDATES

- ▸ GOLD 2023+ recognises abnormal lung development (low peak lung function) as a COPD aetiotype (COPD-D) — trajectory concept from Lange et al.

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Draw the five-stage timeline bar with weeks — easy marks; quote Reid's three laws.
- ▸ Alveolarisation is post-natal — so the lung can partly recover after neonatal injury, but BPD (arrested alveolarisation) persists into adult life.

LANDMARK TRIALS & GUIDELINES

- Lange P et al, NEJM 2015 — ~half of COPD at 65 years arose from low peak FEV1 in early adulthood rather than accelerated decline.

INVESTIGATIONS & TREATMENT AT A GLANCE

1.1 Development of the Lung & Postnatal Growth

‹ Back to topic (p 10)
Next topic 1.2 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Antenatal ultrasound (anomaly scan, 18–22 wk)	Normal lung echogenicity; heart axis 45° ± 20°	Echogenic mass, mediastinal shift, polyhydramnios, hydrops	CPAM, sequestration, CDH; CPAM volume ratio (CVR) > 1.6 predicts hydrops
Lung-to-head ratio (LHR, CDH)	Observed/expected LHR > 45%	O/E LHR < 25%	Severe pulmonary hypoplasia — poor survival
Lecithin/sphingomyelin ratio (amniotic fluid)	> 2.0 = mature lung	< 1.5	High risk of neonatal RDS
Phosphatidylglycerol (amniotic fluid)	Present after ~ 35 weeks	Absent	Immature surfactant system
Chest radiograph (neonate)	Clear lungs, normal volumes	Ground-glass, air bronchograms, low volumes	Respiratory distress syndrome (surfactant deficiency)
Spirometry (school age / adult)	FEV1 and FVC 80–120% predicted; z-score > −1.645	Low peak FEV1 in early adulthood	Abnormal lung-growth trajectory → COPD risk (GOLD COPD-D)

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Betamethasone (antenatal)	12 mg IM × 2 doses 24 h apart	Threatened preterm birth 24–34 weeks	Maternal hyperglycaemia; repeat courses limited
Dexamethasone (antenatal alternative)	6 mg IM 12-hourly × 4 doses	As above	As above
Surfactant (poractant alfa)	200 mg/kg intratracheal (then 100 mg/kg if needed)	Neonatal RDS	Bradycardia, desaturation during dosing; LISA/INSURE technique
Caffeine citrate	20 mg/kg load, then 5–10 mg/kg/day	Apnoea of prematurity; reduces BPD	Tachycardia, feed intolerance
Nasal CPAP	5–8 cmH_2O	Preterm respiratory distress	Pneumothorax, nasal trauma

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Antenatal steroids are most effective when delivery occurs 24 h–7 days after the first dose.
- Low peak lung function in early adulthood is a COPD pathway in its own right — ask about prematurity and childhood infections in every young COPD patient.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Development of the airways and blood vessels. *[DNB Jun 2025 P1]*
2. Post natal lung development. *[DNB Dec 2022 P1]*
3. Briefly describe the stages of development of lung and the airways. *[BFUHS 2022 P1]*

1.2 Airway Structure, Epithelium, Cilia & Alveolar Cells

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-A1 Anatomy and histology
- ✓ Describe the airway wall structure at trachea, bronchus and bronchiole level
- ✓ Describe the respiratory epithelium, cilia ultrastructure and alveolar cells with functions
- ✓ Apply this knowledge to ciliary dyskinesia, mucus hypersecretion and surfactant disorders

IN BRIEF Airways branch dichotomously over ~23 generations: 0–16 conducting zone (anatomical dead space ~150 mL) and 17–23 respiratory zone (acinus). Cartilage disappears at bronchioles (<1 mm), and the epithelium changes from pseudostratified ciliated columnar to cuboidal (club cells) to squamous alveolar cells.

Airway wall at three principal levels

Feature	Trachea / main bronchi	Bronchi	Bronchioles
Cartilage	C-shaped rings, posterior trachealis muscle	Irregular plates	Absent
Epithelium	Pseudostratified ciliated columnar with goblet cells	Same, shorter	Simple ciliated cuboidal → club (Clara) cells
Glands	Abundant submucosal seromucous glands	Present	Absent
Smooth muscle	Posterior only	Spiral bundles	Relatively thickest layer
Diameter	15–25 mm	>1 mm	<1 mm; terminal ~0.5 mm

Cells of the conducting airway epithelium

Epithelial cell types

Cell	Proportion / site	Function
Ciliated columnar	Most numerous in large airways	Mucociliary clearance (~200–300 cilia/cell, 1000–1500 beats/min)
Goblet cells	1 per 5 ciliated cells	Mucin (MUC5AC, MUC5B) secretion; hyperplasia in smokers, asthma
Basal cells	Basement membrane	Stem/progenitor cells for repair
Club (Clara) cells	Bronchioles	CC16 secretion, xenobiotic metabolism (CYP450), progenitor for bronchiolar epithelium
Neuroendocrine (Kulchitsky) cells	Scattered, NEBs	Serotonin, bombesin (GRP), calcitonin; origin of carcinoid and SCLC
Brush cells / ionocytes	Rare	Chemosensation; ionocytes express CFTR highly

Ultrastructure of a cilium

- **Axoneme "9+2":** nine peripheral microtubule doublets around a central pair, enclosed by ciliary membrane, anchored in a basal body (9 triplets, no central pair).
- **Dynein arms** (outer and inner) on the A-tubule of each doublet hydrolyse ATP and slide doublets — producing the effective stroke; **nexin links** convert sliding to bending; **radial spokes** connect to the central sheath.
- Cilia beat in a periciliary (sol) layer ~7 µm deep, their tips engaging the overlying gel (mucus) layer; metachronal waves propel mucus cephalad at 4–20 mm/min.

- **Defects:** absent outer dynein arm (most common in PCD, DNAH5/DNAI1), inner arm, radial spoke or central-pair defects; Kartagener = PCD + situs inversus. Diagnosis by nasal NO, high-speed video microscopy, TEM and genetics.

Bronchial mucus

- Two layers — periciliary sol (water, tethered mucins MUC1, MUC4, MUC16) and gel (secreted polymeric MUC5AC and MUC5B); 97% water, 2–3% mucins, lipids, IgA, lysozyme, lactoferrin, defensins.
- Normal production ~10–100 mL/day, swallowed unnoticed. Hypersecretion (>100 mL/day): chronic bronchitis, bronchiectasis, bronchorrhoea in invasive mucinous adenocarcinoma.
- Dehydration of the sol layer (CF: CFTR loss, ENaC excess) collapses cilia and stalls clearance.

Alveolar epithelium

Pneumocytes and alveolar macrophages

Cell	Features	Functions
Type I pneumocyte	40% of cells, 95% of surface; very thin (0.1–0.2 µm)	Gas exchange; fluid transport (aquaporin-5); cannot divide
Type II pneumocyte	60% of cells, 5% of surface; cuboidal with lamellar bodies, microvilli	Surfactant synthesis & recycling; progenitor of type I after injury; Na+ transport (ENaC) clears alveolar fluid; innate immunity (SP-A, SP-D)
Alveolar macrophage	Free in alveoli; derived from fetal monocytes (self-renewing) and blood monocytes	Phagocytosis, surfactant catabolism (GM-CSF dependent — defect causes PAP), antigen presentation, cytokines

- **Blood–gas barrier** 0.2–0.5 µm: type I cell + fused basement membrane + capillary endothelium. Pores of Kohn and canals of Lambert provide collateral ventilation.

★ **CLINICAL PEARLS & EXAM FOCUS**

- Type II cell = "defender of the alveolus" — surfactant, repair, fluid clearance, immunity. Hyperplasia of type II cells is the hallmark of the repair phase of DAD/ARDS.
- GM-CSF autoantibodies stop macrophage surfactant clearance → autoimmune PAP (90% of PAP).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Airway wall structure at the three principal levels. *[DNB Jun 2025 P1]*
2. Pneumocytes. *[DNB Jun 2025 P1]*
3. Ultrastructure of a cilium with diagram. *[DNB Jun 2025 P1]*
4. Ultrastructure of a cilium. *[DNB Dec 2022 P1]*
5. Epithelium lining of lower respiratory tract. *[DNB Dec 2022 P1]*
6. Type 2 alveolar epithelial cell structure and functions. *[DNB Dec 2021 P1]*
7. Epithelium of conducting airways. *[DNB Dec 2021 P1]*
8. Alveolar epithelium. *[DNB Dec 2021 P1]*

1.3 Bronchopulmonary Segments, Secondary Lobule & Fissures

‹ NMC curriculum
Previous-year Qs ›

◉ **CBME COMPETENCIES — the postgraduate should be able to:**

- ✓ **NMC syllabus area:** I-A1 Anatomy
- ✓ Name the bronchopulmonary segments and their clinical significance (aspiration, bronchoscopy, surgery)
- ✓ Describe the secondary pulmonary lobule and its HRCT interpretation
- ✓ Recognise normal and accessory fissures including the azygos fissure

INVESTIGATIONS & TREATMENT AT A GLANCE

1.2 Airway Structure, Epithelium, Cilia & Alveolar Cells

‹ Back to topic (p 12)
Next topic 1.3 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Nasal nitric oxide (nNO)	> 77 nL/min (adults/children > 5 y)	< 77 nL/min	Strongly suggests primary ciliary dyskinesia (PCD)
High-speed video microscopy (ciliary beat)	Beat frequency 11–20 Hz, coordinated	Immotile or dyskinetic cilia	PCD
Transmission electron microscopy of cilia	Normal 9+2 axoneme with dynein arms	Absent outer ± inner dynein arms, microtubular disorganisation	PCD (≈ 70% have TEM defect)
Saccharin test	Taste within 30 min	> 60 min	Impaired mucociliary clearance (screening only)
Sweat chloride	< 30 mmol/L	≥ 60 mmol/L (30–59 intermediate)	Cystic fibrosis
BAL differential cell count	Macrophages > 85%, lymphocytes 10–15%, neutrophils < 3%, eosinophils < 1%	Lymphocytosis > 15%, neutrophilia > 3%, eosinophils > 25%	Lymphocytic: sarcoid/HP; neutrophilic: IPF/infection; eosinophilic: eosinophilic pneumonia
Serum IgE (total)	< 100 IU/mL	> 417 IU/mL (> 1000 in ABPA criteria 2024: > 500)	Atopy, ABPA

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Hypertonic saline 7% (nebulised)	4 mL twice daily (after salbutamol)	PCD, bronchiectasis, CF — mucus clearance	Bronchospasm — first dose supervised
Dornase alfa	2.5 mg nebulised once daily	Cystic fibrosis	**Not** for non-CF bronchiectasis (harmful)
Azithromycin (long-term)	250 mg daily or 500 mg three times weekly	PCD/bronchiectasis exacerbation prevention	QTc, hearing loss, NTM must be excluded
Airway clearance physiotherapy	Twice daily (ACBT, oscillating PEP)	All mucociliary disorders	Teach technique; adherence
Ivacaftor/tezacaftor/elexacaftor	2 tablets (E 100/T 50/I 75 mg) morning + I 150 mg evening	CF with ≥ 1 F508del	LFTs; CYP3A interactions

★ **PRESCRIBING & INTERPRETATION PEARLS**

- PCD: situs inversus in about half (Kartagener syndrome) — always ask for chronic wet cough from infancy, neonatal respiratory distress and chronic otitis.

IN BRIEF A bronchopulmonary segment is the anatomically and functionally independent unit of lung supplied by a segmental (tertiary) bronchus and segmental pulmonary artery, with intersegmental veins at its periphery — the basis of segmentectomy and bronchoscopic localisation.

Bronchopulmonary segments

Lobe	Right lung (10)	Left lung (8–10)
Upper	Apical (1), Posterior (2), Anterior (3)	Apicoposterior (1+2), Anterior (3); Lingula: Superior (4), Inferior (5)
Middle	Lateral (4), Medial (5)	—
Lower	Superior (6), Medial basal (7), Anterior basal (8), Lateral basal (9), Posterior basal (10)	Superior (6), Anteromedial basal (7+8), Lateral basal (9), Posterior basal (10)

Clinical significance

- **Aspiration:** supine — posterior segment of RUL and superior segments of lower lobes; upright — basal segments of RLL (right main bronchus is wider, shorter, more vertical).
- **Post-primary TB:** apical and posterior segments of upper lobes and superior segment of lower lobe (high V/Q, high PAO2, poor lymphatic clearance).
- **Postural drainage** positions are planned per segment; segmentectomy for small peripheral cancers (JCOG0802 showed survival benefit vs lobectomy for ≤2 cm tumours).
- Right middle lobe syndrome — long narrow RML bronchus surrounded by lymph nodes, poor collateral ventilation → recurrent collapse.

Secondary pulmonary lobule (Miller)

- Smallest unit of lung bounded by connective-tissue septa; polyhedral, 1–2.5 cm; contains 3–24 acini; supplied by a lobular bronchiole and pulmonary artery branch in the **centrilobular core**; drained by veins and lymphatics in the **interlobular septa**.
- **HRCT localisation:** centrilobular (airway/vascular — HP, RB, endobronchial spread of TB, tree-in-bud), perilymphatic (septal, subpleural, peribronchovascular — sarcoidosis, lymphangitic carcinomatosis, silicosis), random (haematogenous — miliary TB, metastases).
- Septal thickening: smooth (pulmonary oedema, lymphangitis early), nodular (lymphangitic carcinomatosis, sarcoidosis), irregular (fibrosis).
- Primary lobule (Reid) = acinus distal to terminal bronchiole (~6–10 mm) — the gas-exchanging unit.

Nodule distribution in the secondary lobule

Pattern	Key HRCT feature	Typical causes
Centrilobular	Spare pleura/fissures by 5–10 mm	HP, RB-ILD, infectious bronchiolitis, endobronchial TB
Perilymphatic	Nodules on fissures, septa, bronchovascular bundles	Sarcoidosis, silicosis/CWP, lymphangitic carcinoma
Random	Uniform, touch pleura and vessels	Miliary TB, fungal, haematogenous metastases

Fissures

- **Oblique (major)** fissures separate upper and lower lobes; **horizontal** (minor) separates RUL and RML at 4th costal cartilage.
- **Azygos fissure** (0.4–1%): four layers of pleura (two parietal + two visceral) invaginated by an aberrant azygos vein that failed to migrate over the right apex — curvilinear line from apex to a "tear-drop" azygos shadow; the medial "azygos lobe" is not a true lobe (no separate bronchus). Clinical relevance: may be mistaken for bulla edge or mass; causes difficulty in thoracoscopy/central line placement.
- Other accessory fissures: inferior accessory (medial basal segment), superior accessory (separates superior segment), left minor fissure.

★ **CLINICAL PEARLS & EXAM FOCUS**

- "Ten on the right, eight on the left" — draw the diagram with numbers; draw the secondary lobule with core and septum and label the three nodule patterns.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Bronchopulmonary segments of human lung. *[DNB Jun 2025 P1]*
2. Discuss anatomy of bronchopulmonary segments with diagram and its clinical significance. *[WBUHS 2022 P1]*
3. Anatomy of secondary pulmonary lobule. *[DNB Oct 2024 P1]*
4. Secondary pulmonary lobule. *[DNB Oct 2023 P1]*
5. Secondary lobule. *[DNB Dec 2021 P1]*
6. Secondary lobule of lung. *[BFUHS 2020 P1]*
7. Azygos fissure. *[DNB Oct 2024 P1]*

1.4 Pulmonary & Bronchial Circulation and Lymphatics

‹ NMC curriculum
Previous-year Qs ›

◉ **CBME COMPETENCIES — the postgraduate should be able to:**

- ✓ **NMC syllabus area:** I-A1 Anatomy
- ✓ Describe the dual blood supply of the lung and its clinical significance in haemoptysis and infarction
- ✓ Describe the lymphatic drainage and the IASLC lymph-node map relevance

IN BRIEF The lung has a dual circulation: the low-pressure, high-flow pulmonary circulation (entire cardiac output, gas exchange) and the high-pressure systemic bronchial circulation (~1–2% of output, nutrition of airways, pleura and vessel walls). Bronchial arteries cause 90% of massive haemoptysis.

Pulmonary versus bronchial circulation

Feature	Pulmonary arteries	Bronchial arteries
Origin	Right ventricle (pulmonary trunk)	Descending aorta T5–T6 (variable; intercostobronchial trunk on right)
Pressure	25/8 mmHg (mean <20)	Systemic
Flow	100% of cardiac output	1–2%
Supplies	Alveolar capillaries	Airways to terminal bronchiole, visceral pleura, vasa vasorum, nodes
Drainage	Pulmonary veins → LA	Proximal → azygos/hemiazygos; distal → pulmonary veins (physiological shunt ~2%)
Response to hypoxia	Vasoconstriction (HPV)	Dilatation, hypertrophy in chronic inflammation

Clinical importance

- **Haemoptysis:** bronchial arteries hypertrophy in bronchiectasis, TB cavity, aspergilloma, CF — targets of bronchial artery embolisation. Beware spinal artery (anterior spinal artery of Adamkiewicz may arise from intercostobronchial trunk) — transverse myelitis complication.
- **Infarction is uncommon after PE** because bronchial collaterals keep parenchyma alive; infarction occurs when bronchial flow is compromised (LV failure, shock).
- **Rasmussen aneurysm** — pulmonary artery pseudoaneurysm in TB cavity wall.
- Pulmonary vascular resistance is ~1/10 of systemic; recruitment and distension lower PVR when flow rises (see 2.15).

Lymphatics

- Superficial (subpleural) and deep (peribronchovascular) networks drain to intrapulmonary → hilar → mediastinal → supraclavicular nodes; right lung drains to right; left lower lobe may cross to the right (contralateral N3 spread).

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INVESTIGATIONS & TREATMENT AT A GLANCE

1.3 Bronchopulmonary Segments, Secondary Lobule & Fissures

‹ Back to topic (p 13)
Next topic 1.4 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Chest radiograph (PA + lateral)	Normal fissures, lobar volumes	Displaced fissures, silhouette sign, segmental opacity	Localises collapse/consolidation to lobe and segment
HRCT secondary lobule assessment	Lobules 1–2.5 cm; centrilobular artery visible, septa barely seen	Interlobular septal thickening; centrilobular nodules; tree-in-bud	Septal: oedema, lymphangitis; centrilobular: bronchiolitis, HP, TB spread
Bronchoscopy — segmental nomenclature	All segmental orifices patent	Endobronchial lesion, extrinsic compression	Plan biopsy/BAL by segment
Ventilation/perfusion scan	Matched normal segmental perfusion	Segmental mismatched defects	Pulmonary embolism
Accessory fissures on CT	Azygos fissure 0.4–1%; inferior accessory 30–50% (non-pathological)	Misinterpreted as pleural line/ pneumothorax	Recognise as normal variants

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Antibiotics by segment involvement	Per pneumonia protocol (e.g. co-amoxiclav 1.2 g IV 8-hourly)	Aspiration in posterior segment of upper lobe / superior segment of lower lobe	Cover anaerobes in aspiration
Postural drainage	Position by segment, 5–10 min per segment	Localised bronchiectasis/lung abscess	Avoid head-down in GERD, raised ICP, recent haemoptysis
Segmentectomy	Surgical	Peripheral NSCLC ≤ 2 cm (JCOG0802), localised bronchiectasis	Pre-op PFT/DLCO; air leak

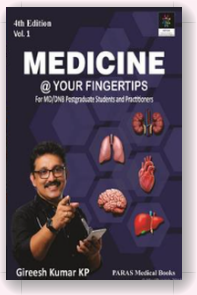
★ **PRESCRIBING & INTERPRETATION PEARLS**

- Aspiration supine → posterior segment of right upper lobe and superior segment of right lower lobe; upright → basal segments of right lower lobe.

- IASLC lymph-node map stations 1–14 (2009) guide N staging and EBUS sampling (see 10.4).
- Thoracic duct crosses from right to left at T5 — injury above this level causes left chylothorax, below causes right.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Blood supply of lung. *[DNB Oct 2023 P1]*
2. The bronchial circulation. *[DNB Jun 2021 P1]*
3. Discuss normal pulmonary circulation. Describe various interventional radiological procedures used in respiratory medicine. *[VNSGU 2025 P4]*



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1.5 Mediastinum & Pleura: Anatomy and Innervation

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-A1, I-B9 Anatomy and innervation
- ✓ Describe the divisions and contents of the mediastinum (traditional and ITMIG)
- ✓ Describe the pleura, its blood supply and nerve supply, and their clinical relevance

IN BRIEF The mediastinum is the space between the pleural sacs, from thoracic inlet to diaphragm. The ITMIG (2014) CT-based classification into prevascular, visceral and paravertebral compartments is now preferred for localising masses.

Mediastinal compartments and masses

Compartment (ITMIG)	Traditional	Contents	Common masses
Prevascular	Anterior	Thymus, fat, lymph nodes, left brachiocephalic vein	Thymoma, teratoma/germ cell, lymphoma, thyroid (the 4 Ts)
Visceral	Middle	Heart, great vessels, trachea, oesophagus, thoracic duct, nodes	Lymphadenopathy, bronchogenic/pericardial cysts, aneurysm, oesophageal lesions
Paravertebral	Posterior	Paravertebral tissue, sympathetic chain, nerve roots	Neurogenic tumours (schwannoma, neuroblastoma), extramedullary haematopoiesis

Investigating an anterior mediastinal mass

- CECT chest; MRI for cystic vs solid and invasion; tumour markers (AFP, β-hCG, LDH) in young men; PET-CT in lymphoma.
- Biopsy: CT-guided core, EBUS/EUS for visceral compartment, mediastinoscopy/VATS; avoid biopsy of suspected resectable thymoma (direct resection). Check for myasthenia (anti-AChR) before anaesthesia.

Pleura

- **Visceral pleura:** covers lung including fissures; supplied by bronchial arteries; autonomic nerves only — **insensitive to pain**.
- **Parietal pleura:** costal, mediastinal, diaphragmatic, cervical; supplied by intercostal, internal thoracic arteries; somatic nerves — **pain-sensitive**.

INVESTIGATIONS & TREATMENT AT A GLANCE

1.4 Pulmonary & Bronchial Circulation and Lymphatics

‹ Back to topic (p 15)
Next topic 1.5 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
CT pulmonary angiography	No filling defects; main PA diameter < 29 mm	Filling defect; PA ≥ 29 mm (or PA:Ao ratio > 1)	PE; pulmonary hypertension
Bronchial artery CT angiography	Bronchial arteries < 2 mm	Hypertrophied > 2 mm, tortuous; non-bronchial systemic collaterals	Source of haemoptysis — guides embolisation
Echocardiography (TR velocity)	≤ 2.8 m/s	> 2.8 m/s (PH probable > 3.4)	Pulmonary hypertension screening
Right heart catheterisation	mPAP ≤ 20 mmHg; PAWP ≤ 15; PVR ≤ 2 WU	mPAP > 20 mmHg; PVR > 2 WU (precapillary)	Defines PH (ESC/ERS 2022)
Lymphoscintigraphy / MR lymphangiography	Normal thoracic duct flow	Leak, obstruction	Chylothorax, lymphatic disorders
Pleural/chyle triglycerides	< 50 mg/dL	> 110 mg/dL (chylomicrons present)	Chylothorax

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Tranexamic acid	1 g IV 8-hourly or 500 mg nebulised 8-hourly	Haemoptysis	Thrombosis risk; avoid in active VTE
Bronchial artery embolisation	Interventional	Massive/recurrent haemoptysis	Spinal artery origin — paraplegia risk
Octreotide	50–100 µg SC 8-hourly (or 1–4 µg/kg/h infusion)	Chylothorax (reduce lymph flow)	Gallstones, hyperglycaemia
Medium-chain triglyceride diet	Low long-chain fat diet	Chylothorax	Nutrition deficits

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Bronchial arteries (systemic pressure) cause > 90% of massive haemoptysis; pulmonary arteries (Rasmussen) far less often.

- **Nerve supply of parietal pleura:** costal and peripheral diaphragmatic pleura — intercostal nerves (pain to chest wall/abdomen); central diaphragmatic and mediastinal — phrenic nerve (C3–5; pain referred to shoulder tip).
- **Pleural fluid:** 0.1–0.3 mL/kg; formed from parietal systemic capillaries; absorbed via parietal pleural stomata to lymphatics (capacity up to 20× formation).

★ CLINICAL PEARLS & EXAM FOCUS

▸ Shoulder-tip pain in pleurisy or subphrenic abscess = central diaphragmatic pleura via phrenic nerve; abdominal pain in lower-lobe pneumonia = lower intercostal nerves.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Anatomy of mediastinum. *[DNB Jun 2021 P1]*
2. Anatomy of the anterior mediastinum and investigations for its mass lesion. *[DNB Oct 2023 P1]*
3. Nerve supply of pleura. *[BFUHS 2020 P1]*

1.6 Diaphragm, Respiratory Muscles & Phrenic Nerve

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-A2 Applied anatomy
- ✓ Describe the development, structure, innervation and action of the diaphragm
- ✓ Describe the respiratory muscles and their roles in quiet and forced breathing
- ✓ Interpret the effects of phrenic nerve injury and respiratory muscle weakness

IN BRIEF The diaphragm is the principal inspiratory muscle (≈70% of tidal volume at rest). It develops from four components, is supplied by the phrenic nerve (C3–5) and acts as a piston and by its zone of apposition to lift and expand the lower rib cage.

Development (four components)

- Septum transversum (central tendon), pleuroperitoneal membranes, dorsal mesentery of oesophagus (crura), and body-wall muscle. Failure of pleuroperitoneal closure → Bochdalek hernia (posterolateral, left 85%); Morgagni hernia is retrosternal.
- Cervical origin (C3–5 myotomes) explains phrenic innervation and shoulder-tip referred pain.

Structure

- Peripheral muscle (sternal, costal, crural parts) inserts into the central tendon. Openings: IVC (T8, with right phrenic nerve), oesophagus (T10, vagi), aorta (T12, thoracic duct, azygos).
- **Zone of apposition** — costal diaphragm in contact with inner lower rib cage; lost in hyperinflation (COPD) → paradoxical inward lower rib motion (Hoover's sign).

Respiratory muscles

Group	Muscles	Role
Primary inspiratory	Diaphragm, parasternal and external intercostals, scalenes	Quiet breathing
Accessory inspiratory	Sternocleidomastoid, trapezius, pectorals, serratus	Increased demand; sign of distress
Expiratory	Abdominal (rectus, obliques, transversus), internal intercostals	Active expiration, cough, Valsalva
Upper airway	Genioglossus, laryngeal abductors	Patency; failure in sleep → OSA

Phrenic nerve

- C3–C5 (mainly C4); descends on scalenus anterior, enters thorax anterior to subclavian artery; right runs along SVC and right atrium, left crosses the aortic arch; both pass anterior to the lung hilum.

INVESTIGATIONS & TREATMENT AT A GLANCE

1.5 Mediastinum & Pleura: Anatomy and Innervation

‹ Back to topic (p 16)
Next topic 1.6 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
CT chest with contrast (ITMIG compartments)	No mediastinal mass; nodes < 10 mm short axis	Mass in prevascular / visceral / paravertebral compartment	Localises differential (thymoma/ lymphoma vs nodes vs neurogenic)
Serum AFP	< 10 ng/mL	Raised	Non-seminomatous germ cell tumour
Serum β-hCG	< 5 mIU/mL (non-pregnant)	Raised	Germ cell tumour (seminoma — mild rise)
Serum LDH	140–280 U/L	Markedly raised	Lymphoma, germ cell tumour
Anti-acetylcholine receptor antibodies	Negative	Positive	Thymoma-associated myasthenia — check before thymectomy
Pleural fluid analysis (Light's criteria)	Transudate: fluid/serum protein ≤ 0.5, LDH ratio ≤ 0.6, LDH ≤ ⅔ ULN	Any one criterion met	Exudate — needs further work-up

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Pyridostigmine	30–60 mg 4–6-hourly	Myasthenia with thymoma before surgery	Cholinergic crisis
Thymectomy	Surgical	Thymoma, generalised AChR+ myasthenia	Peri-operative myasthenic crisis
Chemotherapy (e.g. ABVD/ R-CHOP)	Oncology protocol	Mediastinal lymphoma	Bleomycin lung toxicity (ABVD)
Intercostal nerve block	Bupivacaine 0.25% 3–5 mL per space	Pleuritic pain, rib procedures	Pneumothorax, LA toxicity (max 2 mg/kg)

★ PRESCRIBING & INTERPRETATION PEARLS

- Parietal pleura is pain-sensitive (intercostal/phrenic nerves); visceral pleura is not — pleuritic pain means parietal involvement.

- **Injury:** cardiac surgery (cold cardioplegia), neck surgery, tumours (lung cancer, thymoma), neuralgic amyotrophy, C-spine trauma, interscalene block.
- **Unilateral palsy:** often asymptomatic; elevated hemidiaphragm; paradoxical movement on sniff test (fluoroscopy/USG); VC falls ~25% supine. **Bilateral:** orthopnoea, VC falls >50% supine, hypoventilation in REM sleep.
- **Tests:** USG thickening fraction (<20% = paralysis), sniff test, phrenic nerve conduction, Pdi/twitch, maximal inspiratory pressure (MIP).

Intercostal muscles

- External intercostals (inspiratory, especially upper and dorsal), internal interosseous (expiratory), parasternal intercartilaginous (inspiratory). Nerve supply: intercostal nerves T1–T11.

★ **CLINICAL PEARLS & EXAM FOCUS**

▸ A 25% fall in VC from sitting to supine suggests diaphragmatic weakness; >50% suggests bilateral palsy.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Discuss structure, innervation and action of diaphragm. *[DNB Oct 2024 P1]*
2. Role of intercostal muscles. *[DNB Jun 2025 P1]*
3. Phrenic nerve. *[DNB Oct 2023 P1]*
4. Respiratory muscles. *[DNB Oct 2023 P1]*

1.7 Developmental Anomalies I — Agenesis, Sequestration, CPAM, Bronchogenic Cyst

‹ NMC curriculum
Previous-year Qs ›

◉ **CBME COMPETENCIES — the postgraduate should be able to:**

- ✓ **NMC syllabus area:** I-A3 Developmental anomalies
- ✓ Classify congenital lung malformations by embryological stage
- ✓ Describe the clinical features, imaging and management of sequestration, CPAM and bronchogenic cyst

IN BRIEF Congenital thoracic malformations arise from arrested or aberrant foregut budding. Many present antenatally on ultrasound; in adults they present as recurrent infection, a cystic or mass lesion, or incidentally.

Classification of pulmonary agenesis (Schneider-Boyden)

Type	Description
Agenesis	Complete absence of lung, bronchus and vessels
Aplasia	Rudimentary bronchus, no parenchyma or vessels
Hypoplasia	All elements present but reduced in number/size (secondary: CDH, oligohydramnios)

- Right agenesis has worse prognosis (heart and mediastinum shift, associated cardiac anomalies). CXR: opaque hemithorax with ipsilateral shift — differentiate from collapse and pneumonectomy.

Pulmonary sequestration

- Non-functioning lung tissue with no normal bronchial communication and **systemic arterial supply** (usually descending thoracic or abdominal aorta).

Intralobar vs extralobar sequestration

Feature	Intralobar (75%)	Extralobar (25%)
Pleura	Within normal visceral pleura	Own pleural investment
Venous drainage	Pulmonary veins (left-to-left)	Systemic (azygos, IVC)

INVESTIGATIONS & TREATMENT AT A GLANCE

1.6 Diaphragm, Respiratory Muscles & Phrenic Nerve

‹ Back to topic (p 17)
Next topic 1.7 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Chest radiograph (inspiratory)	Right hemidiaphragm ~ 1–2 cm higher than left	Elevated hemidiaphragm	Paralysis, eventration, subphrenic process, volume loss
Fluoroscopic sniff test	Both domes descend on sniff	Paradoxical upward movement	Unilateral diaphragmatic paralysis
Diaphragm ultrasound — excursion	Quiet breathing 1–2.5 cm; deep breath > 4.7 cm	< 1 cm or paradoxical	Diaphragmatic weakness/paralysis
Diaphragm thickening fraction (US)	> 20%	< 20%	Weakness; predicts weaning failure in ICU (< 30%)
Supine vs upright FVC	Fall < 10%	Fall > 20–25% (bilateral palsy > 30%)	Diaphragmatic weakness
MIP / MEP	MIP > −80 cmH_2O (men), > −60 (women); MEP > 90	MIP less negative than −60	Respiratory muscle weakness
SNIP (sniff nasal pressure)	> 70 cmH_2O (men), > 60 (women)	< 40 cmH_2O	Neuromuscular weakness — predicts NIV need
Phrenic nerve conduction / EMG	Latency 6–8 ms	Prolonged / absent	Neuropathic phrenic palsy

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Non-invasive ventilation (nocturnal)	IPAP 12–16, EPAP 4–5 cmH_2O	Bilateral diaphragm paralysis, NMD with hypercapnia	Interface tolerance, aspiration
Diaphragmatic plication	Surgical (VATS)	Symptomatic unilateral paralysis/eventration	Only after 6–12 months if no recovery
Diaphragm pacing	Phrenic nerve stimulator	High cervical cord injury, central hypoventilation	Needs intact phrenic nerve
Inspiratory muscle training	30–50% of MIP, 15 min twice daily	COPD, weaning	Fatigue

★ PRESCRIBING & INTERPRETATION PEARLS

- Bilateral diaphragm paralysis → orthopnoea and paradoxical abdominal movement; supine FVC drop > 30% is the bedside clue.

Feature	Intralobar (75%)	Extralobar (25%)
Site	Posterior basal segment, left > right	Left, between lower lobe and diaphragm; may be subdiaphragmatic
Age	Adolescents/adults, recurrent infection	Neonates; associated anomalies (CDH, CPAM, cardiac) in 60%
Presentation	Recurrent pneumonia, haemoptysis, cystic mass	Antenatal mass, respiratory distress, high-output failure

- **Diagnosis:** CT angiography demonstrating systemic feeding artery is key; Doppler USG, MRA.
- **Management:** intralobar — surgical resection (lobectomy/segmentectomy) after infection controlled; ligate feeding artery carefully (may arise below diaphragm). Endovascular embolisation is an option for small/haemorrhagic lesions. Asymptomatic extralobar may be observed.
- **Hybrid lesions** = CPAM with systemic supply.

CPAM (Congenital pulmonary airway malformation) — Stocker classification

Stocker classification (5 types)

Type	Origin	Cyst size	Features
0	Trachea/bronchus (acinar dysplasia)	Solid, tiny	Incompatible with life (<3%)
1 (60–70%)	Distal bronchus/bronchiole	Large >2 cm, few	Best prognosis; rare mucinous adenocarcinoma risk
2 (15–20%)	Bronchiole	0.5–2 cm	Associated renal, cardiac anomalies
3 (5–10%)	Bronchiole/alveolar duct	Solid, microcystic <0.5 cm	Mass effect, hydrops, poor prognosis
4 (10%)	Distal acinus	Large peripheral thin-walled	Overlaps with type I pleuropulmonary blastoma

- Antenatal CVR (CPAM volume ratio) >1.6 predicts hydrops. Symptomatic → resection; asymptomatic → resection usually at 3–6 months (infection, malignancy risk).

Bronchogenic cyst

- Abnormal budding of ventral foregut (weeks 4–6); lined by respiratory epithelium with cartilage, glands, smooth muscle. 2/3 **mediastinal** (subcarinal, paratracheal — middle/visceral compartment), 1/3 intrapulmonary (lower lobes).
- Presentation: incidental, chest pain, dysphagia, airway compression (infants), infection, haemorrhage, air-fluid level if communicates.
- CT: well-defined round lesion, water attenuation (0–20 HU) — may be high if proteinaceous; MRI T2 bright. Differentials: oesophageal duplication cyst, pericardial cyst (cardiophrenic angle), neurenteric cyst (vertebral anomaly), cystic thymoma, lymphangioma, necrotic node.
- Management: surgical excision (VATS) for symptomatic and most asymptomatic cysts; EBUS aspiration risks infection and recurrence.

Tracheal bronchus

- Bronchus arising from the trachea (usually right, within 2 cm of carina) supplying the RUL apical segment ("pig bronchus" if entire RUL); 0.1–2%. Causes recurrent RUL pneumonia, atelectasis; important in intubation (ETT may occlude it).

★ CLINICAL PEARLS & EXAM FOCUS

- Systemic arterial supply on CT angiography = sequestration; quote "intralobar — pulmonary venous drainage; extralobar — systemic venous drainage".

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INVESTIGATIONS & TREATMENT AT A GLANCE

1.7 Developmental Anomalies I — Agenesis, Sequestration, CPAM, Bronchogenic Cyst

‹ Back to topic (p 18)
Next topic 1.8 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Antenatal ultrasound / fetal MRI	Normal lung	Cystic or echogenic mass, systemic feeding vessel on Doppler	CPAM vs sequestration
CT angiography chest	No aberrant systemic artery	Systemic artery from aorta to lung segment	**Pulmonary sequestration** (intralobar: pulmonary venous drainage; extralobar: systemic)
CT chest (CPAM)	—	Multicystic lesion communicating with airway (Stocker types 0–4)	CPAM; type 4 overlaps with pleuropulmonary blastoma
CT chest (bronchogenic cyst)	—	Well-defined fluid-density (0–20 HU) mediastinal/subcarinal cyst, no enhancement	Bronchogenic cyst (may be high HU if proteinaceous)
CXR in agenesis/aplasia	Two normal lungs	Opaque hemithorax, mediastinal shift **towards** side, absent pulmonary artery	Agenesis vs collapse (no air bronchogram, no hyperinflation)
Echocardiography	Normal heart	Associated cardiac anomalies (~ 50% in agenesis)	Prognosis

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Surgical resection (lobectomy / segmentectomy)	Elective at 3–6 months of age (asymptomatic CPAM varies)	Symptomatic CPAM, sequestration, bronchogenic cyst	Infection risk, malignancy potential (pleuropulmonary blastoma)
Endovascular embolisation	Interventional	Sequestration with heart failure from shunt	Infarction
Antibiotics for infected cyst	e.g. co-amoxiclav 1.2 g IV 8-hourly	Infected CPAM/sequestration/ bronchogenic cyst	Definitive surgery after infection settles
Maternal betamethasone	12 mg IM × 2 doses	Large microcystic CPAM with hydrops	Specialist fetal-medicine decision

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Intralobar sequestration (75%) presents later with recurrent lower-lobe pneumonia (left > right); extralobar is diagnosed in infancy and associated with other anomalies.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Definition and types of pulmonary sequestration. *[DNB Oct 2024 P1]*
2. Congenital agenesis of lung. *[DNB Oct 2023 P1]*
3. Stocker's classification of congenital pulmonary airway malformation (CPAM). *[DNB Oct 2023 P1]*
4. Bronchogenic cyst. *[DNB Oct 2023 P1]*
5. Tracheal bronchus. *[DNB Oct 2023 P1]*
6. Clinical manifestation of pulmonary sequestration. *[DNB Dec 2022 P2]*
7. Management of intrapulmonary sequestration. *[DNB Dec 2022 P2]*
8. Lung sequestration. *[DNB Jun 2021 P1]*

1.8 Developmental Anomalies II — TEF, Scimitar, PAVM, Unilateral Hyperlucent Lung

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-A3 Developmental anomalies
- ✓ Describe the types and features of tracheo-oesophageal fistula
- ✓ Describe vascular anomalies — scimitar syndrome, PAVM — and their management
- ✓ Approach a radiologically unilateral hyperlucent lung including Swyer-James-MacLeod syndrome

IN BRIEF Vascular and foregut anomalies may present in adults with recurrent aspiration, cyanosis, embolic stroke or an unexplained hyperlucent hemithorax — recognising their imaging signatures is a frequent viva theme.

Tracheo-oesophageal fistula (Gross / Vogt classification)

Type	Description	Frequency
A	Oesophageal atresia without fistula	8%
B	Atresia with proximal TEF	1%
C	Atresia with distal TEF	85%
D	Atresia with proximal and distal TEF	2%
E (H-type)	TEF without atresia	4% — presents late with recurrent aspiration

- **Clinical:** neonatal frothing, choking/cyanosis with feeds, inability to pass NG tube (coils on X-ray), gasless abdomen in type A/B; H-type → recurrent pneumonia, cough with drinking, presents in childhood/adults. VACTERL associations.
- **Acquired TEF in adults:** oesophageal/lung cancer (malignant, stented), prolonged cuffed intubation, TB, trauma. Diagnose with bronchoscopy + oesophagoscopy, CT; methylene blue test.

Scimitar (congenital venolobar / hypogenetic lung) syndrome

- Partial anomalous pulmonary venous return of right lung to the IVC — curved "Turkish sword" shadow along right heart border; right lung hypoplasia, dextroposition of heart, systemic arterial supply to right lower lobe; ± ASD.
- Left-to-right shunt (Qp:Qs); may cause PAH. Repair if Qp:Qs >1.5:1 or PAH; otherwise follow-up.

Pulmonary arteriovenous malformation (PAVM)

- Direct artery–vein communication bypassing capillaries → right-to-left shunt. **80–90% associated with HHT** (ENG, ACVRL1 mutations); screen every PAVM patient for HHT (Curaçao criteria).
- Features: dyspnoea, cyanosis, clubbing, orthodeoxia/platypnoea (basal lesions), haemoptysis, haemothorax; paradoxical emboli → stroke, brain abscess.
- Diagnosis: transthoracic contrast (bubble) echo (screening), CT angiography; 100% oxygen shunt study.
- **Treatment:** transcatheter embolisation (coils/plugs) when feeding artery ≥2–3 mm or any symptomatic lesion; antibiotic prophylaxis before dental procedures; avoid scuba diving; follow-up CT for recanalisation.

Unilateral hyperlucent lung — approach

Causes of unilateral hyperlucent hemithorax

Category	Causes
Technical / chest wall	Rotation, mastectomy, Poland syndrome (absent pectoralis), scoliosis
Pleura	Pneumothorax
Airway	Endobronchial obstruction with check-valve (foreign body, tumour), congenital lobar emphysema, bronchial atresia
Parenchyma	Swyer-James-MacLeod syndrome, giant bulla, compensatory hyperinflation after lobectomy/ collapse
Vascular	Pulmonary artery agenesis/hypoplasia, large PE (Westermark sign), PA compression (tumour, fibrosing mediastinitis)

- **Swyer-James-MacLeod syndrome:** post-infectious obliterative bronchiolitis (adenovirus, measles, mycoplasma) in childhood affecting one lung/lobe → small or normal-sized hyperlucent lung, reduced vascularity, **air trapping on expiration** (mediastinum shifts away on expiratory film), matched V/Q defects, bronchiectasis on HRCT. Treat infections; resection rarely.
- **Work-up:** inspiratory–expiratory CXR, HRCT with expiratory images, CT angiography, bronchoscopy for obstruction, V/Q scan.

★ CLINICAL PEARLS & EXAM FOCUS

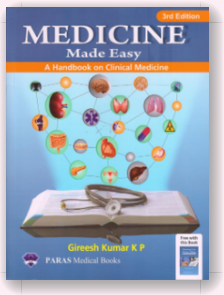
▸ Small hyperlucent lung with air trapping = Swyer-James; normal-sized hyperlucent lung with no air trapping = vascular cause (PA agenesis, PE).

LANDMARK TRIALS & GUIDELINES

- Shovlin CL et al, Curaçao criteria for HHT (2000); BTS clinical statement on PAVMs (Shovlin, Thorax 2017).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Tracheo-esophageal fistula (TEF): types and clinical features. *[DNB Oct 2024 P1; DNB Oct 2023 P1]*
2. Scimitar syndrome. *[DNB Oct 2023 P1]*
3. Macleod syndrome. *[DNB Jun 2021 P1]*
4. Management of a radiologically unilateral hyperlucent lung. *[DNB Jun 2021 P2]*
5. Pulmonary arterio-venous malformation. *[DNB Dec 2025 P2]*



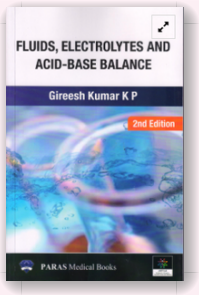
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INVESTIGATIONS & TREATMENT AT A GLANCE

1.8 Developmental Anomalies II — TEF, Scimitar, PAVM, Unilateral Hyperlucent Lung

‹ Back to topic (p 20)
Next topic 2.1 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Contrast echocardiography (bubble study)	No bubbles in left heart	Bubbles in LA after **3–8 cardiac cycles**	Intrapulmonary shunt (PAVM); < 3 cycles = intracardiac shunt
CT angiography (PAVM)	No AV malformation	Feeding artery ≥ 2–3 mm with draining vein	Embolise if feeding artery ≥ 3 mm (or any size in HHT)
Shunt fraction (100% O_2 test)	Shunt < 5%	> 5%	Quantifies right-to-left shunt
ABG / orthodeoxia	SpO_2 stable on standing	SpO_2 falls > 5% upright	Basal PAVM, hepatopulmonary syndrome
CT chest — scimitar syndrome	Normal pulmonary venous drainage	Curvilinear vein to IVC, right lung hypoplasia, dextroposition	Partial anomalous venous return
Bronchoscopy / contrast swallow	—	H-type tracheo-oesophageal fistula	Recurrent aspiration pneumonia in infants
V/Q scan (Swyer-James/ MacLeod)	Normal ventilation & perfusion	Matched reduced V and Q; air-trapping on expiratory CT	Unilateral hyperlucent lung (post-infectious bronchiolitis obliterans)

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Transcatheter embolisation (coils/plugs)	Interventional	PAVM with feeding artery ≥ 3 mm	Paradoxical embolism during procedure; recanalisation
Antibiotic prophylaxis before dental/surgical procedures	Amoxicillin 2 g orally 1 h before	All patients with PAVM	Brain abscess risk from right-to-left shunt
Iron replacement / bevacizumab	Ferrous sulphate 200 mg 1–3 times daily; bevacizumab per HHT protocol	HHT epistaxis and anaemia	Bevacizumab: hypertension, proteinuria
Surgical repair of TEF / scimitar	Surgical	Symptomatic lesions	Anastomotic leak, stenosis

★ **PRESCRIBING & INTERPRETATION PEARLS**

- In HHT, screen every first-degree relative with bubble echocardiography; IV lines in PAVM patients need air filters.

SECTION 2

Respiratory Physiology & Pulmonary Function

Paper I

Assessment of pulmonary function; control of ventilation and mechanics; ventilation, perfusion, gas exchange and transport; non-respiratory functions; inhalation kinetics; acid–base and electrolytes; defence mechanisms; exercise physiology; pregnancy, altitude and ageing; physiological basis of symptoms (NMC syllabus I-B 1–13). The single most examined area of Paper I.

2.1 Lung Volumes & Capacities and Their Measurement 22
2.2 Spirometry & Flow–Volume Loops 24
2.3 Diffusion & DLCO 26
2.4 Respiratory Mechanics — Compliance, Resistance, Surfactant & Atelectasis 28
2.5 Ventilation, Dead Space, V/Q Matching & Mechanisms of Hypoxaemia 29
2.6 Gas Transport — Oxygen and Carbon Dioxide Dissociation Curves 32
2.7 Control of Ventilation, Chemoreceptors & Reflexes 33
2.8 Acid–Base Balance & ABG Interpretation; Electrolytes 35
2.9 Non-Respiratory Functions & Pulmonary Defence Mechanisms 37
2.10 Exercise Physiology, CPET & Six-Minute Walk Test 39
2.11 Bronchoprovocation, Oscillometry, Small Airway Tests, FeNO & Respiratory Muscle Tests 40
2.12 Physiological Changes in Pregnancy, Ageing & High Altitude 41
2.13 Physiological Basis of Pulmonary Symptoms & Signs — Dyspnoea, Cough, Clubbing 43
2.14 Aerosol Kinetics & Inhaled Drug Delivery Devices 44
2.15 Pulmonary Circulation Physiology — West Zones, Starling Resistor, HPV & Fluids 45
2.16 Principles of Electrocardiography & Cardiac Changes in Lung Disease 47

2.1 Lung Volumes & Capacities and Their Measurement

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-B1 Assessment of pulmonary functions
- ✓ Define static lung volumes and capacities and give normal values
- ✓ Describe measurement of FRC, RV and TLC by helium dilution, nitrogen washout and body plethysmography
- ✓ Explain closing volume and its clinical significance

IN BRIEF Four volumes (TV, IRV, ERV, RV) combine into four capacities (IC, FRC, VC, TLC). Spirometry measures all except RV, so FRC, RV and TLC need gas dilution or plethysmography. Restriction is defined by TLC <LLN (5th percentile), not by FVC.

Static lung volumes (70-kg adult)

Volume / capacity	Definition	Normal
Tidal volume (TV)	Air moved in quiet breath	500 mL (6–8 mL/kg)
Inspiratory reserve volume (IRV)	Maximal inspiration beyond tidal	3000 mL
Expiratory reserve volume (ERV)	Maximal expiration below FRC	1100 mL
Residual volume (RV)	Air left after maximal expiration	1200 mL (RV/TLC 20–35%)
Inspiratory capacity (IC)	TV + IRV	3500 mL
Functional residual capacity (FRC)	ERV + RV — equilibrium of lung recoil and chest-wall outward recoil	2300 mL
Vital capacity (VC)	IRV + TV + ERV	4600 mL
Total lung capacity (TLC)	VC + RV	5800 mL

Determinants

- **TLC:** inspiratory muscle strength vs lung and chest wall recoil — reduced in ILD, neuromuscular disease, obesity, pleural disease; increased in emphysema.
- **FRC:** relaxation volume where lung inward recoil = chest wall outward recoil; falls supine (~0.5–1 L), with obesity, anaesthesia, pregnancy; rises in hyperinflation (dynamic in COPD).
- **RV:** in young people limited by expiratory muscle strength; in older people by airway closure — rises with age and air trapping (RV/TLC >40% = air trapping).

Methods of measuring FRC/RV/TLC

Method	Principle	Comment
Helium dilution (closed circuit)	C1V1 = C2(V1+FRC); inert insoluble gas equilibrates	Measures only communicating gas — underestimates in bullae/severe COPD
Nitrogen washout (open circuit)	Breathe 100% O2, collect exhaled N2 (≈79% of FRC)	Also underestimates; gives washout index of ventilation inhomogeneity (LCI)
Body plethysmography	Boyle's law: P1V1 = P2V2 during panting against closed shutter	Measures all thoracic gas (TGV) incl. trapped; gold standard; also gives Raw
Imaging (CT/CXR planimetry)	Volumetric estimation	Research, when patient cannot perform

Closing volume

- Volume above RV at which dependent small airways begin to close during expiration; measured by **single-breath nitrogen washout** — phase IV inflection.
- Closing capacity = CV + RV. Normally CC < FRC; CC rises with age (exceeds FRC supine at ~44 years, upright at ~66 years), smoking, small airway disease, obesity → V/Q mismatch and hypoxaemia.
- Phase III slope reflects ventilation inhomogeneity — an early marker of small airway disease.

Minute ventilation & vital capacity

- Minute ventilation (VE) = TV × RR ≈ 6–8 L/min; alveolar ventilation VA = (TV − VD) × RR ≈ 4.2 L/min.
- VC falls in restriction, neuromuscular weakness (serial VC <15–20 mL/kg or fall >30% predicts need for ventilation in GBS/ myasthenia) and in severe obstruction (air trapping).

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★ CLINICAL PEARLS & EXAM FOCUS

- Restrictive pattern on spirometry (low FVC, normal ratio) must be confirmed by TLC; up to half are not truly restricted (PRISm, poor effort, obesity).
- Plethysmographic TLC − helium TLC = volume of trapped gas (bullae).

LANDMARK TRIALS & GUIDELINES

- ATS/ERS standardisation of lung volume measurement (2005; updated technical standard 2023).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Measurement of residual volume (RV). *[DNB Jun 2025 P1]*
2. Static and dynamic lung volumes. *[DNB Oct 2023 P1]*
3. Functional residual capacity (FRC) and residual volume (RV). *[DNB Dec 2021 P1]*
4. Measurement of lung volume. *[DNB Dec 2021 P1]*
5. Closing volume. *[DNB Dec 2021 P1; VNSGU 2025 P1; Manipal 2014 P1]*
6. Vital capacity. *[Manipal 2014 P1]*
7. Minute ventilation. *[Manipal 2014 P1]*

2.2 Spirometry & Flow–Volume Loops

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-B1 Assessment of pulmonary functions
- ✓ State indications, contraindications and the technique (manoeuvres, acceptability, repeatability) of spirometry
- ✓ Interpret spirometry using LLN and z-scores (ATS/ERS 2022) and recognise patterns including PRISm
- ✓ Interpret flow–volume loops in upper airway obstruction

IN BRIEF Spirometry measures volume against time (FEV1, FVC) and flow against volume. The 2022 ATS/ERS interpretation standard uses GLI reference equations, z-scores (LLN = z −1.645) and grades severity by FEV1 z-score.

Indications and contraindications

Indications	Relative contraindications
Diagnose obstructive/restrictive disease; quantify severity	MI or ACS within 1 week; unstable cardiovascular status
Monitor disease and drug effect (asthma, COPD, ILD, NM disease)	Recent eye, thoracic, abdominal or brain surgery
Pre-operative assessment, disability, occupational surveillance	Pneumothorax, haemoptysis of unknown origin, large aneurysm
Screening smokers; drug toxicity (amiodarone, bleomycin)	Active TB/respiratory infection (infection control), late pregnancy, confusion

Pre-test instructions and manoeuvres

- Avoid smoking 1 h, vigorous exercise 1 h, heavy meal 2 h; withhold SABA 4–6 h, LABA 24–36 h, LAMA 36–48 h if testing reversibility; tight clothing removed; sit upright, nose clip.
- **FVC manoeuvre:** rapid full inspiration to TLC → maximal blast → continue ≥6 s (or plateau) → rapid full inspiration (loop).
- **Acceptability:** back-extrapolated volume <5% of FVC or 100 mL; no cough in first second; no leak or obstruction; end-of-forced-expiration criteria. **Repeatability:** best two FVC and FEV1 within 150 mL (100 mL if FVC <1 L); up to 8 attempts.
- **Bronchodilator response (2022):** >10% of predicted value change in FEV1 or FVC (replaces the 12% and 200 mL rule).
- **Slow vital capacity (SVC):** relaxed full expiration — larger than FVC in obstruction (airway collapse during forced expiration); used for FEV1/VC ratio and IC.

INVESTIGATIONS & TREATMENT AT A GLANCE

2.1 Lung Volumes & Capacities and Their Measurement

‹ Back to topic (p 22)
Next topic 2.2 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Total lung capacity (TLC)	80–120% predicted (≈ 6 L adult male); LLN by GLI z-score > −1.645	< LLN	Restriction (confirm restriction only by TLC)
Functional residual capacity (FRC)	~ 2.4 L; 75–120% predicted	> 120%	Hyperinflation (COPD, asthma)
Residual volume (RV)	~ 1.2 L; 65–120% predicted	> 140%	Air trapping
RV/TLC ratio	20–35% (rises with age, < 40%)	> 40%	Air trapping
Vital capacity (VC)	~ 4.8 L; > 80% predicted	< 80% / LLN	Restriction or obstruction with trapping
Body plethysmography vs He dilution	Agree within 10%	Plethysmography > dilution	Non-communicating air spaces (bullae)

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Bronchodilator (salbutamol) before PFT	400 µg via spacer, test after 15 min	Assess reversibility	Withhold SABA 4–6 h, LABA 24–36 h, LAMA 36–48 h before baseline test
Ipratropium (reversibility test)	160 µg via spacer, test after 30 min	Alternative bronchodilator	Glaucoma, urinary retention
Supplemental O_2 during testing	Titrate to SpO_2 ≥ 90%	Hypoxaemic patients	Note FiO_2 on report

★ **PRESCRIBING & INTERPRETATION PEARLS**

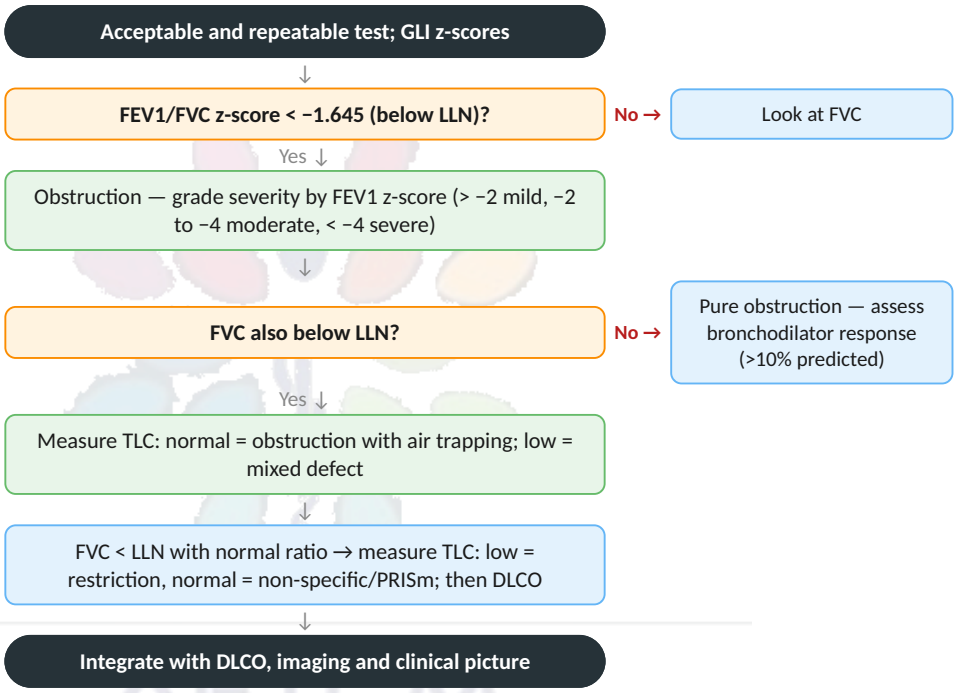
- Restriction is proven by TLC < LLN — a low FVC with normal FEV1/FVC may be air trapping or poor effort.

Spirometric patterns

Pattern	FEV1/FVC	FVC	Other
Obstruction	<LLN (GOLD uses <0.70 post-BD)	Normal or ↓ (air trapping)	Scooped expiratory loop; ↑ RV, TLC
Restriction	Normal or ↑	↓	Confirm TLC <LLN; tall narrow loop
Mixed	<LLN	↓	TLC <LLN with obstruction
PRISm (preserved ratio impaired spirometry)	≥0.70 / ≥LLN	FEV1 <80% (or <LLN)	10–20% of population; obesity, early COPD; higher mortality; 20–30% progress to COPD
Non-specific	Normal	↓	Normal TLC — often obesity, early airway disease

Interpretation algorithm

Algorithm 2.1 · Interpretation of spirometry (ATS/ERS 2022)



Flow–volume loops in upper airway obstruction

Central/upper airway obstruction patterns

Type	Loop shape	Examples	FEF50/FIF50
Fixed (intra- or extrathoracic)	Flattening of both inspiratory and expiratory limbs (box)	Tracheal stenosis post-intubation, goitre encircling trachea	≈1
Variable extrathoracic	Inspiratory limb flattened (negative intraluminal pressure collapses airway)	Vocal cord paralysis, VCD, laryngeal tumour, OSA	>1 (often >2)
Variable intrathoracic	Expiratory limb flattened (positive pleural pressure compresses airway)	Tracheomalacia, tumour of lower trachea/ main bronchus	<0.3

- Empey index FEV1 (mL)/PEF (L/min) >10 suggests UAO; PEF disproportionately low compared with FEV1. Spirometry detects UAO only when lumen <8–10 mm — CT and bronchoscopy are needed.

Other indices

- **FEF25–75 (MMEF):** effort-independent mid-flows, variable; reduced early in small airway disease but not recommended for diagnosis.
- **FEV1/FEV6** surrogate for FEV1/FVC in office spirometry; **PEF** for home monitoring.
- **Office (handheld) spirometry:** screening in primary care (COPD-6, PIKO-6), case-finding; must still meet quality standards.

✦ RECENT ADVANCES & GUIDELINE UPDATES

- ATS/ERS 2022 interpretation: GLI-2012 (and race-neutral GLI Global 2022) equations, z-scores, severity by FEV1 z-score, BDR >10% of predicted.
- GOLD 2025 recognises PRISm and "pre-COPD" as at-risk states needing follow-up.

★ CLINICAL PEARLS & EXAM FOCUS

- Draw three flow-volume loops (fixed, variable extra-, variable intrathoracic) — one of the most repeated short notes.

LANDMARK TRIALS & GUIDELINES

- Stanojevic S et al, ERS/ATS technical standard on interpretive strategies, Eur Respir J 2022; Graham BL et al, Standardization of spirometry 2019 update.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Slow vital capacity. *[DNB Oct 2024 P1]*
2. Interpretation of spirometry in obstructive and restrictive lung disease. *[DNB Oct 2024 P1]*
3. Office spirometry. *[DNB Oct 2023 P1]*
4. Flow volume loops in diagnosis of upper airway diseases. *[DNB Oct 2023 P1]*
5. Describe indications, contraindications, pre-procedure instructions and different manoeuvres for spirometry. *[DNB May 2024 P2]*
6. Draw and describe important features of flow volume loops. How are they useful in differentiating airway obstruction at different levels? *[WBUHS 2022 P1]*
7. Write a short note on indications and contraindications of spirometry. *[BFUHS 2020 P1]*
8. Characteristic patterns of disordered respiratory function. *[DNB Jun 2025 P4]*

2.3 Diffusion & DLCO

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-B1 Assessment of pulmonary functions
- ✓ Explain Fick's law and the factors affecting diffusion across the alveolar–capillary membrane
- ✓ Describe methods of DLCO measurement and the factors that influence it
- ✓ Interpret patterns of DLCO and KCO in disease

IN BRIEF DLCO (TLCO) measures the transfer of CO from alveolar gas to haemoglobin. It depends on the membrane (DM), capillary blood volume (Vc) and haemoglobin: 1/DL = 1/DM + 1/(θ·Vc). Normal ~25–30 mL/min/mmHg.

Diffusion of gases

- **Fick's law:** $\dot{V}gas = (A \times D \times (P1 - P2)) / T$, where D ∝ solubility/√MW. CO2 diffuses ~20× faster than O2 (much higher solubility) — diffusion limitation rarely causes hypercapnia.
- Capillary transit time ~0.75 s; O2 equilibrates in 0.25 s — reserve allows normal saturation at rest even with thickened membrane; limitation appears with exercise, altitude (low driving pressure).
- **Perfusion-limited** gases (N2O, O2 normally) vs **diffusion-limited** (CO). CO is used because Hb's high affinity keeps capillary PCO ≈0.

INVESTIGATIONS & TREATMENT AT A GLANCE

2.2 Spirometry & Flow–Volume Loops

‹ Back to topic (p 24)
Next topic 2.3 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
FEV1/FVC	> LLN (≈ > 0.70 in adults; GOLD fixed ratio 0.70)	< LLN (or < 0.70 post-BD for GOLD)	Airflow obstruction
FEV1 (% predicted / z-score)	> 80%; z > −1.645	z −1.65 to −2.5 mild; −2.5 to −4 moderate; < −4 severe (ATS/ERS 2022)	Grades severity
Bronchodilator response	Change < 10% of predicted FEV1/FVC (ATS/ERS 2022)	> 10% of predicted value	Significant reversibility (asthma likely); old: ≥ 12% and ≥ 200 mL
FVC	> 80% predicted / > LLN	< LLN with normal ratio	Possible restriction or PRISm — confirm with TLC
FEF25–75	> 60–65% predicted	Reduced	Small-airway dysfunction (variable, non-specific)
Peak expiratory flow	Within 20% of predicted; diurnal variation < 10%	Diurnal variability > 10% (adults)	Variable airflow — asthma
Flow–volume loop shape	Smooth triangular expiratory, semicircular inspiratory	Scooped (obstruction); flattened both limbs (fixed UAO); flattened inspiratory (variable extrathoracic)	Pattern recognition
Empey index	FEV1 (mL)/PEF (L/min) < 10	> 10	Upper airway obstruction

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Salbutamol (reversibility)	400 µg MDI + spacer	Bronchodilator response testing	Tachycardia, tremor
Prednisolone trial	30–40 mg daily × 2 weeks	Steroid-reversibility in uncertain asthma/COPD	Hyperglycaemia; FeNO/eosinophils preferred
Methacholine (bronchoprovocation)	Doubling doses to PD20	Suspected asthma with normal spirometry	Not if FEV1 < 60% or < 1.5 L

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Contraindications to spirometry: MI within 1 week, recent eye/thoracic/abdominal surgery, pneumothorax, haemoptysis, aortic aneurysm > 6 cm.

Methods of measurement

Method	Principle	Comment
Single-breath (breath-hold) — standard	Inhale 0.3% CO + tracer (He/CH4) to TLC, hold 10 s, analyse exhaled alveolar sample	VA from tracer dilution; KCO = DLCO/VA; correct for Hb (and COHb, altitude)
Steady state	Breathe low CO for minutes	Exercise testing; affected by V/Q
Rebreathing / intrabreath	Continuous analysis	Patients unable to breath-hold
DLNO–DLCO	NO transfer reflects DM	Partitions membrane vs blood volume

Causes of altered DLCO

Low DLCO	High DLCO
ILD, pulmonary fibrosis (↓ area, ↑ thickness)	Polycythaemia
Emphysema (↓ surface area) — helps differentiate from asthma	Pulmonary haemorrhage (DAH) — serial rise
Pulmonary vascular disease (PAH, CTEPH, PE) — isolated low DLCO	Left-to-right shunt, early CHF (↑ Vc)
Anaemia, raised COHb (smokers)	Asthma (mildly), obesity
Loss of lung volume (pneumonectomy — VA low, KCO high)	Exercise, supine posture

KCO interpretation

- Low VA with high/normal KCO → extraparenchymal restriction (chest wall, neuromuscular, effusion, pneumonectomy).
- Low VA with low KCO → parenchymal disease (ILD, emphysema).
- Normal VA with low KCO → pulmonary vascular disease, anaemia, early emphysema/ILD.

★ CLINICAL PEARLS & EXAM FOCUS

- Isolated reduction in DLCO with normal spirometry/volumes = pulmonary vascular disease (PAH, CTEPH), early ILD, emphysema, anaemia — order echo and CTPA/V/Q.
- DLCO <40% predicted is a high-risk marker before lung resection (ppo-DLCO <30–40%).

LANDMARK TRIALS & GUIDELINES

- ERS/ATS standards for single-breath carbon monoxide uptake in the lung, Eur Respir J 2017.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Factors affecting diffusion of gases from alveolus to pulmonary circulation. *[DNB Jun 2025 P1]*
2. Diffusing capacity for carbon monoxide (DLCO). *[DNB Jun 2025 P1]*
3. Diffusion capacity of carbon monoxide (DLCO). *[DNB Oct 2024 P1]*
4. Significance of changes in DLCO. *[DNB Dec 2021 P1]*
5. Methods to measure and factors influencing DLCO. *[DNB Oct 2023 P4]*
6. Define DLCO. How is it measured? What factors influence it? What is its clinical utility? *[WBUHS 2022 P1]*
7. Mechanism of gas exchange at the alveolar level. *[DNB Jun 2021 P1]*

INVESTIGATIONS & TREATMENT AT A GLANCE

2.3 Diffusion & DLCO

‹ Back to topic (p 26)
Next topic 2.4 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
DLCO (% predicted)	80–120% (LLN by GLI 2017)	Mild 60–LLN; moderate 40–60%; severe < 40%	Gas-exchange impairment
KCO (DLCO/VA)	80–120%	Low	Parenchymal/vascular disease (emphysema, ILD, PVD)
KCO high with low VA	—	KCO > 120%	Extraparenchymal restriction (neuromuscular, chest wall, pneumonectomy)
DLCO raised	—	> 140%	Alveolar haemorrhage, polycythaemia, asthma, obesity, L→R shunt
Haemoglobin correction	Hb 13.4 g/dL (men), 12 (women) reference	Anaemia lowers DLCO	Always correct DLCO for Hb
Isolated low DLCO with normal spirometry	—	DLCO < LLN	Pulmonary vascular disease (PAH, CTEPH), early ILD, emphysema

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Smoking abstinence before test	No smoking for 24 h	Accurate DLCO (COHb lowers DLCO)	Record COHb if possible
Oxygen	Titrate to SpO_2 88–92% (COPD) or 94–98%	Exertional desaturation with low DLCO	Assess with 6MWT
Treat cause	Antifibrotics (ILD), PAH therapy, anaemia correction	Low DLCO disorders	See topic-specific tables

★ **PRESCRIBING & INTERPRETATION PEARLS**

- DLCO < 40% predicted predicts exertional desaturation and is a red flag for PH and post-resection risk.

2.4 Respiratory Mechanics — Compliance, Resistance, Surfactant & Atelectasis

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-B2 Pulmonary mechanics
- ✓ Define static and dynamic compliance and describe their measurement and clinical relevance
- ✓ Explain airway resistance, dynamic compression, equal pressure point and hysteresis
- ✓ Describe surfactant composition and functions; classify atelectasis

IN BRIEF Breathing must overcome elastic recoil (compliance) and flow resistance. Surfactant lowers surface tension, stabilises alveoli and prevents oedema; loss of either property leads to collapse, stiff lungs and increased work of breathing.

Compliance

- Compliance = ΔV/ΔP; lung ~200 mL/cmH2O, chest wall ~200, total respiratory system ~100 mL/cmH2O. Specific compliance = C/FRC.
- **Static compliance** — measured during no flow: oesophageal balloon (pleural pressure) at several volumes, or in ventilated patients **Cstat = VT / (Pplat − PEEP)** (normal 60–100 mL/cmH2O).
- **Dynamic compliance** = VT / (PIP − PEEP) — includes resistance; frequency dependence of compliance (fall at high RR) is a sign of small airway disease.
- **Reduced:** fibrosis, oedema, ARDS, atelectasis, pneumonectomy; chest wall — obesity, kyphoscoliosis, ascites. **Increased:** emphysema (loss of elastic tissue), ageing.
- **Hysteresis:** the inflation and deflation P–V curves differ (deflation volume higher at same pressure) due to surface tension forces (surfactant) and alveolar recruitment–derecruitment; much less when lungs are saline-filled.

Airway resistance

- Raw = (Palv − Pmouth)/flow ≈ 0.5–2 cmH2O/L/s. Poiseuille: $R \propto 8\eta l/\pi r^4$ — halving radius increases resistance 16-fold.
- **Site:** 80% in large and medium bronchi (turbulent flow); small airways <2 mm contribute <20% — the "quiet zone" where disease may be silent on spirometry.
- **Factors:** lung volume (resistance falls as volume rises — radial traction), bronchial smooth-muscle tone (vagal, β2, histamine, leukotrienes), mucus, oedema, gas density (heliox reduces turbulent resistance).
- **Measurement:** body plethysmography (sRaw), forced oscillation/IOS, interrupter technique; in ventilated patient (PIP − Pplat)/flow.

Dynamic compression and the equal pressure point

- During forced expiration, pleural pressure becomes positive; pressure inside the airway falls along its length. At the **equal pressure point (EPP)** intraluminal = pleural pressure; downstream the airway is compressed — flow becomes **effort-independent** (determined by elastic recoil and upstream resistance).
- In emphysema, loss of recoil moves EPP towards alveoli → early collapse, air trapping, scooped loop; pursed-lip breathing moves EPP proximally.

Surfactant

- **Composition:** 90% lipid (dipalmitoyl phosphatidylcholine ~50%, phosphatidylglycerol), 10% proteins: SP-A and SP-D (hydrophilic, collectins — innate immunity), SP-B and SP-C (hydrophobic — spreading, film stability).
- **Functions:** lowers surface tension (to ~0–5 mN/m at low volume) → increases compliance, reduces work of breathing; stabilises alveoli of different sizes (Laplace P = 2T/r); keeps alveoli dry (reduces transudation); opsonisation and host defence.
- **Clinical:** neonatal RDS (exogenous surfactant); ARDS (inactivation by plasma proteins); genetic SP-B/SP-C/ABCA3 deficiency (childhood ILD); PAP (failure of clearance).

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Types of acquired atelectasis

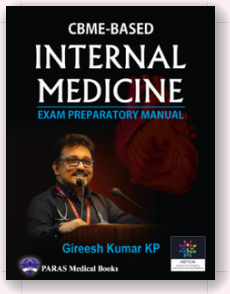
Type	Mechanism	Examples
Resorptive (obstructive)	Airway obstruction; gas absorbed distally (faster with 100% O2)	Mucus plug, tumour, foreign body
Passive (relaxation)	Loss of contact between pleura — lung recoils	Pneumothorax, effusion
Compressive	Space-occupying lesion pushes lung	Large bulla, tumour, massive effusion
Adhesive	Surfactant deficiency/inactivation	Neonatal RDS, ARDS, post-cardiopulmonary bypass, radiation
Cicatrisation	Fibrosis contracts lung	Post-TB, fibrosis
Gravity-dependent / round atelectasis	Dependent regions; pleural folding	Anaesthesia; asbestos-related round atelectasis

★ CLINICAL PEARLS & EXAM FOCUS

- Ventilated patient with high PIP but normal Pplat = airway problem (bronchospasm, secretion, kinked tube); both high = compliance problem (pneumothorax, oedema, intubated right main bronchus).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. What is lung compliance and methods of measuring lung compliance? *[DNB Oct 2023 P1]*
2. Measurement of compliance. *[DNB Dec 2021 P1]*
3. Assessment of lung compliance. *[DNB Jun 2021 P1]*
4. Airways resistance. *[DNB Dec 2021 P1]*
5. Measurement of airway resistance. *[BFUHS 2019 P2]*
6. Dynamic compression of the airways. *[DNB Dec 2021 P1]*
7. What is surfactant? Discuss its clinical utility. *[WBUHS 2022 P1]*
8. Write a short note on surfactant. *[BFUHS 2022 P1]*



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2.5 Ventilation, Dead Space, V/Q Matching & Mechanisms of Hypoxaemia

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-B3 Ventilation and gas exchange
- ✓ Calculate alveolar PO2, A–a gradient, dead space (Bohr) and shunt fraction
- ✓ Describe regional ventilation and perfusion, and causes and effects of V/Q mismatch
- ✓ Classify mechanisms of hypoxaemia and distinguish them at the bedside

IN BRIEF Five mechanisms cause hypoxaemia — low inspired PO2, hypoventilation, V/Q mismatch, shunt and diffusion limitation. A normal A–a gradient points to hypoventilation or low FiO2; failure to correct with 100% O2 points to shunt.

INVESTIGATIONS & TREATMENT AT A GLANCE

2.4 Respiratory Mechanics — Compliance, Resistance, Surfactant & Atelectasis

‹ Back to topic (p 28)
Next topic 2.5 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Static compliance (ventilated)	50–100 mL/cmH_2O (Vt ÷ [Pplat − PEEP])	< 40 mL/cmH_2O	Stiff lungs (ARDS, fibrosis, oedema)
Airway resistance (Raw, plethysmography)	0.6–2.4 cmH_2O/L/s	Raised	Obstructive airway disease
Peak − plateau pressure gap	< 10 cmH_2O	> 10 cmH_2O	High airway resistance (bronchospasm, secretions, tube kink)
Plateau pressure	< 30 cmH_2O	> 30 cmH_2O	Over-distension risk — reduce Vt
Driving pressure (Pplat − PEEP)	< 15 cmH_2O	> 15 cmH_2O	Associated with mortality in ARDS
CXR — atelectasis	Normal	Displaced fissure, crowded vessels, elevated hemidiaphragm	Lobar collapse
Lecithin/sphingomyelin ratio	> 2.0	< 1.5	Surfactant deficiency (neonatal RDS)

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Chest physiotherapy & incentive spirometry	10 breaths hourly while awake	Post-operative atelectasis prevention	Pain control needed
Recruitment manoeuvre / PEEP titration	PEEP 5–15 cmH_2O per table	Ventilated atelectasis/ARDS	Barotrauma, hypotension
Therapeutic bronchoscopy	Procedure	Lobar collapse from mucus plug not resolving	Hypoxaemia
Surfactant	100–200 mg/kg intratracheal	Neonatal RDS	Not effective in adult ARDS

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Obstructive atelectasis shifts the mediastinum towards the lesion; compressive atelectasis (large effusion) shifts it away.

Key equations

- **Alveolar gas equation:** PAO2 = FiO2 (PB − PH2O) − PaCO2/R = 0.21 × (760 − 47) − PaCO2/0.8 ≈ 150 − 1.25 × PaCO2 ≈ 100 mmHg at sea level.
- **A–a gradient** normal 5–10 mmHg (young); age/4 + 4; widens with FiO2. PaO2/FiO2 ratio normal >400.
- **Bohr dead space:** VD/VT = (PaCO2 − PECO2)/PaCO2 ≈ 0.25–0.35; anatomical (Fowler's N2 method) ~150 mL; physiological = anatomical + alveolar dead space.
- **Shunt equation:** Qs/Qt = (CcO2 − CaO2)/(CcO2 − CvO2); normal 2–5% (bronchial and Thebesian veins). Measured on 100% O2.

Regional differences (upright)

- Both ventilation and perfusion increase towards the base, but perfusion more steeply → V/Q ≈3 at apex (high PAO2 — TB reactivation), ≈0.6 at base; overall 0.8.
- West zones (see 2.15).

Mechanisms of hypoxaemia

Mechanism	PaCO2	A–a gradient	Response to 100% O2	Examples
Low FiO2 / altitude	↓ (hyperventilation)	Normal	Corrects	High altitude, aircraft
Hypoventilation	↑	Normal	Corrects	Opioids, NM disease, OHS, chest wall
V/Q mismatch	Normal/↓ (↑ late)	↑	Corrects	COPD, asthma, PE, pneumonia — commonest
Shunt (V/Q = 0)	Normal/↓	↑	Poor correction (shunt >30%)	ARDS, lobar pneumonia, atelectasis, intracardiac/ PAVM
Diffusion limitation	Normal/↓	↑ (exercise)	Corrects	ILD, emphysema on exertion, altitude

Effects of V/Q mismatch

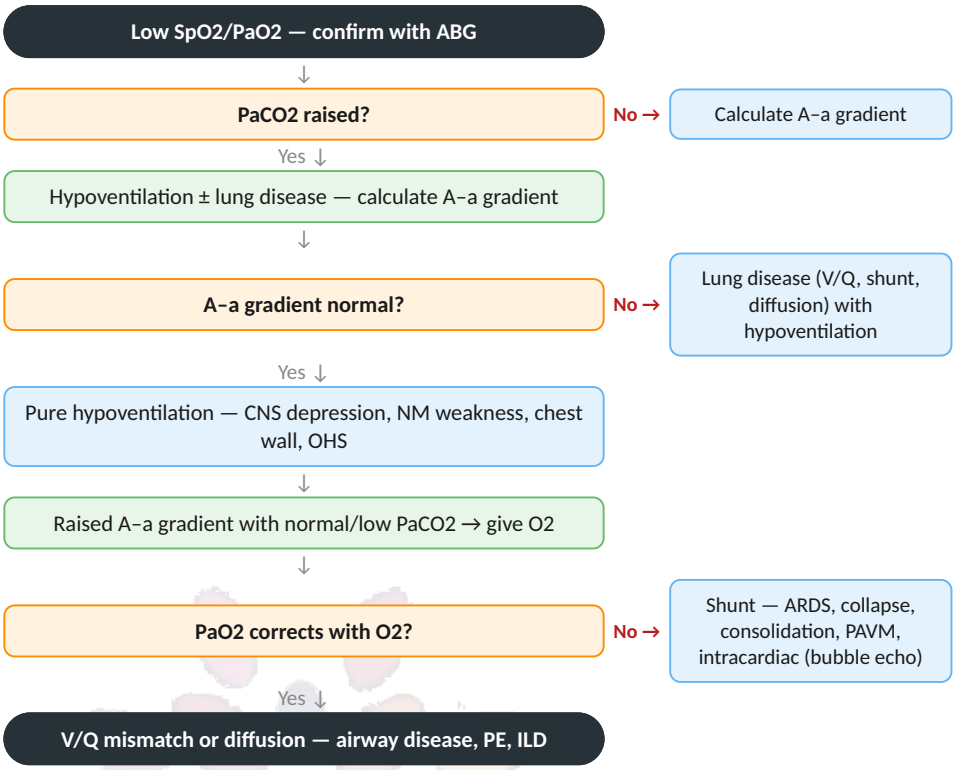
- Low V/Q units → hypoxaemia that cannot be compensated by high V/Q units (flat top of ODC); CO2 is compensated by increased ventilation (linear CO2 curve) until ventilatory capacity is exceeded.
- High V/Q units → wasted ventilation (dead space) — PE, emphysema, positive pressure ventilation, hypovolaemia.
- Measured by MIGET (multiple inert gas elimination), V/Q scintigraphy, EIT.

Hypoxia types

- **Hypoxaemic** (low PaO2), **anaemic** (low Hb, CO poisoning), **stagnant/circulatory** (low cardiac output), **histotoxic** (cyanide — cells cannot use O2; high venous O2).

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Algorithm 2.2 · Bedside approach to hypoxaemia



CO2 narcosis in COPD

- Uncontrolled high-flow O2 in hypercapnic COPD raises PaCO2 via loss of hypoxic pulmonary vasoconstriction (↑ dead-space ventilation — main mechanism), the Haldane effect, and to a lesser extent reduced hypoxic drive.
- Target SpO2 88–92%, controlled O2 (Venturi 24–28%), NIV if pH <7.35 with PaCO2 >45 mmHg.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Normal A–a gradient + high PaCO2 = pure hypoventilation — "the lungs are innocent".

LANDMARK TRIALS & GUIDELINES

- Abdo WF, Heunks LMA. Oxygen-induced hypercapnia in COPD: myths and facts. Crit Care 2012; BTS emergency oxygen guideline 2017.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Describe the causes of ventilation-perfusion (VA/Q) mismatch and its effects. *[DNB Jun 2025 P1]*
2. Alveolar air equation. *[DNB Jun 2025 P1]*
3. Measurement of physiological shunt. *[DNB Jun 2025 P1]*
4. Causes of hypoxaemia. *[DNB Jun 2025 P1]*
5. Alveolar–arterial gradient. *[DNB Dec 2022 P1]*
6. Dead space. *[DNB Dec 2022 P1]*
7. Pathophysiology of arterial hypoxemia. *[DNB Oct 2024 P3]*
8. Describe the pathophysiological mechanisms of hypoxia. *[BFUHS 2022 P1]*

INVESTIGATIONS & TREATMENT AT A GLANCE

2.5 Ventilation, Dead Space, V/Q Matching & Mechanisms of Hypoxaemia

‹ Back to topic (p 29)
Next topic 2.6 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
PaO_2 (room air, sea level)	80–100 mmHg (≈ 100 − age/3)	< 60 mmHg	Hypoxaemia; < 60 = respiratory failure
A–a gradient	(age/4) + 4 mmHg; < 15–20 in young	Raised	V/Q mismatch, shunt or diffusion defect (not hypoventilation)
PaO_2/FiO_2 ratio	> 400	≤ 300 mild, ≤ 200 moderate, ≤ 100 severe (ARDS)	Oxygenation index
Dead-space fraction (Vd/Vt)	0.2–0.35	> 0.6	Severe ARDS/PE — high mortality
Shunt (100% O_2 response)	Rise in PaO_2 to > 500 mmHg	PaO_2 fails to rise	True shunt (> 25–30%)
$PaCO_2$	35–45 mmHg	> 45 mmHg	Alveolar hypoventilation

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Controlled oxygen	Target SpO_2 94–98% (88–92% if hypercapnia risk)	Hypoxaemia	Avoid hyperoxia; reassess ABG in CO_2 retainers
High-flow nasal oxygen	40–60 L/min, FiO_2 titrated	Hypoxaemic respiratory failure	ROX index for failure
Treat mechanism	PEEP (shunt), bronchodilators (V/Q), NIV (hypoventilation)	Mechanism-specific	—

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Hypoventilation: normal A–a gradient with raised $PaCO_2$; shunt: no response to 100% oxygen.

2.6 Gas Transport — Oxygen and Carbon Dioxide Dissociation Curves

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-B3 Gas transport
- ✓ Draw the oxyhaemoglobin dissociation curve with landmarks and factors shifting it
- ✓ Describe CO2 transport and the CO2 dissociation curve; Bohr and Haldane effects
- ✓ Apply to clinical situations — CO poisoning, methaemoglobinaemia, transfusion, altitude

IN BRIEF O2 is carried 98.5% bound to Hb (1.34 mL/g) and 1.5% dissolved (0.003 mL/dL/mmHg). The sigmoid ODC protects saturation at moderate falls in PO2 and unloads O2 in tissues; CO2 travels mainly as bicarbonate.

Oxygen content and delivery

- CaO2 = (1.34 × Hb × SaO2) + (0.003 × PaO2) ≈ 20 mL/dL; DO2 = CO × CaO2 × 10 ≈ 1000 mL/min; VO2 ≈ 250 mL/min; extraction ratio ~25%.

Oxyhaemoglobin dissociation curve

ODC landmarks

Point	PO2 (mmHg)	SaO2
Arterial	100	97–98%
Flat-portion knee	60	90%
Mixed venous	40	75%
P50	26.7	50%

- **Shape:** sigmoid due to cooperative binding (T → R state); flat upper part ensures loading despite PAO2 fall; steep part facilitates unloading.

Factors shifting the ODC

Right shift (↑ P50, ↓ affinity, unloading)	Left shift (↓ P50, ↑ affinity)
↑ H+ (↓ pH) — Bohr effect	Alkalosis
↑ PCO2	↓ PCO2
↑ Temperature	Hypothermia
↑ 2,3-DPG (anaemia, altitude, hypoxia, thyroid)	↓ 2,3-DPG (stored blood)
Exercise, HbS	Fetal Hb, CO (COHb), methaemoglobin, myoglobin (hyperbola)

- **CO poisoning:** affinity 240× O2, reduces carrying capacity and left-shifts curve; SpO2 falsely normal — need co-oximetry. **MetHb:** SpO2 settles ~85%; chocolate blood; methylene blue.

CO2 transport

- Bicarbonate 70% (carbonic anhydrase in RBC; chloride shift — Hamburger), carbamino compounds 23%, dissolved 7%.
- **CO2 dissociation curve** is nearly linear in the physiological range and steeper than ODC; venous and arterial content differ ~4 mL/dL.
- **Haldane effect:** deoxygenated Hb carries more CO2 (better buffer and carbamino formation) — O2 loading in lungs releases CO2; contributes to O2-induced hypercapnia in COPD.
- **Bohr effect:** CO2/H+ reduce Hb O2 affinity — facilitates unloading in tissues; "double Bohr" in placenta.

- **Factors affecting CO2 curve:** oxygen saturation (Haldane), Hb concentration, temperature, pH.

★ **CLINICAL PEARLS & EXAM FOCUS**

▸ Draw ODC with P50, arterial and venous points and arrows for shifts — and state "Bohr: CO2 affects O2 carriage; Haldane: O2 affects CO2 carriage".

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Oxygen dissociation curve. *[DNB Oct 2024 P1]*
2. Oxygen haemoglobin dissociation curve. *[DNB Dec 2022 P1; BFUHS 2020 P1]*
3. Draw and describe oxyhemoglobin dissociation curve. Explain its clinical implications. *[WBUHS 2022 P1]*
4. Factors affecting carbon dioxide dissociation curve. *[DNB Dec 2022 P1]*
5. Carbon dioxide transport. *[DNB Dec 2021 P1]*
6. Blood gas transport in the lung parenchyma. *[DNB Oct 2023 P1]*
7. Define Bohr and Haldane effect. *[BFUHS 2019 P1]*

2.7 Control of Ventilation, Chemoreceptors & Reflexes

‹ NMC curriculum
Previous-year Qs ›

◉ **CBME COMPETENCIES — the postgraduate should be able to:**

- ✓ **NMC syllabus area:** I-B2, I-B9 Control of ventilation, innervation and reflexes
- ✓ Describe the respiratory centres, central and peripheral chemoreceptors and their responses
- ✓ Describe pulmonary reflexes — stretch, J-receptor, cough — and loop gain
- ✓ Explain periodic breathing (Cheyne–Stokes) and hyperventilation syndromes

IN BRIEF Breathing is generated by a central pattern generator in the medulla (pre-Bötzinger complex), modulated by chemoreceptors (CO2/H+, O2), mechanoreceptors, and the cortex. Ventilatory control instability (high loop gain) underlies central apnoea and Cheyne–Stokes respiration.

Components of the ventilatory control system

Control system

Component	Structure	Function
Controller	Pre-Bötzinger complex (rhythm), dorsal respiratory group (NTS, inspiratory), ventral respiratory group (inspiratory & expiratory), pontine respiratory group (pneumotaxic — inspiratory off-switch)	Rhythm and pattern generation
Cortex / limbic	Voluntary, behavioural, emotional control	Speech, breath-holding; wakefulness drive
Sensors	Central and peripheral chemoreceptors; lung stretch, irritant, J-receptors; muscle spindles	Feedback
Effectors	Phrenic, intercostal, upper-airway motor neurones; respiratory muscles	Ventilation

Chemoreceptors

Feature	Central	Peripheral (carotid & aortic bodies)
Location	Ventrolateral medulla (retrotrapezoid nucleus)	Carotid bifurcation (glomus type I cells), aortic arch
Stimulus	H+ in CSF (from PaCO2)	↓ PaO2 (<60 mmHg), ↑ PaCO2, ↓ pH
Contribution	~70–80% of CO2 response; slower	All of hypoxic response; fast; ~20–30% CO2 response
Nerve	—	Carotid sinus nerve (IX); aortic (X)

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INVESTIGATIONS & TREATMENT AT A GLANCE

2.6 Gas Transport — Oxygen and Carbon Dioxide Dissociation Curves

‹ Back to topic (p 32)
Next topic 2.7 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
SpO_2 (pulse oximetry)	95–100%	< 94% (target-dependent)	Hypoxaemia; unreliable in CO poisoning, methaemoglobinaemia, dark skin
P50	26.6 mmHg	Low (left shift: alkalosis, ↓ temp, ↓ 2,3-DPG, HbF, COHb) / high (right shift)	Affinity change
Carboxyhaemoglobin (co-oximetry)	< 3% (non-smokers), < 10% smokers	> 10–25%; > 25% severe	CO poisoning — SpO_2 falsely normal
Methaemoglobin	< 1.5%	> 10% cyanosis; > 30% symptomatic; > 70% fatal	"Saturation gap"; chocolate-brown blood
Oxygen content (CaO_2)	18–20 mL/dL	Low	$(1.34 \times Hb \times SaO_2) + 0.003 \times PaO_2$
$ScvO_2$	70–80%	< 70%	Inadequate oxygen delivery

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
100% oxygen via non-rebreather	15 L/min	CO poisoning — half-life of COHb ~ 90 min (vs 300 min in air)	Continue until COHb < 3%
Hyperbaric oxygen	2.5–3 ATA	CO poisoning with LOC, COHb > 25%, pregnancy > 15%, cardiac ischaemia	Barotrauma, availability
Methylene blue	1–2 mg/kg IV over 5 min (repeat at 1 h)	Methaemoglobinaemia > 20–30% or symptomatic	Contraindicated in G6PD deficiency (use ascorbic acid/exchange); serotonin syndrome with SSRIs
Red cell transfusion	Target Hb 7–9 g/dL	Severe anaemia limiting O_2 delivery	Volume overload

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Pulse oximetry reads ~ 85% in methaemoglobinaemia regardless of true saturation — demand co-oximetry.

Feature	Central	Peripheral (carotid & aortic bodies)
Adaptation	CSF bicarbonate correction in chronic hypercapnia	Hypoxic drive maintained; blunted after carotid endarterectomy

- **Ventilatory responses:** hypercapnic ventilatory response ~2–5 L/min/mmHg (linear); hypoxic response hyperbolic, sharply above PaO2 <60; synergistic (hypoxia + hypercapnia).
- **Chemical factors** affecting respiration: PaCO2 (main), H+, PaO2; also temperature, catecholamines, progesterone (pregnancy), drugs (opioids, benzodiazepines depress; doxapram, almitrine stimulate).

Pulmonary receptors and reflexes

Pulmonary reflexes

Receptor	Location / fibre	Stimulus	Response
Slowly adapting stretch (SAR)	Airway smooth muscle, myelinated vagal	Lung inflation	Hering–Breuer inflation reflex — terminates inspiration (weak in adults)
Rapidly adapting (irritant, RAR)	Epithelium, myelinated	Dust, smoke, histamine, rapid inflation	Cough, bronchoconstriction, Head's paradoxical reflex, sighs
J (juxtacapillary) receptors	Alveolar wall, unmyelinated C-fibres	Interstitial oedema, emboli, capsaicin	Rapid shallow breathing, dyspnoea, apnoea, bradycardia (pulmonary chemoreflex)
Bronchial C-fibres	Airway, unmyelinated	Chemical irritants, inflammation	Cough, mucus secretion, bronchoconstriction
Chest-wall spindles	Intercostals	Length–tension mismatch	Dyspnoea sensation

Cough reflex

- **Afferent:** RARs (larynx, trachea, carina — mechanical) and C-fibres (TRPV1, TRPA1 — chemical) via vagus (superior laryngeal, recurrent laryngeal), also glossopharyngeal, trigeminal, phrenic, auricular branch (Arnold's).
- **Centre:** NTS and brainstem cough generator, cortical modulation.
- **Efferent:** phrenic, spinal motor nerves, recurrent laryngeal (glottic closure).
- **Phases:** inspiratory (to ~50% VC) → compressive (glottic closure, intrathoracic pressure to 300 mmHg) → expulsive (glottis opens; flows up to 12 L/s, airway dynamic compression increases linear velocity).
- Cough hypersensitivity syndrome — central/peripheral sensitisation; P2X3 antagonists (gefapixant, camlipixant) in refractory cough.

Loop gain and periodic breathing

- **Loop gain** = controller gain × plant gain — the ratio of the ventilatory response to a disturbance. LG >1 → unstable, oscillating breathing.
- **High controller gain:** heart failure, hypoxia, altitude; **high plant gain:** low FRC, hypercapnia sensitivity near apnoea threshold; **long circulatory delay** (low cardiac output) adds instability.
- **Cheyne–Stokes respiration:** crescendo–decrescendo cycles (45–90 s in HF) with central apnoeas; seen in HFrEF, stroke, altitude, opioids, renal failure. Prognostic in HF. Managed by optimising HF, CPAP; ASV contraindicated if LVEF ≤45% (SERVE-HF).
- Loop gain is one of the four OSA endotypes (see 13.2).

Hyperventilation syndromes

- Breathing in excess of metabolic need → hypocapnic alkalosis: anxiety/panic disorder (dysfunctional breathing), pain, early asthma/PE, sepsis, hepatic encephalopathy, salicylates, pregnancy, altitude, CNS lesions.
- Features: breathlessness at rest, sighing, paraesthesia, carpopedal spasm, chest tightness, dizziness; Nijmegen questionnaire ≥23.
- **Diagnosis of exclusion** — rule out PE, asthma, metabolic acidosis. Treat with breathing retraining (physiotherapy, Buteyko/Papworth), CBT; avoid rebreathing paper bag (hypoxaemia risk).

★ CLINICAL PEARLS & EXAM FOCUS

▸ Peripheral chemoreceptors respond to PaO2, not O2 content — so anaemia and CO poisoning do not stimulate ventilation.

LANDMARK TRIALS & GUIDELINES

• Cowie MR et al, SERVE-HF, NEJM 2015 — ASV increased mortality in HFrEF with central sleep apnoea.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Discuss anatomy and physiology of the ventilatory control system. *[DNB Oct 2024 P1]*
2. Chemoreceptors. *[DNB Oct 2023 P1]*
3. Chemoreceptors for control of breathing. *[NEET-SS/DNB 2024]*
4. Chemical factors affecting respiration. *[DNB Dec 2022 P1]*
5. Cough reflex. *[NEET-SS/DNB 2024]*
6. Cough reflex pathways. *[Manipal 2014 P1]*
7. J receptors. *[Manipal 2014 P1]*
8. Discuss in short about Cheyne–Stokes respiration. *[BFUHS 2019 P1]*

2.8 Acid–Base Balance & ABG Interpretation; Electrolytes

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-B7 Acid–base and electrolyte balance
- ✓ Apply the Henderson–Hasselbalch equation and a stepwise method to ABG interpretation
- ✓ Describe causes, compensation and management of respiratory acidosis and alkalosis and metabolic alkalosis
- ✓ Manage hyponatraemia relevant to pulmonary disease (SIADH)

IN BRIEF pH = 6.1 + log [HCO3−]/(0.03 × PaCO2). The lungs regulate PaCO2 within minutes; the kidneys adjust HCO3− over 3–5 days. Always check internal consistency, the primary disorder, expected compensation and the anion gap.

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INVESTIGATIONS & TREATMENT AT A GLANCE

2.7 Control of Ventilation, Chemoreceptors & Reflexes

‹ Back to topic (p 33)
Next topic 2.8 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Hypercapnic ventilatory response	1–3 L/min per mmHg $PaCO_2$ rise	Blunted	Obesity hypoventilation, COPD "blue bloater", CCHS
Arterial $PaCO_2$ (awake)	35–45 mmHg	> 45 mmHg awake	Chronic hypoventilation
Serum bicarbonate	22–28 mmol/L	> 27 mmol/L	Screening for chronic hypoventilation (OHS)
Polysomnography	AHI < 5/h	Central apnoeas, Cheyne–Stokes, sleep hypoventilation	Control-of-breathing disorders
PHOX2B testing	Normal	Polyalanine expansion	Congenital central hypoventilation syndrome
MRI brainstem	Normal	Lesion	Central hypoventilation, Ondine's curse

Treatment — drug, dose and caution

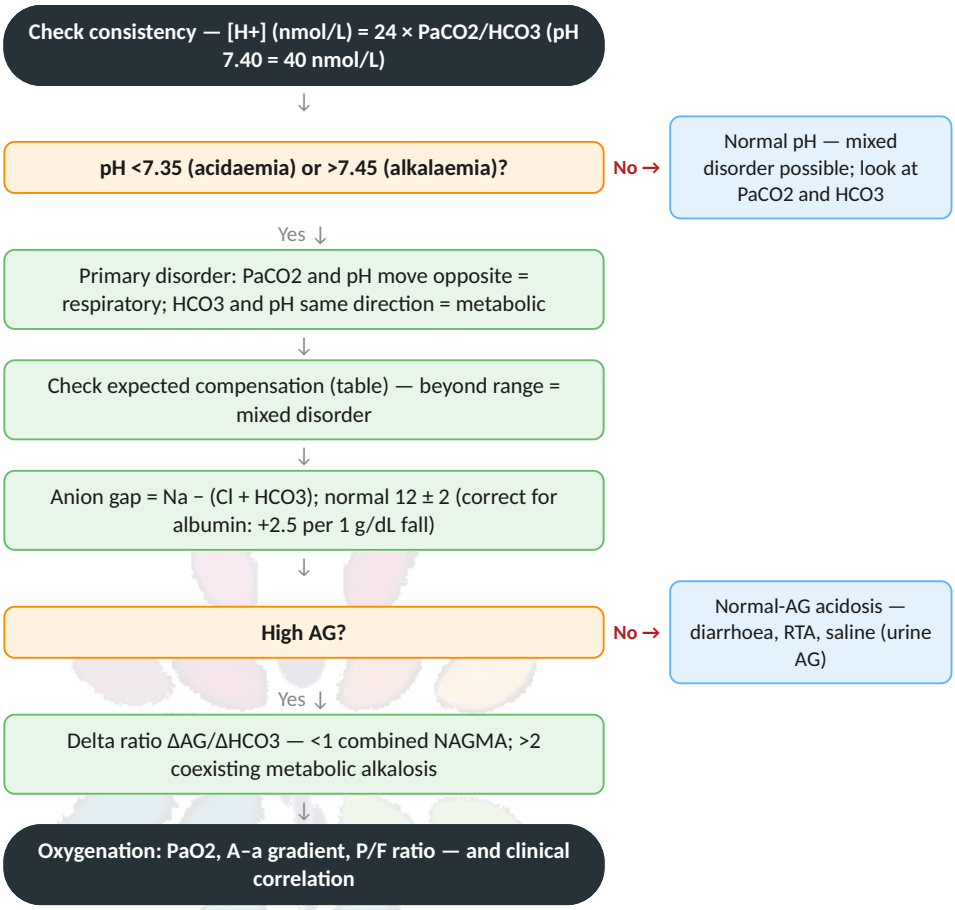
Drug / intervention	Adult dose	Indication	Caution
Naloxone	0.4–2 mg IV, repeat (infusion ⅔ of reversal dose/h)	Opioid respiratory depression	Short half-life — re-narcotisation; withdrawal
Flumazenil	0.2 mg IV, increments to 1 mg	Benzodiazepine overdose (selected)	Seizures in mixed overdose/dependence
Acetazolamide	250 mg twice daily	High-altitude periodic breathing; central apnoea	Paraesthesia, hypokalaemia, sulfa allergy caution
Doxapram	1–4 mg/min infusion	Rarely, post-anaesthetic hypoventilation	Hypertension, seizures; largely obsolete
NIV / adaptive servo-ventilation	Per titration	Hypoventilation; central apnoea (ASV avoided if LVEF ≤ 45%)	SERVE-HF mortality signal

★ PRESCRIBING & INTERPRETATION PEARLS

- Over-oxygenation in COPD raises $PaCO_2$ mainly through worsened V/Q (Haldane effect), not only loss of hypoxic drive.

Stepwise ABG interpretation

Algorithm 2.3 · Six-step ABG analysis



Expected compensation

Primary disorder	Compensation
Metabolic acidosis	PaCO2 = 1.5 × HCO3 + 8 (± 2) (Winter's)
Metabolic alkalosis	PaCO2 rises 0.7 per 1 mmol/L ↑ HCO3
Acute respiratory acidosis	HCO3 ↑ 1 per 10 mmHg ↑ PaCO2; pH ↓ 0.08 per 10
Chronic respiratory acidosis	HCO3 ↑ 3.5 per 10 mmHg ↑ PaCO2; pH ↓ 0.03 per 10
Acute respiratory alkalosis	HCO3 ↓ 2 per 10 mmHg ↓ PaCO2
Chronic respiratory alkalosis	HCO3 ↓ 4–5 per 10 mmHg ↓ PaCO2

Respiratory acidosis

- **Causes:** CNS depression (opioids, sedatives, stroke), neuromuscular (GBS, MG, MND, myopathy, high cord), chest wall (kyphoscoliosis, obesity), airway/lung (COPD, severe asthma, end-stage ILD), ventilator (inadequate MV, permissive hypercapnia).
- **Features:** headache, flapping tremor, drowsiness, papilloedema, warm peripheries, bounding pulse; narcosis.
- **Management:** treat cause; controlled O2 (88–92%); NIV for pH 7.25–7.35 (and selected lower); intubation for failure/coma; avoid rapid correction of chronic hypercapnia (post-hypercapnic metabolic alkalosis — give acetazolamide/KCl).

Metabolic alkalosis

- **Saline-responsive** (urine Cl <20): vomiting, NG suction, diuretics (remote), post-hypercapnia. **Saline-resistant** (urine Cl >20): hyperaldosteronism, Cushing, Bartter/Gitelman, severe hypokalaemia, current diuretics.

- Pulmonary relevance: hypoventilation worsens hypercapnic failure and impairs weaning. Treat with NaCl/KCl, acetazolamide 250–500 mg, stop diuretics.

ABG in COPD

- Stable chronic hypercapnia: pH near normal, high HCO3 (compensated). Acute-on-chronic: pH falls more than chronic compensation predicts; HCO3 high but inadequate.

Hyponatraemia in pulmonary practice

- SIADH in pneumonia (Legionella), TB, SCLC (ectopic ADH), positive pressure ventilation. Euvolaemic, Uosm >100, UNa >30, low serum urate.
- **Treat:** severe symptoms (seizure, coma) — 150 mL 3% saline over 20 min, repeat ×2 to raise Na 4–6 mmol/L; limit rise to ≤10 mmol/L in 24 h (≤8 if high ODS risk); fluid restriction, urea, tolvaptan for SIADH.

★ CLINICAL PEARLS & EXAM FOCUS

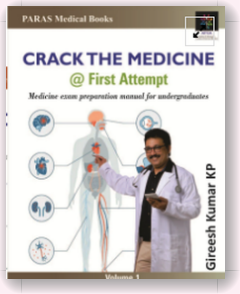
▸ "Acute rise of 10 mmHg PaCO2 → HCO3 up 1; chronic → up 3.5" — the most-asked compensation rule.

LANDMARK TRIALS & GUIDELINES

- Hyponatraemia: European clinical practice guideline (Spasovski, 2014); SALT-ED/SALSA trials — rapid intermittent bolus 3% saline is safe.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Respiratory acidosis. *[DNB Oct 2024 P1; NEET-SS/DNB 2024; Manipal 2014 P1]*
2. Henderson–Hasselbalch equation. *[BFUHS 2020 P1]*
3. Metabolic alkalosis: causes and management. *[WBUHS 2022 P1]*
4. ABGs associated with COPD. *[DNB Oct 2023 P3]*
5. Hyponatraemia. *[Manipal 2014 P2]*
6. Management of hyponatraemia. *[BFUHS 2019 P1]*



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2.9 Non-Respiratory Functions & Pulmonary Defence Mechanisms

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-B4, I-B10 Metabolic functions and defence
- ✓ List the metabolic and endocrine (non-respiratory) functions of the lung
- ✓ Describe mucociliary clearance, macrophage function and the protease–antiprotease balance

IN BRIEF Beyond gas exchange, the lung filters, metabolises vasoactive substances, synthesises surfactant and defends itself through mechanical, innate and adaptive mechanisms against ~10,000 L of inhaled air daily.

INVESTIGATIONS & TREATMENT AT A GLANCE

2.8 Acid–Base Balance & ABG Interpretation; Electrolytes

‹ Back to topic (p 35)
Next topic 2.9 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
pH	7.35–7.45	< 7.35 acidaemia; > 7.45 alkalaemia	Primary disorder
HCO_3^-	22–26 mmol/L	< 22 metabolic acidosis; > 26 metabolic alkalosis	
Anion gap	8–12 mmol/L (add 2.5 per 1 g/dL albumin fall below 4)	> 12	HAGMA (lactate, ketones, renal failure, toxins)
Delta ratio	1–2	< 1 (+ NAGMA); > 2 (+ metabolic alkalosis)	Mixed disorders
Winter's formula (expected $PaCO_2$)	1.5 × HCO_3 + 8 ± 2	Higher/lower	Additional respiratory acidosis/ alkalosis
Lactate	< 2 mmol/L	> 4 mmol/L	Tissue hypoperfusion (sepsis)
Sodium / potassium	Na 135–145; K 3.5–5.0 mmol/L	Hypo/hyper	SIADH (lung cancer, TB, pneumonia); hypokalaemia with β-agonists/steroids
Urine chloride	> 20 mmol/L (chloride-resistant)	< 20 (chloride-responsive)	Metabolic alkalosis cause

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Sodium bicarbonate	1–2 mmol/kg IV (8.4%) when pH < 7.1 with AKI	Severe metabolic acidosis	Hypernatraemia, CO_2 load — ventilate
0.9% saline / balanced crystalloid	20–30 mL/kg bolus	Chloride-responsive alkalosis; volume depletion	Hyperchloraemic acidosis (saline)
Acetazolamide	250–500 mg IV/oral once–twice daily	Post-hypercapnic metabolic alkalosis delaying weaning	Hypokalaemia
Potassium chloride	10–20 mmol/h IV (max 40 central)	Hypokalaemia	Cardiac monitoring at high rates
NIV	IPAP 15–25, EPAP 4–6 cmH_2O	Acute respiratory acidosis (pH 7.25–7.35) in COPD	Monitor pH at 1–2 h

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Expected compensation — acute respiratory acidosis: HCO_3 ↑ 1 per 10 mmHg $PaCO_2$; chronic: ↑ 3.5 per 10.

Non-respiratory functions of the lung

Function	Details
Filtration	Traps microemboli, fibrin, cell aggregates; sieve for blood clots
Reservoir	~500 mL of blood; buffer for LV filling
Metabolic — activation	ACE on endothelium converts angiotensin I → II (70% in one passage)
Metabolic — inactivation	Bradykinin (80%), serotonin, noradrenaline, PGE2/F2α, leukotrienes removed; adrenaline, histamine, dopamine, angiotensin II pass unchanged
Synthesis	Surfactant, mucus, prostacyclin, NO, endothelin, heparin (mast cells), IgA
Fluid & acid–base	Excretes CO2 (volatile acid), water, volatile substances (alcohol, ketones, anaesthetics)
Haemostasis	Thromboplastin, heparin, plasminogen activators; megakaryocytes produce platelets in lung capillaries
Thermoregulation & phonation	Heat and water exchange, speech, cough
Endocrine	Neuroendocrine cells (serotonin, bombesin); ectopic hormone production in tumours

Defence mechanisms

Layers of pulmonary defence

Level	Mechanisms
Upper airway	Nasal hairs, turbinates (particles >10 µm), humidification, sneeze
Airway reflexes	Cough, laryngeal closure, bronchoconstriction
Mucociliary escalator	Particles 2–10 µm trapped and cleared within 24 h
Humoral (innate)	Lysozyme, lactoferrin, defensins, cathelicidin (LL-37), SP-A/SP-D, complement, α1-antitrypsin, secretory IgA (airway), IgG (alveoli)
Cellular (innate)	Alveolar macrophages (particles <2 µm), neutrophils recruited, dendritic cells, NK cells, epithelial TLRs
Adaptive	BALT, Th1 (intracellular pathogens), Th2, Th17, cytotoxic T cells, B cells

Mucociliary clearance

- Effectiveness depends on ciliary function (beat frequency 12–15 Hz), mucus rheology (viscoelasticity) and the periciliary layer depth.
- **Impaired by:** smoking, dehydration, cold dry air, viral infection, PCD, CF, bronchiectasis, anaesthetics, opioids, high FiO2.
- **Measurement:** saccharin test (<30 min), radio-aerosol clearance, nasal NO (PCD screening).

Alveolar macrophages

- Most abundant immune cell in alveoli (~95% of BAL cells); tissue-resident, GM-CSF/PPAR-γ dependent.
- Functions: phagocytosis and killing, surfactant catabolism, antigen presentation, cytokine release (TNF, IL-1, IL-8 → neutrophils), resolution of inflammation (efferocytosis), tissue repair (TGF-β, PDGF — fibrosis).
- **Clinical:** host for M. tuberculosis; GM-CSF autoimmunity → PAP; smoker's macrophages (pigmented) in RB-ILD/DIP; foamy macrophages in amiodarone toxicity; haemosiderin-laden macrophages in DAH.

Protease–antiprotease balance

- Neutrophil elastase, proteinase-3, MMP-9/12 degrade elastin; balanced by α1-antitrypsin (main), SLPI, elafin, TIMPs.
- Imbalance from smoking (oxidants inactivate α1-AT, recruit neutrophils) or α1-AT deficiency → panacinar emphysema.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Particle size decides fate: >10 µm nose, 2–10 µm airways (mucociliary), 0.5–2 µm alveoli (macrophages) — coal dust, silica.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Alveolar macrophages. *[DNB Jun 2025 P1]*
2. Role of macrophages in the lung. *[DNB Oct 2024 P1]*
3. Write a short note on macrophages in the lung. *[BFUHS 2019 P1]*
4. Mucociliary escalator. *[DNB Jun 2025 P1; WBUHS 2022 P1]*
5. Mucociliary clearance mechanism. *[DNB Oct 2024 P1]*
6. Non respiratory functions of the lungs. *[DNB Oct 2023 P1]*
7. Non respiratory functions of lungs. *[DNB Jun 2021 P1]*
8. Antiproteinases and lung. *[DNB Dec 2022 P1]*

2.10 Exercise Physiology, CPET & Six-Minute Walk Test

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-B11 Exercise physiology and testing
- ✓ Describe the physiological response to exercise and anaerobic threshold
- ✓ Describe the protocol and interpretation of CPET
- ✓ Perform and interpret the six-minute walk test

IN BRIEF Exercise testing unmasks limitation not evident at rest. CPET integrates cardiac, ventilatory and metabolic responses to identify why a patient stops; the 6MWT is a simple, reproducible measure of functional capacity and prognosis.

Physiological response to exercise

- VO2 rises linearly with work to VO2max (~35–45 mL/kg/min in adults); cardiac output up to 5×; ventilation up to 15–20× through increased VT then RR.
- **Anaerobic (lactate/ventilatory) threshold:** at ~50–60% VO2max lactate rises; buffering by HCO3 produces extra CO2 → VCO2 and VE rise disproportionately (V-slope method).
- Normal person stops because of cardiovascular limit; breathing reserve (MVV − VEmax) >15 L/min or >15–20%.

CPET patterns

Limitation	Peak VO2	AT	Breathing reserve	HR reserve	Other
Cardiac (HF)	↓	↓ (early)	Normal	Low	Low O2 pulse, high VE/VCO2 slope
Ventilatory (COPD)	↓	Normal/not reached	Low (<15%)	High	Dynamic hyperinflation (falling IC)
Pulmonary vascular	↓	↓	Normal	Low	High VE/VCO2 (>34), desaturation, ↑ VD/VT
Deconditioning	↓	Early	Normal	Normal	Normal pattern overall
Poor effort	↓	Not reached	High	High	RER <1.05

- **Uses:** unexplained dyspnoea, pre-operative risk (VO2max <10 mL/kg/min prohibitive; >20 safe for pneumonectomy), HF transplant listing (<12–14), rehabilitation prescription, disability.

Six-minute walk test (ATS/ERS 2014)

- 30-m flat corridor, standard encouragement every minute, no warm-up, rest 15 min before; record distance, SpO2 (nadir), HR, Borg dyspnoea and fatigue before and after.
- **Normal:** 400–700 m (Enright equations); **MCID:** 25–33 m in COPD, 24–45 m in IPF.
- **Indications:** COPD, ILD (desaturation <88% prognostic), PAH (risk stratification: >440 m low risk, <165 m high risk — ESC 2022), pre-op, rehab, HF.
- **Contraindications:** unstable angina/MI within 1 month; relative — resting HR >120, BP >180/100.
- **Stop if:** chest pain, intolerable dyspnoea, leg cramps, staggering, diaphoresis, SpO2 <80%.
- Alternatives: shuttle walk test (incremental, endurance), sit-to-stand test.

INVESTIGATIONS & TREATMENT AT A GLANCE

2.9 Non-Respiratory Functions & Pulmonary Defence Mechanisms

‹ Back to topic (p 37)
Next topic 2.10 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Serum ACE	8–65 U/L (lab-specific)	Raised	Sarcoidosis (non-specific; ↓ on ACE inhibitors)
α1-antitrypsin level	1.0–2.0 g/L (20–50 µmol/L)	< 0.57 g/L (< 11 µmol/L)	Severe AATD — phenotype/ genotype
Immunoglobulins (IgG, IgA, IgM)	IgG 7–16 g/L; IgA 0.7–4; IgM 0.4–2.3	Low	CVID, hypogammaglobulinaemia → recurrent infection/bronchiectasis
Specific antibody (pneumococcal) response	≥ 1.3 µg/mL to ≥ 70% serotypes	Poor response	Specific antibody deficiency
Nasal NO / ciliary studies	nNO > 77 nL/min	< 77 nL/min	PCD
Mucociliary clearance (saccharin)	< 30 min	> 60 min	Impaired clearance

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
IV immunoglobulin	0.4–0.6 g/kg every 3–4 weeks (trough IgG > 8 g/L)	CVID/hypogammaglobulinaemia with infections	Infusion reactions, thrombosis
AAT augmentation	60 mg/kg IV weekly	Severe AATD with FEV1 30–65%	Cost; IgA deficiency (anaphylaxis)
Pneumococcal vaccines	PCV20 single dose (or PCV15 then PPSV23)	Chronic lung disease, immunodeficiency	Live vaccines avoided in immunodeficiency
Influenza vaccine	Annual 0.5 mL IM	All chronic lung disease	Egg allergy no longer a contraindication

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Recurrent bacterial bronchitis with bronchiectasis → always check immunoglobulins and specific antibody responses.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Desaturation ≥4% to <89% on 6MWT in ILD predicts mortality and may justify ambulatory oxygen.

LANDMARK TRIALS & GUIDELINES

• ATS/ERS technical standard on field walking tests, Eur Respir J 2014; ERS statement on CPET in clinical practice 2019.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Describe in brief cardiopulmonary exercise testing. *[BFUHS 2022 P1]*
2. Six-minute walk test. *[DNB Jun 2021 P1; BFUHS 2022 P2]*

2.11 Bronchoprovocation, Oscillometry, Small Airway Tests, FeNO & Respiratory Muscle Tests

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-B1 Assessment of pulmonary functions
- ✓ Describe bronchial challenge tests and their interpretation
- ✓ Describe impulse oscillometry and evaluation of small airway function
- ✓ Interpret FeNO and other non-invasive markers of airway inflammation; test respiratory muscle strength

IN BRIEF These tests extend spirometry — detecting airway hyperresponsiveness (methacholine/mannitol), small airway dysfunction (oscillometry, MBW), type-2 airway inflammation (FeNO, sputum eosinophils) and respiratory muscle weakness (MIP/MEP).

Bronchoprovocation tests

Type	Agent	Positive	Use
Direct	Methacholine, histamine	PD20 ≤200 µg (PC20 <8 mg/mL)	High sensitivity; negative test rules out current asthma
Indirect	Mannitol, hypertonic saline, adenosine, exercise, eucapnic voluntary hyperventilation (EVH)	≥15% fall (mannitol), ≥10% exercise/EVH	Specific for asthma; athletes (EVH), exercise-induced bronchoconstriction
Specific	Occupational allergen	Late/dual response	Occupational asthma confirmation

- Contraindications: FEV1 <60% predicted or <1.5 L, recent MI/stroke, uncontrolled HTN, aortic aneurysm, pregnancy; resuscitation equipment ready; withhold bronchodilators.

Impulse oscillometry (IOS) / forced oscillation technique

- Pressure oscillations (5–35 Hz) superimposed on tidal breathing; measures respiratory impedance — **resistance (R)** and **reactance (X)**.
- **R5** = total airway resistance; **R20** = central; **R5–R20** = small airway resistance (>0.07 kPa/L/s abnormal); **X5** more negative and **resonant frequency (Fres)** and **AX** (area of reactance) raised in small airway disease.
- **Advantages:** effort-independent, tidal breathing — children, elderly, ventilated, NM disease; detects small airway dysfunction with normal spirometry; within-breath reactance detects expiratory flow limitation.

Evaluation of small airway function

- Spirometry: FEF25–75, FEV3/FVC, air trapping (RV/TLC); N2 single-breath washout (phase III slope, closing volume); multiple-breath washout (LCI); IOS; frequency dependence of compliance; HRCT expiratory air trapping, parametric response mapping.

INVESTIGATIONS & TREATMENT AT A GLANCE

2.10 Exercise Physiology, CPET & Six-Minute Walk Test

‹ Back to topic (p 39)
Next topic 2.11 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Peak VO_2 (CPET)	> 84% predicted (≈ 25–40 mL/kg/min)	< 84%; < 15 mL/kg/min	Exercise limitation; < 10–12 mL/kg/min = high surgical risk
Anaerobic threshold	> 40% of predicted VO_2max	Low (< 11 mL/kg/min)	Cardiac/deconditioning limitation
Breathing reserve (1 – VEmax/MVV)	> 15–20% (MVV ≈ FEV1 × 40)	< 15%	Ventilatory limitation
VE/VCO_2 slope	< 30	> 34 (> 40 poor prognosis)	Ventilatory inefficiency — PH, heart failure, ILD
6MWT distance	400–700 m (age/height adjusted)	< 350 m; < 165 m (PAH high risk)	Functional limitation; prognosis
SpO_2 during 6MWT	No fall > 4% / stays ≥ 90%	Fall ≥ 4% or < 88%	Exertional desaturation — ambulatory O_2
Heart-rate recovery (1 min)	> 12 beats	< 12	Autonomic dysfunction, worse prognosis

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Ambulatory oxygen	Titrate to SpO_2 ≥ 88–90% on exertion	Exertional desaturation with benefit shown	Cylinder weight; adherence
Pulmonary rehabilitation	6–12 weeks, 2–3 sessions/week	COPD, ILD, PH (stable), post-COVID	Exclude unstable angina, severe AS
Pre-test medication	Continue usual bronchodilators	CPET/6MWT	Record on report

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Stop CPET for chest pain, ST depression > 2 mm, SBP drop > 10 mmHg, SpO_2 < 80%, severe dyspnoea.

FeNO

- Nitric oxide from iNOS in airway epithelium, induced by IL-13 — marker of **type-2 inflammation**; measured at 50 mL/s (ATS 2011).

FeNO interpretation (ATS 2011)

FeNO (ppb, adults)	Interpretation
<25	Type-2 eosinophilic inflammation unlikely; steroid response less likely
25–50	Interpret in context
>50	Eosinophilic inflammation likely; steroid-responsive

- **Applications:** supports asthma diagnosis (GINA, NICE), predicts ICS response, adherence check, selects/monitors biologics (dupilumab), differentiates COPD/ACO; lowered by smoking, ICS; raised by atopy, rhinitis.
- **Other markers:** induced sputum eosinophils (>3%), blood eosinophils, exhaled breath condensate, electronic nose (VOCs), periostin.

Respiratory muscle tests

- MIP (PImax) <−80 cmH2O excludes significant weakness; MEP; SNIP (sniff nasal pressure, >70 men, >60 women cmH2O); supine fall in VC; transdiaphragmatic pressure (Pdi); phrenic stimulation; diaphragm USG thickening fraction.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Methacholine is sensitive (good to rule out), mannitol/exercise are specific (good to rule in).

LANDMARK TRIALS & GUIDELINES

- ATS clinical practice guideline on FeNO interpretation (Dweik 2011); ERS technical standard on bronchial challenge testing 2017; ERS oscillometry standards 2020.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Broncho-provocation testing. *[DNB Dec 2021 P1]*
2. Impulse oscillometry. *[DNB Dec 2021 P1; DNB Dec 2025 P2; BFUHS 2022 P4]*
3. Evaluation of small airway function. *[DNB Oct 2023 P4]*
4. Current status of exhaled nitric oxide measurement. *[DNB Dec 2022 P4]*
5. Applications of exhaled breath nitric oxide in respiratory conditions. *[DNB Dec 2025 P4]*
6. FENO. *[BFUHS 2022 P4]*
7. Clinical applications of exhaled breath analysis in respiratory medicine. *[BFUHS 2020 P4]*
8. Inflammatory markers in bronchial asthma. *[BFUHS 2019 P4]*

2.12 Physiological Changes in Pregnancy, Ageing & High Altitude

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-B12 Physiology in pregnancy, altitude, ageing
- ✓ Describe respiratory changes of pregnancy and their implication for ABG interpretation
- ✓ Describe changes in the ageing lung
- ✓ Describe acclimatisation to high altitude and effects of rapid ascent

IN BRIEF Pregnancy produces progesterone-driven hyperventilation with compensated respiratory alkalosis and reduced FRC; ageing reduces elastic recoil and chest wall compliance; altitude lowers inspired PO2 and triggers a cascade of acclimatisation.

INVESTIGATIONS & TREATMENT AT A GLANCE

2.11 Bronchoprovocation, Oscillometry, Small Airway Tests, FeNO & Respiratory Muscle Tests

‹ Back to topic (p 40)
Next topic 2.12 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Methacholine challenge (PC20 / PD20)	PC20 > 16 mg/mL; PD20 > 400 µg	PC20 < 4 mg/mL (PD20 < 100 µg)	Airway hyperresponsiveness — supports asthma (high NPV)
Mannitol challenge	PD15 not reached at 635 mg	15% fall in FEV1	Indirect AHR — exercise/asthma
FeNO	< 25 ppb adults (< 20 children)	> 50 ppb adults (> 35 children)	Type-2 (eosinophilic) airway inflammation; ICS-responsive
Impulse oscillometry R5 – R20	< 0.07 kPa/L/s (approx.)	Raised	Small-airway dysfunction
Reactance area (AX)	< 0.33 kPa/L (adults)	Raised	Peripheral airway obstruction
MIP / MEP	MIP > 80 cmH_2O (men), > 60 (women)	MIP < 60	Respiratory muscle weakness
Sputum eosinophils	< 2–3%	≥ 3%	Eosinophilic airway disease

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Salbutamol (rescue after challenge)	200–400 µg via spacer	Bronchoconstriction after challenge	Monitor FEV1 to recovery
Inhaled corticosteroid trial	Budesonide 400–800 µg/day × 4–8 weeks	High FeNO / sputum eosinophils	Oral thrush — rinse mouth
Withholding medications before challenge	SABA 6 h, LABA 36 h, ICS 12–24 h, antihistamines 72 h	Valid testing	Document

★ **PRESCRIBING & INTERPRETATION PEARLS**

- A negative methacholine test in a symptomatic, untreated patient makes asthma very unlikely.

Respiratory changes of pregnancy

Parameter	Change	Mechanism / implication
Minute ventilation	↑ 30–50% (↑ VT)	Progesterone stimulates respiratory centre
PaCO2	28–32 mmHg	Normal pregnancy — a "normal" 40 mmHg signals impending failure
pH / HCO3	7.40–7.47 / 18–21	Compensated respiratory alkalosis
PaO2	100–105 mmHg	Higher; falls supine late in pregnancy
FRC, ERV, RV	↓ 20%	Elevated diaphragm (4 cm) — rapid desaturation at intubation
TLC, VC, FEV1, FEV1/FVC	Unchanged	Spirometry valid
O2 consumption	↑ 20–30%	Fetus and maternal metabolism
DLCO	↑ early, ↓ later	Blood volume vs anaemia
Upper airway	Oedema, hyperaemia	Difficult airway, epistaxis, rhinitis, snoring

- **Dyspnoea of pregnancy** in 60–70% (from first trimester) — physiological if normal exam and SpO2.

Ageing lung

Change	Consequence
Loss of elastic recoil (senile emphysema — duct ectasia)	↑ RV, ↑ FRC, ↑ closing volume, ↓ FEV1/FVC (use LLN to avoid overdiagnosis)
Stiffer chest wall (calcified costal cartilages, kyphosis)	↓ chest wall compliance, ↑ work of breathing
Weaker respiratory muscles	↓ MIP, ↓ cough effectiveness
↓ Alveolar surface area, capillaries	↓ DLCO; PaO2 falls (~100 − age/3)
Blunted responses	↓ hypoxic/hypercapnic responses, ↓ dyspnoea perception
Immune senescence	Pneumonia risk; atypical presentations

High altitude

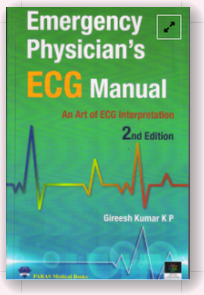
- PB falls ~ halves at 5500 m; PIO2 = 0.21 × (PB − 47).
- **Rapid ascent:** hypoxaemia → hyperventilation (hypoxic ventilatory response) → respiratory alkalosis, limiting further ventilation; HPV → pulmonary hypertension; fluid shifts; sleep periodic breathing.
- **Acclimatisation:** increased ventilation over days (CSF HCO3 excretion, carotid body sensitisation), renal bicarbonaturia, ↑ 2,3-DPG, erythropoietin → polycythaemia (weeks), ↑ capillary density, mitochondrial changes.
- Maladaptations: AMS, HACE, HAPE, chronic mountain sickness (Monge's), high-altitude pulmonary hypertension (see 15.1).

★ CLINICAL PEARLS & EXAM FOCUS

▸ In pregnancy a PaCO2 of 40 mmHg = CO2 retention; also "normal" PaO2 is higher, so ABG targets differ.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Respiratory physiologic changes of pregnancy. *[DNB Dec 2022 P1]*
2. Pulmonary physiological changes during pregnancy. *[BFUHS 2023 P2]*
3. Aging lung. *[DNB Dec 2022 P1]*
4. Effect of high altitude on rapid ascent. *[DNB Dec 2022 P1]*
5. Discuss in brief about high altitude physiology and related clinical disorders. *[BFUHS 2019 P1]*



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2.13 Physiological Basis of Pulmonary Symptoms & Signs — Dyspnoea, Cough, Clubbing

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-B13 Physiological basis of symptoms
- ✓ Explain the mechanisms of dyspnoea and enumerate respiratory causes of breathlessness
- ✓ Approach chronic cough and haemoptysis physiologically
- ✓ Describe clubbing and hypertrophic osteoarthropathy — causes, mechanism and grading

IN BRIEF Dyspnoea arises from a mismatch between central respiratory drive (efferent) and the mechanical response of the respiratory system (afferent feedback) — "neuromechanical dissociation". Clubbing is a sign whose mechanism (megakaryocyte–platelet-derived growth factors) links lung, heart and gut disease.

Mechanisms of dyspnoea

- **Increased drive/effort:** chemoreceptor stimulation (hypoxaemia, hypercapnia, acidosis), J-receptor and C-fibre activation (oedema, ILD, PE).
- **Increased load or weakness:** airway resistance, low compliance, hyperinflation (dynamic), muscle weakness.
- **Neuromechanical (efferent–reafferent) dissociation:** drive not matched by ventilation — central to COPD and ILD dyspnoea.
- Qualitative descriptors: "air hunger" (chemical drive), "work/effort" (load), "chest tightness" (bronchoconstriction).
- Scales: mMRC (0–4), Borg CR10, BDI/TDI, VAS; CAT.

Respiratory causes of breathlessness

Onset	Causes
Acute (minutes)	Pneumothorax, PE, acute asthma, anaphylaxis, foreign body, pulmonary oedema
Subacute (hours–days)	Pneumonia, effusion, exacerbation of COPD/ILD, lobar collapse, ARDS
Chronic	COPD, asthma, ILD, bronchiectasis, PH, chest wall disease, NM weakness, obesity, lung cancer, large effusion
Positional	Orthopnoea (HF, bilateral diaphragm palsy), platypnoea–orthodeoxia (PAVM, HPS, PFO), trepopnoea (unilateral lung disease)

Clubbing

- Bulbous enlargement of the terminal phalanx with loss of the Lovibond angle (>180°), increased nail-fold angle, Schamroth window lost, phalangeal depth ratio (DPD/IPD) >1, fluctuation of nail bed.
- **Grades:** 1 fluctuation/softening; 2 obliteration of angle; 3 parrot-beak/drumstick; 4 HPOA.
- **Mechanism (Dickinson):** megakaryocytes and platelet clumps bypass pulmonary capillaries (shunt or lung disease) → lodge in digital vasculature → release PDGF and VEGF → vascular proliferation, oedema; hypoxia increases VEGF; PGE2 (HPGD/SLCO2A1 mutations in primary HOA).

INVESTIGATIONS & TREATMENT AT A GLANCE

2.12 Physiological Changes in Pregnancy, Ageing & High Altitude

‹ Back to topic (p 41)
Next topic 2.13 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
ABG in pregnancy	$PaCO_2$ 28–32 mmHg; pH 7.40–7.47; HCO_3 18–22	$PaCO_2$ > 35–40 mmHg	"Normal" $PaCO_2$ of 40 in pregnancy = impending failure
Spirometry in pregnancy	FEV1, FVC unchanged; FRC ↓ 20%	Reduced FEV1	Not due to pregnancy — investigate
Spirometry in elderly	FEV1/FVC falls with age (use LLN)	Fixed 0.70 overdiagnoses obstruction in elderly	Use GLI LLN
SpO_2 at altitude (3000 m)	~ 90–94%	< 85% with symptoms	HAPE/AMS risk
Haemoglobin at altitude	Rises 1–2 g/dL	> 21 g/dL men, > 19 g/dL women	Chronic mountain sickness
D-dimer in pregnancy	Physiologically raised	—	Limited value; use CTPA/V/Q per algorithm

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Acetazolamide	125 mg twice daily (start 1 day before ascent)	AMS prevention	Paraesthesia, sulfa caution
Nifedipine SR	30 mg twice daily	HAPE prevention/treatment	Hypotension
Dexamethasone	8 mg then 4 mg 6-hourly	HACE / moderate–severe AMS	Hyperglycaemia; descend
Budesonide/formoterol in pregnancy	Usual doses	Asthma in pregnancy — continue	Uncontrolled asthma is riskier than drugs

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Oxygen and descent are the definitive treatment of all altitude illnesses.

Causes of clubbing

System	Causes
Pulmonary	Lung cancer (NSCLC), bronchiectasis, CF, lung abscess, empyema, IPF/asbestosis, mesothelioma, PAVM — **not uncomplicated COPD or TB**
Cardiac	Cyanotic CHD, infective endocarditis, atrial myxoma
GI/hepatic	IBD, coeliac, cirrhosis (HPS), GI lymphoma
Other	Thyroid acropachy, hereditary, unilateral (aneurysm, AV fistula)

Hypertrophic (pulmonary) osteoarthropathy

- Clubbing + periostitis of long bones (distal tibia, radius) + symmetric arthropathy; painful; 5–10% of NSCLC (adenocarcinoma) and pleural fibromas; bone scan "parallel stripe" sign.
- Regresses after tumour resection or vagotomy; NSAIDs, bisphosphonates, octreotide.

★ CLINICAL PEARLS & EXAM FOCUS

▸ New clubbing in a COPD patient = look for lung cancer or bronchiectasis.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Mechanism of dyspnoea. *[DNB Dec 2021 P1]*
2. Respiratory causes of breathlessness. *[DNB Jun 2025 P2]*
3. Clubbing and hypertrophic osteoarthropathy. *[DNB Jun 2025 P2]*

2.14 Aerosol Kinetics & Inhaled Drug Delivery Devices

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-B6 Inhalation kinetics and aerosol therapy
- ✓ Explain the mechanisms of aerosol deposition and the factors governing it
- ✓ Compare inhaler devices and match device to patient
- ✓ Describe the role of inhaled antibiotics

IN BRIEF Lung deposition depends on particle size (MMAD 1–5 µm ideal), inspiratory flow and breath-hold. Device choice should match the patient's inspiratory flow, coordination and cognition — wrong technique is the commonest cause of "uncontrolled" disease.

Mechanisms of deposition

Mechanism	Particle size	Site	Favoured by
Inertial impaction	>5 µm	Oropharynx, large airway bifurcations	High flow, turbulence
Sedimentation	1–5 µm	Small airways, alveoli	Slow deep breath, breath-hold 10 s
Diffusion (Brownian)	<0.5 µm	Alveoli	Long residence; much exhaled
Interception / electrostatic	Fibres	Airway walls	Asbestos fibres

- **Patient factors:** inspiratory flow, breath-hold, airway calibre (obstruction shifts deposition centrally), tidal vs single breath; **device factors:** MMAD, GSD, velocity, spacer.

INVESTIGATIONS & TREATMENT AT A GLANCE

2.13 Physiological Basis of Pulmonary Symptoms & Signs — Dyspnoea, Cough, Clubbing

‹ Back to topic (p 43)
Next topic 2.14 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
mMRC dyspnoea scale	Grade 0	Grade ≥ 2	"More symptomatic" (GOLD) — prognosis
CAT score	< 10	≥ 10	Symptomatic COPD
Leicester Cough Questionnaire	21 (best)	Low scores	Cough-related quality of life
Haemoglobin & cyanosis	Central cyanosis appears with ≥ 5 g/dL deoxygenated Hb (SaO_2 ≈ 85%)	—	Masked in anaemia; early in polycythaemia
BNP / NT-proBNP	BNP < 100 pg/mL; NT-proBNP < 300	Raised	Cardiac dyspnoea
Clubbing — Schamroth / Lovibond	Diamond window present; angle < 160°	Window obliterated; angle > 180°	Lung cancer, bronchiectasis, IPF, empyema (not COPD alone)

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Opioid for refractory breathlessness	Morphine SR 10 mg/day (max 30 mg)	Chronic breathlessness in advanced disease	Constipation, sedation; low dose safe
Codeine/dextromethorphan	Codeine 15–30 mg 6-hourly; DXM 30 mg 6–8-hourly	Short-term cough suppression	Not in productive cough
Gabapentin	300 mg up to 1800 mg/day	Refractory chronic cough	Sedation, dizziness
Fan therapy, breathing techniques	Handheld fan to face	Breathlessness	—

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Clubbing in COPD → look for lung cancer, bronchiectasis or ILD.

Inhaler devices

Device	Flow needed	Advantages	Disadvantages
pMDI	Slow, deep (<60 L/min); coordination	Portable, cheap, all drugs	Coordination, cold-Freon effect, oropharyngeal deposition (~10–20% lung)
pMDI + spacer/valved holding chamber	Tidal breathing	No coordination; ↓ oral deposition; acute asthma, children, elderly	Bulky; static charge
Breath-actuated MDI	Moderate	Auto-firing	Limited availability
Dry powder inhaler (rotahaler, Accuhaler, Turbuhaler, Ellipta)	Fast, forceful (30–90 L/min; device resistance)	No coordination, counter, no propellant	Needs PIF >30 L/min; humidity-sensitive; not in severe attacks
Soft mist inhaler (Respimat)	Slow	Slow-moving mist, high lung deposition (~40–50%)	Assembly
Nebuliser (jet, ultrasonic, vibrating mesh)	Tidal	Severe disease, high doses, antibiotics, infants, ventilated	Time, cost, infection, inefficient (jet)

- **In ventilated patients:** vibrating mesh or pMDI with in-line spacer before Y-piece; humidification reduces delivery.

Inhaled antibiotics

- **Bronchiectasis / CF with Pseudomonas:** tobramycin (TOBI), colistin (Promixin), aztreonam lysine, amikacin; reduce exacerbations in frequent exacerbators (PROMIS trials of colistin).
- **NTM:** amikacin liposome inhalation suspension (ALIS) for refractory MAC (CONVERT trial).
- **VAP adjunct** for MDR gram-negatives (colistin, amikacin) — limited evidence.
- **Others:** inhaled pentamidine (PJP prophylaxis), amphotericin (fungal prophylaxis in transplant), ribavirin (RSV).
- **Adverse:** bronchospasm (pre-dose bronchodilator), dysphonia, tinnitus, resistance.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Device choice = "Can they generate >30 L/min? Can they coordinate? Can they handle the device?" — check technique at every visit (GINA).

LANDMARK TRIALS & GUIDELINES

- Laube BL et al, ERS/ISAM task force on aerosol delivery 2011; Haworth CS et al, PROMIS-I/II, Lancet Respir Med 2019.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Factors governing airway deposition of aerosols. *[WBUHS 2022 P1]*
2. Advantages and disadvantages of different modes of inhalation therapy. *[WBUHS 2022 P1]*
3. Enumerate various inhaled drug delivery devices and their principal mechanisms. *[DNB May 2024 P2]*
4. Write a note on the role of inhaled antibiotics in various respiratory disorders. *[DNB May 2024 P2]*

2.15 Pulmonary Circulation Physiology — West Zones, Starling Resistor, HPV & Fluids

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-B3 Pulmonary blood flow
- ✓ Describe the pulmonary vascular resistance and its determinants, West zones and the Starling resistor model
- ✓ Explain hypoxic pulmonary vasoconstriction and Starling forces governing lung water
- ✓ Apply to fluid therapy in critically ill patients

INVESTIGATIONS & TREATMENT AT A GLANCE

2.14 Aerosol Kinetics & Inhaled Drug Delivery Devices

‹ Back to topic (p 44)
Next topic 2.15 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Peak inspiratory flow (In-Check dial)	DPI needs ≥ 30–60 L/min; MDI best 30–60 L/min slow	< 30 L/min	Cannot use DPI — choose MDI + spacer or nebuliser
Inhaler technique check	All critical steps correct	Critical errors	Commonest cause of poor control
Particle MMAD	1–5 µm deposits in lower airways	> 5 µm oropharynx; < 1 µm exhaled	Device/aerosol choice
Lung deposition	MDI 10–20%; DPI 15–40%; SMI ~ 40%	—	Device efficiency

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
pMDI + spacer	1 puff per 4–5 breaths or single slow breath + 10 s hold	Children, elderly, acute attacks	Clean spacer weekly; air-dry
Dry-powder inhaler	Quick, deep inhalation	Adults with adequate flow	Do not exhale into device
Soft-mist inhaler (Respimat)	2 puffs once daily (tiotropium 2.5 µg)	Low inspiratory flow	Coordination still needed
Nebuliser (jet)	Salbutamol 2.5–5 mg, ipratropium 0.5 mg	Acute severe attacks; unable to use devices	Infection control; drive with O_2 except hypercapnia (use air)

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Check technique at every visit before escalating therapy — teach-back method.

IN BRIEF The pulmonary circulation is low-pressure, low-resistance and highly distensible. Gravity and alveolar pressure determine regional perfusion (West zones), modelled by the Starling resistor — a collapsible tube whose flow depends on upstream pressure minus the surrounding (alveolar) pressure.

Pulmonary vascular resistance

- PVR = (mPAP − PAWP)/CO = 1–2 Wood units (80–160 dyn·s·cm^{-5}); PH defined by PVR >2 WU (ESC 2022).
- PVR falls with increased flow by **recruitment** (closed capillaries open) and **distension**; lowest at FRC — rises at both low volumes (extra-alveolar vessels narrow) and high volumes (alveolar vessels compressed).

West zones and the Starling resistor

West zones

Zone	Pressure relationship	Flow determined by	Notes
1	PA > Pa > Pv	No flow — alveolar dead space	Only with hypovolaemia, high PEEP
2	Pa > PA > Pv	Pa − PA (waterfall / Starling resistor)	Middle lung; flow independent of venous pressure
3	Pa > Pv > PA	Pa − Pv	Base; PAWP valid only when catheter tip in zone 3
4	Interstitial pressure ↑	Extra-alveolar vessel compression	Very base, low volumes, oedema

- **Starling resistor:** a collapsible tube inside a chamber; when chamber pressure exceeds downstream pressure, flow is governed by upstream minus chamber pressure — the "vascular waterfall". Explains why PEEP increases dead space and why PAWP may reflect alveolar pressure.

Hypoxic pulmonary vasoconstriction (HPV)

- Alveolar hypoxia constricts small pulmonary arteries (mitochondrial redox → K+ channel inhibition → Ca2+ influx) — diverts blood to better ventilated areas, improving V/Q.
- **Blunted by:** vasodilators (nitroprusside, CCBs, inhaled anaesthetics), sepsis, alkalosis — worsening hypoxaemia. Global chronic HPV → PH (COPD, altitude).

Lung water — Starling forces

- Qf = Kf [(Pc − Pi) − σ(πc − πi)]; normal lymph flow ~10–20 mL/h, can increase 10-fold.
- **Hydrostatic oedema** (↑ Pc: LVF, fluid overload, negative pressure) vs **permeability oedema** (↑ Kf, ↓ σ: ARDS) — see 9.6.

Intravenous fluids in critically ill patients

- Phases (ROSE): resuscitation → optimisation → stabilisation → evacuation (de-resuscitation).
- Use balanced crystalloids over 0.9% saline in sepsis/critical illness (SMART); avoid starches (6S, CHEST); albumin in large-volume needs.
- Assess fluid responsiveness (PLR, PPV/SVV, IVC variability, mini-fluid challenge); conservative fluids improve oxygenation and ventilator-free days in ARDS (FACTT).

★ CLINICAL PEARLS & EXAM FOCUS

▸ In ARDS, "dry lungs are happy lungs" — after shock resolves, use a conservative fluid strategy (FACTT).

LANDMARK TRIALS & GUIDELINES

- FACTT (NHLBI ARDSNet, NEJM 2006); SMART (Semler, NEJM 2018); CLASSIC (2022) — restrictive fluids non-inferior in septic shock.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. The Starling resistor model of the pulmonary circulation. *[DNB Oct 2024 P1]*
2. Intravenous fluid therapy in critically ill patients. *[BFUHS 2020 P1]*

INVESTIGATIONS & TREATMENT AT A GLANCE

2.15 Pulmonary Circulation Physiology — West Zones, Starling Resistor, HPV & Fluids

‹ Back to topic (p 45)
Next topic 2.16 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
mPAP (RHC)	≤ 20 mmHg	> 20 mmHg	Pulmonary hypertension
PVR	≤ 2 Wood units	> 2 WU	Pre-capillary PH
PAWP	≤ 15 mmHg	> 15 mmHg	Post-capillary PH (left heart)
Vasoreactivity test (iNO)	—	mPAP ↓ ≥ 10 mmHg to ≤ 40 mmHg with unchanged/↑ CO	Responder → high-dose CCB (idiopathic PAH)
Cardiac index	2.5–4.0 L/min/m²	< 2.0	Poor prognosis in PAH
TAPSE (echo)	> 17 mm	< 17 mm	RV dysfunction

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Inhaled nitric oxide	5–20 ppm	Vasoreactivity testing; refractory hypoxaemia	Methaemoglobin, rebound PH
Amlodipine / nifedipine	Amlodipine up to 20 mg/day; nifedipine up to 240 mg/day	Vasoreactive idiopathic PAH only	Hypotension; harmful in non-responders
Oxygen	Keep SpO_2 > 90%	Hypoxic vasoconstriction in chronic lung disease	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Hypoxic pulmonary vasoconstriction is the reason oxygen lowers PA pressure in COPD and ILD.

2.16 Principles of Electrocardiography & Cardiac Changes in Lung Disease

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-B5, I-B7 Electrocardiography; electrolytes
- ✓ Describe the principles of the ECG and recognise ECG signs of RV strain, cor pulmonale and PE
- ✓ Recognise ECG effects of electrolyte disturbances and drugs used in pulmonology (QT prolongation)

IN BRIEF The ECG records summated myocardial depolarisation vectors through 12 leads. In pulmonary practice it detects RV hypertrophy/strain, arrhythmias (MAT, AF), and QT prolongation from anti-TB and macrolide drugs.

Principles

- Einthoven's triangle, hexaxial reference for frontal leads, precordial horizontal plane; paper 25 mm/s (1 mm = 40 ms), 10 mm/mV.
- Normal axis −30° to +90°; intervals PR 120–200 ms, QRS <120 ms, QTc <450 (men), <470 (women) ms (Bazett/Fridericia).

ECG in pulmonary disease

Condition	ECG features
COPD/emphysema	Low voltage, right axis deviation, P pulmonale (>2.5 mm in II), poor R progression, MAT
Cor pulmonale / RVH	R/S >1 in V1, RV strain (ST–T inversion V1–V3), RBBB, RAD >+110°
Acute PE	Sinus tachycardia (commonest), S1Q3T3, new RBBB, T inversion V1–V4, AF, right axis shift
Pneumothorax (left)	Reduced QRS amplitude, axis shift, mimic of anterior MI
Hypoxia / hypercapnia	Tachyarrhythmias, AF

QT-prolonging drugs in pulmonology

- Bedaquiline, delamanid, clofazimine, moxifloxacin/levofloxacin, macrolides, azoles. NTEP: baseline ECG; monitor QTcF; stop if QTcF >500 ms; caution if >450.
- Correct hypokalaemia, hypomagnesaemia, hypocalcaemia before starting.

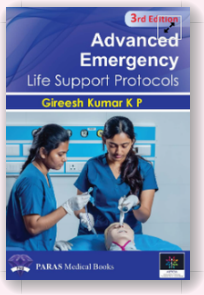
Electrolyte ECG changes

Disturbance	ECG
Hyperkalaemia	Peaked T, flat P, wide QRS, sine wave
Hypokalaemia	U waves, ST depression, flat T, long QU (salbutamol, steroids, theophylline)
Hypercalcaemia	Short QT (sarcoidosis, squamous cancer — PTHrP)
Hypocalcaemia / hypomagnesaemia	Long QT, torsades

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ Repeated nebulised salbutamol + steroids → hypokalaemia and arrhythmias in acute asthma — monitor K+.

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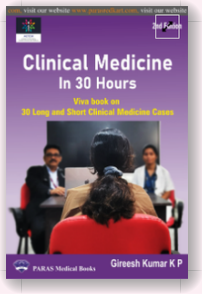
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INVESTIGATIONS & TREATMENT AT A GLANCE

2.16 Principles of Electrocardiography & Cardiac Changes in Lung Disease

‹ Back to topic (p 47)
Next topic 3.1 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
P pulmonale	P wave < 2.5 mm in II	P ≥ 2.5 mm in II, III, aVF	Right atrial enlargement (COPD, PH)
Right axis deviation	−30° to +90°	> +110°	RV hypertrophy/strain
RV strain pattern	—	R/S > 1 in V1, T inversion V1–V3	PH, acute PE
S1Q3T3	—	Present	Acute PE (specific, insensitive) — sinus tachycardia is commonest
QTc	Men < 450 ms; women < 470 ms	> 500 ms	Stop QT-prolonging drugs (bedaquiline, macrolides, FQ)
Multifocal atrial tachycardia	—	≥ 3 P-wave morphologies, rate > 100	COPD exacerbation, theophylline
Low-voltage QRS	> 5 mm limb leads	< 5 mm	Hyperinflation, effusion, obesity

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Verapamil / diltiazem	Diltiazem 0.25 mg/kg IV; verapamil 2.5–5 mg IV	MAT/AF rate control in COPD	Avoid in decompensated heart failure
Magnesium sulphate	2 g IV over 10–20 min	Torsades, QT-related arrhythmia, MAT	Hypotension
Correct K^+ / Mg^{2+}	K > 4.0, Mg > 2.0 mg/dL	QT prolongation on TB/ macrolide therapy	—
Cardioselective β-blocker	Bisoprolol 1.25–10 mg daily	Cardiac indications in COPD	Safe in COPD; caution in asthma

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Baseline ECG is mandatory before bedaquiline, delamanid, clofazimine, moxifloxacin and macrolides.

SECTION 3

Microbiology, Pathology, Immunology & Pharmacology

Paper I

Mycobacteria and their laboratory diagnosis; respiratory bacteria, viruses, fungi and parasites; pathology of inflammation, granuloma and emphysema; hypersensitivity and allergy testing; pharmacology of anti-TB drugs, bronchodilators, anti-inflammatories, antimicrobials, antineoplastics and immunosuppressants; pharmacovigilance (NMC syllabus I-C, I-D, I-F, I-G).

3.1 Mycobacteria — Biology, Staining, Culture & Phenotypic DST 49
3.2 Molecular Diagnosis of TB — CBNAAT, Truenat, LPA & Whole-Genome Sequencing 51
3.3 Respiratory Viruses & Their Laboratory Diagnosis 52
3.4 Respiratory Fungi — Classification & Laboratory Diagnosis 53
3.5 Bacteria, Atypical Organisms, Anaerobes & Biomarkers of Infection 54
3.6 Pathology — Inflammation, Granuloma & Emphysema 56
3.7 Hypersensitivity, Allergy Testing & Immunology of Lung Disease 57
3.8 Pharmacology of First-Line Anti-TB Drugs & Drug-Induced Liver Injury 58
3.9 Second-Line & Newer Anti-TB Drugs — Bedaquiline, Delamanid, Pretomanid, Linezolid, Fluoroquinolones 60
3.10 Bronchodilators & Inhaled Corticosteroids 61
3.11 Antimicrobials, Antifungals & Macrolides in Respiratory Medicine 63
3.12 Antineoplastic, Immunosuppressant & Biologic Drugs; Pharmacovigilance 64

3.1 Mycobacteria — Biology, Staining, Culture & Phenotypic DST

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-C1, I-C4, I-C5 Mycobacteria and laboratory diagnosis
- ✓ Describe the biology, cell wall and virulence of M. tuberculosis
- ✓ Describe AFB staining techniques and their grading under NTEP
- ✓ Describe solid and liquid culture and phenotypic drug susceptibility testing

IN BRIEF M. tuberculosis is a slow-growing (generation time 18–24 h), obligate aerobic, non-motile, acid-fast bacillus. Its lipid-rich mycolic acid wall resists decolourisation by acid-alcohol — the basis of Ziehl–Neelsen staining — and contributes to virulence and drug tolerance.

Biology and virulence

- **M. tuberculosis complex:** M. tuberculosis, M. bovis (and BCG), M. africanum, M. microti, M. canettii, M. orygis.
- **Cell wall:** peptidoglycan – arabinogalactan – mycolic acids; lipoarabinomannan (LAM — urine LAM test), trehalose dimycolate (**cord factor** — serpentine cords, granuloma), sulfolipids, PDIMs.
- **Virulence factors:** ESX-1 secretion system (ESAT-6, CFP-10 — absent in BCG, basis of IGRA), inhibition of phagosome–lysosome fusion, catalase-peroxidase (KatG), dormancy regulon (DosR) in hypoxic granuloma.
- Bacterial populations (Mitchison): rapidly multiplying in cavity (INH kills), slow in acid environment (PZA), spurters (RIF), dormant (none) — basis of combination and duration.

AFB staining methods

Method	Stain / principle	Sensitivity / detection limit	Comments
Ziehl–Neelsen (hot)	Carbol fuchsin with heat, 25% H2SO4 decolouriser, methylene blue counterstain	5,000–10,000 bacilli/mL	Red rods on blue background; cheap
Kinyoun (cold)	Higher phenol, no heat	Similar	Used for Nocardia (1% H2SO4 — modified)
Fluorescent (auramine-O / LED FM)	Auramine binds mycolic acid; potassium permanganate quench	10% more sensitive; faster (×200 field)	NTEP uses LED fluorescence microscopy at DMCs

- **Specimen:** two sputum samples (one early-morning) of 2–5 mL, mucopurulent; induced sputum, gastric aspirate, BAL when non-productive.

Grading of smear (ZN, 100 fields at 1000×)

Finding	Grade
No AFB in 100 fields	Negative
1–9 AFB in 100 fields	Scanty (record number)
10–99 AFB in 100 fields	1+
1–10 AFB per field (50 fields)	2+
>10 AFB per field (20 fields)	3+

Culture

- **Decontamination:** NALC–NaOH (Petroff's) removes contaminants; then centrifuge.
- **Solid:** Löwenstein–Jensen (egg-based, malachite green) — rough, buff, "cauliflower" colonies in 3–8 weeks; Middlebrook 7H10/7H11 (agar). **Liquid:** MGIT 960 (fluorescence quenched by O2; positive in 10–14 days, up to 42 days) — gold standard; 10–100 bacilli/mL detection.
- **Identification:** MPT64 antigen immunochromatography (MTB complex), niacin, nitrate reduction; line probe for NTM species.

Phenotypic DST

- **Liquid MGIT DST** (WHO critical concentrations): first-line and second-line — the reference for bedaquiline, linezolid, clofazimine, delamanid, pretomanid.
- Methods: proportion method (≥1% resistant colonies = resistant), absolute concentration, resistance ratio; MODS, nitrate reductase assay, colorimetric redox (low-resource).
- Turnaround 4–6 weeks; used after molecular tests for drugs lacking reliable molecular markers.

Immunohistochemistry in respiratory diseases

- **Lung cancer typing:** TTF-1, Napsin A (adenocarcinoma); p40, p63, CK5/6 (squamous); synaptophysin, chromogranin, CD56, INSM1 (neuroendocrine/SCLC); Ki-67 (carcinoid grading).
- **Mesothelioma:** calretinin, WT-1, D2-40, CK5/6 positive; BAP1 loss, MTAP loss; adenocarcinoma markers negative (Ber-EP4, MOC-31, claudin-4).

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- **Predictive:** ALK (D5F3), ROS1, PD-L1 (22C3, SP263 — tumour proportion score).
- **Others:** CD1a/langerin (PLCH), HMB-45 and SMA (LAM), S-100, MPT64 in TB granulomas.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Smear needs ~10^4 bacilli/mL, culture 10–100 — explaining why smear-negative culture-positive TB is common.

LANDMARK TRIALS & GUIDELINES

- NTEP Training Modules (2023) for laboratory network; WHO operational handbook on TB — diagnosis (2024).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Staining techniques for acid fast bacilli (AFB). *[DNB Oct 2023 P3]*
2. Liquid culture and DST for acid fast bacilli. *[WBUHS 2022 P3]*
3. Utility of immunohistochemistry in respiratory diseases. *[DNB Jun 2021 P1]*

3.2 Molecular Diagnosis of TB — CBNAAT, Truenat, LPA & Whole-Genome Sequencing

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-C4 Laboratory diagnosis; II-A1 Diagnostic methods
- ✓ Describe the principle, performance and NTEP role of Xpert MTB/RIF (Ultra), Truenat, LPA and WGS/tNGS
- ✓ Interpret genotypic resistance results including trace calls and indeterminate RIF

IN BRIEF WHO-recommended rapid diagnostics (WRDs) detect MTB and resistance within hours. NTEP uses NAAT (CBNAAT/Truenat) as the upfront test for all presumptive TB, followed by LPA and liquid culture DST for drug-resistance work-up.

Molecular tests for TB

Test	Principle	Targets / resistance	Turnaround	Comment
Xpert MTB/RIF (CBNAAT)	Cartridge real-time PCR, molecular beacons	rpoB 81-bp RRDR (RIF)	2 h	Sensitivity ~88% (smear+ 98%, smear− 67%)
Xpert Ultra	IS6110/IS1081 multicopy + rpoB melt curve	RIF; "trace" category	80 min	LOD ~16 CFU/mL; better in paucibacillary, CSF, children
Xpert MTB/XDR	10-colour cartridge	INH (katG, inhA, ahpC), FQ (gyrA/B), AMK/KAN (rrs, eis), ETO	90 min	After RIF-R or INH-R suspicion
Truenat MTB / MTB Plus / MTB-RIF Dx (Molbio, India)	Chip-based micro-PCR, battery-operated	nrdB/IS6110; rpoB	~1 h	Indian point-of-care NAAT; peripheral labs
FL-LPA (GenoType MTBDRplus)	PCR + reverse hybridisation strips	rpoB, katG, inhA	2–3 days	Smear-positive/culture isolates; detects INH mono-R
SL-LPA (MTBDRsl)	Same	gyrA/B (FQ), rrs, eis (SLI)	2–3 days	Guides FQ use; mandatory before shorter regimen
WGS / targeted NGS	Sequencing of whole genome / amplicons	All drugs incl. BDQ, LZD, PZA (pncA)	Days	WHO mutation catalogue (2nd ed 2023); surveillance, transmission
LAMP (TB-LAMP)	Isothermal amplification	MTB only	1 h	Replacement for smear
LF-LAM	Urine lipoarabinomannan lateral flow	—	25 min	HIV with CD4 ≤200 or seriously ill

Interpreting results

- **MTB detected, RIF resistance not detected** → DS-TB regimen; send sample for FL-LPA (INH resistance) under universal DST.

INVESTIGATIONS & TREATMENT AT A GLANCE

3.1 Mycobacteria — Biology, Staining, Culture & Phenotypic DST

‹ Back to topic (p 49)
Next topic 3.2 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Sputum smear (ZN/LED-FM)	Negative	Scanty, 1+, 2+, 3+	Bacillary load; ≥ 5000–10 000 bacilli/mL needed
Liquid culture (MGIT)	No growth at 42 days	Positive (median 10–14 days)	Gold standard; enables phenotypic DST
Solid culture (LJ)	No growth at 8 weeks	Buff, rough colonies 3–8 weeks	Slower, cheaper
MPT64 antigen (isolate)	—	Positive	Identifies MTB complex (vs NTM)
Phenotypic DST (critical concentrations, MGIT)	Susceptible	Growth at R 0.5, H 0.1, Mfx 0.25, Lfx 1.0, Bdq 1.0, Lzd 1.0 µg/mL	Resistance
Contamination rate	MGIT 8–10%; LJ 3–5%	Higher	Specimen processing quality

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Sample collection	2 sputum samples (one early morning), 3–5 mL	All presumptive TB	Collect outdoors/ventilated area
NALC–NaOH decontamination	1–1.5% final NaOH, 15–20 min	Culture processing	Over-decontamination kills bacilli
Biosafety	BSL-3 for culture manipulation	Laboratory safety	N95 + BSC

★ PRESCRIBING & INTERPRETATION PEARLS

- Culture conversion = two consecutive negative cultures ≥ 7 days apart (WHO 2021).

- **RIF resistance detected** → treat as RR-TB; repeat on second specimen if low pre-test probability; send SL-LPA, LC-DST.
- **Trace (Ultra)** — MTB present at low copy; RIF indeterminate. In PLHIV, children, EPTB: treat as TB. In adults with prior TB, interpret clinically (non-viable DNA persists). Repeat test.
- **RIF indeterminate** → repeat test on fresh specimen; send for LPA/culture.
- **Polymerase chain reaction (general):** denaturation–annealing–extension cycles; real-time PCR quantifies via fluorescence; false positives from contamination, dead bacilli (not for treatment monitoring).

Whole-genome sequencing

- Detects mutations for all drugs, novel mutations, heteroresistance (>10%), lineage and transmission clusters (SNP distance ≤5–12).
- Limitations: cost, infrastructure, bioinformatics; uncertain significance of some mutations (Rv0678 for BDQ/CFZ). India's genomic surveillance under NTEP expanding (tNGS endorsed by WHO 2024).

✦ RECENT ADVANCES & GUIDELINE UPDATES

▸ WHO 2024 consolidated guidelines (Module 3) endorse targeted NGS and low-complexity automated NAATs for INH and second-line resistance.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Xpert and LPA remain positive after cure — never use NAAT for follow-up; use smear and culture.

LANDMARK TRIALS & GUIDELINES

- Dorman SE et al, Xpert Ultra, Lancet Infect Dis 2018; WHO catalogue of MTB mutations (2nd edition, 2023).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Rapid molecular drug resistance testing (genotypic tests) for tuberculosis. *[DNB Dec 2021 P3]*
2. Describe rapid molecular tests for diagnosis of drug resistant TB. *[WBUHS 2020 P3]*
3. Write a short note on genotypic tests for drug-resistant tuberculosis. *[BFUHS 2022 P1]*
4. Whole genome sequencing (WGS). *[WBUHS 2022 P3]*
5. Discuss molecular methods in diagnosis of tuberculosis and their clinical utility. *[WBUHS 2022 P3]*
6. Newer molecular methods for assessing drug resistance in tuberculosis. *[BFUHS 2019 P2]*
7. GeneXpert Ultra. *[BFUHS 2023 P2]*
8. Polymerase chain reaction. *[Manipal 2014 P3]*

3.3 Respiratory Viruses & Their Laboratory Diagnosis

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-C8 Respiratory viruses
- ✓ Classify respiratory viruses and the syndromes they produce
- ✓ Describe methods for laboratory diagnosis of respiratory viral infections

IN BRIEF Viruses cause most upper respiratory infections and 20–30% of adult CAP. Multiplex PCR has transformed diagnosis; influenza, SARS-CoV-2 and RSV are the clinically actionable targets in adults.

Respiratory viruses

Virus	Family	Clinical syndromes	Specific therapy / prevention
Influenza A, B	Orthomyxoviridae	Influenza, primary viral and secondary bacterial pneumonia, ARDS	Oseltamivir, baloxavir; annual vaccine

INVESTIGATIONS & TREATMENT AT A GLANCE

3.2 Molecular Diagnosis of TB — CBNAAT, Truenat, LPA & Whole-Genome Sequencing

‹ Back to topic (p 51)
Next topic 3.3 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Xpert MTB/RIF Ultra	MTB not detected	MTB detected (high/medium/low/very low/trace); Rif resistance detected	LOD ~ 16 CFU/mL; trace = treat in PLHIV/children/EPTB
Truenat MTB Plus / MTB-RIF Dx	Not detected	Detected	Chip-based, battery-operated — peripheral labs
Xpert MTB/XDR	Susceptible	Resistance to INH, FQ, ethionamide, amikacin	Reflex test after TB detected
FL-LPA	Wild type bands present	Missing WT / mutation bands (rpoB, katG, inhA)	R and H resistance
SL-LPA	Susceptible	gyrA/gyrB, rrs, eis mutations	FQ and SLID resistance
Targeted NGS / WGS	No resistance mutations	Catalogued mutations	Comprehensive DST incl. Bdq, Lzd (WHO 2025)

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Interpret Rif-resistant Xpert	Start MDR/RR-TB regimen	RR-TB detected	Confirm with second sample in low-risk patients
Repeat Xpert	Fresh sample	Indeterminate / error	—
Hr-TB regimen	6(Lfx)REZ	INH resistance detected, Rif susceptible	Exclude FQ resistance

★ **PRESCRIBING & INTERPRETATION PEARLS**

- NAAT may stay positive after cure — use culture, not Xpert, to diagnose recurrence.

Virus	Family	Clinical syndromes	Specific therapy / prevention
SARS-CoV-2	Coronaviridae	COVID-19 pneumonia, ARDS, thrombosis	Nirmatrelvir/r, remdesivir, dexamethasone; vaccine
RSV	Pneumoviridae	Bronchiolitis in infants; pneumonia in elderly, transplant	Ribavirin (selected); RSV vaccines (RSVPreF3, RSVpreF) ≥60–75 years; nirsevimab (infants)
hMPV	Pneumoviridae	Bronchiolitis, pneumonia	Supportive
Parainfluenza 1–3	Paramyxoviridae	Croup, bronchiolitis	Supportive
Adenovirus	Adenoviridae	Pharyngoconjunctival fever, pneumonia (military recruits), bronchiolitis obliterans	Cidofovir (severe)
Rhinovirus	Picornaviridae	Common cold, asthma/COPD exacerbations	Supportive
Measles, varicella	Paramyxo-/ Herpesviridae	Giant-cell pneumonia; varicella pneumonia (adults, pregnancy)	Acyclovir for VZV
CMV, HSV	Herpesviridae	Pneumonitis in transplant, HIV	Ganciclovir/valganciclovir, letermovir prophylaxis
Hantavirus	Hantaviridae	Hantavirus cardiopulmonary syndrome	Supportive/ECMO
Nipah	Paramyxoviridae	Encephalitis with ARDS (Kerala outbreaks)	Supportive; m102.4 antibody (compassionate)

Diagnostic methods

Method	Principle	Notes
Multiplex RT-PCR panels (BioFire, etc.)	Nucleic acid amplification	Highest sensitivity; 1–2 h; detects co-infection
Rapid antigen tests	Immunochromatography	Low sensitivity for influenza; useful for SARS-CoV-2 early
Viral culture / shell vial	Cell lines with CPE	Slow; research, resistance
Serology	Paired sera fourfold rise	Retrospective; epidemiology
Histology/cytology	Inclusion bodies (CMV owl's-eye, Warthin–Finkeldey cells in measles)	Biopsy or BAL

- Specimens: nasopharyngeal swab (flocked), mid-turbinate, BAL (lower tract), in VTM at 2–8 °C.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Kerala Nipah outbreaks (2018–2024) — know transmission (fruit bats, person-to-person), BSL-4 handling, NIV Pune testing.

3.4 Respiratory Fungi — Classification & Laboratory Diagnosis

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ **NMC syllabus area:** I-C9 Respiratory fungi
✓ Classify fungal diseases of the lung
✓ Describe laboratory methods for diagnosis of pulmonary mycoses

IN BRIEF Pulmonary fungi are either opportunistic moulds and yeasts (Aspergillus, Mucorales, Candida, Cryptococcus, Pneumocystis) or endemic dimorphic fungi (Histoplasma, Blastomyces, Coccidioides, Talaromyces). Actinomyces and Nocardia are bacteria traditionally grouped with fungi.

INVESTIGATIONS & TREATMENT AT A GLANCE

3.3 Respiratory Viruses & Their Laboratory Diagnosis

‹ Back to topic (p 52)
Next topic 3.4 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Multiplex respiratory PCR panel	Not detected	Influenza, RSV, hMPV, SARS-CoV-2, adenovirus etc.	Rapid aetiology; cohorting
SARS-CoV-2 RT-PCR (Ct value)	Not detected	Ct < 25 high load	Infectivity estimate (lab-dependent)
Rapid antigen test (influenza/COVID)	Negative	Positive	High specificity, moderate sensitivity
CMV PCR (blood/BAL)	< 500 IU/mL (lab-specific)	Rising load; BAL high load + histology	CMV pneumonitis in transplant
Procalcitonin	< 0.25 ng/mL	> 0.5 ng/mL	Suggests bacterial co-infection
Lymphocyte count	1.0–4.0 × 10^9/L	< 1.0	Severe viral (COVID) disease marker

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Oseltamivir	75 mg twice daily × 5 days (renal adjust)	Influenza — high-risk or hospitalised (start ≤ 48 h, later if severe)	Nausea; neuropsychiatric (rare)
Remdesivir	200 mg IV day 1, then 100 mg daily × 4 days	Hospitalised COVID-19 needing O_2	LFTs, eGFR
Ganciclovir	5 mg/kg IV 12-hourly	CMV pneumonitis	Myelosuppression, renal dosing
Ribavirin (aerosolised/oral)	Per protocol	Severe RSV in transplant	Teratogenic

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Viral PCR positivity alone does not exclude bacterial co-infection — use clinical picture and procalcitonin.

Classification of pulmonary mycoses

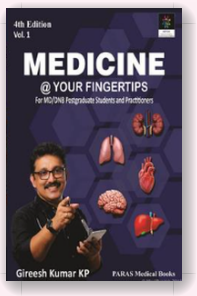
Group	Organisms	Pulmonary disease
Opportunistic moulds	Aspergillus, Mucorales (Rhizopus, Mucor, Lichtheimia), Fusarium, Scedosporium	IPA, CPA, ABPA, mucormycosis
Opportunistic yeasts	Candida, Cryptococcus	Candidaemia (pneumonia rare); cryptococcal pneumonia/meningitis
Atypical fungus	Pneumocystis jirovecii	PJP in HIV, steroids, transplant
Endemic dimorphic	Histoplasma (India — Gangetic plains, NE), Blastomyces, Coccidioides, Paracoccidioides, Talaromyces marneffei (NE India), Sporothrix	Primary pneumonia, chronic cavitary, disseminated
Fungus-like bacteria	Actinomyces (anaerobic, sulphur granules), Nocardia (aerobic, partially acid-fast)	Chronic pneumonia, empyema necessitans, brain abscess

Laboratory diagnosis

Method	Examples	Comments
Direct microscopy	KOH, calcofluor white (fluorescent)	Aspergillus: septate acute-angle branching; Mucor: broad aseptate right-angle ribbons
Stains	GMS, PAS (tissue); India ink, mucicarmine (Cryptococcus); toluidine/Giemsa/IF (Pneumocystis)	Histology proves invasion
Culture	Sabouraud dextrose agar (± antibiotics); BHI for dimorphs	Species and susceptibility; Mucorales may not grow from tissue (grinding)
Antigen	Galactomannan (serum >0.5, BAL ≥1.0); β-D-glucan (not Mucor/Crypto); cryptococcal antigen (LFA); Histoplasma urine antigen	GM sensitivity lower in non-neutropenic
Antibody	Aspergillus-specific IgG (CPA), IgE (ABPA)	Immunocompetent hosts
Molecular	Aspergillus PCR, Mucorales PCR, Pneumocystis qPCR	High NPV
Imaging	Halo sign, air crescent, reversed halo (mucor)	Supportive

★ CLINICAL PEARLS & EXAM FOCUS

- β-D-glucan is negative in mucormycosis and cryptococcosis — "the two Ms don't glucan" (Mucor and Cryptococcus lack/shield it).



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3.5 Bacteria, Atypical Organisms, Anaerobes & Biomarkers of Infection

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-C2, I-C3 Bacteria and laboratory diagnosis
- ✓ List bacteria, atypical organisms and anaerobes causing respiratory infection
- ✓ Describe specimens and tests for non-tuberculous respiratory infection
- ✓ Use biomarkers (procalcitonin, CRP) to guide antibiotic therapy

INVESTIGATIONS & TREATMENT AT A GLANCE

3.4 Respiratory Fungi — Classification & Laboratory Diagnosis

‹ Back to topic (p 53)
Next topic 3.5 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Serum galactomannan	Index < 0.5	≥ 0.5 (serum), ≥ 1.0 (BAL)	Invasive aspergillosis
β-D-glucan	< 60 pg/mL (< 80 indeterminate)	≥ 80 pg/mL	Pan-fungal (not Mucorales/ Cryptococcus)
Aspergillus-specific IgG	Below kit cut-off (e.g. < 27 mg/L ImmunoCAP)	Raised	Chronic pulmonary aspergillosis
Cryptococcal antigen (serum/CSF)	Negative	Positive (titre)	Cryptococcosis; screen if CD4 < 100
KOH / calcofluor microscopy	No fungi	Septate acute-angle branching (Aspergillus); broad aseptate right-angle (Mucorales)	Rapid morphology
Histoplasma urinary antigen	Negative	Positive	Disseminated histoplasmosis

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Voriconazole	6 mg/kg IV 12-hourly × 2 then 4 mg/kg; oral 200 mg 12-hourly	Invasive aspergillosis	TDM trough 1–5.5 mg/L; visual, hepatic, skin; rifampicin contraindicated
Liposomal amphotericin B	3–5 mg/kg/day (5–10 for mucormycosis)	Mucormycosis, cryptococcosis, severe histoplasmosis	Nephrotoxicity, hypokalaemia
Itraconazole	200 mg twice daily	CPA, ABPA, histoplasmosis	TDM > 0.5–1 mg/L; heart failure
Isavuconazole	200 mg 8-hourly × 6 doses, then 200 mg daily	Aspergillosis/mucormycosis (alternative)	QT shortening

★ PRESCRIBING & INTERPRETATION PEARLS

- Galactomannan is less sensitive in non-neutropenic patients and those on mould-active prophylaxis.

IN BRIEF Diagnosis of bacterial pneumonia relies on good-quality sputum, blood cultures, urinary antigens and molecular panels; procalcitonin helps distinguish bacterial from viral infection and guides antibiotic duration.

Respiratory pathogens

Group	Organisms	Key features
Typical bacteria	S. pneumoniae, H. influenzae, M. catarrhalis, S. aureus (MSSA/MRSA), K. pneumoniae	Lobar pneumonia; Klebsiella in alcoholics (red-currant jelly sputum, bulging fissure)
Gram-negative (nosocomial)	P. aeruginosa, Acinetobacter, Enterobacterales (ESBL, CRE)	HAP/VAP, bronchiectasis
Atypical	Mycoplasma, Chlamydophila pneumoniae, Legionella, C. psittaci, Coxiella	Extrapulmonary features; β-lactams ineffective
Tropical	Burkholderia pseudomallei, Orientia tsutsugamushi, Leptospira	Melioidosis (diabetics), scrub typhus ARDS, leptospiral pulmonary haemorrhage
Anaerobes	Peptostreptococcus, Prevotella, Fusobacterium, Bacteroides	Aspiration, lung abscess, empyema (putrid)

Laboratory diagnosis

Test	Comment
Sputum Gram stain & culture	Good sample: >25 PMN and <10 squamous cells per LPF (Bartlett/Murray–Washington)
Blood cultures	Positive in 5–15% of CAP; take before antibiotics in severe CAP
Urinary antigen	Pneumococcal (C-polysaccharide); Legionella serogroup 1
Multiplex PCR	Atypicals, viruses; quantitative bacterial panels in BAL
Quantitative cultures	BAL ≥10^4 CFU/mL, PSB ≥10^3, ETA ≥10^5–10^6 (VAP)
Serology	Mycoplasma IgM, scrub typhus IgM ELISA, leptospira MAT
Pleural fluid	pH, glucose, culture in blood-culture bottles

Biomarkers

- **Procalcitonin:** calcitonin precursor released from parenchymal cells under bacterial endotoxin/IL-6 (suppressed by IFN-γ in viral infection); rises in 3–6 h, peaks 12–24 h. <0.25 ng/mL bacterial unlikely; >0.5 likely; stopping antibiotics when PCT falls ≥80% or <0.5 safely reduces exposure (SAPS, ProHOSP). Not to withhold antibiotics in severe CAP (IDSA 2019).
- **CRP:** sensitive, non-specific; >100 mg/L supports bacterial pneumonia; NICE uses CRP <20 to avoid antibiotics in primary care.
- Others: presepsin, sTREM-1, IL-6, MR-proADM (prognosis), lactate.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Procalcitonin is falsely raised in trauma, surgery, burns, medullary thyroid cancer, SCLC, cardiogenic shock; may be low in early or localised infection.

LANDMARK TRIALS & GUIDELINES

- de Jong E et al, SAPS trial, Lancet Infect Dis 2016 — PCT-guided stopping reduced antibiotic days and mortality.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Biomarkers of infection. *[NEET-SS/DNB 2024]*
2. Procalcitonin. *[VNSGU 2025 P2]*

INVESTIGATIONS & TREATMENT AT A GLANCE

3.5 Bacteria, Atypical Organisms, Anaerobes & Biomarkers of Infection

‹ Back to topic (p 54)
Next topic 3.6 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Procalcitonin	< 0.1 ng/mL	> 0.25 (bacterial likely); > 2 (sepsis)	Antibiotic start/stop guidance
CRP	< 5 mg/L	> 100 mg/L	Bacterial pneumonia likely
Sputum Gram stain quality	< 10 epithelial cells and > 25 PMNs per LPF	> 10 epithelial cells	Rejected (saliva)
Blood culture	No growth	Positive	Bacteraemia — in all severe CAP
Urinary antigens	Negative	Pneumococcal or Legionella serogroup 1 positive	Targeted therapy
Mycoplasma PCR / IgM	Negative	Positive	Atypical pneumonia
Melioidosis culture (Ashdown medium)	No growth	*B. pseudomallei*	Diabetics, monsoon season (India)

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Amoxicillin	1 g 8-hourly oral	Non-severe CAP	Penicillin allergy
Ceftriaxone + azithromycin	2 g IV daily + 500 mg daily	Hospitalised CAP	QTc with macrolide
Doxycycline	100 mg twice daily (200 mg first dose)	Atypical pathogens, CAP alternative	Photosensitivity
Ceftazidime / meropenem	Ceftazidime 2 g 6–8-hourly; meropenem 1 g 8-hourly	Melioidosis intensive phase (≥ 2 weeks)	Then co-trimoxazole eradication 3–6 months
Metronidazole / clindamycin	Metronidazole 500 mg 8-hourly; clindamycin 600 mg 8-hourly	Anaerobic infection	C. difficile

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Procalcitonin < 0.25 ng/mL supports stopping antibiotics in stable patients.

3.6 Pathology — Inflammation, Granuloma & Emphysema

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-D1, I-D2, I-D4 Pathology
- ✓ Describe pathogenetic mechanisms of acute and chronic inflammation in the lung
- ✓ Describe granuloma formation in tuberculosis and the types of granulomas
- ✓ Describe types of emphysema and the pathology of chronic bronchitis

IN BRIEF Lung injury is followed by inflammation — neutrophil-predominant acute exudation, or chronic inflammation with macrophages, lymphocytes and fibrosis. The granuloma is a specialised chronic response to persistent antigens; emphysema is destruction of alveolar walls distal to terminal bronchioles without obvious fibrosis.

Granuloma formation in tuberculosis

- Alveolar macrophages phagocytose bacilli → cytokines (TNF-α, IL-12) → recruit monocytes, T cells → Th1 response (IFN-γ) activates macrophages → transform to **epithelioid cells**, fuse into **Langhans giant cells** (peripheral horseshoe nuclei) → lymphocyte cuff, fibroblasts → central **caseous necrosis** (lipid-rich, hypoxic).
- TNF-α is essential to maintain granulomas — anti-TNF therapy reactivates TB.
- Outcomes: healing with fibrosis and calcification (Ghon focus), liquefaction and cavitation (bacilli multiply in cavity — transmission), dissemination.

Granulomas in the lung

Type	Examples	Clue
Necrotising (caseating)	TB, histoplasmosis, cryptococcus, NTM	Infection — stain for AFB/fungi
Non-necrotising (naked, well-formed)	Sarcoidosis, berylliosis, MAC hot-tub lung	Perilymphatic, asteroid/Schaumann bodies
Poorly formed	Hypersensitivity pneumonitis, CVID-GLILD, drug reactions	Bronchiolocentric
Necrotising with vasculitis	GPA (geographic necrosis), EGPA (eosinophils)	ANCA
Foreign body	Aspiration, talc (IV drug use)	Birefringent particles

Emphysema

Types of emphysema

Type	Location	Association
Centriacinar (centrilobular)	Respiratory bronchioles; upper lobes	Smoking, CWP
Panacinar (panlobular)	Entire acinus; lower lobes	α1-antitrypsin deficiency, IV Ritalin
Paraseptal (distal acinar)	Subpleural, along septa	Spontaneous pneumothorax, bullae in young
Irregular (paracicatricial)	Adjacent to scars	TB, silicosis

- **Pathogenesis:** protease–antiprotease and oxidant–antioxidant imbalance, apoptosis of alveolar cells, autoimmunity, impaired repair.

Chronic bronchitis and asthma pathology

- **Chronic bronchitis:** mucous gland hypertrophy (**Reid index** >0.4 — gland thickness/wall thickness), goblet cell hyperplasia, squamous metaplasia, small airway fibrosis and plugging.
- **Asthma:** epithelial shedding (Creola bodies, Curschmann spirals), thickened basement membrane (sub-epithelial collagen), eosinophils, Charcot–Leyden crystals, smooth muscle hypertrophy, mucus plugs.

INVESTIGATIONS & TREATMENT AT A GLANCE

3.6 Pathology — Inflammation, Granuloma & Emphysema

‹ Back to topic (p 56)
Next topic 3.7 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Histology — granuloma	—	Caseating (TB) vs non-caseating (sarcoid)	Granulomatous inflammation pattern
AFB / GMS / PAS stains	Negative	Organisms seen	Infectious granuloma
Emphysema on CT	LAA-950 < 5%	> 5–10% low-attenuation area	Emphysema quantification
α1-antitrypsin	1.0–2.0 g/L	< 0.57 g/L	Panlobular basal emphysema
ESR	< 20 mm/h	> 100 mm/h	TB, malignancy, vasculitis, myeloma

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Corticosteroids (granulomatous inflammation)	Prednisolone 20–40 mg/day	Sarcoidosis, HP	Exclude TB/fungus first
Smoking cessation	Varenicline 1 mg twice daily × 12 weeks	Emphysema progression	Neuropsychiatric (rare)
Antimicrobials for infectious granuloma	ATT / antifungals per organism	TB, fungal	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Always send tissue for culture/NAAT along with histology — granulomas are not specific.

★ CLINICAL PEARLS & EXAM FOCUS

- Reid index >0.4 = chronic bronchitis; Langhans giant cell nuclei are peripheral horseshoe; foreign-body giant cell nuclei are scattered.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Types of lung emphysema. *[DNB Dec 2022 P1]*
2. Granuloma formation in tuberculosis. *[DNB Dec 2022 P1]*

3.7 Hypersensitivity, Allergy Testing & Immunology of Lung Disease

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-F1–3 Allergy and immunology
- ✓ Classify hypersensitivity reactions with pulmonary examples
- ✓ Describe in-vivo and in-vitro allergy tests including skin prick testing
- ✓ Explain the role of eosinophils and the hygiene hypothesis

IN BRIEF The Gell–Coombs scheme classifies immune injury into four types; most pulmonary immunological diseases combine types (e.g., ABPA I + III + IV). Allergy testing identifies specific IgE sensitisation that must be correlated with clinical history.

Hypersensitivity reactions in lung disease

Type	Mechanism	Pulmonary examples
I (immediate)	IgE on mast cells → histamine, leukotrienes	Allergic asthma, rhinitis, anaphylaxis, ABPA (part)
II (cytotoxic)	IgG/IgM against tissue antigen	Goodpasture (anti-GBM), transfusion lung injury (TRALI — anti-HLA)
III (immune complex)	Antigen–antibody deposits, complement	Hypersensitivity pneumonitis (acute), SLE, ABPA, cryoglobulinaemia
IV (delayed, T-cell)	Th1 / CD8 cells, macrophages	Tuberculosis, sarcoidosis, HP (chronic), berylliosis, Mantoux test

Allergy tests

Diagnostic tests in allergic lung disease

Test	Principle	Interpretation / comments
Skin prick test (SPT)	Allergen drop + lancet; histamine (+) and saline (–) controls; read at 15 min	Wheal ≥3 mm over negative control = sensitisation; stop antihistamines 3–7 days
Intradermal test	0.02 mL intradermal	More sensitive, more false positives; drugs, venom
Patch test	Delayed type IV	Contact allergy
Total IgE	ELISA	>1000 IU/mL suggests ABPA (criterion >500 in newer criteria)
Specific IgE (ImmunoCAP)	Fluoroenzyme immunoassay	A. fumigatus sIgE >0.35 kUA/L; component-resolved diagnosis
Precipitins / specific IgG	Immunodiffusion, ELISA	HP antigens, CPA
Basophil activation test	Flow cytometry	Drug allergy research
Provocation tests	Bronchial, nasal, oral	Gold standard; specialist settings

Eosinophils in lung disease

- Develop under IL-5 (GM-CSF, IL-3); recruited by eotaxin (CCR3); granule proteins (MBP, ECP, EPO, EDN) damage epithelium; release leukotrienes, cytokines; also tissue repair roles.
- Diseases: asthma (type-2 high), ABPA, EGPA, eosinophilic pneumonias, TPE, HES, drug reactions, parasitic (Löffler), eosinophilic COPD phenotype.
- Targets: IL-5 (mepolizumab, reslizumab), IL-5R (benralizumab — depletes eosinophils), IL-4Rα (dupilumab), TSLP (tezepelumab).

Hygiene hypothesis

- Reduced early microbial exposure (small families, urban living, antibiotics, caesarean birth) → Th2-skewed immunity and impaired regulatory T cells → allergy and asthma (Strachan 1989).
- Evolved into the "old friends"/microbiome hypothesis — farm exposure (Amish vs Hutterite children) protects.

Immunology of tuberculosis

- Th1 (IFN-γ, TNF-α, IL-12) protective; IL-10 and Tregs downregulate; Th17 in early recruitment; CD8 cytotoxicity; B cells/antibodies minor role. Latency = containment, not sterilisation.
- Defects predisposing to TB: HIV (CD4 loss), anti-TNF, steroids, IL-12/IFN-γ axis defects (MSMD), diabetes, malnutrition.

★ CLINICAL PEARLS & EXAM FOCUS

▸ ABPA involves type I (IgE), III (precipitins) and IV (granulomas) — say so in exam answers.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. What is the role of eosinophil in lung disease? *[BFUHS 2022 P1]*
2. Skin prick allergy testing. *[BFUHS 2020 P4]*
3. Hygiene hypothesis. *[Manipal 2014 P4]*

3.8 Pharmacology of First-Line Anti-TB Drugs & Drug-Induced Liver Injury

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-G2 Antitubercular drugs
- ✓ Describe mechanism, doses, adverse effects and interactions of first-line anti-TB drugs
- ✓ Manage anti-TB drug-induced hepatotoxicity as per NTEP

IN BRIEF Isoniazid, rifampicin, pyrazinamide and ethambutol form the backbone of drug-susceptible TB treatment. Hepatotoxicity (H, R, Z) is the most important adverse effect and needs structured reintroduction.

First-line anti-TB drugs

Drug	Mechanism	Dose (daily)	Key adverse effects	Notes
Isoniazid (H)	Prodrug activated by KatG; inhibits InhA (mycolic acid synthesis); bactericidal for rapidly dividing	5 mg/kg (max 300 mg); high-dose 10–15 mg/kg	Hepatitis, peripheral neuropathy (pyridoxine 10–25 mg), psychosis, SLE-like, seizures	Slow acetylators (NAT2) → toxicity; inhibits CYP2C19/3A4 (phenytoin)
Rifampicin (R)	Binds β-subunit of RNA polymerase (rpoB); sterilising	10 mg/kg (max 600); high dose 20–35 under study	Hepatitis (cholestatic), flu-like, thrombocytopenia, haemolysis, renal failure (intermittent), orange secretions	Potent CYP3A4/P-gp inducer — OCPs, warfarin, PIs, steroids, azoles
Pyrazinamide (Z)	Pyrazinoic acid (pncA) disrupts membrane energetics; acid pH semi-dormant	25 mg/kg	Hepatitis (dose-related), hyperuricaemia/gout, arthralgia, rash	Most hepatotoxic of the three

INVESTIGATIONS & TREATMENT AT A GLANCE

3.7 Hypersensitivity, Allergy Testing & Immunology of Lung Disease

‹ Back to topic (p 57)
Next topic 3.8 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Total IgE	< 100 IU/mL	> 500 IU/mL (ABPA)	Atopy, ABPA, parasites
Specific IgE (e.g. Aspergillus)	< 0.35 kUA/L	≥ 0.35 kUA/L	Sensitisation
Skin-prick test	Wheal < 3 mm	≥ 3 mm larger than negative control	Sensitisation (stop antihistamines 72 h)
Blood eosinophils	< 300/µL	≥ 300 (type-2 asthma); > 500 ABPA; > 1500 hypereosinophilia	Phenotyping, biologics
Serum precipitins (HP panel)	Negative	Positive	Exposure (not disease) in HP
ANA / RF / anti-CCP / ANCA	Negative	Positive	CTD-ILD, vasculitis
Complement C3/C4	C3 0.9–1.8; C4 0.1–0.4 g/L	Low	SLE, immune complex disease

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Antihistamine (cetirizine)	10 mg daily	Allergic rhinitis	Sedation (mild)
Adrenaline	0.5 mg IM (1:1000) anterolateral thigh, repeat 5 min	Anaphylaxis	Monitor; second-line antihistamine/ steroid
Omalizumab	Dose by IgE (30–1500 IU/mL) and weight, SC every 2–4 weeks	Severe allergic asthma	Anaphylaxis (observe)
Allergen immunotherapy	SCIT/SLIT 3–5 years	Allergic rhinitis ± controlled asthma	Uncontrolled asthma — contraindication

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Precipitins indicate exposure — HP needs compatible HRCT, BAL lymphocytosis or histology.

Drug	Mechanism	Dose (daily)	Key adverse effects	Notes
Ethambutol (E)	Inhibits arabinosyl transferase (embB)	15 mg/kg	Optic neuritis (red-green colour, acuity)	Bacteriostatic; renal dose adjust; check vision
Streptomycin (S)	30S ribosome	15 mg/kg IM	Ototoxicity (vestibular), nephrotoxicity	Rarely used

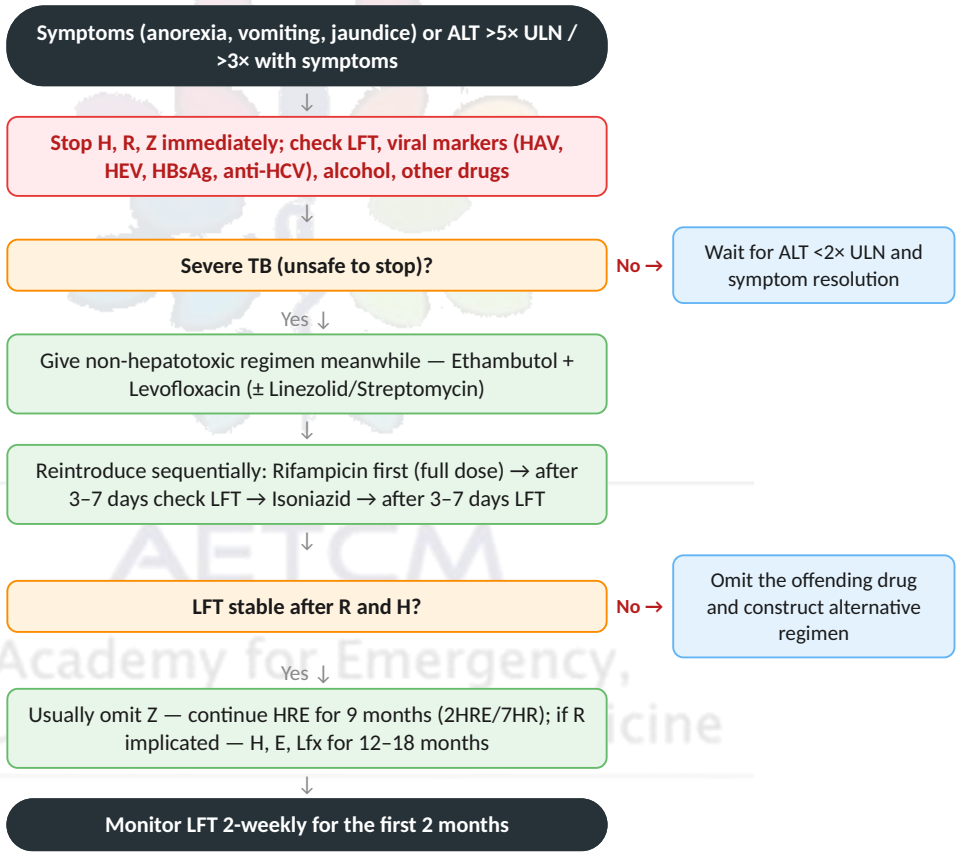
Isoniazid in detail

- Pharmacokinetics: oral, crosses CSF; acetylated by NAT2 — slow acetylators (~50–60% Indians) have higher hepatotoxicity and neuropathy.
- Toxicity: hepatitis (age >35, alcohol, HBV/HCV, pregnancy/postpartum), neuropathy (pyridoxine), overdose → seizures, lactic acidosis (pyridoxine gram for gram).
- Uses: treatment, TPT (6H, 3HP, 1HP), high-dose for inhA mutation.

DILI — definition and NTEP management

- **Definition (ATS):** ALT >3× ULN with symptoms (or jaundice), or >5× ULN without symptoms; bilirubin >2× ULN.

Algorithm 3.1 · Management of anti-TB drug-induced hepatotoxicity



★ CLINICAL PEARLS & EXAM FOCUS

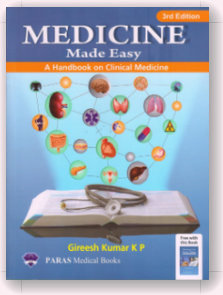
- Pyridoxine to all on INH under NTEP if risk (pregnancy, HIV, diabetes, alcohol, malnutrition, CKD) — many programmes give universally.

LANDMARK TRIALS & GUIDELINES

- Saukkonen JJ et al, ATS statement on hepatotoxicity of anti-TB therapy, AJRCCM 2006; NTEP Guidelines for Programmatic Management of TB (2021, updates 2024).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Isoniazid. *[Manipal 2014 P3]*
2. How will you manage a case of antitubercular drug induced jaundice? *[WBUHS 2022 P3]*



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3.9 Second-Line & Newer Anti-TB Drugs — Bedaquiline, Delamanid, Pretomanid, Linezolid, Fluoroquinolones

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-G2 Antitubercular drugs
- ✓ Describe mechanism, dose, adverse effects and monitoring of newer and repurposed anti-TB drugs
- ✓ Explain the role of fluoroquinolones in TB treatment

IN BRIEF Bedaquiline, pretomanid, delamanid, linezolid, fluoroquinolones and clofazimine have transformed DR-TB into an all-oral, shorter, more successful treatment. QT prolongation, myelosuppression and neuropathy require structured monitoring.

Key second-line and newer drugs

Drug	Class / mechanism	Adult dose	Major toxicity	Monitoring
Bedaquiline (Bdq)	Diarylquinoline — inhibits mycobacterial ATP synthase (atpE)	400 mg OD ×2 weeks then 200 mg thrice weekly ×22 weeks (or 200 mg OD ×8 weeks then 100 mg OD)	QT prolongation, hepatitis, arthralgia	ECG, K/Mg/Ca; long half-life (5.5 months)
Pretomanid (Pa)	Nitroimidazo-oxazine; inhibits mycolic acid synthesis and respiratory poison (NO release)	200 mg OD	Hepatotoxicity, myelosuppression (with LZD), neuropathy, ovarian/testicular effects (animal)	LFT
Delamanid (Dlm)	Nitro-dihydro-imidazooxazole; mycolic acid (methoxy-/keto-) inhibition	100 mg BD (or 300 OD)	QT prolongation (mild), psychiatric	ECG
Linezolid (Lzd)	Oxazolidinone — 50S ribosome initiation	600 mg OD (reduced to 300 if toxicity)	Myelosuppression, peripheral and optic neuropathy, lactic acidosis, serotonin syndrome	CBC weekly initially, vision
Levofloxacin / Moxifloxacin	FQ — DNA gyrase (gyrA/B)	Lfx 750–1000 mg; Mfx 400 mg (high-dose 600–800)	QT (Mfx>Lfx), tendinopathy, dysglycaemia, CNS	ECG, glucose
Clofazimine (Cfz)	Riminophenazine — membrane, ROS	100 mg OD	Skin discolouration (pink–brown), ichthyosis, QT, GI	ECG
Cycloserine / terizidone	D-alanine analogue — cell wall	10–15 mg/kg	Psychosis, depression, seizures	Mental status; pyridoxine
Ethionamide	InhA (ethA-activated)	15–20 mg/kg	GI upset, hypothyroidism, hepatitis	TFT
Amikacin	Aminoglycoside	15 mg/kg	Ototoxicity, nephrotoxicity	Audiometry; rarely used now

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INVESTIGATIONS & TREATMENT AT A GLANCE

3.8 Pharmacology of First-Line Anti-TB Drugs & Drug-Induced Liver Injury

‹ Back to topic (p 58)
Next topic 3.9 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
LFT baseline (ALT/AST, bilirubin)	ALT < 40 U/L; bilirubin < 1.2 mg/dL	ALT > 3× ULN with symptoms or > 5× without; bilirubin > 2×	Stop hepatotoxic ATT (DILI)
Visual acuity & colour vision (Ishihara)	6/6, all plates read	Reduced acuity, red-green defect	Ethambutol optic neuritis — stop
Serum uric acid	3.5–7.2 mg/dL	Raised (asymptomatic common)	Pyrazinamide — treat only if gout
Serum creatinine / eGFR	> 60 mL/min	< 30 mL/min	Adjust E, Z (thrice weekly)
Platelet count	150–400 × 10^9/L	< 100	Rifampicin thrombocytopenia — stop permanently
HBsAg / anti-HCV	Negative	Positive	Higher DILI risk — closer monitoring

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Isoniazid	5 mg/kg (max 300 mg)	DS-TB	Pyridoxine 10–25 mg; hepatitis, neuropathy
Rifampicin	10 mg/kg (max 600 mg)	DS-TB	CYP inducer; orange secretions; flu-like
Pyrazinamide	25 mg/kg	DS-TB intensive phase	Hepatotoxic, gout
Ethambutol	15 mg/kg	DS-TB	Optic neuritis; renal dosing
DILI reintroduction	R first (full dose) → H after 3–7 days → Z only if mild	After ALT < 2× ULN	Interim E + Lfx (± Lzd) if severe TB

★ **PRESCRIBING & INTERPRETATION PEARLS**

- NTEP 4FDC (H75/R150/Z400/E275): 25–34 kg 2 tabs, 35–49 kg 3, 50–64 kg 4, 65–75 kg 5, > 75 kg 6.

Fluoroquinolones in tuberculosis

- Group A drugs for DR-TB; Mfx in 4-month rifapentine regimen (4HPMZ — Study 31) for DS-TB; Lfx in TPT for MDR contacts (V-QUIN, TB-CHAMP).
- Resistance (gyrA) defines **pre-XDR-TB** (RR + FQ resistance). Avoid empirical FQ for CAP without excluding TB — masks diagnosis and breeds resistance.

Pretomanid

- Approved 2019 in BPaL (Nix-TB); BPaLM (TB-PRACTECAL) with moxifloxacin; India (NTEP) introduced BPaLM in 2024 for MDR/RR-TB ≥14 years.
- Not for children <14, pregnancy (limited data); lactation caution.

✦ RECENT ADVANCES & GUIDELINE UPDATES

▸ WHO 2025: BDLLfxC 6-month regimen (Bdq, Dlm, Lzd, Lfx, Cfz) for MDR/RR-TB including children and pregnancy — from BEAT-Tuberculosis and endTB trials.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Bedaquiline + delamanid + clofazimine + moxifloxacin together = additive QT effect — baseline and monthly ECG; stop if QTcF >500 ms.

LANDMARK TRIALS & GUIDELINES

- Conradie F et al, Nix-TB NEJM 2020; ZeNix NEJM 2022 (lower LZD 600 mg); Nyang'wa BT et al, TB-PRACTECAL NEJM 2022.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Bedaquiline and delamanid. *[DNB Oct 2024 P3]*
2. Delamanid. *[WBUHS 2020 P3]*
3. Pretomanid. *[WBUHS 2022 P3; VNSGU 2025 P1]*
4. Fluoroquinolones in tuberculosis. *[Manipal 2014 P3]*
5. New anti-tuberculosis drugs. *[Manipal 2014 P3]*

3.10 Bronchodilators & Inhaled Corticosteroids

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ **NMC syllabus area:** I-G4 Bronchodilator and anti-inflammatory drugs
✓ Classify bronchodilators and describe the pharmacology of β2-agonists (incl. ultra-LABA), antimuscarinics and methylxanthines
✓ Describe the pharmacology, benefits and adverse effects of inhaled corticosteroids

IN BRIEF β2-agonists relax airway smooth muscle via cAMP; antimuscarinics block M3-mediated bronchoconstriction and mucus secretion; ICS suppress type-2 inflammation. Ultra-long-acting once-daily agents and fixed-dose combinations improve adherence.

Classification of bronchodilators

Class	Short-acting	Long-acting (12 h)	Ultra-long-acting (24 h)
β2-agonist	Salbutamol, terbutaline, levosalbutamol	Salmeterol, formoterol (fast onset)	Indacaterol, olodaterol, vilanterol
Antimuscarinic	Ipratropium	Aclidinium, glycopyrronium (BD in some)	Tiotropium, umeclidinium, glycopyrronium, revefenacin
Methylxanthine	Aminophylline	Theophylline SR, doxofylline	—

INVESTIGATIONS & TREATMENT AT A GLANCE

3.9 Second-Line & Newer Anti-TB Drugs — Bedaquiline, Delamanid, Pretomanid, Linezolid, Fluoroquinolones

‹ Back to topic (p 60)
Next topic 3.10 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
ECG QTcF	< 450 ms (men), < 470 (women)	> 500 ms	Stop QT drugs (Bdq, Dlm, Cfz, Mfx)
Serum K^+, Mg^{2+}, Ca^{2+}	K 3.5–5; Mg 1.7–2.4 mg/dL	Low	Correct before QT drugs
CBC (linezolid)	Hb > 10 g/dL; platelets > 150	Hb < 8, platelets < 75, ANC < 1000	Linezolid myelosuppression — hold/reduce
Peripheral neuropathy screen (BPNS)	Normal	Grade ≥ 2	Linezolid/cycloserine — reduce/stop
Visual acuity/colour vision	Normal	Optic neuropathy	Stop linezolid
TSH	0.4–4.0 mIU/L	> 10	Ethionamide/PAS hypothyroidism
Audiometry	Normal	Hearing loss	Aminoglycoside toxicity

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Bedaquiline	400 mg daily × 2 wk, then 200 mg thrice weekly × 22 wk (or 200 mg daily × 8 wk then 100 mg daily)	MDR/RR-TB	QT, hepatotoxicity; take with food
Pretomanid	200 mg daily	BPaL(M)	Hepatotoxicity; not in pregnancy
Delamanid	100 mg twice daily	BDLLfxC, longer regimens	QT
Linezolid	600 mg daily	BPaLM, BDLLfxC	Myelosuppression, neuropathy, lactic acidosis
Clofazimine	100 mg daily	MDR regimens	Skin discolouration, QT
Levofloxacin / moxifloxacin	Lfx 750–1000 mg; Mfx 400 mg daily	MDR regimens	Tendinopathy, QT, dysglycaemia

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Cycloserine 10–15 mg/kg (max 1 g) needs pyridoxine; watch for psychosis and seizures.

Class	Short-acting	Long-acting (12 h)	Ultra-long-acting (24 h)
PDE3/4 inhibitor	—	Ensifentrine (nebulised, 2024)	—

β2-agonists

- β2 → Gs → adenylyl cyclase → cAMP → PKA → myosin light chain kinase inhibition, K+ channel opening → relaxation; also inhibit mast cell mediators, increase ciliary beat.
- **Ultra-LABA** (indacaterol, olodaterol, vilanterol): high intrinsic efficacy, lipid raft anchoring, once daily, rapid onset (indacaterol 5 min); combined with LAMA (IND/GLY, UMEC/VI, TIO/OLO) and ICS (FF/UMEC/VI). Role: COPD maintenance (GOLD group B/E LABA+LAMA); vilanterol/FF in asthma.
- **Adverse effects:** tremor, tachycardia, hypokalaemia, hyperglycaemia, lactic acidosis (IV), tachyphylaxis; LABA monotherapy in asthma ↑ mortality (SMART study) — always with ICS.

Antimuscarinics

- M3 blockade (bronchial smooth muscle, glands); kinetic M3 selectivity of tiotropium (slow dissociation) — UPLIFT showed fewer exacerbations.
- **Umeclidinium** 62.5 µg once daily (Ellipta DPI), quaternary ammonium; with vilanterol (Anoro) or FF/VI (Trelegy). Side effects: dry mouth, urinary retention, glaucoma (nebulised ipratropium with mask).

Methylxanthines — pleiotropic actions of theophylline

- Non-selective PDE inhibition (bronchodilation at high levels), adenosine receptor antagonism (side effects — seizures, arrhythmia), **HDAC2 activation** at low doses (restores steroid sensitivity in COPD/smokers), improved diaphragmatic contractility, mucociliary clearance, respiratory drive, anti-inflammatory (↓ IL-8, neutrophils), diuresis.
- Narrow therapeutic index 10–20 mg/L (5–15 for anti-inflammatory); metabolised by CYP1A2 — levels ↑ with ciprofloxacin, macrolides, cimetidine, HF, cirrhosis; ↓ with smoking, rifampicin, phenytoin.
- Toxicity: nausea, tachyarrhythmias, seizures, hypokalaemia; treatment — charcoal, haemoperfusion. Doxofylline (no adenosine antagonism) safer.
- Role: add-on in COPD where inhalers unaffordable; apnoea of prematurity (caffeine); not in acute exacerbations routinely (TWICS: low-dose theophylline did not reduce COPD exacerbations).

Inhaled corticosteroids

- Bind cytosolic GR → nuclear translocation → transactivation (β2-receptor, anti-inflammatory genes) and **transrepression** (recruit HDAC2 to switch off NF-κB/AP-1 inflammatory genes).
- Agents: beclomethasone, budesonide, fluticasone propionate/furoate, ciclesonide (prodrug), mometasone.
- **Local adverse:** oral candidiasis, dysphonia (rinse, spacer). **Systemic (dose-related):** adrenal suppression, osteoporosis, cataract, glaucoma, skin bruising, diabetes; **pneumonia in COPD** (fluticasone > budesonide), mycobacterial infection risk.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Theophylline + ciprofloxacin/erythromycin in a smoker who quits during admission = toxicity risk (CYP1A2 inhibition + loss of induction).

LANDMARK TRIALS & GUIDELINES

- UPLIFT (Tashkin, NEJM 2008); TWICS (Devereux, JAMA 2018); SMART salmeterol (Nelson, Chest 2006).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Role of ultra-LABA in obstructive airway diseases. *[DNB Oct 2024 P2]*
2. Pleiotropic actions of theophylline. *[DNB Oct 2024 P2]*
3. Methylxanthines use in respiratory disease. *[BFUHS 2020 P1]*
4. Umeclidinium. *[VNSGU 2025 P4]*

INVESTIGATIONS & TREATMENT AT A GLANCE

3.10 Bronchodilators & Inhaled Corticosteroids

‹ Back to topic (p 61)
Next topic 3.11 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Spirometry reversibility	Δ FEV1 < 10% predicted	≥ 10% predicted (ATS/ERS 2022)	Bronchodilator responsiveness
Serum potassium	3.5–5.0 mmol/L	< 3.5	High-dose β-agonist hypokalaemia
Heart rate / ECG	< 100/min	Tachyarrhythmia	β-agonist toxicity
Theophylline level	10–20 mg/L (now 5–15 targeted)	> 20 mg/L	Toxicity — seizures, arrhythmias
Blood glucose	70–140 mg/dL	Hyperglycaemia	Systemic steroids/β-agonists
Bone density (DEXA)	T-score > −1	≤ −2.5	Long-term high-dose ICS/OCS

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Salbutamol	100–200 µg as needed; nebulised 2.5–5 mg	Reliever	Tremor, tachycardia, hypokalaemia
Budesonide/formoterol (MART)	200/6 µg 1 puff as needed (max 12/day)	GINA Track 1	Thrush — rinse
Tiotropium	18 µg DPI or 2.5 µg × 2 SMI daily	COPD; asthma step 5	Dry mouth, urinary retention
Fluticasone furoate/ vilanterol	100/25 µg once daily	Asthma/COPD	Pneumonia risk in COPD
Theophylline SR	200–300 mg twice daily	Add-on (limited)	Interactions (ciprofloxacin, macrolides ↑ levels)

★ PRESCRIBING & INTERPRETATION PEARLS

- ICS in COPD: most benefit when blood eosinophils ≥ 300/µL; avoid when < 100 with repeated pneumonia.

3.11 Antimicrobials, Antifungals & Macrolides in Respiratory Medicine

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-G1, I-G5 Antimicrobial, antiviral, antifungal drugs
- ✓ Describe the pharmacology of antifungal drugs used in respiratory disorders
- ✓ Describe the antibacterial, anti-inflammatory and immunomodulatory roles of macrolides
- ✓ Describe drugs used for Pneumocystis pneumonia

IN BRIEF Antifungals target ergosterol (polyenes, azoles), β-glucan synthesis (echinocandins) or nucleic acids (flucytosine). Macrolides act beyond their antibacterial spectrum, reducing exacerbations in COPD, bronchiectasis and asthma.

Antifungal drugs in respiratory disease

Class / drug	Mechanism	Spectrum / pulmonary use	Key toxicity
Amphotericin B (deoxycholate, liposomal 3–5 mg/kg; 5–10 for mucor)	Binds ergosterol — pores	Mucormycosis (first-line), cryptococcosis, histoplasmosis severe, IPA alternative	Nephrotoxicity, hypoK/Mg, infusion reactions
Voriconazole	Triazole — inhibits lanosterol 14α-demethylase	IPA first-line; CPA	Visual disturbance, hepatotoxicity, photosensitivity, periostitis (fluoride); TDM 1–5.5 mg/L
Isavuconazole	Triazole (prodrug)	IPA (non-inferior, fewer AEs — SECURE), mucormycosis (VITAL)	Shortens QT (unique), hepatotoxicity
Posaconazole	Triazole	Prophylaxis (AML, GVHD), mucormycosis step-down, IPA	GI, QT, pseudoaldosteronism
Itraconazole	Triazole	ABPA, CPA, histoplasmosis, talaromycosis	Negative inotropy (HF), variable absorption (SUBA formulation better)
Fluconazole	Triazole	Cryptococcus (consolidation), Candida	Narrow spectrum; QT
Echinocandins (caspofungin, micafungin, anidulafungin)	β-1,3-glucan synthase inhibitors	Candidaemia; salvage IPA	Well tolerated; not for Cryptococcus/ Mucor
Flucytosine	Converted to 5-FU in fungus	With amphotericin for cryptococcal meningitis	Marrow suppression

- **Drug interactions:** azoles inhibit CYP3A4 (raise tacrolimus, steroids, vincristine); rifampicin drastically lowers azole levels — avoid together.

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Macrolides in respiratory medicine

- **Antibacterial:** atypicals, S. pneumoniae (resistance high in India), H. influenzae; MAC (clarithromycin/azithromycin core), pertussis.
- **Immunomodulatory:** ↓ neutrophil recruitment and IL-8, ↓ mucus hypersecretion, inhibition of Pseudomonas quorum sensing and biofilm, ↑ efferocytosis; motilin agonism.
- **Evidence:** COPD exacerbation prevention (azithromycin 250 mg daily — Albert 2011), non-CF bronchiectasis (EMBRACE, BAT, BLESS), asthma (AMAZES — 500 mg thrice weekly reduces exacerbations), diffuse panbronchiolitis (dramatic), BOS after transplant, CF.
- Adverse: QT prolongation, hearing loss, GI, macrolide resistance (exclude NTM before long-term use — monotherapy breeds resistant MAC).
- **Severe CAP:** adding macrolide to β-lactam reduces mortality (observational; ACCESS trial — clarithromycin reduced treatment failure in severe CAP 2023).

Drugs for Pneumocystis pneumonia

Drug	Dose	Notes
Co-trimoxazole (TMP-SMX)	15–20 mg/kg/day TMP in 3–4 doses ×21 days	First-line; prophylaxis 1 DS daily or 3×/week
Prednisolone	40 mg BD ×5 d, 40 OD ×5 d, 20 OD ×11 d	HIV with PaO2 <70 or A–a gradient >35
Primaquine + clindamycin	30 mg + 600 mg q8h	Moderate–severe alternative (check G6PD)
Pentamidine IV	4 mg/kg/day	Severe, intolerant; hypoglycaemia, pancreatitis
Atovaquone	750 mg BD	Mild–moderate; prophylaxis
Dapsone + trimethoprim	100 mg + 15 mg/kg/day	Mild; check G6PD

★ CLINICAL PEARLS & EXAM FOCUS

- Before long-term azithromycin: ECG (QTc), hearing check and sputum AFB/NTM culture.

LANDMARK TRIALS & GUIDELINES

- Albert RK et al, NEJM 2011; Gibson PG et al, AMAZES Lancet 2017; Maertens J et al, SECURE Lancet 2016.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Antifungal drugs in respiratory disorders. *[DNB Dec 2025 P1]*
2. Role of macrolides in respiratory medicine. *[BFUHS 2022 P2]*
3. Drugs for Pneumocystis pneumonia. *[DNB Oct 2023 P2]*

3.12 Antineoplastic, Immunosuppressant & Biologic Drugs; Pharmacovigilance

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-G3, I-G6, I-G7 Antineoplastic drugs, interactions, pharmacovigilance
- ✓ Outline classes of antineoplastic, immunosuppressant and biologic drugs used in pulmonology with pulmonary toxicities
- ✓ Describe important drug interactions and the pharmacovigilance programme (PvPI)

IN BRIEF Pulmonologists prescribe chemotherapy, targeted agents, immunotherapy, steroids and steroid-sparing immunosuppressants. Many cause lung toxicity, infection and interactions — all must be reported through the Pharmacovigilance Programme of India.

Drugs and their pulmonary relevance

Class	Examples	Pulmonary use	Notable pulmonary toxicity
Platinum / taxanes / others	Cisplatin, carboplatin, paclitaxel, docetaxel, pemetrexed, gemcitabine, etoposide	Lung cancer	Gemcitabine and docetaxel pneumonitis
Targeted TKIs	Osimertinib (EGFR), alectinib/lorlatinib (ALK), crizotinib/entrectinib (ROS1), sotorasib (KRAS G12C)	NSCLC with drivers	ILD (EGFR TKIs 1–5%)
Immune checkpoint inhibitors	Pembrolizumab, nivolumab, atezolizumab, durvalumab	NSCLC, SCLC, mesothelioma	Pneumonitis (3–5%)
Anti-VEGF	Bevacizumab	Non-squamous NSCLC	Haemoptysis (squamous, cavitating — avoid), PE, BPF
Immunosuppressants	Azathioprine, mycophenolate, methotrexate, cyclophosphamide, rituximab, tacrolimus	ILD, vasculitis, sarcoidosis, transplant	MTX pneumonitis; PJP, TB reactivation

INVESTIGATIONS & TREATMENT AT A GLANCE

3.11 Antimicrobials, Antifungals & Macrolides in Respiratory Medicine

‹ Back to topic (p 63)
Next topic 3.12 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Culture and sensitivity	—	MIC-guided susceptibility	Narrow therapy
Vancomycin trough / AUC	AUC 400–600 mg·h/L (trough 10–20)	> 600	Nephrotoxicity
Aminoglycoside trough	Amikacin < 2.5 mg/L	Raised	Oto/nephrotoxicity
Voriconazole trough	1–5.5 mg/L	< 1 (failure) / > 5.5 (toxicity)	TDM
QTc on macrolides	< 450 ms	> 500 ms	Arrhythmia risk
Hearing assessment (long-term azithro)	Normal	Loss	Stop macrolide

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Azithromycin (long-term)	250 mg daily or 500 mg three times weekly	Bronchiectasis, COPD exacerbation prevention	Exclude NTM first; QTc; hearing
Piperacillin–tazobactam	4.5 g IV 6–8-hourly (extended infusion)	HAP/VAP, Pseudomonas	Hypokalaemia; AKI with vancomycin
Meropenem	1 g IV 8-hourly (2 g in meningitis)	Resistant Gram-negatives	Seizures (less than imipenem)
Vancomycin	15–20 mg/kg IV 8–12-hourly (load 25 mg/kg)	MRSA	AUC monitoring
Linezolid	600 mg 12-hourly	MRSA pneumonia	Thrombocytopenia > 14 days

★ PRESCRIBING & INTERPRETATION PEARLS

- Do not give fluoroquinolones for pneumonia when TB has not been excluded — masks TB and selects FQ resistance.

Class	Examples	Pulmonary use	Notable pulmonary toxicity
Antifibrotics	Nintedanib, pirfenidone	IPF, PPF	GI, hepatotoxicity
Biologics	Omalizumab, mepolizumab, benralizumab, dupilumab, tezepelumab	Severe asthma, EGPA, COPD (dupilumab, mepolizumab)	Eosinophilic pneumonia/EGPA unmasking on steroid taper (dupilumab — hypereosinophilia)
Bleomycin, amiodarone, nitrofurantoin	—	—	Classic drug-induced lung disease (see 7.9)

Important drug interactions in pulmonology

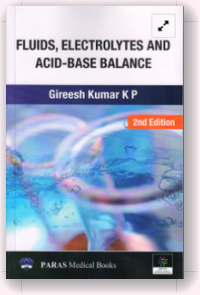
- **Rifampicin** — induces CYP3A4, 2C9, P-gp: reduces dolutegravir (double dose), PIs (avoid; use rifabutin), warfarin, OCPs, steroids (double dose), azoles, tacrolimus, methadone, DOACs.
- **Azoles / macrolides** inhibit CYP3A4 — raise theophylline, statins, tacrolimus, BDQ.
- **QT stacking** — Bdq, Dlm, Cfz, FQs, macrolides, azoles, ondansetron, antipsychotics.
- **Linezolid** — serotonin syndrome with SSRIs; **theophylline** — FQ/macrolide; **INH** — phenytoin, carbamazepine.

Pharmacovigilance

- PvPI (2010) — national coordination centre at Indian Pharmacopoeia Commission, Ghaziabad; ADR monitoring centres (AMCs) in medical colleges; report with **Suspected ADR Reporting Form** (any HCP, also patients via mobile app/helpline 1800-180-3024).
- NTEP has an active drug safety monitoring and management (**aDSM**) system for DR-TB regimens; ADRs reported in Ni-kshay.
- Causality assessment: WHO-UMC scale (certain, probable, possible, unlikely), Naranjo algorithm; severity: Hartwig scale; preventability: Schumock–Thornton.
- Materiovigilance (MvPI) covers devices — e.g., NIV masks, inhalers, stents.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Any serious unexpected ADR in a DR-TB patient → aDSM report within 24 h via Ni-kshay.



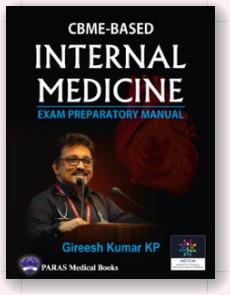
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INVESTIGATIONS & TREATMENT AT A GLANCE

3.12 Antineoplastic, Immunosuppressant & Biologic Drugs; Pharmacovigilance

‹ Back to topic (p 64)
Next topic 4.1 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
CBC before chemotherapy	ANC > 1.5 × 10⁹/L; platelets > 100	ANC < 0.5 (febrile neutropenia risk)	Delay chemotherapy
HBV serology before immunosuppression	HBsAg/anti-HBc negative	Positive	Antiviral prophylaxis
IGRA/TST before anti-TNF / JAK inhibitors	Negative	Positive	TPT before starting
DLCO before/after bleomycin	> 80%	Fall > 15–20%	Bleomycin toxicity — stop
Methotrexate monitoring (CBC, LFT, creatinine)	Normal	Transaminases > 3× ULN; cytopenia	Dose adjust; pneumonitis
TPMT activity before azathioprine	Normal	Low	Myelosuppression risk

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Prednisolone	0.5–1 mg/kg/day, taper	Immune lung disease	PCP prophylaxis if ≥ 20 mg > 4 weeks; bone protection
Mycophenolate mofetil	1–1.5 g twice daily	CTD-ILD	Cytopenia, teratogenic
Azathioprine	2 mg/kg/day	ILD/vasculitis maintenance	TPMT; myelosuppression
Co-trimoxazole prophylaxis	960 mg three times weekly or 480 mg daily	PCP prophylaxis on immunosuppression	Rash, hyperkalaemia
Pembrolizumab	200 mg IV every 3 weeks	NSCLC PD-L1 ≥ 50%	Immune pneumonitis — steroids

★ PRESCRIBING & INTERPRETATION PEARLS

- Report suspected adverse drug reactions to PvPI (helpline 1800-180-3024).

SECTION 4

Tuberculosis

Paper II / III

Aetiopathogenesis, diagnosis, differential diagnosis and management of pulmonary TB under the National TB Elimination Programme (NTEP) and the Standards for TB Care in India; complications; TB in children, elderly, pregnancy, diabetes, HIV and organ failure; pleural, pericardial and extrapulmonary TB; NTM; MDR/XDR-TB; prevention (NMC syllabus II-A1, I-E3). The core of Paper III in DNB and most university MD papers.

4.1 Epidemiology of TB, End TB Strategy & the National TB Elimination Programme 66
4.2 Pathogenesis — Primary & Post-Primary TB, Wallgren Timetable 68
4.3 Diagnosis of Pulmonary TB — NTEP Algorithm, Radiology & Differential Diagnosis 70
4.4 Latent TB Infection — TST, IGRA & TB Preventive Treatment 71
4.5 Treatment of Drug-Susceptible TB under NTEP 74
4.6 Drug-Resistant TB — Definitions, Risk Factors, Surveillance & Diagnostic Algorithm 76
4.7 Treatment of MDR/RR-TB — BPaLM, Shorter & Longer Oral Regimens 78
4.8 TB in Special Situations — Pregnancy, Renal Failure, Liver Disease, Diabetes & the Elderly 79
4.9 HIV–TB Co-infection, ART Interactions & IRIS 81
4.10 Paediatric TB & Childhood Contact Management 82
4.11 Miliary TB & TB Meningitis 84
4.12 Pleural & Pericardial Tuberculosis 86
4.13 Extrapulmonary TB — Lymph Node, Abdominal, Spinal, Genitourinary, Endobronchial & Cutaneous 87
4.14 Complications & Post-TB Lung Disease; Corticosteroids and Surgery in TB 89
4.15 Patient Support, Infection Control, Stigma, BCG & TB Vaccines 91
4.16 Non-Tuberculous Mycobacteria 92

4.1 Epidemiology of TB, End TB Strategy & the National TB Elimination Programme

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-E2, I-E3, II-A1(4) Epidemiology and national programme
- ✓ Describe the global and Indian burden of TB and its determinants
- ✓ Describe the End TB Strategy targets and the evolution of the Indian programme (NTP → RNTCP → NTEP)
- ✓ Explain the components of the National Strategic Plan, active case finding and TB-free initiatives

IN BRIEF India carries about a quarter of the world's TB burden (~27 lakh incident cases annually). The NTEP (renamed from RNTCP in 2020) aims to end TB ahead of the SDG target of 2030 through the Detect–Treat–Prevent–Build (DTPB) framework.

Burden and determinants

- **Global (WHO Global TB Report 2024):** ~10.8 million incident cases and 1.25 million deaths in 2023 — TB again the leading single infectious killer; 30 high-burden countries account for 87%.
- **India:** ~26% of global cases; incidence estimated ~195 per lakh in 2023 (≈18% decline since 2015) and mortality ~22 per lakh; record notification (>26 lakh in 2024); ~1.1 lakh MDR/RR-TB cases estimated.
- **Determinants:** undernutrition (largest attributable risk in India — RATIONS trial), diabetes, HIV, smoking, alcohol, silicosis, overcrowding, poverty, indoor air pollution.
- **Economic impact:** ~50% of TB-affected families face catastrophic costs (>20% of household income); loss of productive years (15–55 age group); TB costs India an estimated USD 32 billion/year (TB economic burden study).

History of the Indian programme

Milestones

Year	Milestone
1962	National TB Programme (NTP) — district TB centres, BCG
1993/1997	RNTCP — DOTS strategy; nationwide by 2006
2007	DOTS-Plus for MDR-TB (PMDT)
2012	TB notification made mandatory; Ni-kshay case-based web portal
2017	Daily FDC regimen; National Strategic Plan 2017–25 (DTPB); universal DST
2018	Ni-kshay Poshan Yojana (DBT ₹500/month)
2020	Renamed National TB Elimination Programme (NTEP)
2021	Guidelines for programmatic management of TB preventive treatment
2022	Pradhan Mantri TB Mukt Bharat Abhiyaan; Ni-kshay Mitra
2024	BPaLM regimen; NPY raised to ₹1000/month; 100-day intensified TB Mukt Bharat campaign (Dec 2024)

End TB Strategy (WHO, 2015)

End TB targets (baseline 2015)

Indicator	2025 milestone	2030 (SDG)	2035
Reduction in TB deaths	75%	90%	95%
Reduction in incidence rate	50%	80%	90%
TB-affected families with catastrophic costs	0%	0%	0%

- **Three pillars:** (1) integrated patient-centred care and prevention; (2) bold policies and supportive systems (UHC, social protection, regulation); (3) intensified research and innovation.
- **Four principles:** government stewardship, civil society engagement, human rights/ethics/equity, adaptation at country level.

National Strategic Plan 2017–25 — Detect, Treat, Prevent, Build

- **Detect:** NAAT for all presumptive TB, universal DST, private sector engagement (PPSA), **active case finding (ACF)** in vulnerable groups (slums, tribals, prisons, PLHIV, diabetics, elderly, migrants), chest X-ray screening (incl. AI-CXR), mandatory notification.
- **Treat:** daily FDC, patient support (99DOTS, MERM, family DOT, Ni-kshay Sampark), DR-TB centres, aDSM, nutrition support (NPY).
- **Prevent:** TPT to contacts and high-risk groups, airborne infection control, BCG, addressing determinants.
- **Build (RESPOND):** human resources, Ni-kshay IT system, multisectoral TB Forums, research (India TB Research Consortium).

Active case finding and TB-free certification

- ACF = provider-initiated systematic screening of high-risk groups (symptom + CXR) vs passive case finding (patient walks in). In India, ACF campaigns (since 2017) detect ~5–8% additional cases.
- **TB-free status (sub-national certification):** districts/states assessed for ≥20–80% decline in incidence (bronze to gold) vs 2015 through drug-sales data, notification, community surveys — awarded since 2020; **TB Mukt Panchayat** initiative (2023).

✦ RECENT ADVANCES & GUIDELINE UPDATES

▸ India TB Report 2025 and 100-day TB Mukt Bharat Abhiyaan (Dec 2024 – Mar 2025): vulnerability mapping, handheld X-ray, NAAT for all presumptive TB, nutrition for patients.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Remember End TB targets as "90-80 by 2030, 95-90 by 2035, zero catastrophic cost".

LANDMARK TRIALS & GUIDELINES

- WHO End TB Strategy (WHA 2014); Bhargava A et al, RATIONS trial, Lancet 2023 — nutritional support to household contacts reduced TB incidence by ~40%.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Magnitude of TB burden in India. *[DNB Jun 2020 P3]*
2. The WHO End TB strategy, with targets for 2030 (SDG) and 2035. *[WBUHS 2022 P3]*
3. End TB strategy. *[BFUHS 2020 P4]*
4. Active case finding. *[WBUHS 2020 P3]*
5. Economic impact of tuberculosis. *[BFUHS 2020 P3]*
6. TB-free district. *[WBUHS 2022 P3]*

4.2 Pathogenesis — Primary & Post-Primary TB, Wallgren Timetable

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A1(1) Aetiopathogenesis; I-D2
- ✓ Describe the pathogenesis of primary infection, latency and post-primary (reactivation/reinfection) TB
- ✓ Describe the radiological manifestations of primary and post-primary TB
- ✓ Describe the Wallgren timetable

IN BRIEF Inhaled droplet nuclei (1–5 µm) reach alveoli; ~30% of exposed persons become infected, and 5–10% of infected develop disease, half within 2 years. Primary TB is a disease of the hilar nodes and lower/middle zones; post-primary TB is cavitary and apical.

Primary tuberculosis

- Bacilli multiply in alveolar macrophages for 3–8 weeks (no immunity) → lymphatic spread to hilar nodes and silent haematogenous seeding (apices, kidneys, bones, meninges — Simon foci).
- Cell-mediated immunity develops (TST/IGRA positive at 3–8 weeks) → granuloma, caseation.
- **Ghon focus** (subpleural, mid/lower zone) + lymphangitis + hilar node = **Ghon (primary) complex**; calcified = **Ranke complex**.
- **Progressive primary** (infants, immunosuppressed): enlarging nodes compress bronchi (epituberculosis, middle lobe syndrome), node erosion (endobronchial TB), progressive consolidation and cavity, pleural effusion (3–7 months), miliary TB and meningitis (within 6 months).

INVESTIGATIONS & TREATMENT AT A GLANCE

4.1 Epidemiology of TB, End TB Strategy & the National TB Elimination Programme

‹ Back to topic (p 66)

Next topic 4.2 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
TB incidence (WHO 2025 report)	—	India 187/lakh (2024); global 131/100 000	Programme indicator
Treatment success rate	Target ≥ 90%	< 85%	Programme performance
Notification rate (public + private)	—	Gap vs estimated incidence	"Missing cases"
Universal DST coverage	100% of diagnosed	< 90%	Programme gap
TPT coverage among contacts	Target ≥ 90%	Low	Prevention gap

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Ni-kshay Poshan Yojana	₹1000/month DBT for full treatment	All notified TB patients	Bank account seeding
Energy-dense nutrition supplement	Per NTEP	BMI < 18.5	—
Ni-kshay Mitra food basket	Monthly	Patients and household contacts	Consent
Notification	On Ni-kshay within 7 days	Every diagnosed case incl. private	Mandatory

★ PRESCRIBING & INTERPRETATION PEARLS

- End TB targets (2035): 90% fall in incidence and 95% fall in deaths vs 2015.



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Post-primary (secondary) tuberculosis

- Reactivation of dormant foci (or exogenous reinfection — common in high-burden settings) in previously sensitised host; Koch phenomenon — vigorous DTH → caseous necrosis, liquefaction, cavitation.
- Apical/posterior segments of upper lobes and superior segment of lower lobes (high PAO2, poor lymphatic flow).
- **Lipid pneumonia hypothesis (Hunter):** early post-primary TB starts as obstructive lipid pneumonia that caseates and is coughed out to form cavities — explains infectiousness of cavities.
- Spread: bronchogenic (tree-in-bud), haematogenous (miliary), lymphatic, contiguous (pleura).

Radiology of primary vs post-primary TB

Feature	Primary TB	Post-primary TB
Location	Any lobe; middle/lower zones, often unilateral	Apical/posterior upper lobe, superior lower lobe
Lymphadenopathy	Hilar/paratracheal — hallmark (esp. children)	Uncommon
Parenchyma	Homogeneous consolidation	Patchy heterogeneous consolidation, fibronodular
Cavitation	Uncommon	Common (thick-walled) with air–fluid level rare
Pleural effusion	Common (young adults)	Less common; empyema
Other	Collapse from node compression, miliary	Tree-in-bud (endobronchial spread), fibrosis, volume loss, tuberculoma, bronchiectasis

Wallgren timetable of primary TB

Wallgren timetable (after infection)

Time	Event
3–8 weeks	Primary complex; tuberculin conversion; erythema nodosum, phlyctenular conjunctivitis
2–6 months	Miliary TB and TB meningitis (haematogenous spread)
3–7 months	Pleural effusion (5–10% of children >5 years)
Up to 1–2 years	Bronchial complications (collapse–consolidation, endobronchial TB)
1–3 years	Bone and joint TB (spine within 1–2 years)
5–8 years	Renal/genitourinary TB
Lifetime	Reactivation (post-primary)

★ CLINICAL PEARLS & EXAM FOCUS

▸ "Ghon + node = complex; Ranke = calcified complex; Simon = apical seeding; Assmann = infraclavicular early post-primary focus; Puhl = apical reinfection lesion."

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Primary pulmonary tuberculosis. *[DNB Jun 2021 P3]*
2. Wallgren timetable of tuberculosis. *[DNB Jun 2021 P3]*
3. Describe pathophysiology of post primary tuberculosis. Discuss complications, sequelae and management of post primary tuberculosis. *[VNSGU 2025 P1]*
4. Radiological changes in primary and post primary tuberculosis. *[WBUHS 2022 P3]*

INVESTIGATIONS & TREATMENT AT A GLANCE

4.2 Pathogenesis — Primary & Post-Primary TB, Wallgren Timetable

‹ Back to topic (p 68)

Next topic 4.3 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
TST / IGRA	TST < 10 mm; IGRA negative	TST ≥ 10 mm (≥ 5 mm in HIV/contacts); IGRA positive	TB infection (not disease)
CXR in primary TB	Normal	Hilar/paratracheal nodes, Ghon focus, effusion	Primary TB
CXR in post-primary TB	Normal	Apical/posterior infiltrates, cavities	Reactivation TB
Sputum Xpert	Not detected	Detected	Confirms disease
ESR / CRP	ESR < 20 mm/h	Raised	Non-specific inflammation

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Standard DS-TB regimen	2HRZE/4HRE (NTEP)	Disease	See 4.5
TPT (3HP / 6H)	3HP weekly × 12; 6H daily	Infection without disease	Rule out disease first
Prednisolone (airway compression in children)	1–2 mg/kg/day × 4 weeks	Node compression, effusion in children	With ATT only

★ PRESCRIBING & INTERPRETATION PEARLS

- Wallgren: primary complex 3–8 weeks; effusion 3–7 months; miliary/TBM within 1 year; bone 1–3 years; renal 5+ years.

4.3 Diagnosis of Pulmonary TB — NTEP Algorithm, Radiology & Differential Diagnosis

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

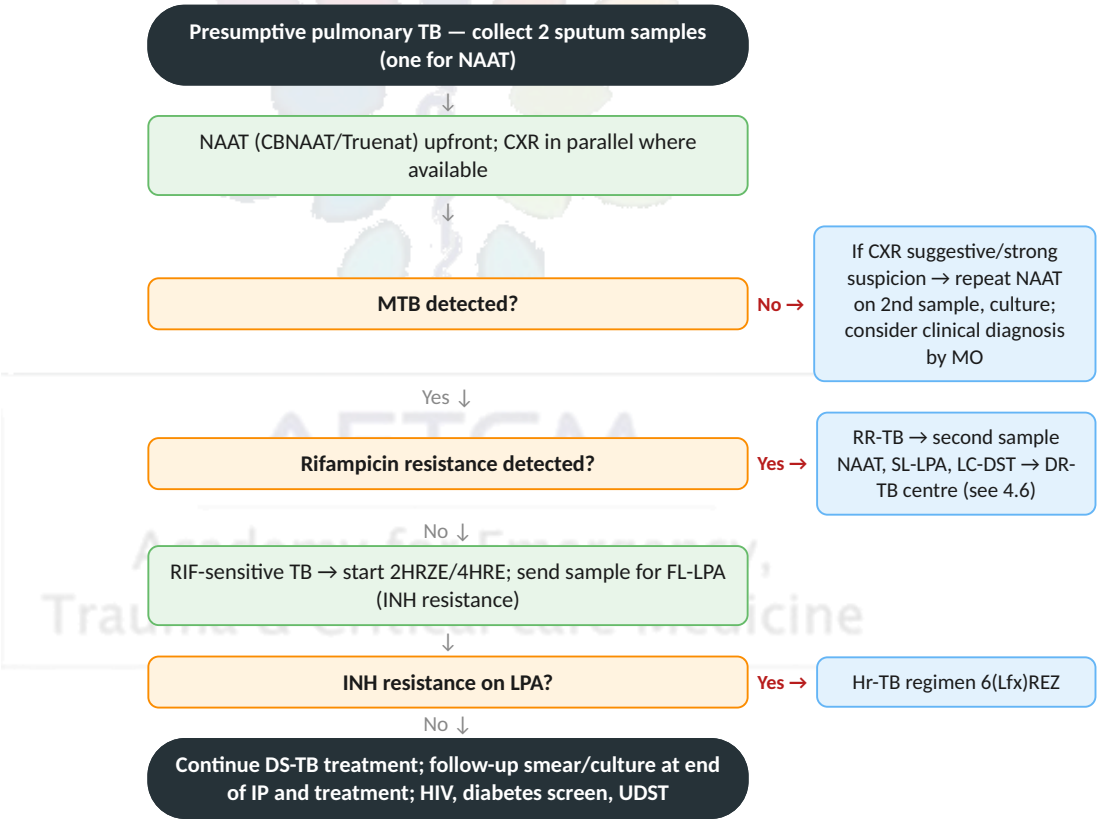
- ✓ **NMC syllabus area:** II-A1(2,3) Diagnostic methods, differential diagnosis
- ✓ Define presumptive TB and apply the NTEP diagnostic algorithm
- ✓ Interpret chest radiology in pulmonary TB and list differential diagnoses
- ✓ Define NTEP case definitions and treatment outcomes

IN BRIEF Every presumptive TB patient should receive a WHO-recommended rapid diagnostic (NAAT: CBNAAT/Truenat) as the initial test. Microbiologically confirmed TB is the goal; clinical diagnosis (radiology, clinical response) is allowed where tests are negative but suspicion is high.

Presumptive TB (NTEP)

- Cough ≥2 weeks (any duration in PLHIV, contacts, diabetics, malnourished), fever ≥2 weeks, significant weight loss, haemoptysis, night sweats, any abnormality on CXR. Also anyone with a positive symptom screen in ACF.

Algorithm 4.1 · NTEP integrated diagnostic algorithm for pulmonary TB



Radiology

- **CXR:** sensitive (~90%) but non-specific; NTEP uses CXR as screening tool (incl. CAD-AI software — CAD4TB, qXR). Suggestive: upper-zone infiltrates, cavities, nodes, miliary pattern, effusion.
- **CT/HRCT:** centrilobular nodules and tree-in-bud (active endobronchial spread), cavities, consolidation, necrotic nodes (central low attenuation with rim enhancement), miliary nodules; differentiates active from sequelae (fibrosis, calcification, bronchiectasis).

Differential diagnosis of pulmonary TB

Presentation	Differentials
Cavitary lesion	Lung abscess, cavitating carcinoma (squamous), NTM, CPA, GPA, melioidosis, nocardiosis, Klebsiella, paragonimiasis, septic emboli
Upper zone fibrosis	Sarcoidosis, silicosis, ABPA, AS, PPFE, radiation, healed TB
Miliary pattern	Sarcoidosis, silicosis, metastases (thyroid, melanoma), fungal (histoplasma), varicella, PAP/PLCH
Hilar lymphadenopathy	Lymphoma, sarcoidosis, metastases
Non-resolving consolidation	Organising pneumonia, adenocarcinoma, lymphoma, fungal

NTEP case definitions

- **Microbiologically confirmed:** positive smear, culture or WRD (NAAT/LPA). **Clinically diagnosed:** not confirmed but diagnosed by MO on clinical/radiological grounds and treated.
- **New** (never treated or <1 month); **previously treated** (recurrent, treatment after failure, after loss to follow-up, other).
- **Outcomes:** cured (bacteriologically negative at end), treatment completed, treatment success (cured + completed), failed, lost to follow-up (≥1 month interruption, now NTEP uses 1 month), died, not evaluated, treatment regimen changed.

Extrapulmonary specimens

- Pleural fluid ADA, LN aspirate/biopsy (NAAT — Xpert sensitivity ~85% in LN), CSF (Ultra), urine, gastric aspirate/stool (children — stool Xpert Ultra), tissue histopathology with NAAT.

✦ RECENT ADVANCES & GUIDELINE UPDATES

▸ WHO 2024: CXR with CAD software for screening ≥15 years; targeted NGS for DR detection; low-complexity NAATs (Truenat) recommended as initial tests.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Never treat presumptive TB with fluoroquinolones as a "therapeutic trial" — it delays diagnosis and creates FQ resistance.

LANDMARK TRIALS & GUIDELINES

- Standards for TB Care in India (STCI, 2014); NTEP Guidelines for PMDT 2021 and Training Modules 1–4 (2023).

4.4 Latent TB Infection — TST, IGRA & TB Preventive Treatment

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A1, I-F3 Diagnosis and prevention
- ✓ Compare tuberculin skin test and IGRA, including causes of false results
- ✓ Identify eligible and expanded populations for TPT under NTEP and choose a regimen
- ✓ List contraindications to TPT and rule out active TB before starting

IN BRIEF TB infection (TBI/LTBI) is a state of persistent immune response to M. tuberculosis antigens without active disease. India's 2021 TPT guidelines expanded preventive treatment to all household contacts of pulmonary TB, PLHIV and other high-risk groups, after excluding active TB.

INVESTIGATIONS & TREATMENT AT A GLANCE

4.3 Diagnosis of Pulmonary TB — NTEP Algorithm, Radiology & Differential Diagnosis

‹ Back to topic (p 70)

Next topic 4.4 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Xpert MTB/RIF Ultra (sputum)	Not detected	Detected ± Rif resistance	First test for all presumptive TB
Sputum smear	Negative	Positive (scanty to 3+)	Infectiousness; monitoring
Liquid culture	No growth at 42 days	Growth	Confirmation, DST
CXR	Normal	Upper-lobe infiltrates, cavities, nodes, effusion	Screening (CAD in ≥ 15 y)
HIV test	Non-reactive	Reactive	All TB patients
Blood glucose / HbA1c	FBS < 100 mg/dL; HbA1c < 5.7%	FBS ≥ 126; HbA1c ≥ 6.5%	Diabetes — bidirectional screening
Urine LF-LAM (PLHIV, CD4 ≤ 200 / ill)	Negative	Positive	Concurrent with NAAT

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
4FDC (HRZE)	Weight band (2–6 tablets)	Intensive phase 2 months	See 4.5
Avoid fluoroquinolone antibiotic trials	—	Presumptive TB with negative NAAT	Masks TB, FQ resistance
Antibiotic trial (non-FQ)	Amoxicillin 500 mg 8-hourly × 7 days	NAAT-negative, possible pneumonia	Re-evaluate at 2 weeks

★ PRESCRIBING & INTERPRETATION PEARLS

- Presumptive TB (NTEP): cough ≥ 2 weeks, fever, weight loss, night sweats, haemoptysis, any CXR abnormality — any duration in vulnerable groups.

TST versus IGRA

Feature	Tuberculin skin test (Mantoux)	IGRA (QuantiFERON-TB Gold Plus, T-SPOT.TB)
Antigen	PPD (2 TU RT-23 / 5 TU PPD-S) intradermal	ESAT-6, CFP-10 (RD1 region) in vitro
Reading	48–72 h; induration (not erythema)	Single blood draw; result 24 h
Cut-offs	≥5 mm (HIV, contacts, immunosuppressed, fibrotic lesions); ≥10 mm (others); ≥15 mm (no risk)	IFN-γ ≥0.35 IU/mL (QFT)
BCG / NTM cross-reaction	Yes	No (except M. kansasii, marinum, szulgai)
Booster phenomenon	Yes (two-step test)	No
Cost / logistics	Cheap; two visits	Expensive; laboratory
Limitations	Operator variability	Indeterminate in lymphopenia

- Neither distinguishes latent from active TB or predicts progression well (PPV 1–3%). Newer: C-Tb and Diaskintest (ESAT-6/ CFP-10 skin tests — WHO 2022 endorsed TBST).

False-positive and false-negative TST

False positive	False negative
BCG vaccination (especially recent/ booster)	HIV, immunosuppressants, steroids
NTM infection	Severe/disseminated TB (miliary, TBM), malnutrition
Incorrect reading (erythema), cross-reactivity	Live vaccines (MMR) within 4 weeks, viral infection (measles)
Boosting in repeated tests	Extremes of age, sarcoidosis, lymphoma, poor technique (subcutaneous injection)

TPT eligibility (NTEP 2021)

Target populations for TPT

Population	Testing for infection	Comment
PLHIV (adults, adolescents, children >12 months)	Not required	After ruling out TB by symptom screen; also infants <12 months contact
Household contacts <5 years of bacteriologically confirmed pulmonary TB	Not required	Highest priority
Household contacts ≥5 years (expanded)	TST/IGRA where available — not mandatory	Rule out active TB by symptoms ± CXR
Other risk groups	TBI test recommended	Anti-TNF therapy, dialysis, organ/haematological transplant, silicosis

TPT regimens

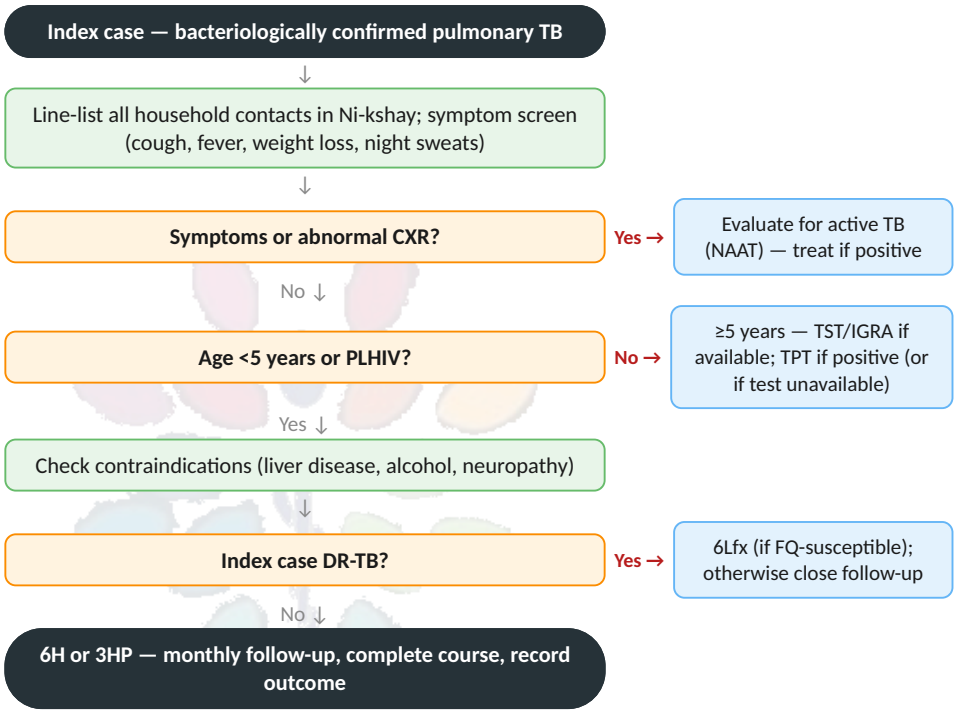
Preventive regimens

Regimen	Description	Use
6H	INH daily ×6 months (adults 5 mg/kg, children 10 mg/kg)	All groups; PLHIV standard
3HP	Weekly INH + rifapentine ×12 doses	Adults and children ≥2 years; not with PIs/some ART (DTG OK)
1HP	Daily INH + rifapentine ×28 days	≥13 years (BRIEF-TB in PLHIV)
3RH / 4R	Rifampicin-based	Alternatives (WHO)

Regimen	Description	Use
6Lfx	Levofloxacin daily ×6 months	Contacts of MDR/RR-TB (FQ-susceptible source) — V-QUIN, TB-CHAMP

- **Contraindications:** active TB (must exclude), acute or chronic hepatitis, regular heavy alcohol, peripheral neuropathy symptoms (for INH), allergy, previous TPT/TB treatment in contacts (relative); rifapentine — interactions with PIs, nevirapine; pregnancy (3HP limited data).
- Monitoring: monthly symptom review, adherence (Ni-kshay TPT module), LFT if risk; isoniazid preventive therapy (IPT) for PLHIV under NACO since 2016.

Algorithm 4.2 · TPT in a household contact



✦ RECENT ADVANCES & GUIDELINE UPDATES

- WHO 2024 consolidated guidelines (Module 1): TB antigen-based skin tests (TBST) acceptable; 6Lfx for contacts of MDR-TB.

★ CLINICAL PEARLS & EXAM FOCUS

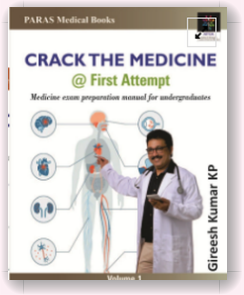
- TPT is pointless — and harmful — if active TB is not excluded first (monotherapy → resistance).

LANDMARK TRIALS & GUIDELINES

- NTEP Guidelines for Programmatic Management of TB Preventive Treatment in India (2021); Swindells S, BRIEF-TB (1HP), NEJM 2019; Fox GJ et al, V-QUIN NEJM 2024.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Eligible population and strategy for tuberculosis preventive therapy (TPT). *[DNB Jun 2025 P3]*
2. Expanded population and strategy for tuberculosis preventive therapy (TPT). *[DNB Jun 2025 P3]*
3. Contraindications to tuberculosis preventive therapy. *[DNB Dec 2025 P3]*
4. What are the newer diagnostic methods of latent tuberculosis? *[DNB May 2024 P2]*
5. Compare tuberculin skin test (TST) and IGRA. *[WBUHS 2022 P3]*
6. What is Mantoux test and its role in primary pulmonary tuberculosis? *[DNB Oct 2023 P3]*
7. What are the causes of false positive tuberculin skin test? *[BFUHS 2022 P3]*
8. Criteria to start TPT (tuberculosis preventive treatment). *[BFUHS 2022 P3]*



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4.5 Treatment of Drug-Susceptible TB under NTEP

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A1(4) Management of pulmonary TB
- ✓ Prescribe the NTEP daily FDC regimen by weight band and monitor treatment
- ✓ Explain principles of anti-TB chemotherapy and causes of acquired drug resistance
- ✓ Manage common adverse drug reactions and treatment interruptions

IN BRIEF New and previously treated drug-sensitive TB receive 2HRZE/4HRE daily as weight-band FDCs for 6 months, with universal DST, HIV and diabetes screening, adherence support and nutrition. Continuation phase may be extended in severe extrapulmonary TB.

Principles of chemotherapy

- Combination therapy prevents emergence of resistance (spontaneous mutation rates 10^{-6}–10^{-8} per drug; cavities contain 10^{7}–10^{9} bacilli).
- Intensive phase kills rapidly multiplying bacilli (sputum conversion); continuation phase sterilises persisters (R, H).
- Daily dosing (not thrice weekly) reduces failure, relapse and acquired resistance.

NTEP regimen for drug-sensitive TB (adults)

Weight band	Intensive phase (8 weeks): HRZE 75/150/400/275	Continuation phase (16 weeks): HRE 75/150/275
25–34 kg	2 tablets	2 tablets
35–49 kg	3 tablets	3 tablets
50–64 kg	4 tablets	4 tablets
65–75 kg	5 tablets	5 tablets
>75 kg	6 tablets	6 tablets

- Paediatric dispersible FDCs (HRZ 50/75/150 + E 100) by weight bands (4–7, 8–11, 12–15, 16–24 kg; 25+ adult).
- **Extension:** CP extended by 3–9 months for TB meningitis, osteoarticular, disseminated TB, or slow responders (total 9–12 months).
- **Shorter 4-month regimens (WHO):** 2HPMZ/2HPM (rifapentine–moxifloxacin, ≥12 years — Study 31/A5349); 4-month HRZ(E) for non-severe TB in children 3 months–16 years (SHINE).

Monitoring

- Clinical (monthly weight, symptoms, ADR), sputum smear/culture at end of IP and end of treatment (follow-up culture where available), LFT if risk, vision (E), blood glucose, HIV.
- **Post-treatment follow-up** at 6, 12, 18, 24 months (clinical ± sputum) for relapse.

INVESTIGATIONS & TREATMENT AT A GLANCE

4.4 Latent TB Infection — TST, IGRA & TB Preventive Treatment

‹ Back to topic (p 71)

Next topic 4.5 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Mantoux TST (5 TU PPD-S / 2 TU RT23)	< 10 mm (< 5 mm in HIV)	≥ 10 mm; ≥ 5 mm in HIV, contacts, immunosuppressed	TB infection
IGRA (QFT-Plus)	TB antigen − Nil < 0.35 IU/mL	≥ 0.35 IU/mL	TB infection (no BCG cross-reaction)
T-SPOT.TB	≤ 4 spots	≥ 8 spots (5–7 borderline)	TB infection
Cy-TB / TBST	Induration < 5 mm	≥ 5 mm	TB infection (WHO conditional)
CXR before TPT	Normal	Any abnormality	Exclude disease first
LFT baseline (risk groups)	ALT < 40 U/L	> 3× ULN	Avoid/monitor INH regimens

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
6H	Isoniazid 5 mg/kg (300 mg) daily × 6 months (+ pyridoxine)	PLHIV, contacts	Hepatotoxicity, neuropathy
3HP	Isoniazid 900 mg + rifapentine 900 mg weekly × 12 doses (≥ 30 kg)	Contacts ≥ 2 years, PLHIV on DTG	Flu-like reactions; interactions
3HR	H 5 + R 10 mg/kg daily × 3 months	Children	Rifampicin interactions
1HP	H 300 + rifapentine 600 mg daily × 28 days	≥ 13 years	Interactions
6Lfx	Levofloxacin 750–1000 mg (15–20 mg/kg children) daily × 6 months	Contacts of MDR/RR-TB (WHO 2024)	Tendinopathy, QT

★ **PRESCRIBING & INTERPRETATION PEARLS**

- TST/IGRA should not be used to diagnose active TB in high-burden countries (WHO Module 3).

- Adherence technologies: 99DOTS (pill pack + missed calls), MERM boxes, video-observed therapy, family DOT; Ni-kshay Sampark helpline 1800-11-6666.

Common ADRs and management

Adverse reactions to first-line drugs

Reaction	Likely drug	Management
Gastric upset	R, Z, H	Take with light food/at bedtime; antiemetic; check LFT
Hepatitis	H, R, Z	Stop and reintroduce (see 3.8)
Rash, pruritus	Any (Z, R, H)	Mild — antihistamine; severe/SJS — stop all, desensitise/reintroduce one by one
Peripheral neuropathy	H	Pyridoxine 50–100 mg
Arthralgia, gout	Z	NSAIDs; allopurinol not effective (urate), stop Z if gout
Visual impairment	E	Stop E; ophthalmology
Flu-like syndrome, thrombocytopenia, renal failure	R (intermittent)	Stop R permanently if thrombocytopenia/haemolysis/AKI

Treatment interruption (NTEP)

- Interruption <1 month — trace, continue, extend to complete doses. ≥1 month — "lost to follow-up"; re-evaluate with NAAT/UDST; restart full regimen if positive/resistant change.

Factors promoting acquired drug resistance

- **Programme/provider:** inappropriate regimens, monotherapy, irregular supply, poor-quality drugs, no DST, unregulated private sector prescriptions.
- **Patient:** non-adherence, ADRs, malabsorption (HIV, diabetes, diarrhoea), stigma, poverty, alcohol, migration.
- **Drug:** sub-therapeutic levels (low weight band dosing, interactions — rifampicin with PIs), cavitary high bacillary load.

✦ RECENT ADVANCES & GUIDELINE UPDATES

▸ Dorman SE et al, Study 31/A5349 (NEJM 2021): 4-month rifapentine–moxifloxacin non-inferior; Paton NI et al, TRUNCATE-TB (NEJM 2023): 2-month strategy with extended follow-up.

★ CLINICAL PEARLS & EXAM FOCUS

▸ The FDC weight bands and "2HRZE/4HRE daily" are the most-quoted NTEP facts — write the table.

LANDMARK TRIALS & GUIDELINES

- WHO consolidated guidelines Module 4: Treatment of drug-susceptible TB (2022); NTEP Technical and Operational Guidelines (2016, updated 2021–24).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Factors promoting emergence of drug resistance during anti-tubercular therapy. *[BFUHS 2020 P3]*

INVESTIGATIONS & TREATMENT AT A GLANCE

4.5 Treatment of Drug-Susceptible TB under NTEP

‹ Back to topic (p 74)

Next topic 4.6 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Sputum smear end of IP (2 months)	Negative	Positive	Do NOT extend IP; do NAAT/LPA + culture DST
Sputum smear end of treatment	Negative	Positive	Failure work-up
Weight monthly	Gain	Loss / no gain	Adjust weight band; non-response
ALT/bilirubin	Normal	ALT > 3× ULN + symptoms or > 5×	DILI
Visual acuity/colour	Normal	Defect	Ethambutol toxicity
FL-LPA / Xpert XDR (Rif-sensitive)	INH susceptible	INH resistant	Switch to Hr-TB regimen

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
4FDC HRZE (75/150/400/275)	25–34 kg: 2; 35–49: 3; 50–64: 4; 65–75: 5; > 75: 6 tabs	Intensive phase 2 months	Daily; with/after food
3FDC HRE (75/150/275)	Same weight bands	Continuation phase 4 months (NTEP)	Extend 3–6 months in selected EPTB
Pyridoxine	10–25 mg daily	Risk of neuropathy (DM, HIV, alcohol, pregnancy, malnutrition)	—
4-month HPMZ (WHO)	Rifapentine 1200 mg + Mfx 400 + H 300 + Z × 2 months, then HPM × 2	≥ 12 years, DS-PTB (WHO; not NTEP policy)	Interactions; not < 40 kg, pregnancy, EPTB

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Thrice-weekly regimens are no longer recommended (WHO Module 4, Rec. 1.3).

4.6 Drug-Resistant TB — Definitions, Risk Factors, Surveillance & Diagnostic Algorithm

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A1(13) MDR and XDR-TB
- ✓ Define mono-, poly-, MDR, pre-XDR and XDR-TB (WHO 2021)
- ✓ Describe risk factors, NDRS findings and the NTEP integrated DR-TB diagnostic algorithm
- ✓ Describe the structure and functions of DR-TB centres

IN BRIEF Drug resistance arises from chromosomal mutations selected by inadequate treatment (acquired) and is transmitted to others (primary/transmitted — now the majority of MDR-TB). Universal DST under NTEP ensures every TB patient is tested for at least rifampicin and isoniazid.

Definitions (WHO 2021)

Category	Definition
Hr-TB	Resistance to isoniazid, susceptible to rifampicin
RR-TB	Resistance to rifampicin (with or without other drugs)
MDR-TB	Resistance to both isoniazid and rifampicin
Pre-XDR-TB	MDR/RR-TB + resistance to any fluoroquinolone
XDR-TB	MDR/RR-TB + FQ + at least one Group A drug (bedaquiline or linezolid)
Poly-resistant	>1 first-line drug other than H+R combination

Risk factors for drug-resistant TB

- Previous TB treatment (especially failure, relapse, lost to follow-up), contact of DR-TB, non-response at end of IP, private-sector or irregular treatment, HIV, diabetes, healthcare workers, prisoners, migrants, residence in high-MDR area.

National Drug Resistance Survey (NDRS) 2014–16 — salient findings

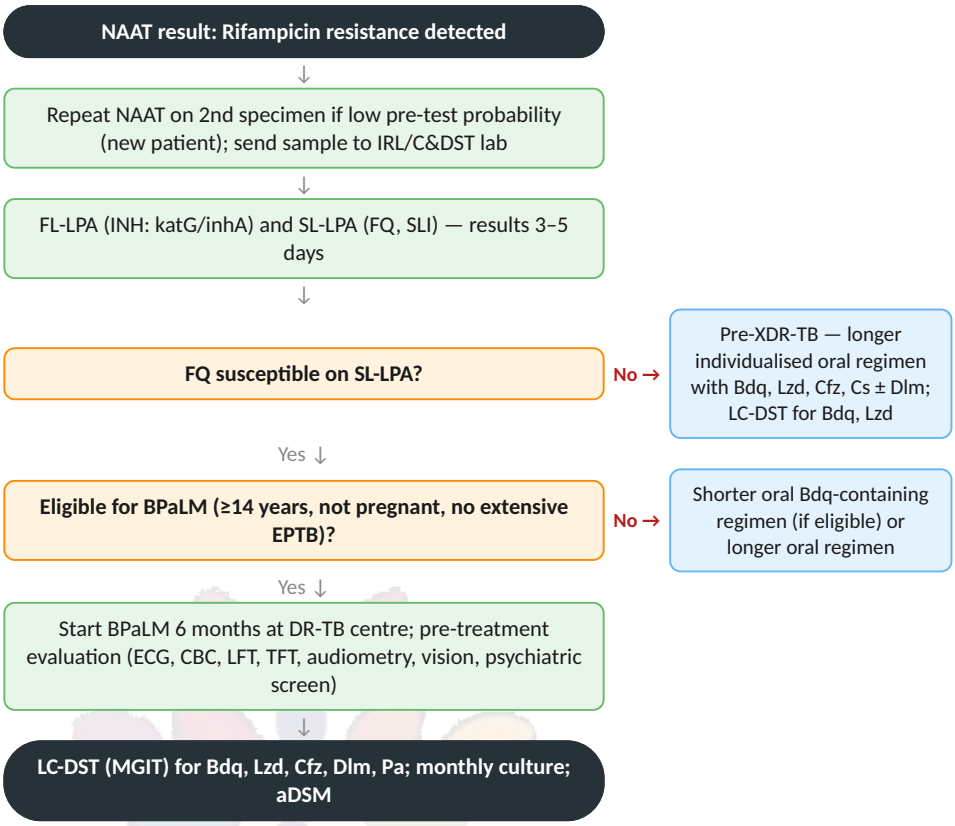
NDRS key results (first-ever national survey, 5,214 patients)

Parameter	New	Previously treated
MDR-TB	2.84%	11.62%
Any isoniazid resistance	11.06%	25.09%
Any rifampicin resistance	3.22%	13.06%

- Any drug resistance overall 28%; FQ resistance among MDR-TB ~21.8%; high INH mono-resistance justified universal INH testing (FL-LPA) and Hr-TB regimen; findings shaped the shorter-regimen eligibility (SL-LPA).

Academy for Emergency, Trauma & Critical care Medicine

Algorithm 4.3 · NTEP integrated DR-TB diagnostic pathway



DR-TB centres (NTEP)

- **Nodal DR-TB centre** (medical college/large hospital with indoor facility): initiation of DR-TB treatment, management of complicated cases, ADR management, DR-TB committee, training, operational research.
- **District DR-TB centre:** initiation and follow-up of uncomplicated patients, decentralised care; **Intermediate Reference Labs (IRL)** and **C&DST labs** for LPA/culture; National Reference Labs (NITRD Delhi, NIRT Chennai, NTI Bengaluru).
- Responsibilities: pre-treatment evaluation, counselling, regimen decision, admission when needed, aDSM, linkage to NPY and Ni-kshay, follow-up cultures, outcome reporting.

★ CLINICAL PEARLS & EXAM FOCUS

▸ XDR now requires FQ + Bdq or Lzd resistance — injectables are no longer part of the definition (WHO 2021).

LANDMARK TRIALS & GUIDELINES

- Report of the First National Anti-TB Drug Resistance Survey, India 2014–16 (MoHFW, 2018); WHO definitions update (2021).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Integrated diagnostic and treatment algorithm for drug resistant tuberculosis. *[DNB Jun 2025 P3]*
2. Various diagnostic methods (phenotypic and genotypic) used in the diagnosis of drug resistant tuberculosis and their role in programmatic management of DR-TB. *[DNB Oct 2024 P3]*
3. Risk factors for drug resistant tuberculosis. *[DNB Jun 2020 P3]*
4. Salient findings of National Drug Resistance Survey (NDRS) 2014–16. *[DNB Jun 2020 P3]*
5. Drug resistant TB centres under RNTCP – roles and responsibilities. *[DNB Dec 2022 P3]*
6. Discuss in detail the drug resistance in tuberculosis. *[Manipal 2014 P3]*
7. XDR tuberculosis. *[Manipal 2014 P3]*

INVESTIGATIONS & TREATMENT AT A GLANCE

4.6 Drug-Resistant TB — Definitions, Risk Factors, Surveillance & Diagnostic Algorithm

‹ Back to topic (p 76)
Next topic 4.7 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Xpert Rif resistance	Not detected	Detected	RR-TB — treat as MDR
FL-LPA	Wild-type	katG (high-level INH), inhA (low-level INH + ethionamide) mutations	Guides H use
SL-LPA / Xpert XDR	Susceptible	FQ resistance	Pre-XDR — use BPaL/BDLC
Liquid culture DST (Bdq, Lzd, Cfz, Dlm)	Susceptible	Resistant	XDR definition (FQ + Bdq/Lzd)
tNGS	No mutation	Resistance mutations	Comprehensive DST (WHO 2025)

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
MDR/RR-TB regimen	BPaLM / BDLLfxC / 9-month	RR-TB	See 4.7
Hr-TB regimen	6(Lfx)REZ	INH-resistant, Rif-susceptible	Rule out FQ resistance
Contact TPT	6Lfx	Contacts of MDR	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Risk factors for DR-TB: previous treatment, contact of DR-TB, non-conversion, private-sector irregular treatment, HIV, prisons.

4.7 Treatment of MDR/RR-TB — BPaLM, Shorter & Longer Oral Regimens

‹ NMC curriculum

Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A1(13) Management of MDR and XDR TB
- ✓ Classify second-line drugs into WHO groups for designing longer regimens
- ✓ Describe BPaLM, the shorter oral bedaquiline-containing regimen and their exclusion criteria
- ✓ Monitor patients on DR-TB treatment and manage ADRs

IN BRIEF All-oral regimens have replaced injectables. NTEP (2024) recommends the 6-month BPaLM regimen as first choice for MDR/RR-TB in eligible patients ≥14 years, with the 9–11-month shorter oral regimen and the 18–20-month longer oral regimen for those not eligible.

WHO grouping of drugs for longer MDR-TB regimens

Group	Drugs	Rule
A	Levofloxacin or moxifloxacin; bedaquiline; linezolid	Include all three
B	Clofazimine; cycloserine or terizidone	Add one or both
C	Ethambutol, delamanid, pyrazinamide, imipenem–cilastatin or meropenem (with clavulanate), amikacin (streptomycin), ethionamide/prothionamide, p-aminosalicylic acid	Add to complete regimen when A and B cannot be used

NTEP MDR/RR-TB regimens

Regimen	Composition	Duration	Eligibility / exclusions
BPaLM	Bedaquiline + Pretomanid + Linezolid (600 mg) + Moxifloxacin	6 months (26 weeks; extend to 9 if culture positive at 4 months)	MDR/RR-TB ≥14 years; FQ-susceptible (BPaL if FQ-resistant). Excluded: pregnancy/lactation, <14 years, TB meningitis/ osteoarticular/disseminated, >1 month prior exposure to Bdq/ Lzd/Pa/Dlm, known resistance
Shorter oral Bdq-containing	4–6 Bdq(6) Lfx Cfz Z E Hh Eto / 5 Lfx Cfz Z E	9–11 months	FQ-susceptible; no extensive disease; no resistance to component (inhA + katG mutations both → exclude); not severe EPTB; children and pregnancy allowed
Longer oral (individualised)	18–20 months: Lfx/Mfx + Bdq (6 months or longer) + Lzd + Cfz + Cs ± others	18–20 months	Pre-XDR/XDR, failures, not eligible for above
Hr-TB	6 (Lfx) R E Z	6 months	INH-resistant, RIF-susceptible

Pre-treatment evaluation and monitoring

- Baseline: weight, CBC, LFT, RFT, electrolytes (K, Mg, Ca), TSH, blood sugar, HIV, pregnancy test, ECG (QTcF), audiometry (if amikacin), visual acuity and colour vision (Lzd, E), psychiatric screen (Cs), UPT, CXR.
- Monthly: smear/culture (to month 6, then per regimen), weight, CBC (Lzd), ECG (Bdq/Dlm/Cfz/Mfx — at 2, 12, 24 weeks and when symptoms), LFT, peripheral neuropathy screen (BPNS).
- **Outcomes:** culture conversion = 2 consecutive negative cultures ≥7 days apart.

ADR management in DR-TB

- **Myelosuppression / neuropathy (Lzd):** reduce to 300 mg or stop; vitamin B6. **QTcF >500 ms:** stop QT drugs (Bdq, Cfz, Mfx), correct electrolytes, ECG.
- **Hepatotoxicity (Pa, Bdq, Z):** stop and reintroduce. **Psychosis (Cs):** reduce/stop, antipsychotics. **Hypothyroidism (Eto, PAS):** levothyroxine.

✦ RECENT ADVANCES & GUIDELINE UPDATES

- WHO 2025 rapid communication: 6-month BDLLfxC regimen (bedaquiline, delamanid, linezolid, levofloxacin, clofazimine) for all ages and in pregnancy; and 9-month BLMZ / BLLfxCZ / BDLLfxZ options from endTB.

★ CLINICAL PEARLS & EXAM FOCUS

- BPaLM exclusions — "PEPPER": Pregnancy, EPTB severe, Paediatric <14, Prior exposure (>1 month) to new drugs, Extensive resistance (Bdq/Lzd/Pa), and Relapse after these drugs.

LANDMARK TRIALS & GUIDELINES

- Nyang'wa BT et al, TB-PRACTECAL NEJM 2022 (BPaLM 89% favourable vs 52% standard); NTEP Guidelines for Programmatic Management of Drug-Resistant TB (2021) and BPaLM addendum (2024).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Classification of drugs for drug resistant tuberculosis under NTEP. *[DNB Dec 2025 P1]*
2. Grouping of anti-TB drugs for designing longer all oral MDR-TB regimen. *[DNB Dec 2021 P3]*
3. Shorter oral regimen and its exclusion criteria for MDR/RR-TB patients. *[DNB Dec 2021 P3]*
4. Shorter MDR-TB regimen and its exclusion criteria as per NTEP of India. *[DNB Dec 2022 P4]*
5. Describe classification of second line anti-TB drugs for management of MDR/RR-TB as per WHO guidelines. *[WBUHS 2020 P3]*
6. BPaL regimen. *[BFUHS 2022 P4]*
7. Newer regimens for MDR and XDR tuberculosis including injection-free regimens. *[BFUHS 2019 P4]*

4.8 TB in Special Situations — Pregnancy, Renal Failure, Liver Disease, Diabetes & the Elderly

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A1(7,12) Geriatric TB; diabetes and TB
- ✓ Manage TB in pregnancy and lactation including drug-resistant TB
- ✓ Modify anti-TB therapy in renal failure and chronic liver disease
- ✓ Manage TB–diabetes comorbidity and TB in the elderly

IN BRIEF Most first-line drugs are safe in pregnancy; renal failure needs dose-interval adjustment of renally cleared drugs (E, Z, FQs, aminoglycosides); liver disease needs regimens with fewer hepatotoxic drugs; and diabetes doubles to triples TB risk and worsens outcomes.

Pregnancy and lactation

- Untreated TB is far more dangerous than treatment. HRZE safe in pregnancy; add pyridoxine 25–50 mg; **avoid streptomycin/aminoglycosides** (fetal ototoxicity) and ethionamide (teratogenic).
- **DR-TB in pregnancy:** bedaquiline, linezolid, levofloxacin, clofazimine, cycloserine, delamanid considered acceptable (WHO 2022/2025 — BDLLfxC); avoid injectables, ethionamide; BPaLM not recommended (pretomanid data lacking); shorter oral regimen with modifications (replace Eto with Lzd).
- **Lactation:** continue breastfeeding; infant receives TPT (6H) after excluding TB (BCG at birth); mother wears mask if sputum-positive in first weeks.
- **Congenital TB** (Cantwell criteria): proven TB in the infant within the first week, primary complex in liver, maternal genital/placental TB, or exclusion of postnatal transmission.
- 16-week pregnant woman with **Xpert trace positive, RIF indeterminate:** treat as TB (trace = MTB detected in high-risk), send fresh sample for repeat NAAT/LPA/culture-DST; start DS-TB regimen while awaiting DST; screen HIV/diabetes; contact tracing.

INVESTIGATIONS & TREATMENT AT A GLANCE

4.7 Treatment of MDR/RR-TB — BPaLM, Shorter & Longer Oral Regimens

‹ Back to topic (p 78)

Next topic 4.8 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
ECG QTcF	< 450 ms	> 500 ms	Stop QT-prolonging drugs
CBC monthly	Hb > 10; platelets > 150	Hb < 8; platelets < 50	Linezolid toxicity
Monthly smear & culture	Negative	Persistently positive at 4 months	Failure work-up
LFT	Normal	ALT > 3× ULN	Bdq/Pa/Z hepatotoxicity
Visual acuity, colour, BPNS	Normal	Abnormal	Linezolid optic/peripheral neuropathy
Pregnancy test	Negative	Positive	BPaLM not used — choose BDLLfxC

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
BPaLM	Bdq 400 mg × 2 wk → 200 mg TIW; Pa 200 mg; Lzd 600 mg; Mfx 400 mg daily × 26 weeks	MDR/RR-TB ≥ 14 years	QT, neuropathy, myelosuppression
BPaL	As above without Mfx	Pre-XDR-TB	—
BDLLfxC	Bdq + Dlm 100 mg bd + Lzd 600 + Lfx 750–1000 + Cfz 100 mg × 6 months	All ages incl. pregnancy (WHO 2025)	Adjust by FQ result
9-month oral regimen (NTEP shorter oral)	Bdq 6 months + Lfx, Cfz, Z, E, Hh, Eto (4–6 m) / Lfx, Cfz, Z, E (5 m)	FQ-susceptible, not eligible for 6-month	Eto hypothyroidism, GI
Longer regimen	Lfx/Mfx + Bdq + Lzd + Cfz/Cs ± C drugs, 18–20 months	XDR, intolerance, severe EPTB	aDSM

★ PRESCRIBING & INTERPRETATION PEARLS

- Linezolid toxicity: reduce to 300 mg for grade 2 neuropathy/myelosuppression; stop for optic neuropathy.

Anti-TB drugs in renal failure (CrCl <30 mL/min or dialysis)

Drug	Adjustment
Isoniazid, rifampicin	No change (pyridoxine)
Pyrazinamide	25–35 mg/kg three times weekly (after dialysis)
Ethambutol	15–25 mg/kg three times weekly
Levofloxacin	750–1000 mg three times weekly
Moxifloxacin, bedaquiline, linezolid, clofazimine	No change (Lzd — watch myelosuppression)
Amikacin / streptomycin	Avoid (or 12–15 mg/kg 2–3 times weekly with levels)
Cycloserine	250 mg OD or 500 mg thrice weekly

TB with chronic liver disease (WHO/NTEP)

Regimens by severity of liver disease

Liver status	Regimen
Stable, ALT <3× ULN, Child A	Standard regimen with close LFT monitoring; or 2 hepatotoxic drugs: 2HRE + Lfx? (9HRE)
Child B (advanced)	One hepatotoxic drug: 9 months RE + Lfx ± H avoided; e.g., 2RE-Lfx/10RE
Child C (decompensated)	No hepatotoxic drugs: 18–24 months E + Lfx + Lzd (± Cs/aminoglycoside)
Acute hepatitis	Defer if possible; otherwise E + Lfx + Lzd until hepatitis resolves

Diabetes and TB

- Diabetes triples risk of active TB; more cavities, lower lobe disease, higher bacillary load, slower conversion, more failure, relapse and death.
- **Bidirectional screening** (NTEP): all TB patients screened for diabetes (RBS/HbA1c); diabetics screened for TB symptoms at every visit.
- Rifampicin induces sulfonylureas, DPP-4, insulin need ↑; prefer insulin in severe hyperglycaemia; INH neuropathy risk — pyridoxine; consider extending CP if slow response.

Geriatric TB

- Atypical presentation (less fever, more confusion, weight loss), lower-zone and non-cavitary disease, more ADRs (hepatotoxicity, renal), polypharmacy interactions; higher mortality. Needs careful dosing, nutritional support, adherence assistance.

★ CLINICAL PEARLS & EXAM FOCUS

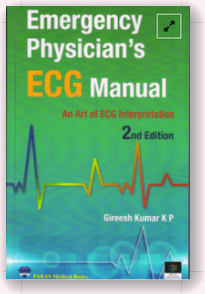
▸ Streptomycin is the only first-line drug to avoid in pregnancy; in CKD, give E and Z three times weekly after dialysis.

LANDMARK TRIALS & GUIDELINES

- WHO consolidated guidelines Module 4 (2022); Collaborative Framework for Care and Control of TB and Diabetes (WHO/IUATLD); NTEP National Framework for joint TB–diabetes collaborative activities (2017).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Management of tuberculosis in pregnant and lactating women. *[DNB Dec 2021 P3]*
2. Management of tuberculosis in pregnancy. *[WBUHS 2020 P3; WBUHS 2022 P3]*
3. Management of MDR-TB in pregnancy. *[DNB Jun 2020 P3]*
4. Management of TB in patients with liver disorders. *[DNB Jun 2020 P3]*
5. Management of TB in patients with renal failure and severe renal insufficiency. *[DNB Jun 2020 P3]*
6. Management of TB in patients with severe renal insufficiency. *[DNB Dec 2022 P3]*
7. Describe management of TB in renal failure. *[WBUHS 2020 P3]*
8. A 16-week pregnant woman with persistent cough: Xpert MTB trace positive, rifampicin resistance indeterminate. Discuss further management. *[BFUHS 2022 P2]*



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4.9 HIV–TB Co-infection, ART Interactions & IRIS

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A1(11) HIV and TB; I-C11
- ✓ Describe the magnitude and clinical/radiological features of HIV–TB
- ✓ Time ART in TB and DR-TB, and manage rifampicin–ART interactions
- ✓ Diagnose and manage paradoxical TB-IRIS

IN BRIEF HIV increases TB risk 18-fold; TB is the leading cause of death among PLHIV. Management integrates early ART (within 2 weeks; delayed in TB meningitis), co-trimoxazole prophylaxis, TPT and dolutegravir-based ART adjusted for rifampicin.

Magnitude

- ~6% of global TB cases are PLHIV (WHO 2024); India ~2–3% of notified TB patients HIV-positive (higher in NE states, Andhra/Karnataka/Maharashtra). Intensified TB case finding at ART centres ("4-symptom screen") and HIV testing of all TB patients (single-window services).

Clinical and radiological features

TB in early vs advanced HIV

Feature	CD4 >350	CD4 <200
Presentation	Typical post-primary	Primary-like, disseminated, extrapulmonary (LN, pleura, meninges)
CXR	Upper lobe cavities	Lower/middle zone infiltrates, adenopathy, miliary, effusion; normal CXR in 10–20%
Sputum smear	Usually positive	Often negative (paucibacillary) — use Ultra, urine LF-LAM, culture
Mortality	Similar to HIV-negative	High

INVESTIGATIONS & TREATMENT AT A GLANCE

4.8 TB in Special Situations — Pregnancy, Renal Failure, Liver Disease, Diabetes & the Elderly

‹ Back to topic (p 79)

Next topic 4.9 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
eGFR	> 60 mL/min	< 30 mL/min	Renal dosing (Z, E, Lfx thrice weekly)
LFT / INR / albumin	Normal	Child–Pugh B/C	Liver-sparing regimen
HbA1c	< 6.5% (target < 7% on treatment)	> 8%	Poor TB outcomes — intensify glycaemic control
Pregnancy test / obstetric scan	—	Positive	Safe regimen choice
HBsAg, anti-HCV	Negative	Positive	DILI risk

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Pyrazinamide (CKD)	25 mg/kg three times weekly after dialysis	CrCl < 30 / HD	—
Ethambutol (CKD)	15 mg/kg three times weekly	CrCl < 30 / HD	Optic toxicity
Liver-sparing regimen	2HRE/7HR (no Z) or R+E+Lfx (12–18 m)	Liver disease	Hepatology input
Insulin	Basal-bolus as needed	DM with TB (rifampicin ↓ OHAs)	Hypoglycaemia after rifampicin stops
HRZE in pregnancy	Standard + pyridoxine	Pregnancy	Avoid aminoglycosides, ethionamide, pretomanid

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Rifampicin reduces sulfonylurea and DPP-4 inhibitor levels — doses may need adjustment.

ART in TB

- **Timing:** start ART within 2 weeks of anti-TB treatment regardless of CD4 (as soon as tolerated); **TB meningitis — delay ART 4–8 weeks** (higher mortality with early ART).
- **DR-TB:** start ART within 2 weeks of DR-TB treatment; avoid efavirenz with bedaquiline (lowers Bdq) — use dolutegravir.
- **Preferred ART (NACO):** TLD (tenofovir + lamivudine + dolutegravir); with rifampicin give **dolutegravir 50 mg twice daily** (extra dose 12 h later) until 2 weeks after rifampicin stops.
- PIs (lopinavir/r, atazanavir/r) — avoid with rifampicin; use rifabutin 150 mg daily instead.
- Co-trimoxazole prophylaxis for all HIV–TB; TPT after completion for PLHIV per guidelines.

TB-IRIS and paradoxical reactions

- **Paradoxical TB-IRIS:** worsening of TB features (fever, enlarging nodes, effusion, new infiltrates, tuberculomas) 1–4 weeks after ART initiation in a patient improving on ATT. **Unmasking IRIS:** new TB appearing after ART.
- **Risk:** low CD4 (<100), high viral load, short ATT–ART interval, disseminated TB.
- **Diagnosis (INSHI criteria):** after excluding treatment failure, drug resistance, another OI, non-adherence.
- **Management:** continue ATT and ART; NSAIDs for mild; **prednisolone 1.5 mg/kg/day ×2 weeks then 0.75 mg/kg ×2 weeks** (Meintjes) for moderate–severe; aspirate abscesses/nodes; prophylactic prednisone 40 mg/day ×14 days then 20 mg reduces IRIS in high-risk (PredART). Stop ART only if life-threatening (CNS).
- **Paradoxical reaction in HIV-negative patients:** 10–25% (nodes, CNS, pleura) — continue ATT, steroids for severe.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Rifampicin + dolutegravir → DTG 50 mg BD; rifampicin + PI → swap to rifabutin.

LANDMARK TRIALS & GUIDELINES

- Abdool Karim SS et al, SAPiT NEJM 2010/2011; Meintjes G et al, PredART NEJM 2018; NACO National Guidelines for HIV Care and Treatment (2021).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. HIV co-infection among TB patients – magnitude and management. *[DNB Dec 2022 P3]*
2. HIV co-infection among TB patients. *[DNB Jun 2020 P3]*
3. Anti-retroviral therapy in HIV infected TB patients. *[DNB Jun 2020 P3]*
4. Initiating ART in patients with DR-TB. *[DNB Dec 2021 P3]*
5. Immune reconstitution inflammatory syndrome. *[DNB Oct 2024 P3]*
6. Paradoxical reaction. *[DNB Jun 2025 P3]*
7. Discuss the radiological features of HIV–TB co-infection. *[WBUHS 2020 P3]*
8. Discuss TB in HIV patient with respect to epidemiology, diagnosis and treatment. *[WBUHS 2022 P3]*

4.10 Paediatric TB & Childhood Contact Management

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A1(6) Tuberculosis in children
- ✓ Diagnose paediatric TB using the NTEP algorithm and appropriate specimens
- ✓ Treat paediatric TB with dispersible FDCs and newer drugs including DR-TB in children
- ✓ Evaluate and manage child contacts of TB patients

IN BRIEF Children have paucibacillary disease and cannot expectorate; diagnosis combines exposure history, symptoms, CXR, TST/IGRA and microbiology from gastric aspirate, induced sputum, stool (Ultra) or nasopharyngeal aspirate. Bacteriological confirmation is achieved in only 30–40%.

INVESTIGATIONS & TREATMENT AT A GLANCE

4.9 HIV–TB Co-infection, ART Interactions & IRIS

‹ Back to topic (p 81)
Next topic 4.10 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
CD4 count	> 500 cells/µL	< 200 (high risk); < 50 (urgent ART)	Timing/IRIS risk
HIV viral load	< 50 copies/mL on ART	> 1000	Treatment failure
Urine LF-LAM	Negative	Positive	TB in advanced HIV
CrAg (serum)	Negative	Positive	Pre-emptive fluconazole before ART
Xpert on sputum + extra-pulmonary samples	Not detected	Detected	Concurrent testing

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Dolutegravir	50 mg twice daily with rifampicin (and 2 weeks after)	ART with rifampicin	Insomnia; metformin interaction
Efavirenz	600 mg daily	ART with rifampicin (no adjustment)	CNS effects
Rifabutin	150 mg daily	With protease inhibitors	Uveitis, neutropenia
Co-trimoxazole prophylaxis	960 mg daily	All HIV–TB	Rash, cytopenia
Prednisone (IRIS prevention)	40 mg × 2 weeks, then 20 mg × 2 weeks	CD4 ≤ 100 starting ART (PredART)	Exclude Kaposi, resistance

★ PRESCRIBING & INTERPRETATION PEARLS

- ART within 2 weeks of ATT (any CD4); defer 4–8 weeks in TB meningitis; DR-TB within 8 weeks.

Diagnosis

- **Presumptive TB in a child:** fever ≥2 weeks, persistent cough ≥2 weeks, weight loss/no gain (or failure to thrive), lethargy; contact history.
- **Specimens:** gastric aspirate (early morning, 2 days), induced sputum, nasopharyngeal aspirate, stool (Xpert Ultra), BAL; tissue/EPTB samples.
- **Tests:** NAAT (Ultra preferred), culture; TST/IGRA supportive; CXR (hilar nodes); WHO treatment decision algorithms (2022) where tests unavailable.

Paediatric TB treatment (NTEP)

Situation	Regimen
Non-severe TB (3 months–16 years) — peripheral LN, non-cavitary disease confined to 1 lobe, no airway obstruction	4-month 2HRZ(E)/2HR (SHINE)
Pulmonary TB / most EPTB	2HRZE/4HRE (dispersible FDCs by weight band)
TB meningitis, osteoarticular	2HRZE/10HRE; or 6-month intensive HRZ-Eto regimen (WHO 2022)
DR-TB	Bdq (all ages) and Dlm (all ages) allowed; shorter oral regimen; BDLLfxC (WHO 2025)

Newer drugs in children

- **Bedaquiline:** approved for all ages (WHO 2022) with weight-based dosing (20 mg dispersible tablets). **Delamanid:** all ages (25 mg tablets). Child-friendly formulations of Lzd, Lfx, Cfz; avoid injectables (hearing loss).
- Pretomanid not yet for <14 years.

Child contacts

- All child contacts (household) screened; symptomatic → evaluate; asymptomatic <5 years → TPT (6H/3HP if ≥2 years); ≥5 years TPT after testing where available; MDR contacts → 6Lfx; follow up 2 years.
- **BCG** at birth protects against severe forms (TBM, miliary) in infants.

Congenital and neonatal TB

- Cantwell criteria (see 4.8); neonate of mother with active TB: evaluate; if well → 6H then BCG (or BCG at birth per NTEP with TPT); breastfeeding continues.

★ CLINICAL PEARLS & EXAM FOCUS

▸ SHINE trial — 4 months enough for non-severe childhood TB.

LANDMARK TRIALS & GUIDELINES

- Turkova A et al, SHINE NEJM 2022; WHO consolidated guidelines Module 5: Management of TB in children and adolescents (2022).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Consideration of newer drugs for treatment of tuberculosis in children. *[DNB Dec 2021 P3]*
2. Evaluation and management of children in contact with tuberculosis patient. *[DNB Jun 2020 P3]*
3. Discuss diagnosis and management of paediatric TB under RNTCP. *[WBUHS 2020 P3]*
4. Congenital tuberculosis. *[Manipal 2014 P3]*

INVESTIGATIONS & TREATMENT AT A GLANCE

4.10 Paediatric TB & Childhood Contact Management

‹ Back to topic (p 82)
Next topic 4.11 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Xpert Ultra (respiratory + stool)	Not detected	Detected	Concurrent testing (WHO 2025)
Gastric aspirate / induced sputum	—	NAAT/culture positive	Microbiological confirmation
TST / IGRA	Negative	TST ≥ 10 mm (≥ 5 mm in HIV/ malnourished)	Infection/support
CXR	Normal	Hilar/paratracheal nodes, miliary, consolidation	Primary TB
Weight-for-age / BMI z-score	> −2 SD	< −2 SD; failure to thrive	Malnutrition — nutritional support

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Paediatric FDC (H50/R75/ Z150) + E100	4–7 kg: 1; 8–11: 2; 12–15: 3; 16–24: 4 tablets (dispersible)	Childhood TB (NTEP)	Dose by weight band; adjust monthly
4-month regimen 2HRZ(E)/ 2HR	Weight band	Non-severe TB, 3 months–16 years (SHINE)	Not for severe/miliary/TBM
TPT for child contacts	6H, 3HP (≥ 2 y), 3HR	All < 5 years after excluding disease	—
6Lfx	15–20 mg/kg daily × 6 months	Child contacts of MDR-TB	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Paediatric doses: H 10, R 15, Z 35, E 20 mg/kg/day.

4.11 Miliary TB & TB Meningitis

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A1(10) Extrapulmonary TB
- ✓ Describe pathogenesis, diagnosis, complications and management of miliary TB
- ✓ Approach a patient with altered sensorium and miliary shadows
- ✓ Describe TBM staging, sequelae and treatment including steroids

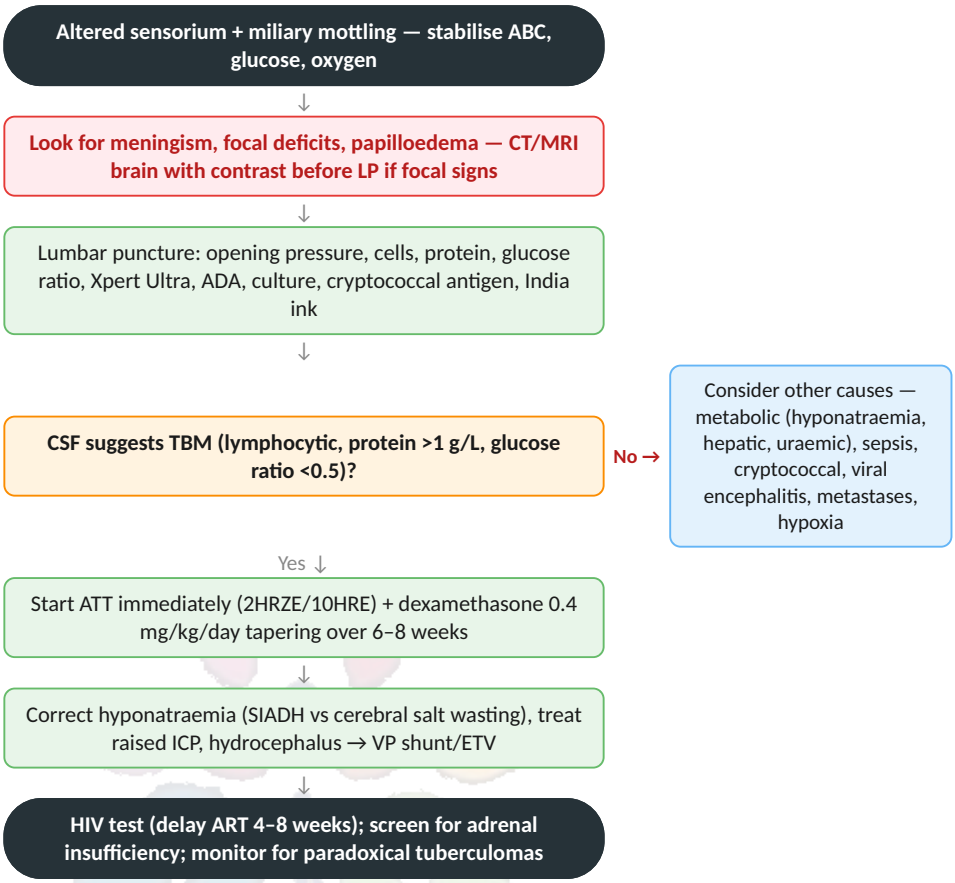
IN BRIEF Miliary TB is haematogenous dissemination producing 1–3 mm uniform nodules; up to 30% have concomitant TB meningitis. Prompt empirical treatment is life-saving because microbiological confirmation is often delayed.

Miliary TB

- **Pathogenesis:** progressive primary infection or erosion of a caseous focus into a vein (Weigert's focus) → massive bacteraemia; risk — infancy, HIV, malnutrition, diabetes, steroids/anti-TNF, pregnancy/postpartum, CKD, elderly (cryptic miliary).
- **Clinical:** fever (often PUO), weight loss, hepatosplenomegaly, choroid tubercles (fundus — pathognomonic), meningism, ARDS, adrenal insufficiency, DIC.
- **Investigations:** CXR (2–3 weeks to appear; 30% normal), HRCT (random nodules), sputum/BAL/gastric NAAT and culture, urine Ultra/LF-LAM, bone marrow and liver biopsy (granulomas, NAAT, culture), CSF (all patients), fundoscopy, blood culture (MGIT myco), CBC (anaemia, pancytopenia), hyponatraemia (SIADH/adrenal), LFT (cholestatic).
- **Complications:** ARDS (mortality high), TBM, DIC, HLH, adrenal crisis, myocarditis, pericarditis, renal failure.
- **Treatment:** 2HRZE/4HRE (extend to 9–12 months if meningitis); steroids if TBM, pericarditis, adrenal insufficiency, ARDS (selected), IRIS.

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Algorithm 4.4 · Altered sensorium with miliary shadows on chest X-ray



TB meningitis — staging and sequelae

MRC (modified BMRC) staging of TBM

Stage	Features	Mortality
I	GCS 15, no focal deficit	~10–20%
II	GCS 11–14, or 15 with focal deficit	~30%
III	GCS ≤10	~50–60%

- **Sequelae:** hydrocephalus (communicating > obstructive), cranial nerve palsies (VI, III, VII), hemiplegia (vasculitis — MCA perforators), optochiasmatic arachnoiditis (blindness), cognitive impairment, epilepsy, hypothalamic–pituitary dysfunction (SIADH, diabetes insipidus), tuberculomas, spinal arachnoiditis (radiculomyelitis), learning disability in children.
- High-dose rifampicin and adjunctive aspirin under study (INTENSE-TBM, ACT HIV).

★ CLINICAL PEARLS & EXAM FOCUS

▸ Choroid tubercles on fundoscopy in a febrile patient = miliary TB — always examine the fundus.

LANDMARK TRIALS & GUIDELINES

- Thwaites GE et al, dexamethasone in TBM, NEJM 2004; Donovan J et al, ACT HIV (dexamethasone in HIV-TBM showed no benefit), NEJM 2023.

INVESTIGATIONS & TREATMENT AT A GLANCE

4.11 Miliary TB & TB Meningitis

‹ Back to topic (p 84)
Next topic 4.12 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
CSF cells	< 5 cells/µL	50–500, lymphocytic	TBM
CSF protein	15–45 mg/dL	100–500 mg/dL (> 1 g/L)	TBM (very high in spinal block)
CSF/serum glucose ratio	> 0.6	< 0.5 (often < 0.3)	TBM
CSF Xpert Ultra	Not detected	Detected (sensitivity ~ 70%)	Initial test (WHO)
CSF ADA	< 10 U/L	> 10 U/L	Supportive
MRI brain	Normal	Basal enhancement, hydrocephalus, infarcts, tuberculomas	TBM
CT chest (miliary)	Normal	Diffuse 1–3 mm random nodules	Miliary TB
Serum sodium	135–145 mmol/L	< 130	SIADH / cerebral salt wasting

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
ATT for TBM	2HRZE / 10HRE (12 months)	TBM, miliary with CNS involvement	—
Dexamethasone	0.4 mg/kg/day IV week 1, taper over 6–8 weeks	TBM (HIV-negative mortality benefit)	Hyperglycaemia; no mortality benefit in HIV (ACT-HIV)
Acetazolamide + furosemide	50–100 mg/kg/day acetazolamide; furosemide 1 mg/kg/day	Communicating hydrocephalus (trial)	Metabolic acidosis; VP shunt if failing
Hypertonic saline / fludrocortisone	3% saline; fludrocortisone 0.1–0.4 mg/day	Cerebral salt wasting	Correct Na ≤ 8–10 mmol/L/day

★ **PRESCRIBING & INTERPRETATION PEARLS**

- MRC TBM grade: I GCS 15; II GCS 11–14 or focal deficit; III GCS ≤ 10.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Management of miliary tuberculosis patient. *[DNB Dec 2021 P3]*
2. Management of a patient with altered sensorium with miliary shadow on chest X-ray. *[DNB Oct 2024 P3]*
3. Discuss miliary TB, its etiopathogenesis, diagnosis, complication and management. *[VNSGU 2025 P1]*
4. Sequelae of tuberculous meningitis. *[DNB Oct 2023 P3]*

4.12 Pleural & Pericardial Tuberculosis

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A1(8) Pleural and pericardial effusion and empyema
- ✓ Diagnose adult pleural tuberculosis using fluid biochemistry, ADA and biopsy
- ✓ Describe pathogenesis, features and management of tuberculous pericarditis

IN BRIEF TB pleural effusion is a delayed hypersensitivity reaction to bacilli entering the pleural space; fluid is a lymphocytic exudate with high ADA but low bacillary yield. Pleural biopsy (thoracoscopic) remains the gold standard.

Pleural TB

- Commonest cause of exudative effusion in young adults in India; unilateral, moderate; fever, pleuritic pain, cough.
- **Fluid:** straw-coloured exudate, lymphocytes >50% (neutrophils early), protein >5 g/dL, glucose moderately low, pH <7.3 in some, **mesothelial cells <5%** (suppressed), ADA high, IFN-γ high.
- **Microbiology:** smear <5%, culture 20–30% (MGIT higher), Xpert ~30–50% (Ultra better); sputum culture positive in ~40% even with normal CXR (induced sputum).
- **Pleural biopsy:** closed (Abrams/Cope) granulomas 60–80%, with culture/NAAT ~90%; thoracoscopic biopsy ~100% ("sago-grain" nodules).
- **Treatment:** standard 6-month regimen; therapeutic aspiration for large effusions; steroids not recommended routinely (no long-term benefit); residual pleural thickening in ~50% — physiotherapy; complication — tuberculous empyema (ICD, decortication; drug levels low in thick pleura).

Adenosine deaminase — diagnostic utility

- Enzyme of purine catabolism in lymphocytes (ADA2 isoform from monocytes/macrophages in TB).
- **Pleural fluid ADA ≥40 U/L** — sensitivity ~92%, specificity ~90% in high-prevalence settings; ADA + lymphocyte/neutrophil ratio >0.75 increases specificity.
- **False positives:** empyema/parapneumonic (neutrophilic), lymphoma, RA, mesothelioma, brucellosis, Q fever. False negatives: early, HIV, elderly.
- **Other fluids:** pericardial ≥40 U/L, peritoneal ≥39 U/L (lower in cirrhosis), CSF ≥10 U/L (less reliable).

Tuberculous pericarditis

- **Pathogenesis:** retrograde lymphatic spread from mediastinal nodes or haematogenous; stages — dry (fibrinous), effusive, absorptive, constrictive.
- **Clinical:** fever, chest pain, dyspnoea, pericardial rub, tamponade (Beck's triad, pulsus paradoxus), raised JVP, hepatomegaly; constriction (Kussmaul's sign, pericardial knock).
- **Diagnosis:** echo (effusion, fibrinous strands), CT (thickening, nodes), pericardiocentesis — exudate, high ADA (≥40), unstimulated IFN-γ, Xpert; **Tygerberg score** (fever, night sweats, weight loss, globulin >40 g/L, TLC <10,000) ≥6 suggests TB.
- **Management:** standard ATT; pericardiocentesis for tamponade/large effusions; steroids (prednisolone tapering 6 weeks) reduce constriction (IMPI) but not death — WHO recommends in HIV-negative; pericardiectomy for constriction persisting after 6–8 weeks ATT.

★ CLINICAL PEARLS & EXAM FOCUS

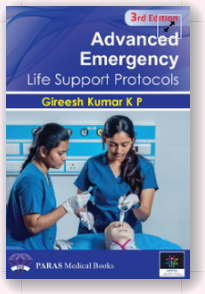
- ▸ High ADA + lymphocytic exudate + low mesothelial cells in a young Indian adult = treat as pleural TB without biopsy (high pretest probability); biopsy if atypical.

LANDMARK TRIALS & GUIDELINES

- Mayosi BM et al, IMPI trial, NEJM 2014 — prednisolone reduced constrictive pericarditis and hospitalisation.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Diagnosis of adult pleural tuberculosis. *[DNB Jun 2025 P3]*
2. Adenosine deaminase – diagnostic utility. *[DNB Jun 2021 P3]*
3. Discuss pathogenesis, clinical features, diagnosis and management of tuberculosis of pericardium. *[WBUHS 2022 P3]*



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4.13 Extrapulmonary TB — Lymph Node, Abdominal, Spinal, Genitourinary, Endobronchial & Cutaneous

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A1(10) Extrapulmonary tuberculosis
- ✓ Diagnose and manage common forms of extrapulmonary TB
- ✓ Differentiate tuberculous lymphadenopathy from other causes and manage recurrent lymphadenitis
- ✓ Recognise endocrine, metabolic and rheumatic manifestations of TB

IN BRIEF EPTB forms 15–25% of TB in India (higher in HIV). Diagnosis requires tissue or fluid for NAAT, culture and histology; treatment is the standard regimen, extended for CNS and bone disease, with surgery for complications.

Overview of extrapulmonary TB (INDEX-TB guidelines)

Site	Key diagnostics	Treatment duration / adjuncts
Lymph node	FNAC (granulomas, caseation, AFB), Xpert on aspirate/ biopsy, culture	6 months; aspirate fluctuant nodes; excision rarely
Pleura	ADA, biopsy	6 months
Abdominal (ileocaecal, peritoneal)	CECT, colonoscopy biopsy (Xpert, culture, histology), ascitic ADA	6 months; surgery for obstruction/ perforation
Spine (Pott's)	MRI (paradiscal, skip lesions, abscess), CT-guided biopsy Xpert	12 months (2HRZE/10HRE); surgery for deficit/instability
Genitourinary	Early-morning urine ×3 culture/Xpert, CT urography (putty kidney, strictures)	6 months; stenting/surgery for strictures
CNS	CSF Ultra, MRI	12 months + steroids
Pericardium	Echo, fluid ADA	6 months ± steroids
Skin	Biopsy	6 months

INVESTIGATIONS & TREATMENT AT A GLANCE

4.12 Pleural & Pericardial Tuberculosis

‹ Back to topic (p 86)
Next topic 4.13 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Pleural fluid ADA	< 40 U/L	> 40 U/L (lymphocytic exudate)	TB pleurisy (high-prevalence setting)
Pleural fluid lymphocytes	—	> 50% (often > 80%)	TB, lymphoma, malignancy
Pleural biopsy (thoracoscopic)	—	Caseating granulomas (> 90%), NAAT/ culture	Highest yield
Pericardial fluid ADA	< 40 U/L	> 40 U/L	TB pericarditis
Unstimulated IFN-γ (pericardial/pleural)	Low	High	Best single test for TB pericarditis
Echo	No effusion	Effusion, fibrin strands, tamponade	Pericardial TB

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Standard ATT	6 months	Pleural/pericardial TB	—
Therapeutic thoracentesis	Up to 1–1.5 L per session	Symptomatic large effusion	Re-expansion oedema
Prednisolone (pericarditis)	60 mg/day tapered over 6 weeks	Pericardial TB (conditional; avoid in HIV)	IMPI trial
Pericardiocentesis / pericardiectomy	Procedure	Tamponade / constriction after 6–8 weeks ATT	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Steroids are not recommended for TB pleural effusion.

Tuberculous lymphadenitis

- Commonest EPTB; cervical (posterior triangle, matted, cold abscess, collar-stud abscess, sinus) — scrofula.
- **Differential diagnosis of lymphadenopathy:** reactive (infection), lymphoma, metastatic (head-neck SCC, thyroid, lung), sarcoidosis, NTM (children, unilateral submandibular), HIV, Kikuchi, Rosai–Dorfman, Castleman, toxoplasma, cat-scratch, IgG4-RD, SLE, drugs (phenytoin).
- **Recurrent/persistent cervical lymphadenitis:** paradoxical reaction (20%, new or enlarging nodes during ATT — not failure), drug resistance (repeat NAAT/culture-DST from aspirate), NTM, wrong diagnosis (lymphoma), non-adherence. Manage: re-aspirate for NAAT/culture/DST, continue ATT, aspirate abscess, steroids for severe paradoxical reaction, excision biopsy if diagnosis uncertain.

Abdominal TB

- **Forms:** gastrointestinal (ileocaecal 75% — ulcerative, hypertrophic, ulcerohypertrophic), peritoneal (wet-ascitic, fibrotic-fixed, dry-plastic "doughy abdomen"), nodal, visceral (liver, spleen).
- **Clinical:** pain, weight loss, fever, subacute obstruction, palpable RIF mass, ascites, diarrhoea.
- **Investigations of ileocaecal TB:** CECT (asymmetric ileocaecal thickening, necrotic nodes, pulled-up contracted caecum), small bowel follow-through (Fleischner's inverted umbrella, Stierlin sign, string sign), colonoscopy (transverse ulcers, patulous IC valve) with biopsy — caseating granulomas, AFB, Xpert, culture; ascitic fluid ADA (>39 U/L); diagnostic laparoscopy.
- **Crohn's vs ITB:** ITB — short segment, transverse ulcers, patulous valve, necrotic nodes, caseation, positive IGRA (not specific); Crohn's — long segments, skip lesions, aphthous/longitudinal ulcers, comb sign, perianal disease. If doubt — 2-month ATT trial with endoscopic response assessment.

Pott's spine

- Paradiscal (commonest), central (vertebral body — collapse, mimics tumour), anterior (subperiosteal), posterior (neural arch); thoracolumbar; disc involvement late; cold abscess (psoas), gibbus, paraplegia (early — abscess/sequestrum; late — healed disease with stenosis).
- **MRI:** subligamentous spread, skip lesions, thin-walled abscess with smooth margins (vs pyogenic). CT-guided biopsy for Xpert/culture/histology.
- **Management:** 12 months ATT, bracing, rest; surgery (decompression + stabilisation) for neurological deficit not improving in 3–4 weeks, instability, deformity >40° kyphosis, large abscess, diagnostic uncertainty. Middle path regimen (Tuli).

Genitourinary TB

- Haematogenous seeding of renal cortex; latency 5–20 years. Sterile pyuria, haematuria, frequency (small "thimble" bladder), epididymo-orchitis ("beaded vas"), infertility (female genital TB — tubal blockage, Asherman).
- Investigations: three early-morning urine samples for culture/Xpert, CT urography (papillary necrosis, calcification, hydrocalyx, putty kidney — autonephrectomy), endometrial biopsy for genital TB.
- 6 months ATT; ureteric stenting during treatment (stricture risk with healing), nephrectomy for non-functioning infected kidney, augmentation cystoplasty.

Endobronchial TB

- Spread from parenchyma via sputum or node erosion; young women; cough, wheeze (localised monophonic), haemoptysis; often smear-positive with normal CXR.
- **Chung's bronchoscopic types:** actively caseating, oedematous–hyperaemic, fibrostenotic, tumorous, granular, ulcerative, non-specific bronchitic.
- Complications: bronchostenosis → collapse, post-obstructive pneumonia; treat with ATT ± steroids (controversial), bronchoscopic dilatation/stent for strictures, surgery (sleeve resection).

Cutaneous TB and tuberculids

- **True cutaneous TB:** lupus vulgaris (apple-jelly nodules), scrofuloderma (over nodes), TB verrucosa cutis (inoculation — "prosector's wart"), tuberculous chancre, orificial TB.

Academy for Emergency, Trauma & Critical care Medicine

- **Tuberculids** (hypersensitivity to bacillary antigens, AFB-negative, strongly TST-positive, respond to ATT): papulonecrotic tuberculid, lichen scrofulosorum, erythema induratum (Bazin's — calves, nodular vasculitis); erythema nodosum and phlyctenular conjunctivitis are "facultative" tuberculids.

Endocrine, metabolic and rheumatic manifestations

- **Endocrine/metabolic:** SIADH (hyponatraemia, especially TBM, miliary), adrenal insufficiency (Addison's — bilateral adrenal enlargement then calcification), hypercalcaemia (macrophage 1α-hydroxylase), hypothyroidism (ethionamide/PAS), hypogonadism, pituitary dysfunction after TBM, stress hyperglycaemia/diabetes unmasking, cachexia.
- **Rheumatic syndromes:** Pott's spine, tuberculous arthritis (monoarticular — hip, knee; Phemister triad: juxta-articular osteoporosis, peripheral erosions, gradual joint-space narrowing), Poncet's disease (reactive polyarthritis during active TB), tenosynovitis, osteomyelitis (spina ventosa — dactylitis), drug-induced (Z — gout/arthralgia, INH — lupus, FQ — tendinitis, R — flu-like), BCG-associated reactive arthritis.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Paradoxical enlargement of nodes during ATT is common — continue treatment; it is not failure unless DST shows resistance.

LANDMARK TRIALS & GUIDELINES

- INDEX-TB guidelines — Guidelines on extrapulmonary TB for India (ICMR, 2016).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Differential diagnosis of lymphadenopathy. *[DNB Jun 2025 P3]*
2. Management of recurrent cervical lymphadenitis. *[DNB Oct 2024 P3]*
3. Tuberculous lymphadenitis. *[DNB Oct 2023 P3]*
4. Clinical manifestation and diagnosis of abdominal tuberculosis. *[DNB Oct 2024 P3]*
5. Investigations of ileocaecal tuberculosis. *[DNB Oct 2023 P3]*
6. Gastrointestinal tuberculosis. *[Manipal 2014 P3]*
7. Endocrine and metabolic manifestations of tuberculosis. *[DNB Oct 2023 P3]*
8. Endobronchial TB. *[DNB Oct 2023 P3]*

4.14 Complications & Post-TB Lung Disease; Corticosteroids and Surgery in TB

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A1(5) Complications in tuberculosis
- ✓ Enumerate complications of pulmonary TB and post-TB sequelae
- ✓ State evidence-based indications for corticosteroids in TB
- ✓ State the role of surgery in pleuropulmonary TB

IN BRIEF Even after microbiological cure, up to half of patients have post-TB lung disease (obstructive, restrictive or mixed impairment, bronchiectasis, CPA, PH). Steroids benefit TB meningitis and pericarditis; surgery is reserved for complications and selected DR-TB.

Complications of pulmonary TB

Category	Complications
Pulmonary (acute)	Massive haemoptysis (Rasmussen aneurysm, bronchial arteries), pneumothorax, empyema, bronchopleural fistula, ARDS, endobronchial stenosis
Pulmonary (chronic / post-TB)	Bronchiectasis (upper lobe, dry), fibrosis, destroyed lung, CPA/aspergilloma, broncholithiasis, post-TB obstructive airway disease (TOPD), chronic respiratory failure, PH/cor pulmonale, lung cancer (scar carcinoma)

INVESTIGATIONS & TREATMENT AT A GLANCE

4.13 Extrapulmonary TB — Lymph Node, Abdominal, Spinal, Genitourinary, Endobronchial & Cutaneous

‹ Back to topic (p 87)
Next topic 4.14 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
FNAC + Xpert (lymph node)	Reactive	Granulomas, AFB, MTB detected	TB lymphadenitis
Ascitic ADA	< 39 U/L	> 39 U/L	Peritoneal TB
SAAG	—	< 1.1 g/dL with high protein	Exudative ascites (TB, malignancy)
MRI spine	Normal	Paradiscal destruction, abscess, cord compression	Pott's spine
Early-morning urine (× 3) NAAT/culture	Negative	Positive	Genitourinary TB
Morning cortisol / ACTH test	> 18 µg/dL post-ACTH	< 18 µg/dL	Adrenal TB

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Standard ATT	6 months (most EPTB)	Lymph node, abdominal, GU	Paradoxical node enlargement — continue
Extended ATT	12 months	Spinal/osteoarticular, CNS	—
Hydrocortisone + fludrocortisone	15–25 mg/day (double on rifampicin) + 50–100 µg	Adrenal insufficiency	Sick-day rules
Surgery	Decompression; stricturoplasty; DJ stent	Neurological deficit; obstruction	After/with ATT

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Residual lymph nodes after treatment are common — not treatment failure.

Category	Complications
Systemic	Amyloidosis (AA), SIADH, adrenal insufficiency, disseminated TB, malnutrition
Pleural	Fibrothorax, trapped lung, calcification, chronic empyema
Treatment-related	DILI, drug resistance, ADRs

Post-TB lung disease (PTLD)

- Recognised by First International Post-TB Symposium (2019); prevalence 18–87% of survivors; spirometry abnormal in ~50%; increased mortality (3–4× general population).
- **Management:** end-of-treatment assessment (symptoms, spirometry, 6MWT, CXR), pulmonary rehabilitation, bronchodilators (bronchodilator trial; ICS for overlapping asthma), airway clearance for bronchiectasis, vaccines, smoking cessation, treat CPA, LTOT for respiratory failure, recognise recurrence.

Corticosteroids in TB

Steroid indications

Indication	Evidence and regimen
TB meningitis	Strong — reduces mortality (HIV-negative); dexamethasone 0.4 mg/kg/day (stage II/III) tapered over 6–8 weeks
TB pericarditis	Reduces constriction (IMPI); prednisolone 120 mg → taper over 6 weeks (HIV-negative)
Paradoxical reaction / IRIS	Prednisolone 1.5 mg/kg tapering 4 weeks
Adrenal insufficiency	Replacement (hydrocortisone; double dose with rifampicin)
Severe miliary / ARDS, laryngeal TB with obstruction, endobronchial TB (selected), spinal TB with cord compression, ureteric TB (stricture prevention), drug hypersensitivity, TB-HLH	Weak/individual
Pleural TB	Not routinely (symptom relief only)

Surgery in pleuropulmonary TB

- **Diagnostic:** biopsy of nodules, mediastinal nodes.
- **Therapeutic (complications):** massive/recurrent haemoptysis not controlled by BAE, destroyed lobe/lung with recurrent infection, aspergilloma, bronchopleural fistula, chronic empyema (decortication), trapped lung, bronchostenosis, broncholithiasis.
- **DR-TB:** localised cavitary disease with persistent culture positivity or high relapse risk — partial lung resection (lobectomy/segmentectomy) after ≥2 months of effective therapy, continue treatment post-surgery (WHO conditional recommendation).
- Historical: thoracoplasty, phrenic crush, plombage, artificial pneumothorax (collapse therapy).

★ CLINICAL PEARLS & EXAM FOCUS

▸ Steroids are "TBM and pericardium — yes; pleura — no".

LANDMARK TRIALS & GUIDELINES

- Allwood BW et al, Post-TB lung health: perspectives from the First International Symposium, IJTLD 2020; Migliori GB et al, ERS/WHO clinical standards for post-TB lung disease 2021.

INVESTIGATIONS & TREATMENT AT A GLANCE

4.14 Complications & Post-TB Lung Disease; Corticosteroids and Surgery in TB

‹ Back to topic (p 89)
Next topic 4.15 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Spirometry after TB	Normal	Obstruction / restriction / mixed	Post-TB lung disease
Aspergillus IgG	Negative	Positive	Chronic pulmonary aspergillosis
CT angiography	—	Bronchial artery hypertrophy, Rasmussen aneurysm	Haemoptysis planning
Sputum culture (recurrence)	Negative	Positive	Recurrent TB (NAAT may be falsely positive)
6MWT / SpO_2	> 400 m; no desaturation	Desaturation	Rehabilitation, O_2 need

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Pulmonary rehabilitation	6–12 weeks	PTLD	—
Itraconazole	200 mg twice daily ≥ 6 months	CPA	No rifampicin co-administration
Bronchial artery embolisation	Procedure	Haemoptysis	Spinal artery risk
Steroids in TB	Dexamethasone (TBM), prednisolone (pericarditis, IRIS)	Specific indications	Not for pleural TB
Surgery	Lobectomy/pneumonectomy	Destroyed lung, localised MDR, aspergilloma	PFT assessment

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Inhaled bronchodilators help post-TB airflow obstruction.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Complications of pulmonary TB. *[DNB Oct 2023 P3]*
2. Post-TB sequelae. *[WBUHS 2020 P3]*
3. Discuss role of surgery in pleuro-pulmonary tuberculosis. *[WBUHS 2020 P3]*
4. Role of corticosteroids in management of tuberculosis. *[DNB Dec 2021 P3]*
5. Role of steroids in tuberculosis. *[DNB Jun 2021 P3]*
6. Role of steroids in the management of TB. *[DNB Jun 2020 P3]*
7. Role of corticosteroids in management of various extrapulmonary tuberculosis. *[DNB Oct 2024 P3]*
8. Role of corticosteroid in management of tuberculosis. *[WBUHS 2022 P3]*

4.15 Patient Support, Infection Control, Stigma, BCG & TB Vaccines

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-E3, II-A1 Programme components and prevention
- ✓ Describe Ni-kshay, Ni-kshay Poshan Yojana and Ni-kshay Mitra under PMTBMBA
- ✓ Describe airborne infection control measures including AIIR and cough etiquette
- ✓ Describe BCG vaccination and newer TB vaccines

IN BRIEF Tuberculosis is a social disease. NTEP couples biomedical treatment with direct benefit transfer for nutrition, community sponsorship (Ni-kshay Mitra), stigma reduction, infection control in facilities and vaccination.

Ni-kshay ecosystem

- **Ni-kshay** — web/mobile case-based surveillance and patient management (notification, DST, treatment, adherence, DBT, TPT, aDSM).
- **Ni-kshay Poshan Yojana (NPY, April 2018):** direct benefit transfer of **₹500/month** for nutrition to every notified TB patient for the duration of treatment; **enhanced to ₹1000/month from November 2024**, with energy-dense nutrition support (EDNS) for underweight patients; incentives to private providers (₹500 notification + ₹500 outcome) and treatment supporters (₹1000 DS-TB, ₹5000 DR-TB); tribal support ₹750.
- **Pradhan Mantri TB Mukt Bharat Abhiyaan (PMTBMBA, Sept 2022)** — **Ni-kshay Mitra**: individuals, corporates, NGOs, elected representatives adopt consenting TB patients for 6–36 months, providing monthly food baskets (cereal, pulses, oil, milk), vocational/diagnostic support; extended to household contacts (2024).

Nutrition in TB

- Undernutrition (BMI <18.5) is the largest attributable risk factor for TB in India; malnourished patients have higher mortality, relapse, DILI.
- **Assessment:** BMI, MUAC, weight change, serum albumin, dietary recall, clinical signs, BIA; children — weight-for-height z-score.
- **RATIONS trial (Jharkhand):** nutritional support to patients improved weight gain and reduced mortality; to contacts reduced incidence by 39–48%.

Airborne infection control

- **Administrative (most important):** triage and fast-tracking of coughers ("separate, cough etiquette, fast-track"), early diagnosis and treatment, minimise hospitalisation, staff screening, IC committee and plan.
- **Environmental:** natural ventilation (≥12 ACH; cross-ventilation), mixed-mode ventilation, **Airborne Infection Isolation Room (AIIR)** — negative pressure (−2.5 Pa), ≥12 air changes/hour, exhaust away from people, anteroom, door closed; upper-room UVGI; HEPA filters.
- **Personal protective:** N95 respirators for HCWs in high-risk areas (fit-tested); surgical masks for patients.
- **Cough etiquette:** cover mouth/nose with tissue or elbow, dispose tissue, hand hygiene, mask for symptomatic patients, maintain 1 m distance.

- National Airborne Infection Control Guidelines (MoHFW 2010).

Stigma

- Enacted (discrimination), perceived and internalised stigma; causes delayed care-seeking, non-disclosure, non-adherence, especially in women.
- Measures: counselling, TB Champions/survivor networks, Ni-kshay Sampark, community engagement (TB Forums), media (TB Harega Desh Jeetega), legal protections, workplace policies; WHO/Stop TB stigma assessment tool.

BCG

- Live attenuated M. bovis (Danish 1331 strain in India); 0.05 mL (infants <1 month) / 0.1 mL intradermal, left upper arm, at birth (up to 1 year).
- Protects against disseminated TB and TBM in children (70–80%); variable protection (0–80%) against adult pulmonary TB (Chingleput trial: no protection).
- Adverse: BCG adenitis, abscess, keloid, osteitis, disseminated BCGosis (SCID, HIV — contraindicated in symptomatic HIV).
- Non-specific "trained immunity"; adult BCG revaccination trial (ICMR) under evaluation.

Newer TB vaccines

- **M72/AS01E** (subunit) — 50% efficacy in IGRA+ adults (phase 2b), phase 3 ongoing (2024–28).
- **MTBVAC** (live attenuated M. tuberculosis), **VPM1002** (recombinant BCG, Serum Institute), **Immuvac/MIP** (Mycobacterium indicus pranii — Indian), **H56:IC31**, **BCG revaccination**.

✦ RECENT ADVANCES & GUIDELINE UPDATES

▸ 2024: NPY ₹1000/month and EDNS; adult BCG vaccination campaign in high-risk adults (elderly, diabetics, smokers, malnourished, past TB) under evaluation in selected states.

★ CLINICAL PEARLS & EXAM FOCUS

▸ NPY: ₹500 (2018) → ₹1000 per month (Nov 2024) — via DBT to Aadhaar-seeded bank account.

LANDMARK TRIALS & GUIDELINES

- Tait DR et al, M72/AS01E NEJM 2019; Bhargava A et al, RATIONS Lancet 2023 and Lancet Glob Health 2023.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Ni-kshay Poshan Yojana (NPY). *[DNB Jun 2025 P3]*
2. Ni-kshay Mitra. *[DNB Jun 2025 P3]*
3. Discuss role of nutrition in tuberculosis and Nikshay Poshan Yojana. *[VNSGU 2025 P1]*
4. What is airborne infection control and describe AIIR? *[BFUHS 2022 P3]*
5. Airborne infection control measures in TB. *[BFUHS 2019 P3]*
6. Stigma related to pulmonary tuberculosis. *[BFUHS 2020 P3]*
7. Cough etiquette for reducing transmission of respiratory pathogens. *[BFUHS 2020 P3]*
8. BCG vaccination. *[DNB Jun 2021 P1]*

4.16 Non-Tuberculous Mycobacteria

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A1(9) Mycobacteria other than tuberculosis
- ✓ Classify NTM (Runyon) and describe the spectrum of disease
- ✓ Apply ATS/ERS/ESCMID/IDSA diagnostic criteria for NTM pulmonary disease
- ✓ Treat MAC, M. kansasii and M. abscessus pulmonary disease

INVESTIGATIONS & TREATMENT AT A GLANCE

4.15 Patient Support, Infection Control, Stigma, BCG & TB Vaccines

‹ Back to topic (p 91)

Next topic 4.16 ›

Most important investigations — reference and abnormal values

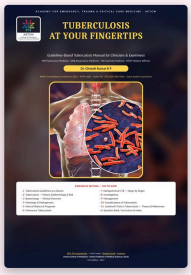
Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Adherence (99DOTS/MERM records)	> 90% doses	Missed doses > 10%	Counselling, support
Air changes per hour (isolation)	≥ 12 ACH	Lower	Infection control gap
N95 fit test	Pass	Fail	Respirator programme
BCG scar	Present	Absent	Vaccination history

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Ni-kshay Poshan Yojana	₹1000/month	All TB patients	DBT
BCG	0.05 mL (< 1 month) / 0.1 mL ID left deltoid	At birth	Not in symptomatic HIV/ immunodeficiency
N95 respirator	Fit-tested	Staff in high-risk areas	—
Surgical mask for patient	—	Coughing patients	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Effective treatment rapidly reduces infectiousness — the most important infection-control measure.



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IN BRIEF NTM are environmental mycobacteria (water, soil) that cause chronic lung disease in structural lung disease (bronchiectasis, post-TB, COPD) and disseminated disease in immunosuppression. Diagnosis needs repeated culture isolation plus clinical and radiological criteria; treatment is long (≥12 months after culture conversion).

Runyon classification

Group	Characteristics	Examples
I — Photochromogens	Slow; yellow pigment on light exposure	M. kansasii, M. marinum, M. simiae
II — Scotochromogens	Slow; pigment in dark	M. scrofulaceum, M. gordonae, M. szulgai, M. xenopi
III — Non-chromogens	Slow; no pigment	M. avium complex (MAC), M. ulcerans, M. malmoense, M. xenopi
IV — Rapid growers	<7 days	M. abscessus, M. fortuitum, M. chelonae

Clinical syndromes

- **Pulmonary:** fibrocavitary (upper lobe — older male smokers, COPD) or nodular-bronchiectatic (RML/lingula — "Lady Windermere syndrome" — slender postmenopausal women), hypersensitivity-like "hot-tub lung".
- **Lymphadenitis** (children, MAC/scrofulaceum), **skin/soft tissue** (M. marinum — **swimming pool/fish-tank granuloma**: sporotrichoid nodules on hands; M. fortuitum/abscessus — post-surgical, cosmetic, injection abscesses; M. ulcerans — Buruli ulcer), **disseminated** (HIV CD4 <50 — MAC), catheter infections.

Diagnostic criteria (ATS/ERS/ESCMID/IDSA 2020)

NTM pulmonary disease criteria — all required

Domain	Criteria
Clinical	Pulmonary or systemic symptoms
Radiological	Nodular or cavitary opacities on CXR, or multifocal bronchiectasis with multiple small nodules on HRCT
Exclusion	Appropriate exclusion of other diagnoses (TB, malignancy)
Microbiological	Positive culture from ≥2 separate expectorated sputum samples; OR ≥1 bronchial wash/BAL; OR transbronchial/lung biopsy with mycobacterial histopathology + positive culture (biopsy or sputum/BAL)

- M. gordonae usually a contaminant; species identification (LPA, MALDI-TOF, sequencing) and macrolide (erm41, rrl) and amikacin (rrs) susceptibility essential.

Treatment of NTM pulmonary disease

Organism	Regimen	Duration
MAC — nodular/ bronchiectatic, macrolide-susceptible	Azithromycin 500 mg + rifampicin 600 + ethambutol 25 mg/kg three times weekly	≥12 months after culture conversion
MAC — cavitary/severe	Daily azithromycin 250–500 + rifampicin + ethambutol + IV amikacin/ streptomycin first 2–3 months	≥12 months after conversion
MAC — refractory (culture-positive after 6 months)	Add amikacin liposome inhalation suspension (ALIS)	CONVERT trial
M. kansasii	Rifampicin + ethambutol + isoniazid or azithromycin	12 months (fixed)
M. abscessus	Induction: IV amikacin + imipenem/cefoxitin + tigecycline + azithromycin (if no inducible erm41); continuation: inhaled amikacin + oral (clofazimine, linezolid, azithromycin)	≥12 months after conversion; surgery for localised disease

- **Never macrolide monotherapy** — resistance. Resection surgery for localised refractory disease.

★ CLINICAL PEARLS & EXAM FOCUS

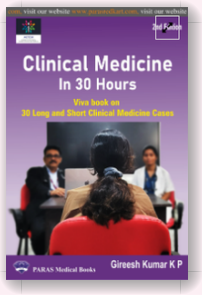
▸ Lady Windermere = MAC nodular bronchiectasis of RML/lingula in thin elderly women (voluntary cough suppression hypothesis).

LANDMARK TRIALS & GUIDELINES

• Daley CL et al, ATS/ERS/ESCMID/IDSA NTM guideline, Clin Infect Dis/Eur Respir J 2020; Griffith DE et al, CONVERT AJRCCM 2018.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Criteria of diagnosis of NTM lung disease. *[DNB Jun 2025 P3]*
2. Treatment of pulmonary NTM disease. *[DNB Jun 2025 P3]*
3. Diagnosis criteria of non-tubercular mycobacterial pulmonary disease (NTM-PD). *[DNB Oct 2024 P3]*
4. Describe the treatment guidelines of Mycobacterium avium complex in pulmonary diseases. *[DNB May 2024 P2]*
5. Describe non-tuberculous mycobacteria, its diagnosis and management. *[WBUHS 2020 P3]*
6. Classification of non-tubercular mycobacteria. *[BFUHS 2020 P1]*
7. Swimming pool granuloma. *[VNSGU 2025 P1]*



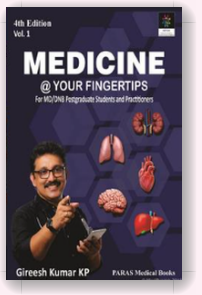
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INVESTIGATIONS & TREATMENT AT A GLANCE

4.16 Non-Tuberculous Mycobacteria

‹ Back to topic (p 92)
Next topic 5.1 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Sputum NTM culture	—	≥ 2 positive sputum cultures (or 1 BAL)	NTM-PD microbiological criterion (ATS/ERS 2020)
Species identification (LPA/ sequencing)	—	MAC, M. abscessus, M. kansasii	Guides therapy
Macrolide susceptibility (erm(41), rrl)	Susceptible	Inducible/mutational resistance	M. abscessus treatment
HRCT	Normal	Nodular-bronchiectatic or fibrocavitary disease	Radiological criterion
Xpert MTB	Not detected	—	Distinguishes NTM from TB

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
MAC (nodular, non-cavitary)	Azithromycin 500 mg + rifampicin 600 mg + ethambutol 25 mg/kg three times weekly	MAC-PD	12 months after conversion
MAC (cavitary/severe)	Daily azithromycin 250 mg + R + E ± amikacin IV/inhaled ALIS 590 mg	Severe MAC	Ototoxicity
M. kansasii	Rifampicin + ethambutol + azithromycin/isoniazid × 12 months	M. kansasii	—
M. abscessus	Initial: amikacin + imipenem/ cefoxitin + tigecycline + azithromycin (if susceptible)	M. abscessus	Expert care

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Never use macrolide monotherapy — rapid resistance.

SECTION 5

Non-Tuberculous Infections of the Lung

Paper II

Approach to pulmonary infection and principles of antibiotic use; community-acquired, hospital-acquired and ventilator-associated pneumonia; atypical, viral, fungal, parasitic, rickettsial and anaerobic infections; aspiration, lung abscess and bronchiectasis; infections in the immunocompromised host; zoonoses (NMC syllabus II-A2, I-C).

5.1 Approach to Pulmonary Infection, Antibiotic Principles & Bronchoalveolar Lavage 95
5.2 Community-Acquired Pneumonia 97
5.3 Hospital-Acquired & Ventilator-Associated Pneumonia 99
5.4 Atypical & Tropical Pneumonias — Mycoplasma, Legionella, Melioidosis, Scrub Typhus, Leptospirosis 100
5.5 Viral Pneumonias — Influenza, COVID-19, RSV — and Vaccines 101
5.6 Aspiration Pneumonia, Lung Abscess & Lipoid Pneumonia 103
5.7 Bronchiectasis, Cystic Fibrosis & Primary Ciliary Dyskinesia 104
5.8 Pulmonary Aspergillosis Spectrum & Mucormycosis 106
5.9 Pneumocystis, Cryptococcosis, Histoplasmosis & Other Fungal Pneumonias 108
5.10 Parasitic Lung Disease — Hydatid, Amoebiasis, Paragonimiasis & Zoonoses 109
5.11 Pulmonary Infections in the Immunocompromised Host (HIV, Transplant, Immunosuppressants) 110
5.12 Recurrent & Non-Resolving Pneumonia; MRSA Pneumonia 113

5.1 Approach to Pulmonary Infection, Antibiotic Principles & Bronchoalveolar Lavage

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A2 Approach to pulmonary infection; principles of antibiotic use
- ✓ Approach a patient with suspected pulmonary infection by host, setting and radiological pattern
- ✓ Apply principles of antimicrobial stewardship and PK/PD-based dosing
- ✓ Describe the diagnostic utility of BAL

IN BRIEF Diagnosis of a lung infection is built on three questions — who is the host (immunocompetent, immunocompromised, structural lung disease), where was it acquired (community, hospital, travel, occupation) and what is the radiological pattern. Antibiotics are chosen empirically from these, then de-escalated using microbiology.

Radiological patterns and likely causes

Pattern	Likely causes
Lobar consolidation	S. pneumoniae, Klebsiella, Legionella

Pattern	Likely causes
Bronchopneumonia	S. aureus, H. influenzae, gram-negatives
Interstitial / diffuse	Viral, Mycoplasma, PJP, miliary TB
Cavitation	TB, anaerobes, S. aureus, Klebsiella, melioidosis, fungi, Nocardia
Nodules with halo	Invasive aspergillosis, mucor (reversed halo), septic emboli
Pneumatocoeles	S. aureus, PJP

Principles of antibiotic use

- Obtain cultures before antibiotics (without delaying in sepsis — within 1 h).
- Empirical therapy guided by local antibiogram; **de-escalate** at 48–72 h; **shortest effective duration** (CAP 5 days if stable; HAP/VAP 7 days).
- **PK/PD:** time-dependent (β-lactams — prolonged/continuous infusions, T>MIC); concentration-dependent (aminoglycosides, FQ — Cmax/MIC, AUC/MIC; once-daily aminoglycoside); TDM for vancomycin (AUC 400–600), aminoglycosides, voriconazole.
- **IV-to-oral switch** when afebrile, haemodynamically stable, taking orally.
- **Stewardship (ICMR AMSP):** restrict reserve antibiotics (WHO AWaRe: Access, Watch, Reserve), audit and feedback, procalcitonin-guided stopping, infection control. India's Kerala Antimicrobial Resistance Strategic Action Plan (KARSAP) is a model.

Bronchoalveolar lavage — diagnostic utility

- Wedge bronchoscope in segmental bronchus; instil 100–300 mL saline in 20–50 mL aliquots; recovery >30% adequate.

BAL interpretation

Finding	Interpretation
Lymphocytes >15% (CD4/CD8 >3.5 in sarcoid)	Sarcoidosis, HP (CD4/CD8 low, lymphocytes >30–50%), NSIP, COP, drug
Neutrophils >3% (>50%)	IPF, infection, ARDS, aspiration
Eosinophils >25%	Eosinophilic pneumonia (acute, chronic), ABPA, drug, EGPA
Progressively bloodier aliquots, haemosiderin-laden macrophages >20%	Diffuse alveolar haemorrhage
Milky fluid, PAS-positive material	Pulmonary alveolar proteinosis
CD1a cells >5%	PLCH
Quantitative culture ≥10^4 CFU/mL	Bacterial pneumonia (VAP)
Galactomannan ≥1.0	Invasive aspergillosis
Pneumocystis (IF/PCR), CMV, AFB/NAAT, fungi	Opportunistic infection
Malignant cells	Lymphangitic carcinomatosis, adenocarcinoma
Asbestos bodies >1/mL	Asbestos exposure

- Safety: hypoxaemia, fever (post-BAL), bleeding (rare); caution if PaO2 <60 on O2, unstable.

★ CLINICAL PEARLS & EXAM FOCUS

▸ BAL is diagnostic in infection, DAH, PAP, eosinophilic pneumonia and lymphangitis; supportive in sarcoid and HP.

LANDMARK TRIALS & GUIDELINES

- Meyer KC et al, ATS clinical practice guideline on BAL cellular analysis in ILD, AJRCCM 2012.

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INVESTIGATIONS & TREATMENT AT A GLANCE

5.1 Approach to Pulmonary Infection, Antibiotic Principles & Bronchoalveolar Lavage

‹ Back to topic (p 95)
Next topic 5.2 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Sputum Gram stain & culture	Normal flora	Predominant organism with PMNs	Targeted therapy
BAL quantitative culture	< 10^4 CFU/mL	≥ 10^4 CFU/mL	Pneumonia (≥ 10^3 for PSB)
BAL cell count	Macrophages > 85%	Neutrophils > 50%	Bacterial infection
Procalcitonin	< 0.1 ng/mL	> 0.5 ng/mL	Bacterial — antibiotic guidance
Blood cultures	No growth	Positive	Bacteraemia

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Empirical antibiotic by severity	Per CURB-65 / setting	Lower respiratory infection	De-escalate at 48–72 h
Antibiotic stewardship	5–7 days for CAP	Uncomplicated pneumonia	Avoid prolonged courses
Bronchoscopy safety	SpO_2 > 90% on O_2; platelets > 50 000	BAL	Hypoxaemia, bleeding

★ PRESCRIBING & INTERPRETATION PEARLS

- "Start smart, then focus" — review cultures at 48–72 hours.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Discuss the diagnostic utility of bronchoalveolar lavage. *[BFUHS 2022 P2]*

5.2 Community-Acquired Pneumonia

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A2 Community-acquired pneumonia
- ✓ Describe the aetiology of CAP in India and assess severity using CURB-65, PSI and ATS/IDSA criteria
- ✓ Select empirical antibiotics for outpatient, ward and ICU CAP
- ✓ Describe the role of corticosteroids and macrolides in severe CAP

IN BRIEF CAP is an acute infection of the lung parenchyma acquired outside hospital, with new radiographic infiltrate. S. pneumoniae remains the commonest pathogen; severity scores decide site of care and empiric therapy covers typical and atypical organisms.

Aetiology of CAP

Setting	Common organisms
Outpatient	S. pneumoniae, M. pneumoniae, H. influenzae, C. pneumoniae, respiratory viruses
Ward	Above + Legionella, aspiration (anaerobes), K. pneumoniae (diabetes, alcohol)
ICU (severe)	S. pneumoniae, S. aureus (incl. CA-MRSA post-influenza), Legionella, gram-negatives, P. aeruginosa (bronchiectasis, steroids)
India-specific	M. tuberculosis (must exclude), Burkholderia pseudomallei, scrub typhus, leptospirosis, influenza H1N1, dengue

Severity assessment

CURB-65 (BTS)

Criterion (1 point each)	Score → management
Confusion	0–1: low risk (mortality <3%) — home
Urea >7 mmol/L (BUN >20 mg/dL)	2: moderate (9%) — short admission/ward
RR ≥30/min	3–5: severe (15–40%) — hospital, consider ICU (4–5)
BP SBP <90 or DBP ≤60	CRB-65 for primary care (no urea)
Age ≥65	—

- **PSI (PORT):** 20 variables, classes I–V — better for identifying low-risk; IDSA prefers PSI.

ATS/IDSA 2007/2019 severe CAP criteria

Major (1 needed)	Minor (≥3 needed)
Septic shock needing vasopressors	RR ≥30; PaO2/FiO2 ≤250; multilobar infiltrates; confusion
Respiratory failure needing mechanical ventilation	BUN ≥20 mg/dL; WBC <4000; platelets <1 lakh; hypothermia <36 °C; hypotension needing aggressive fluids

- Others: SMART-COP (need for IRVS), A-DROP (Japan), qSOFA.

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Empirical antibiotics (IDSA/ATS 2019, adapted to Indian guidelines — ICMR/NCCP 2012)

Setting	Regimen
Outpatient, no comorbidity	Amoxicillin 1 g TDS or doxycycline 100 mg BD (macrolide only if pneumococcal resistance <25% — not in India)
Outpatient with comorbidity (COPD, DM, CKD, CLD, alcohol)	Amoxicillin-clavulanate or cefuroxime/cefpodoxime + macrolide or doxycycline (avoid respiratory FQ in India — TB masking)
Ward	IV ceftriaxone 1–2 g / cefotaxime / amoxicillin-clavulanate + azithromycin (or doxycycline)
ICU	IV β-lactam (ceftriaxone, amox-clav) + azithromycin (or respiratory FQ)
Pseudomonas risk (prior isolation, bronchiectasis, recent IV antibiotics)	Piperacillin-tazobactam / cefepime / meropenem ± amikacin
MRSA risk (prior MRSA, post-influenza necrotising)	Add vancomycin or linezolid
Aspiration	Amoxicillin-clavulanate (anaerobic cover only with abscess/empyema)

- Duration: ≥5 days and clinically stable for 48 h; longer for S. aureus bacteraemia, Pseudomonas, Legionella (7–14 days), abscess, empyema.

Corticosteroids in severe pneumonia

- **CAPE COD (2023):** hydrocortisone 200 mg/day (4–8 days, tapered) within 24 h in severe CAP in ICU reduced 28-day mortality (6.2% vs 11.9%); excluded septic shock and influenza.
- **ATS 2024 guideline:** suggests systemic corticosteroids for adults hospitalised with severe CAP; not for non-severe CAP; avoid in influenza (harm), uncertain in aspergillosis risk.
- Methylprednisolone in severe CAP with high inflammation (Torres, 2015) reduced treatment failure.

Macrolides in severe CAP and sepsis

- Dual therapy (β-lactam + macrolide) associated with lower mortality in severe/bacteraemic pneumococcal CAP — immunomodulatory effects.
- **ACCESS (2023):** clarithromycin added to standard care in CAP with SIRS and organ dysfunction improved early clinical response and reduced inflammatory burden.
- Caution: QT, macrolide resistance; azithromycin preferred.

Non-responding pneumonia — causes

- Wrong organism (TB, fungi, resistant), complications (empyema, abscess), wrong diagnosis (OP, PE, vasculitis, eosinophilic pneumonia, cancer), host factors, drug fever, C. difficile.

✦ RECENT ADVANCES & GUIDELINE UPDATES

▸ ATS clinical practice guideline on corticosteroids in CAP (2024 update); IDSA/ATS CAP guideline 2019 (no HCAP category; obtain cultures only if severe or MRSA/Pseudomonas risk).

★ CLINICAL PEARLS & EXAM FOCUS

▸ Avoid respiratory fluoroquinolones as first-line CAP therapy in India — they partially treat TB and delay its diagnosis.

LANDMARK TRIALS & GUIDELINES

- Dequin PF et al, CAPE COD NEJM 2023; Giamarellos-Bourboulis EJ et al, ACCESS Lancet Respir Med 2024; Metlay JP et al, ATS/IDSA CAP guideline AJRCCM 2019.

INVESTIGATIONS & TREATMENT AT A GLANCE

5.2 Community-Acquired Pneumonia

‹ Back to topic (p 97)
Next topic 5.3 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
CURB-65	0–1	2 (consider admission); ≥ 3 (severe, ICU consider)	Severity
Chest radiograph	Clear	Consolidation	Confirms pneumonia
Procalcitonin	< 0.25 ng/mL	> 0.5	Bacterial likely
Pneumococcal / Legionella urinary antigen	Negative	Positive	Targeted therapy
SpO_2 / PaO_2	≥ 94%	< 92% or PaO_2/FiO_2 < 250	Severe CAP (IDSA/ATS minor criterion)
Blood urea	< 20 mg/dL (7 mmol/L)	> 20 mg/dL	CURB-65 point

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Amoxicillin	1 g 8-hourly × 5 days	Non-severe outpatient CAP	—
Doxycycline / azithromycin	Doxy 100 mg bd; azithro 500 mg day 1 then 250 mg × 4 days	Outpatient, atypical cover	Local macrolide resistance
Ceftriaxone + azithromycin	1–2 g IV daily + 500 mg IV/oral daily	Hospitalised CAP	—
Hydrocortisone	200 mg/day infusion × 4–7 days then taper	Severe CAP in ICU (CAPE COD)	Not in influenza; hyperglycaemia
Oseltamivir	75 mg bd × 5 days	Influenza season with CAP	—

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Respiratory fluoroquinolones are reserved in India — avoid where TB is possible.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Aetiology and severity assessment of community acquired pneumonia. *[DNB Dec 2022 P2]*
2. Management of CAP in out-patient setting. *[DNB Dec 2022 P2]*
3. Empiric antibiotic selection in adult outpatients with community-acquired pneumonia. *[DNB Jun 2025 P2]*
4. Corticosteroids in severe pneumonia. *[DNB Oct 2023 P3]*
5. Macrolides in severe community-acquired pneumonia and sepsis. *[DNB Oct 2023 P3]*

5.3 Hospital-Acquired & Ventilator-Associated Pneumonia

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A2 HAP and VAP
- ✓ Define HAP and VAP; list predisposing factors and organisms
- ✓ Diagnose VAP and select empirical antibiotics based on MDR risk and local antibiogram
- ✓ Implement VAP prevention bundles

IN BRIEF HAP occurs ≥48 h after admission (not incubating); VAP occurs ≥48 h after intubation. In Indian ICUs, MDR gram-negatives — Acinetobacter, Klebsiella (carbapenem-resistant), Pseudomonas — dominate, making local antibiograms essential.

Predisposing factors of nosocomial pneumonia

Host	Intervention / iatrogenic
Age >65, COPD, immunosuppression, coma, malnutrition	Endotracheal intubation (6–21× risk), reintubation, tracheostomy
Recent surgery (thoracic/upper abdominal), trauma, burns	Supine position, sedation, NG tube, enteral feeds with high gastric volumes
Prior antibiotics (MDR colonisation)	Stress ulcer prophylaxis (PPI), contaminated equipment, poor hand hygiene
Aspiration risk (stroke, dysphagia)	Prolonged ventilation, ventilator circuit changes

Diagnosis of VAP

- Clinical: new/progressive infiltrate + ≥2 of fever >38 °C, leucocytosis/leucopenia, purulent secretions, worsening oxygenation.
- **CPIS** (Clinical Pulmonary Infection Score) >6 suggests VAP (limited accuracy). CDC ventilator-associated events (VAC → IVAC → PVAP) for surveillance.
- Microbiology: endotracheal aspirate semi-quantitative (≥10^5 CFU/mL) — IDSA 2016 prefers non-invasive; BAL ≥10^4, PSB ≥10^3; blood cultures.

Empirical therapy (IDSA/ATS 2016 adapted)

Risk	Regimen
Low MDR risk, no septic shock	Single agent: piperacillin-tazobactam or cefepime or levofloxacin
MDR risk (IV antibiotics in 90 days, septic shock, ARDS, ≥5 days hospitalisation, dialysis) or unit with >10% resistance	Two anti-pseudomonal agents from different classes ± MRSA cover (vancomycin/linezolid) if MRSA >10–20%
Carbapenem-resistant Enterobacterales (CRE)	Ceftazidime-avibactam ± aztreonam (for MBL — NDM common in India), polymyxin-based
CR-Acinetobacter	Polymyxin B/colistin + high-dose ampicillin-sulbactam/tigecycline; cefiderocol; sulbactam-durlobactam

- Duration 7 days; de-escalate by culture; procalcitonin to stop.

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VAP prevention bundle

- Head-end elevation 30–45°; daily sedation interruption and spontaneous breathing trial; subglottic secretion drainage ETT; cuff pressure 20–30 cmH2O; oral care (toothbrushing; chlorhexidine — conflicting mortality data); avoid unnecessary reintubation; NIV/HFNC to avoid intubation; early mobilisation; avoid routine circuit changes; hand hygiene; DVT and stress ulcer prophylaxis as indicated; early weaning.

★ CLINICAL PEARLS & EXAM FOCUS

- Indian ICU VAP = think CR-Klebsiella (NDM/OXA-48) and Acinetobacter — know your unit's antibiogram before writing "meropenem".

LANDMARK TRIALS & GUIDELINES

- Kalil AC et al, IDSA/ATS HAP/VAP guideline, Clin Infect Dis 2016; ICMR Treatment Guidelines for Antimicrobial Use in Common Syndromes (2019, 2022).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Predisposing factors of nosocomial pneumonia. *[DNB Jun 2020 P3]*
2. Ventilator associated pneumonia and its management. *[BFUHS 2020 P2; BFUHS 2022 P4]*

5.4 Atypical & Tropical Pneumonias — Mycoplasma, Legionella, Melioidosis, Scrub Typhus, Leptospirosis

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A2 Unusual and atypical pneumonias
- ✓ Recognise clinical clues of atypical and tropical pneumonias
- ✓ Diagnose and treat Mycoplasma, Legionella, melioidosis and rickettsial pneumonia

IN BRIEF Atypical pathogens lack a cell wall or are intracellular, so β-lactams are ineffective; they often produce extrapulmonary features. In India, melioidosis and scrub typhus are under-recognised causes of severe pneumonia and ARDS.

Atypical pneumonias

Organism	Clues	Diagnosis	Treatment
Mycoplasma pneumoniae	Young adults, outbreaks; dry cough, headache; bullous myringitis; cold agglutinin haemolysis; SJS/mucositis (MIRM), encephalitis	PCR, IgM serology, cold agglutinins	Azithromycin 5 days or doxycycline; macrolide resistance (Asia)
Legionella pneumophila	Water systems, AC; high fever, relative bradycardia, diarrhoea, confusion, hyponatraemia, raised LFT/CK	Urinary antigen (serogroup 1), culture on BCYE, PCR	Levofloxacin or azithromycin 7–14 days
Chlamydophila psittaci	Bird exposure; splenomegaly, Horder spots	Serology, PCR	Doxycycline
Coxiella burnetii (Q fever)	Livestock; hepatitis, endocarditis	Serology	Doxycycline

Melioidosis

- **Burkholderia pseudomallei** — soil/water saprophyte (rice fields, monsoon); "Vietnam time bomb"; endemic in coastal Karnataka, Kerala, Odisha, TN.
- **Risk:** diabetes (>50%), alcohol, CKD, thalassaemia, steroids.
- **Clinical:** acute septicaemic pneumonia (upper lobe consolidation/cavities mimicking TB), multiple abscesses (liver, spleen, prostate, parotid in children), septic arthritis; chronic form mimics TB.

INVESTIGATIONS & TREATMENT AT A GLANCE

5.3 Hospital-Acquired & Ventilator-Associated Pneumonia

‹ Back to topic (p 99)
Next topic 5.4 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Clinical Pulmonary Infection Score (CPIS)	≤ 6	> 6	VAP likely (limited accuracy)
Endotracheal aspirate semi-quantitative culture	Light growth	Moderate/heavy growth (≥ 10^5 CFU/mL quantitative)	VAP organism
Procalcitonin trend	Falling	Rising	Guides duration
MRSA nasal PCR	Negative (NPV > 95%)	Positive	Stop/withhold MRSA cover if negative
Local antibiogram	—	Carbapenem resistance	Guides empirical choice

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Piperacillin–tazobactam	4.5 g IV 6-hourly (extended infusion)	HAP/VAP without MDR risk	AKI
Meropenem	1–2 g IV 8-hourly (extended infusion)	MDR risk / ESBL	Seizures (low)
Colistin (polymyxin E)	Load 9 MIU, then 4.5 MIU 12-hourly	Carbapenem-resistant GNB	Nephro/neurotoxicity
Ceftazidime–avibactam (+ aztreonam for MBL)	2.5 g IV 8-hourly	CRE/KPC/OXA-48	Renal dosing
Vancomycin / linezolid	Vanco 15–20 mg/kg 8–12-hourly; linezolid 600 mg 12-hourly	MRSA risk	Nephrotoxicity / thrombocytopenia

★ **PRESCRIBING & INTERPRETATION PEARLS**

- 7 days of therapy is enough for most HAP/VAP (IDSA/ATS 2016).

- **Diagnosis:** culture (Ashdown's medium) — gram-negative bipolar "safety-pin" bacilli; gentamicin/colistin-resistant; latex agglutination, PCR.
- **Treatment:** intensive phase IV ceftazidime 2 g q6–8h or meropenem 1 g q8h ≥2 weeks (4–8 weeks for deep abscess); eradication phase co-trimoxazole (high dose) ×3–6 months. Mortality 20–50%.

Scrub typhus and leptospirosis

- **Scrub typhus** (Orientia tsutsugamushi, chigger mite): eschar, fever, lymphadenopathy, interstitial pneumonitis, ARDS, myocarditis, meningoencephalitis, AKI; IgM ELISA; doxycycline 100 mg BD 7 days or azithromycin; IV doxycycline + azithromycin in severe (INTREST trial, 2024).
- **Leptospirosis** (Weil's disease): pulmonary haemorrhage syndrome (high mortality), ARDS, AKI, jaundice; IgM ELISA, MAT, PCR; penicillin/ceftriaxone or doxycycline; plasmapheresis/steroids debated.

★ CLINICAL PEARLS & EXAM FOCUS

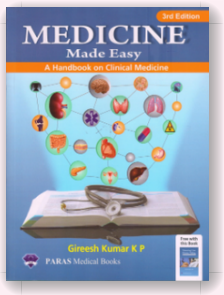
▸ Diabetic farmer from coastal India with cavitating pneumonia, splenic abscesses and no response to standard antibiotics → melioidosis.

LANDMARK TRIALS & GUIDELINES

- Varghese GM et al, INTREST trial (combination IV doxycycline + azithromycin in severe scrub typhus), NEJM 2023.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Mycoplasma pneumonia. *[DNB Jun 2021 P2]*
2. Melioidosis. *[Manipal 2014 P2]*



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5.5 Viral Pneumonias — Influenza, COVID-19, RSV — and Vaccines

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A2 Viral pneumonias; I-C8
- ✓ Diagnose and treat influenza and COVID-19 pneumonia
- ✓ Manage critically ill patients with COVID-19
- ✓ Describe COVID-19 vaccine types, side effects and vaccine hesitancy

IN BRIEF Viral pneumonias range from mild bronchitis to ARDS. Antivirals work best early; in severe COVID-19 the inflammatory phase responds to dexamethasone and IL-6/JAK inhibition; secondary bacterial and fungal infection drive late mortality.

Influenza

- Antigenic drift (seasonal epidemics) and shift (pandemics — H1N1 2009); high-risk: pregnancy, elderly, chronic lung/heart disease, diabetes, obesity, immunosuppressed.

INVESTIGATIONS & TREATMENT AT A GLANCE

5.4 Atypical & Tropical Pneumonias — Mycoplasma, Legionella, Melioidosis, Scrub Typhus, Leptospirosis

‹ Back to topic (p 100)
Next topic 5.5 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Mycoplasma PCR / IgM	Negative	Positive; cold agglutinins ≥ 1:64	Mycoplasma pneumonia
Legionella urinary antigen	Negative	Positive (serogroup 1)	Legionnaires' disease
Serum sodium	135–145 mmol/L	< 130	Legionella clue
Scrub typhus IgM ELISA	OD < 0.5	OD > 0.5; eschar	Scrub typhus pneumonitis (India)
Leptospira IgM / MAT	Negative	MAT ≥ 1:400	Pulmonary haemorrhage syndrome
B. pseudomallei culture	No growth	Positive	Melioidosis
Q fever / psittacosis serology	Negative	4-fold rise	Zoonotic pneumonia

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Azithromycin	500 mg daily × 3–5 days	Mycoplasma, Legionella (with or without levofloxacin)	QTc
Doxycycline	100 mg bd × 7 days	Scrub typhus, Q fever, psittacosis, leptospirosis (mild)	Not in pregnancy (azithro for scrub)
Ceftazidime / meropenem then co-trimoxazole	2 g 6-hourly / 1 g 8-hourly × ≥ 2 weeks; then TMP-SMX 3–6 months	Melioidosis	Relapse if eradication incomplete
Penicillin G / ceftriaxone	1.5 MU 6-hourly / 1 g daily	Severe leptospirosis	Jarisch-Herxheimer

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Pneumonia with eschar, thrombocytopenia and transaminitis in India → think scrub typhus.

- **Complications:** primary viral pneumonia/ARDS, secondary bacterial pneumonia (S. aureus incl. MRSA, pneumococcus), myocarditis, encephalitis, influenza-associated aspergillosis.
- **Treatment (MoHFW categories A/B/C):** oseltamivir 75 mg BD ×5 days (double in severe, renal adjustment) started early (within 48 h ideally, but in all hospitalised regardless); baloxavir (single dose) for uncomplicated; zanamivir/peramivir; avoid steroids.

COVID-19

- **Pathophysiology:** ACE2 entry, viral phase then immune/inflammatory phase (cytokine storm), endotheliitis, microthrombosis; "silent hypoxaemia".

Management of COVID-19 by severity

Severity	Management
Mild (no hypoxia)	Symptomatic; nirmatrelvir/ritonavir within 5 days if high risk (check interactions); molnupiravir alternative
Moderate (SpO2 <94%, RR >24)	Oxygen (target 92–96%), dexamethasone 6 mg ×10 days, prophylactic anticoagulation (therapeutic heparin in non-ICU — ATTACC), remdesivir (5 days), awake prone positioning
Severe/critical (HFNC, NIV, MV)	Dexamethasone + tocilizumab or baricitinib (RECOVERY), HFNC/NIV/helmet, lung-protective ventilation, prone, ECMO in selected; prophylactic-dose anticoagulation

- **Caring for the critically ill patient:** early HFNC/awake prone, avoid excessive fluids, monitor for secondary infections (CAPA, mucormycosis — CAM), thrombosis, delirium, pressure injuries; lung-protective MV; family communication.

COVID-19 vaccines (India)

Types and adverse effects

Platform	Example	Notable adverse effects
Viral vector (ChAdOx1)	Covishield	Fever, myalgia; rare TTS (VITT), GBS
Inactivated whole virion	Covaxin	Local pain, fever; generally mild
Protein subunit	Corbevax, Covovax	Local reactions
mRNA	Comirnaty, Spikevax; Gemcovac (self-amplifying, India)	Myocarditis (young males), anaphylaxis
DNA	ZyCoV-D (needle-free)	Local

- **Vaccine hesitancy** (WHO 3C model — complacency, convenience, confidence; 5C adds calculation and collective responsibility): addressed by transparent communication, trusted HCPs, community leaders, countering misinformation, easy access.

RSV

- Older adults and immunocompromised: pneumonia, exacerbations of COPD/HF; RSV vaccines (RSVPreF3-AS01, RSVpreF, mRNA-1345) for ≥60–75 years and at-risk 50–59; maternal RSVpreF; nirsevimab for infants. Ribavirin (aerosolised/oral) in HSCT recipients.

✦ RECENT ADVANCES & GUIDELINE UPDATES

▸ WHO 2023/2024 COVID-19 therapeutics: nirmatrelvir/r for high-risk non-severe; corticosteroids + IL-6 or JAK inhibitor for severe/critical; against ivermectin, convalescent plasma, hydroxychloroquine.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Dexamethasone helps COVID patients on oxygen but harms those not needing oxygen (RECOVERY).

LANDMARK TRIALS & GUIDELINES

- RECOVERY Collaborative Group, dexamethasone NEJM 2021; tocilizumab Lancet 2021; Uyeki TM et al, IDSA influenza guideline 2018.

INVESTIGATIONS & TREATMENT AT A GLANCE

5.5 Viral Pneumonias — Influenza, COVID-19, RSV — and Vaccines

‹ Back to topic (p 101)
Next topic 5.6 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Influenza/SARS-CoV-2 RT-PCR	Not detected	Detected	Diagnosis
Lymphocyte count	> 1.0 × 10^9/L	< 0.8	Severe COVID
D-dimer	< 0.5 µg/mL	> 1–2 µg/mL	Thrombosis risk/severity
Ferritin / CRP / IL-6	Ferritin < 300; CRP < 10	Ferritin > 1000; CRP > 75	Hyperinflammation — tocilizumab consideration
SpO_2	≥ 94%	< 94%	O_2 / dexamethasone indication

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Oseltamivir	75 mg bd × 5 days	Influenza	—
Dexamethasone	6 mg daily × up to 10 days	COVID-19 needing O_2 (RECOVERY)	Not in mild disease
Tocilizumab	8 mg/kg IV (max 800 mg) single dose	COVID with rising O_2 needs and CRP ≥ 75	Infection, LFTs
Enoxaparin	40 mg SC daily (prophylaxis)	Hospitalised COVID	Bleeding
Vaccines	Influenza annually; COVID per national schedule; RSV (≥ 60 y) single dose	Prevention	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Avoid steroids in non-hypoxaemic COVID — RECOVERY showed possible harm.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Vaccination against COVID-19: types of vaccines and side effects. *[DNB Jun 2021 P1]*
2. Vaccine hesitancy. *[DNB Jun 2021 P1]*
3. Caring for the critically ill patient with COVID-19. *[DNB Oct 2023 P4]*
4. Respiratory syncytial virus. *[Manipal 2014 P2]*

5.6 Aspiration Pneumonia, Lung Abscess & Lipoid Pneumonia

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A2 Lung abscess; II-B4 Aspiration
- ✓ Differentiate aspiration pneumonitis from aspiration pneumonia and manage both
- ✓ Describe microbiology, diagnosis, differential diagnosis and treatment of lung abscess
- ✓ Recognise exogenous lipoid pneumonia

IN BRIEF Aspiration of oropharyngeal or gastric contents causes chemical pneumonitis (Mendelson) or bacterial pneumonia; untreated, anaerobic infection necroses lung to form an abscess — a cavity >2 cm with an air–fluid level.

Aspiration pneumonitis vs pneumonia

Feature	Pneumonitis (Mendelson)	Aspiration pneumonia
Material	Sterile acidic gastric contents (pH <2.5, >0.3 mL/kg)	Colonised oropharyngeal secretions
Risk	Decreased consciousness (overdose, anaesthesia, seizure)	Dysphagia, stroke, poor dentition, alcohol, elderly
Onset	Hours — acute hypoxaemia, wheeze, fever, infiltrate	Days; typical pneumonia features
Treatment	Suction, supportive, O2; antibiotics not routinely (unless persisting >48 h); no steroids	Antibiotics (amoxicillin-clavulanate, ceftriaxone ± metronidazole if abscess)

Lung abscess

- **Primary** (aspiration in predisposed host — anaerobes, polymicrobial; right lower/posterior segments) vs **secondary** (bronchial obstruction — tumour, foreign body; septic emboli; immunosuppression; bronchiectasis; spread from subphrenic/amoebic abscess).
- **Microbiology:** anaerobes (Peptostreptococcus, Prevotella, Fusobacterium, Bacteroides), S. milleri group, S. aureus, Klebsiella (K1 hypervirulent), Pseudomonas, Nocardia, Actinomyces, melioidosis, Entamoeba (right lower lobe from liver).
- **Clinical:** fever, putrid sputum (anaerobes), haemoptysis, weight loss, clubbing (chronic); poor dentition.
- **Investigations:** CXR/CT (thick-walled cavity, air–fluid level, makes acute angle with chest wall vs empyema's obtuse angle, "split pleura sign" in empyema), sputum culture, blood culture, bronchoscopy (exclude obstruction — mandatory in smokers >45, atypical sites, no response), CT-guided aspiration.

Differential diagnosis of lung abscess (cavity with air–fluid level)

Condition	Distinguishing features
Empyema with BPF	Lenticular, obtuse angle with chest wall, split pleura sign, air–fluid level length differs on PA and lateral
Cavitating carcinoma	Thick irregular wall (>15 mm), older smoker, eccentric cavity, nodular inner wall
Tuberculosis	Upper lobe, no air–fluid level usually, satellite nodules
Infected bulla/cyst	Thin wall, minimal surrounding consolidation
GPA	Multiple cavitating nodules, ANCA, sinus/renal involvement
Hydatid cyst	Water-lily sign, membranes
Fungal (aspergilloma, mucor)	Air crescent, immunosuppression

Condition	Distinguishing features
Septic emboli	Multiple peripheral nodules, feeding vessel sign, IV drug use, endocarditis
Pulmonary infarct, sequestration, hiatus hernia	Clinical context, CTA

- **Treatment:** amoxicillin-clavulanate or ampicillin-sulbactam, or ceftriaxone + metronidazole/clindamycin; 3–6 weeks (until cavity resolves or small stable scar); postural drainage; percutaneous drainage for large (>6 cm) non-responding; surgery for failure, massive haemoptysis, suspected cancer.

Lipoid pneumonia

- **Exogenous:** aspiration of mineral oil (laxatives, nasal drops), ghee/oil instillation in infants (India — oil massage), fire-eaters; **endogenous** (obstructive — distal to tumour; PAP, lipid storage).
- CT: consolidation with **fat attenuation (−30 to −150 HU)**, crazy paving; BAL — lipid-laden macrophages (Oil red O); treatment — stop exposure, whole lung lavage in severe, steroids debated.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Acute angle with chest wall and a spherical cavity = abscess; obtuse angle and lenticular shape = empyema.

LANDMARK TRIALS & GUIDELINES

- Mandell LA, Niederman MS. Aspiration pneumonia, NEJM 2019.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Diagnosis and management of aspiration pneumonia. *[DNB Dec 2021 P2]*
2. Differential diagnosis of lung abscess. *[DNB Jun 2025 P2]*
3. Discuss the microbiology, diagnosis and treatment of lung abscess. *[Manipal 2014 P2]*
4. Lipoid pneumonia. *[Manipal 2014 P2]*

5.7 Bronchiectasis, Cystic Fibrosis & Primary Ciliary Dyskinesia

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A2 Bronchiectasis; II-B4 Cystic fibrosis and ciliary disorders
- ✓ Describe aetiology and evaluation of bronchiectasis (incl. young male with bilateral disease)
- ✓ Manage bronchiectasis per ERS 2017/BTS 2019 and EMBARC-India data
- ✓ Describe complications, reversible bronchiectasis, CF and PCD

IN BRIEF Bronchiectasis is permanent dilatation of bronchi with a vicious vortex of infection, inflammation, impaired clearance and structural damage. In India it is younger, more severe, often post-TB, with Pseudomonas in ~30% (EMBARC-India registry).

Aetiology of bronchiectasis

Category	Causes
Post-infective	TB (commonest in India), measles, pertussis, severe pneumonia, NTM
Obstructive	Foreign body, tumour, node compression (middle lobe)
Immunodeficiency	CVID, hypogammaglobulinaemia, HIV, IgG subclass deficiency
Mucociliary	Cystic fibrosis, PCD, Young's syndrome
Allergic	ABPA (central bronchiectasis)

INVESTIGATIONS & TREATMENT AT A GLANCE

5.6 Aspiration Pneumonia, Lung Abscess & Lipoid Pneumonia

‹ Back to topic (p 103)
Next topic 5.7 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
CT chest	—	Thick-walled cavity with air-fluid level, dependent segments	Lung abscess
Sputum anaerobic culture	—	Polymicrobial	Aspiration
Bronchoscopy	—	Obstructing lesion / foreign body	Abscess in non-dependent site or no response
BAL lipid-laden macrophages (Oil Red O)	Absent	Present	Lipoid pneumonia
CT (lipoid pneumonia)	—	Fat-density (−30 to −150 HU) consolidation	Exogenous lipoid pneumonia

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Co-amoxiclav	1.2 g IV 8-hourly → oral 625 mg 8-hourly	Aspiration pneumonia/abscess	3–6 weeks for abscess
Clindamycin	600 mg IV 8-hourly	Penicillin allergy	C. difficile
Metronidazole + β-lactam	500 mg 8-hourly + ceftriaxone	Alternative	Metronidazole alone inadequate
Percutaneous / endobronchial drainage	Procedure	Abscess > 6 cm not responding	Pneumothorax, empyema

★ PRESCRIBING & INTERPRETATION PEARLS

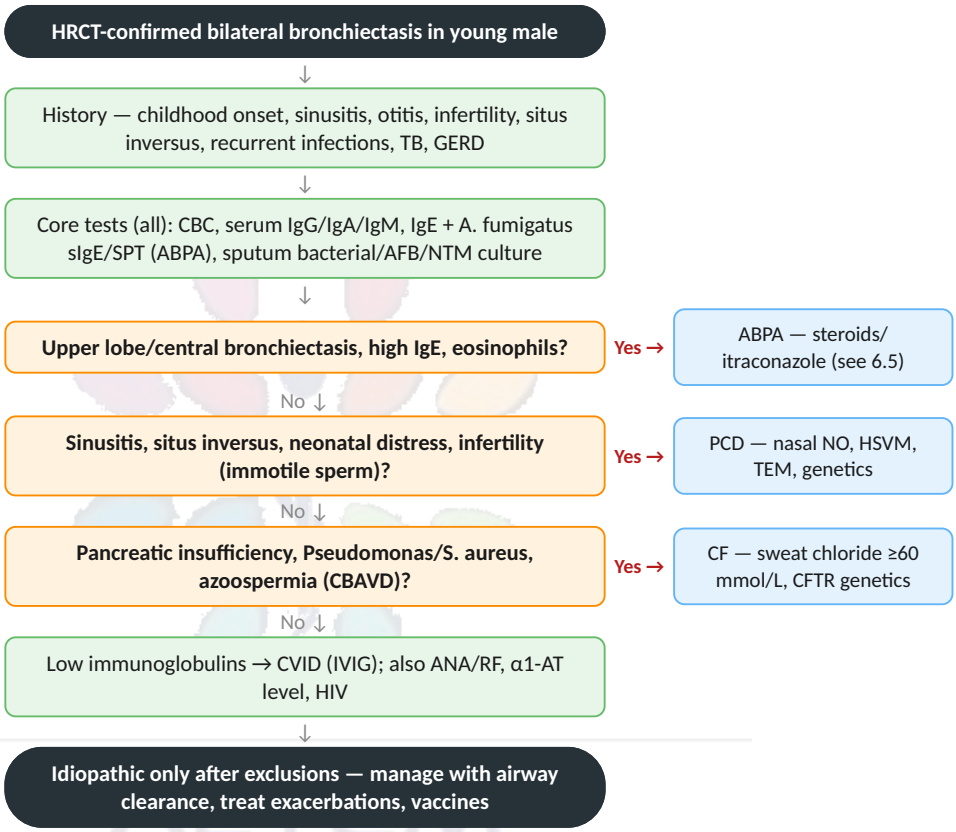
- Lung abscess usually resolves with antibiotics alone; drainage in < 10–20%.

Category	Causes
Systemic	RA, Sjögren, IBD, α1-ATD, yellow nail syndrome
Aspiration/GERD; congenital (Mounier-Kuhn, Williams–Campbell)	Idiopathic in ~30–40%

- **Morphology (Reid):** cylindrical, varicose, cystic (saccular). HRCT: broncho-arterial ratio >1 (signet ring), lack of tapering, visible bronchi within 1 cm of pleura.

Evaluation of a young male with bilateral bronchiectasis

Algorithm 5.1 · Aetiological work-up of bronchiectasis (young male, bilateral)



Management (ERS 2017, BTS 2019)

- **Airway clearance:** ACBT, oscillating PEP (Acapella, flutter), postural drainage, hypertonic saline (7%) or mannitol; carbocisteine; **no rhDNase in non-CF** (harm).
- **Exacerbations:** 14 days antibiotics guided by sputum culture (Pseudomonas: ciprofloxacin or IV antipseudomonal).
- **Pseudomonas eradication** on first isolation: ciprofloxacin 2 weeks ± inhaled colistin/tobramycin ×3 months.
- **≥3 exacerbations/year:** long-term macrolide (azithromycin 250 mg daily/500 mg thrice weekly) if no Pseudomonas or as alternative; inhaled antibiotics if chronic Pseudomonas.
- Others: pulmonary rehabilitation, influenza and pneumococcal vaccines, bronchodilators if co-existing airflow obstruction, ICS only for eosinophilic/asthma overlap, surgery for localised disease, BAE for haemoptysis, transplant.
- **Severity:** BSI (Bronchiectasis Severity Index), FACED/E-FACED.

Complications of bronchiectasis

- Haemoptysis (massive), recurrent pneumonia, lung abscess, empyema, pneumothorax, chronic respiratory failure, PH/cor pulmonale, NTM and Aspergillus infection, AA amyloidosis, brain abscess, depression; social isolation from cough/sputum.

Reversible (pseudo-) bronchiectasis

- Transient bronchial dilatation during acute pneumonia/atelectasis that resolves within 3–6 months; also ABPA (reversible early). HRCT for diagnosis should be done when clinically stable (≥6–8 weeks after infection).

Cystic fibrosis

- Autosomal recessive CFTR mutations (F508del commonest; classes I–VI); rare in Indians (often missed; atypical mutations). Sweat chloride ≥60 mmol/L diagnostic (30–59 intermediate).
- **Management:** airway clearance, dornase alfa, hypertonic saline, inhaled antibiotics (tobramycin, aztreonam), azithromycin, nutrition (pancreatic enzymes, fat-soluble vitamins, high-calorie), CFTR modulators — **elexacaftor/tezacaftor/ivacaftor** (≥1 F508del; dramatic FEV1 gains), CF-related diabetes, transplant.

Primary ciliary dyskinesia

- Autosomal recessive (>50 genes: DNAH5, DNAI1, CCDC39/40); neonatal respiratory distress, year-round wet cough and rhinitis, otitis media, bronchiectasis (middle/lower lobes), situs inversus (Kartagener ~50%), infertility.
- **Diagnosis (ERS 2017):** nasal NO <77 nL/min (screen), high-speed video microscopy, TEM (ultrastructure), genetics, immunofluorescence. **PICADAR** score for referral.
- Treatment: airway clearance, antibiotics, azithromycin (BESTCILIA), ENT care.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Bronchiectasis in India — exclude ABPA in every patient (IgE + A. fumigatus sIgE); TB sequelae is the commonest cause.

LANDMARK TRIALS & GUIDELINES

- Polverino E et al, ERS bronchiectasis guidelines 2017; Dhar R et al, EMBARC-India registry, Lancet Glob Health 2019; Middleton PG et al, elexacaftor–tezacaftor–ivacaftor NEJM 2019.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Management of bronchiectasis. *[DNB Dec 2021 P2]*
2. Reversible bronchiectasis. *[DNB Oct 2023 P2]*
3. Management of a case of cystic fibrosis of lung. *[DNB Oct 2023 P2]*
4. Describe the approach of evaluating and managing a young male with bilateral bronchiectasis. *[DNB May 2024 P2]*
5. Complications of bronchiectasis. *[DNB Dec 2025 P3]*
6. Ciliary dyskinesia. *[VNSGU 2025 P2]*

5.8 Pulmonary Aspergillosis Spectrum & Mucormycosis

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A2 Fungal pneumonias; I-C9
- ✓ Classify the spectrum of pulmonary aspergillosis by host immunity
- ✓ Diagnose and treat CPA, aspergilloma and invasive aspergillosis including CAPA
- ✓ Diagnose and treat pulmonary and rhino-orbital mucormycosis and fungal sinusitis

IN BRIEF Aspergillus disease depends on host immunity: hypersensitivity (ABPA) in atopy, saprophytic and chronic (aspergilloma, CPA) in structural lung disease, and invasive in neutropenia/immunosuppression. India has a large burden of post-TB CPA and COVID-associated mucormycosis.

INVESTIGATIONS & TREATMENT AT A GLANCE

5.7 Bronchiectasis, Cystic Fibrosis & Primary Ciliary Dyskinesia

‹ Back to topic (p 104)
Next topic 5.8 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
HRCT	Bronchus-artery ratio < 1	Ratio > 1 (signet ring), lack of tapering, visible peripheral bronchi	Bronchiectasis
Sputum culture	Normal flora	*P. aeruginosa*, *H. influenzae*	Pseudomonas → eradication, worse prognosis
Total IgE / Aspergillus-specific IgE	IgE < 500 IU/mL	IgE > 500 + specific IgE positive	ABPA
Immunoglobulins	Normal	Low IgG/IgA	Immunodeficiency
Sweat chloride	< 30 mmol/L	≥ 60 mmol/L	Cystic fibrosis
Nasal NO	> 77 nL/min	< 77 nL/min	PCD
Bronchiectasis Severity Index	0–4 mild	≥ 9 severe	Prognosis

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Airway clearance + hypertonic saline	7% saline 4 mL bd	All with sputum	Bronchospasm test dose
Azithromycin maintenance	250 mg daily or 500 mg TIW	≥ 3 exacerbations/year	NTM exclusion; QTc
Inhaled colistin / tobramycin	Colistin 1 MU bd; tobramycin 300 mg bd	Chronic Pseudomonas	Bronchospasm
Ciprofloxacin (eradication)	750 mg bd × 2 weeks	New Pseudomonas isolation	Tendinopathy
Exacerbation antibiotics	14 days per culture	Exacerbation	—

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Do not use dornase alfa in non-CF bronchiectasis.

Spectrum of pulmonary aspergillosis

Entity	Host	Key features
ABPA	Asthma, CF	Hypersensitivity; see 6.5
Aspergillus sensitisation / SAFS	Severe asthma	Positive sIgE without ABPA criteria
Simple aspergilloma	Pre-existing cavity (post-TB), normal immunity	Fungal ball, air crescent, mobile on decubitus; stable
Chronic cavitary PA (CCPA)	Post-TB, COPD, NTM, sarcoid	≥3 months symptoms, new/expanding cavities, pleural thickening, Aspergillus IgG
Chronic fibrosing PA	Untreated CCPA	Lobe destruction
Aspergillus nodule	Mild immunosuppression	Mimics cancer
Subacute invasive (CNPA)	Mild immunosuppression (diabetes, steroids, alcohol, COPD)	<3 months, progressive cavitation
Invasive pulmonary aspergillosis (IPA)	Neutropenia, HSCT, SOT, high-dose steroids, ICU, influenza/COVID	Angio-invasion: halo sign, air crescent (recovery), GM positive

Chronic pulmonary aspergillosis

- **Diagnosis (ERS/ESCMID 2016):** compatible imaging for ≥3 months + Aspergillus IgG (or precipitins) or direct evidence (culture/histology) + exclusion of other diagnoses (TB!). In India, smear-negative "TB" relapse after ATT is often CPA.
- **Treatment:** oral itraconazole 200 mg BD or voriconazole 150–200 mg BD for 6 months (12 months if response partial); surgery for simple aspergilloma (lobectomy) and localised disease with haemoptysis.
- **Non-surgical treatment of mycetoma (aspergilloma):** observation if asymptomatic; tranexamic acid for minor haemoptysis; **bronchial artery embolisation** for massive haemoptysis (recurrence common); systemic antifungals (limited effect on ball); intracavitary instillation of amphotericin B/iodine via percutaneous catheter; radiotherapy (selected); endobronchial removal rarely.

Invasive aspergillosis

- **EORTC/MSGERC 2020:** host factors + clinical (dense nodules ± halo, air crescent, cavity, wedge consolidation) + mycology (GM, culture, PCR) → probable; histology → proven.
- **Treatment:** voriconazole 6 mg/kg q12h ×2 then 4 mg/kg q12h (TDM) or isavuconazole 200 mg q8h ×6 doses then daily; liposomal amphotericin for azole intolerance; ≥6–12 weeks; reduce immunosuppression.
- **COVID-19-associated pulmonary aspergillosis (CAPA)** and influenza-associated (IAPA): ECMM/ISHAM 2020 criteria — BAL GM ≥1.0, culture; mortality ~50%; voriconazole/isavuconazole.

Mucormycosis

- **Agents:** Rhizopus arrhizus (commonest), Mucor, Lichtheimia, Apophysomyces (cutaneous, India), Rhizopus microsporus.
- **Risk:** uncontrolled diabetes/DKA (India ~70%), steroids (COVID-associated mucormycosis epidemic 2021), haematological malignancy, iron overload/deferoxamine.
- **Forms:** rhino-orbito-cerebral (black eschar on palate/nasal turbinate, proptosis, ophthalmoplegia), pulmonary (reversed halo, thick-walled cavity, vessel occlusion sign), cutaneous, GI, disseminated.
- **Diagnosis:** KOH/calcofluor — broad aseptate ribbon-like hyphae with right-angle branching; histopathology (tissue invasion); culture; PCR; GM and BDG negative.
- **Treatment (ECMM/MSG 2019):** urgent surgical debridement + liposomal amphotericin B 5 mg/kg/day (10 mg/kg CNS) → step-down posaconazole/isavuconazole; control diabetes and ketoacidosis, stop steroids. Mortality 30–60%.

Fungal sinusitis

- **Non-invasive:** allergic fungal rhinosinusitis (Bent and Kuhn criteria — nasal polyps, eosinophilic mucin, Charcot–Leyden crystals; FESS + steroids), fungal ball (maxillary; FESS).
- **Invasive:** acute fulminant (mucor, Aspergillus in neutropenia/diabetes — surgical emergency), chronic invasive, granulomatous invasive (A. flavus — Sudan/India, immunocompetent).

★ CLINICAL PEARLS & EXAM FOCUS

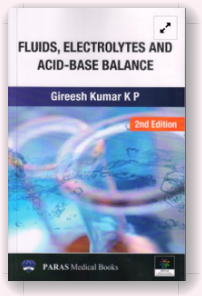
- ▸ Post-TB patient with "relapse" but negative NAAT/culture, new pleural thickening and cavity = CPA until proved otherwise — send Aspergillus IgG.

LANDMARK TRIALS & GUIDELINES

- Denning DW et al, ERS/ESCMID CPA guideline Eur Respir J 2016; Cornely OA et al, global mucormycosis guideline Lancet Infect Dis 2019; Sen M et al, COSMIC study (India CAM) 2021.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Non-surgical treatments of mycetoma lung. *[DNB Jun 2025 P2]*
2. COVID-19 associated fungal infections. *[BFUHS 2022 P4]*
3. Fungal sinusitis. *[Manipal 2014 P2]*



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5.9 Pneumocystis, Cryptococcosis, Histoplasmosis & Other Fungal Pneumonias

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A2 Unusual pneumonias; I-C9
- ✓ Diagnose and treat PJP in HIV and non-HIV hosts
- ✓ Describe pulmonary cryptococcosis, histoplasmosis, talaromycosis, nocardiosis and actinomycosis

IN BRIEF Fungal pneumonias in India include PJP (HIV, steroids, transplant), cryptococcosis, endemic histoplasmosis (Gangetic plains, NE) and talaromycosis (Manipur). Non-HIV PJP is more fulminant with lower fungal load.

Pneumocystis jirovecii pneumonia

- **Risk:** HIV with CD4 <200; non-HIV — prednisolone ≥20 mg >4 weeks, rituximab, haematological malignancy, transplant, fludarabine.
- **Features:** subacute dyspnoea, dry cough, fever, exertional desaturation, high LDH; CXR bilateral perihilar ground glass (may be normal); HRCT diffuse GGO with crazy paving, cysts (pneumatoceles) — pneumothorax.
- **Diagnosis:** induced sputum/BAL immunofluorescence, PCR (qPCR), BDG (high NPV).
- **Treatment:** see 3.11 — co-trimoxazole 21 days + steroids if PaO2 <70 (HIV); prophylaxis for CD4 <200, prolonged steroids, transplant.

Other fungal pneumonias

Infection	Epidemiology / host	Pulmonary features	Treatment
Cryptococcosis (C. neoformans, gattii)	HIV, transplant; pigeon droppings	Nodules, masses, consolidation; check CSF always	Mild pulmonary: fluconazole 400 mg 6–12 months; severe/CNS: amphotericin + flucytosine induction

INVESTIGATIONS & TREATMENT AT A GLANCE

5.8 Pulmonary Aspergillosis Spectrum & Mucormycosis

‹ Back to topic (p 106)
Next topic 5.9 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Aspergillus IgG	Negative	Positive	CPA
Total IgE (ABPA)	< 500 IU/mL	≥ 500 IU/mL	ABPA criterion (ISHAM 2024)
Serum/BAL galactomannan	< 0.5 / < 1.0	≥ 0.5 serum; ≥ 1.0 BAL	Invasive aspergillosis
CT halo / air-crescent sign	—	Halo (early IPA), air crescent (recovery)	IPA
Tissue KOH/histology (mucor)	—	Broad, ribbon-like aseptate hyphae, right-angle branching	Mucormycosis
HbA1c / ketones	Normal	Uncontrolled DM / DKA	Mucormycosis risk

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Voriconazole	6 mg/kg IV 12-hourly × 2 then 4 mg/kg; oral 200 mg bd	Invasive aspergillosis	TDM; visual, LFTs
Itraconazole	200 mg bd × ≥ 6 months	CPA; ABPA	TDM; avoid with rifampicin
Prednisolone (ABPA)	0.5 mg/kg/day × 2 weeks, tapered over 3–4 months	ABPA	Steroid toxicity
Liposomal amphotericin B	5–10 mg/kg/day	Mucormycosis + surgical debridement	Nephrotoxicity
Posaconazole / isavuconazole	Posa 300 mg bd day 1 then 300 mg daily	Mucor step-down	TDM (posa > 1 mg/L)

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Mucormycosis needs three things together: antifungal, surgical debridement and control of the predisposition (DM, steroids).

Infection	Epidemiology / host	Pulmonary features	Treatment
Histoplasmosis	Bird/bat droppings, caves; endemic India (WB, UP, Assam)	Acute (flu-like, hilar nodes), chronic cavitary (mimics TB), disseminated (adrenal enlargement, oral ulcers), mediastinal granuloma/fibrosing mediastinitis	Itraconazole; liposomal amphotericin for severe/ disseminated
Talaromycosis (T. marneffei)	HIV in Manipur, SE Asia	Umbilicated skin papules, fever, pneumonia	Amphotericin then itraconazole
Blastomycosis, coccidioidomycosis	North America (travel)	Pneumonia, skin, bone	Itraconazole/amphotericin
Nocardiosis	Steroids, transplant	Nodules/cavities, empyema, brain abscess	Co-trimoxazole ± imipenem/ amikacin 6–12 months
Actinomycosis	Poor dentition, alcohol	Mass crossing fissures/chest wall, sinuses, sulphur granules	High-dose penicillin 6–12 months

★ **CLINICAL PEARLS & EXAM FOCUS**

- Non-HIV PJP — sicker, faster, lower organism load; PCR/BDG help when stains are negative.

LANDMARK TRIALS & GUIDELINES

- Limper AH et al, ATS statement on fungal infections 2011; NACO/IAP guidelines on opportunistic infections.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Diagnosis and management of fungal pneumonias. *[BFUHS 2019 P4]*

5.10 Parasitic Lung Disease — Hydatid, Amoebiasis, Paragonimiasis & Zoonoses

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A2 Parasitic infections, zoonosis; I-C12
- ✓ Describe the clinical and radiological features and management of pulmonary hydatid disease
- ✓ Describe thoracic amoebiasis, paragonimiasis and other parasitic causes of lung disease

IN BRIEF Parasites reach the lung by haematogenous spread (hydatid, larval migration), direct extension (amoebic liver abscess) or by residing in the lung (Paragonimus). Eosinophilia is a frequent but not universal clue.

Pulmonary hydatid disease

- **Echinococcus granulosus** — dog definitive host, sheep/humans intermediate; lung is second commonest site (25–30%) after liver; children/young adults.
- **Clinical:** often asymptomatic; cough, chest pain, haemoptysis; rupture → expectoration of salty fluid and membranes ("grape skins"), anaphylaxis, infection, pneumothorax.

Radiological signs of hydatid cyst

Sign	Description
Intact cyst	Well-defined round/oval homogeneous opacity, "cannon-ball"
Meniscus / crescent sign	Air between pericyst and endocyst (impending rupture)
Cumbo sign (onion peel)	Air within endocyst and between layers
Water-lily (camalote) sign	Floating collapsed membranes in fluid
Rising sun sign	Membrane remnants on fluid surface

INVESTIGATIONS & TREATMENT AT A GLANCE

5.9 Pneumocystis, Cryptococcosis, Histoplasmosis & Other Fungal Pneumonias

‹ Back to topic (p 108)
Next topic 5.10 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
LDH	< 250 U/L	> 350 U/L	PCP (non-specific)
β-D-glucan	< 60 pg/mL	> 80 pg/mL	PCP (high sensitivity)
Induced sputum / BAL PCR or IF for *Pneumocystis*	Negative	Positive	PCP
CD4 count	> 200	< 200	PCP risk in HIV
Serum CrAg	Negative	Positive	Cryptococcosis — LP to exclude meningitis
Histoplasma antigen (urine)	Negative	Positive	Histoplasmosis

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Co-trimoxazole (treatment)	15–20 mg/kg/day TMP in 3–4 doses × 21 days	PCP	Rash, hyperkalaemia, cytopenia
Prednisolone (PCP)	40 mg bd × 5 d, 40 mg daily × 5 d, 20 mg daily × 11 d	PaO_2 < 70 mmHg / A–a > 35	—
Fluconazole	400 mg daily (pulmonary crypto)	Cryptococcal pneumonia without CNS disease	Interactions
Liposomal amphotericin + flucytosine	AmB 3–4 mg/kg/day + 5-FC 100 mg/kg/day	Cryptococcal meningitis / severe disease	Nephrotoxicity, marrow
Itraconazole	200 mg tds × 3 days then bd	Histoplasmosis (mild–moderate)	TDM

★ **PRESCRIBING & INTERPRETATION PEARLS**

- PCP prophylaxis: CD4 < 200 in HIV or prednisolone ≥ 20 mg/day for > 4 weeks.

Sign	Description
Mass within cavity / dry cyst	Collapsed membranes after complete evacuation
Inverse crescent	Air at base

- **Diagnosis:** CT, USG (liver), serology (ELISA, IHA, Western blot — less sensitive in lung), eosinophilia. Avoid percutaneous aspiration.
- **Treatment:** surgery is definitive — cystotomy with capitonnage (lung-sparing), enucleation, rarely lobectomy; albendazole 400 mg BD for 1–3 months pre- and post-operatively (or alone for small/multiple inoperable cysts); treat concomitant liver cysts (PAIR for liver).

Thoracic amoebiasis

- Rupture of amoebic liver abscess through diaphragm (right lower lobe): empyema, lung abscess, hepatobronchial fistula ("anchovy sauce" sputum), pericardial involvement (left lobe abscess — tamponade).
- Diagnosis: USG/CT liver, serology, trophozoites in sputum/pus; treatment metronidazole 750 mg TDS ×10 days + luminal agent (diloxanide), drainage of empyema.

Paragonimiasis

- Lung fluke (Paragonimus westermani, heterotremus) from raw crabs/crayfish — NE India (Manipur, Arunachal, Nagaland).
- Chronic cough, rusty/chocolate sputum, haemoptysis — mimics TB (smear-negative); CXR nodules, ring shadows, cysts, effusion (eosinophilic); eggs in sputum; praziquantel 25 mg/kg TDS ×2–3 days.

Other parasites and zoonoses

- **Löffler syndrome** (Ascaris, hookworm, Strongyloides larval migration), **tropical pulmonary eosinophilia** (filarial, see 7.8), **strongyloides hyperinfection** (steroids — gram-negative sepsis, ARDS; ivermectin), dirofilariasis (coin lesion), toxocariasis, schistosomiasis (PH).
- **Zoonotic pneumonias:** psittacosis, Q fever, tularaemia, plague (pneumonic), anthrax (inhalational — mediastinal widening), leptospirosis, brucellosis, melioidosis, hantavirus.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Never aspirate a suspected hydatid cyst — anaphylaxis and spillage.

LANDMARK TRIALS & GUIDELINES

- WHO-IWGE expert consensus on echinococcosis (Brunetti, 2010).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Hydatid cyst. *[WBUHS 2020 P2]*
2. Radiological findings in hydatid disease of lung. *[BFUHS 2020 P2]*
3. Hydatid disease of lung. *[BFUHS 2019 P3]*
4. Thoracic amoebiasis. *[Manipal 2014 P2]*

5.11 Pulmonary Infections in the Immunocompromised Host (HIV, Transplant, Immunosuppressants)

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A2 AIDS and opportunistic infections; I-C10
- ✓ Approach pulmonary infiltrates in the immunocompromised host by type of immune defect and timing
- ✓ Manage a post-HSCT patient with cough and dyspnoea
- ✓ Prevent respiratory infections in patients receiving immunosuppressants

INVESTIGATIONS & TREATMENT AT A GLANCE

5.10 Parasitic Lung Disease — Hydatid, Amoebiasis, Paragonimiasis & Zoonoses

‹ Back to topic (p 109)
Next topic 5.11 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Hydatid serology (ELISA/IHA)	Negative	Positive	Echinococcosis (may be negative in lung cysts)
CT chest (hydatid)	—	Water-lily sign, meniscus sign, cyst	Hydatid cyst
Amoebic serology	Negative	Positive	Amoebic liver abscess with pleuropulmonary extension
Sputum/BAL for *Paragonimus* ova	Negative	Operculated ova	Paragonimiasis (North-East India, crabs)
Absolute eosinophil count	< 500/µL	> 500; > 3000 TPE	Parasitic lung disease
Filarial IgG / antibodies	Low	Very high IgE + filarial antibodies	Tropical pulmonary eosinophilia

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Albendazole	400 mg bd (10–15 mg/kg/day) with meals; cycles	Hydatid (with surgery)	LFTs, neutropenia; teratogenic
Surgery (cystectomy, capitonnage)	Procedure	Pulmonary hydatid	Avoid rupture/anaphylaxis
Metronidazole	750 mg 8-hourly × 7–10 days + luminal agent	Amoebiasis	Disulfiram-like reaction
Praziquantel	25 mg/kg tds × 2 days	Paragonimiasis	Headache, dizziness
Diethylcarbamazine	6 mg/kg/day × 21 days	Tropical pulmonary eosinophilia	Mazzotti-like reactions

★ PRESCRIBING & INTERPRETATION PEARLS

- Paragonimiasis mimics TB (haemoptysis, cavities) — check sputum for ova in NE India.

IN BRIEF The type of immune defect predicts the pathogen: neutropenia → bacteria and moulds; T-cell defect (HIV, transplant, steroids) → PJP, TB, CMV, Nocardia, Cryptococcus; humoral defect → encapsulated bacteria. Non-infectious mimics must always be considered.

Immune defect and likely pathogens

Defect	Conditions	Pathogens
Neutropenia / neutrophil dysfunction	Chemotherapy, AML, CGD	Gram-negatives, S. aureus, Aspergillus, Mucor, Candida
Cell-mediated (T-cell)	HIV, SOT, HSCT, steroids, anti-TNF, lymphoma	PJP, TB/NTM, CMV, HSV, VZV, Cryptococcus, Histoplasma, Nocardia, Legionella, Toxoplasma
Humoral (B-cell) / asplenia	CVID, myeloma, CLL, rituximab, splenectomy	S. pneumoniae, H. influenzae, Neisseria
Mixed	Allogeneic HSCT with GVHD	All

HIV — pulmonary infections by CD4 count

- **Any CD4:** bacterial pneumonia (pneumococcus — 10× risk), TB, influenza.
- **CD4 <200:** PJP, cryptococcosis, disseminated TB, histoplasmosis/talaromycosis. **CD4 <50:** CMV, MAC, Aspergillus, Pseudomonas. Non-infectious: Kaposi sarcoma, lymphoma, LIP (children), PAH, emphysema, lung cancer.

Timeline after HSCT

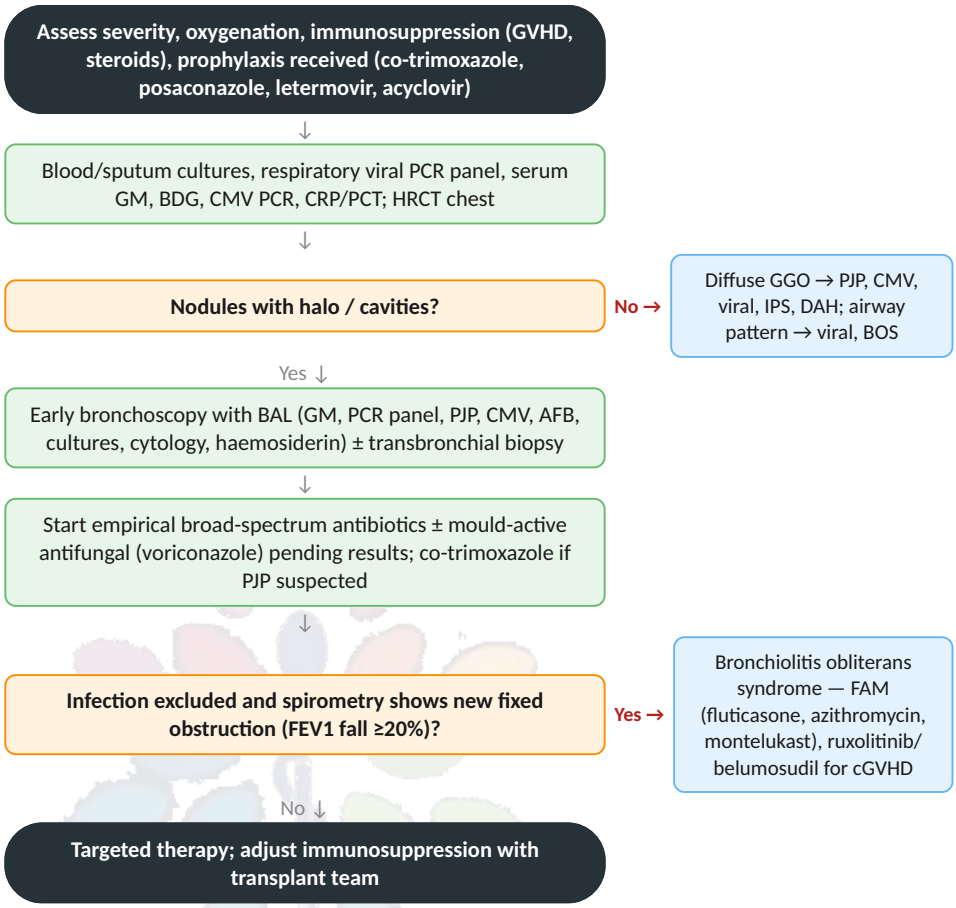
Pulmonary complications after haematopoietic stem cell transplant

Phase	Infectious	Non-infectious
Pre-engraftment (0–30 days)	Bacteria, Aspergillus, Candida, HSV	Engraftment syndrome, DAH, IPS, fluid overload, drug toxicity
Early post-engraftment (30–100 days)	CMV, PJP, Aspergillus, respiratory viruses, adenovirus	IPS, DAH, PERDS
Late (>100 days)	Encapsulated bacteria, VZV, Aspergillus, community viruses, TB	Bronchiolitis obliterans syndrome (chronic GVHD), organising pneumonia, PTLD

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Approach — 40-year-old woman 5 months after allogeneic HSCT with cough and dyspnoea

Algorithm 5.2 · Pulmonary infiltrate after HSCT



Preventing infections in patients on immunosuppressants

- **Screening before biologics/immunosuppressants:** TB (IGRA/TST + CXR → TPT), HBV (HBsAg, anti-HBc → prophylaxis tenofovir/ entecavir), HCV, HIV, strongyloides (endemic — ivermectin), VZV serology.
- **Vaccines** before starting (≥2 weeks for inactivated, ≥4 weeks for live): pneumococcal (PCV20 or PCV13/15 → PPSV23), influenza annually, recombinant zoster, COVID-19, hepatitis B; avoid live vaccines during immunosuppression.
- **Chemoprophylaxis:** co-trimoxazole for PJP (prednisolone ≥20 mg ≥4 weeks with second immunosuppressant, rituximab, cyclophosphamide); posaconazole in AML/GVHD; acyclovir (HSV/VZV); letermovir (CMV) in HSCT.
- Hand hygiene, avoid construction sites (Aspergillus), food safety, mask in crowds, pet precautions.

★ CLINICAL PEARLS & EXAM FOCUS

▸ In the immunocompromised, early BAL beats empirical guessing — yield falls sharply after 24–72 h of antimicrobials.

LANDMARK TRIALS & GUIDELINES

- Azoulay E et al, ERS/ESCMID statement on acute respiratory failure in immunocompromised (2020); IDSA guidelines on vaccination of the immunocompromised host.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. HIV and pulmonary infections. *[DNB Jun 2021 P2]*
2. A 40-year-old lady who underwent allogeneic stem cell transplantation five months ago presents with cough and dyspnoea of three weeks. How will you manage her? *[BFUHS 2023 P2]*
3. Prevention of respiratory infections in patients receiving immunosuppressants. *[BFUHS 2020 P3]*

INVESTIGATIONS & TREATMENT AT A GLANCE

5.11 Pulmonary Infections in the Immunocompromised Host (HIV, Transplant, Immunosuppressants)

‹ Back to topic (p 110)
Next topic 5.12 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
ANC	> 1.5 × 10⁹/L	< 0.5	Neutropenic pneumonia (bacteria, moulds)
CD4 count	> 500	< 200 (PCP), < 50 (MAC, CMV)	HIV-associated pathogens
Galactomannan / β-D-glucan	Negative	Positive	Fungal infection
CMV PCR	Undetectable	Rising	CMV pneumonitis
BAL multiplex PCR, culture, cytology	Negative	Pathogen	Essential early in immunocompromised infiltrates
CT chest	—	Nodules ± halo (mould), GGO (PCP/CMV), tree-in-bud (bacterial/TB)	Pattern-based differential

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Cefepime / piperacillin–tazobactam	Cefepime 2 g 8-hourly	Febrile neutropenia with pneumonia	—
Voriconazole	As in 5.8	Suspected mould	—
Co-trimoxazole	Treatment dose	PCP	—
Ganciclovir	5 mg/kg 12-hourly	CMV pneumonitis	Myelosuppression
G-CSF	5 µg/kg/day SC	Profound neutropenia (selected)	Bone pain

★ PRESCRIBING & INTERPRETATION PEARLS

- Do bronchoscopy within 24–48 h — empirical therapy without samples lowers diagnostic yield.

5.12 Recurrent & Non-Resolving Pneumonia; MRSA Pneumonia

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-A2 Other pneumonias
- ✓ Define and evaluate recurrent and non-resolving pneumonia
- ✓ Diagnose and treat MRSA pneumonia

IN BRIEF Recurrent pneumonia (≥2 episodes in 1 year or ≥3 ever, with radiological clearing between) points to a local (same site) or systemic (different sites) predisposition. Non-resolving pneumonia — failure of radiological resolution by 4–8 weeks — needs exclusion of TB, cancer and non-infectious mimics.

Causes of recurrent pneumonia

Same lobe (local)	Different lobes (systemic)
Endobronchial obstruction — tumour, foreign body, broncholith, stricture	Immunodeficiency (CVID, HIV, IgG subclass, complement), asplenia
Bronchiectasis (localised), sequestration, bronchogenic cyst	Aspiration (dysphagia, GERD, alcohol, seizures, achalasia)
Middle lobe syndrome	CF, PCD, COPD, asthma
Tracheal bronchus, TEF	Heart failure, ILD, drug-induced, vasculitis, OP

Non-resolving pneumonia — differential

Category	Causes
Infection	TB, NTM, fungi (CPA, mucor), melioidosis, nocardia, resistant bacteria, empyema/abscess
Neoplastic	Invasive mucinous adenocarcinoma, lymphoma, bronchial obstruction by cancer
Inflammatory	Organising pneumonia, eosinophilic pneumonia, vasculitis/DAH, HP, sarcoid
Others	Pulmonary infarction, lipoid pneumonia, drug toxicity, pulmonary oedema

- **Normal resolution:** 50% clear by 2 weeks, 64% by 4 weeks, 73% by 6 weeks; slower in elderly, smokers, multilobar, bacteraemic pneumococcal, Legionella.
- **Work-up:** repeat CT, bronchoscopy (BAL, biopsy), immunoglobulins, HIV, swallow study, echo; biopsy if persistent.

MRSA pneumonia

- **CA-MRSA** (PVL toxin): previously healthy young, post-influenza, necrotising/cavitating pneumonia, haemoptysis, leucopenia, high mortality. **HA-MRSA:** HAP/VAP, prior colonisation.
- Diagnosis: sputum/BAL culture, blood cultures, nasal MRSA PCR (high NPV — de-escalate if negative).
- **Treatment:** vancomycin (AUC-guided, trough 15–20 historically) or **linezolid** 600 mg BD (ZEPHyR — better clinical cure, inhibits toxin production); add clindamycin for PVL toxin; duration 7–21 days; drain empyema.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Necrotising pneumonia with haemoptysis after influenza in a young adult — think PVL-MRSA; add linezolid.

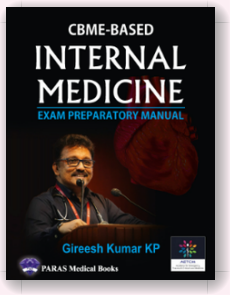
LANDMARK TRIALS & GUIDELINES

- Wunderink RG et al, ZEPHyR trial, Clin Infect Dis 2012.

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PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Recurrent pneumonia. *[DNB Oct 2023 P2]*
2. Methicillin resistant staphylococcal pneumonia. *[DNB Oct 2023 P2]*



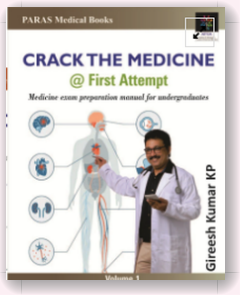
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INVESTIGATIONS & TREATMENT AT A GLANCE

5.12 Recurrent & Non-Resolving Pneumonia; MRSA Pneumonia

‹ Back to topic (p 113)
Next topic 6.1 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
CXR / CT at 6–8 weeks	Resolution	Persisting > 6–8 weeks	Non-resolving pneumonia
Sputum Xpert / AFB	Negative	Positive	TB as cause
Bronchoscopy (BAL, biopsy)	Normal	Obstruction, malignancy, organism	Persistent infiltrate
MRSA screen / culture (PVL)	Negative	MRSA (PVL-positive necrotising)	MRSA pneumonia
Immunoglobulins, HIV	Normal	Low / reactive	Immunodeficiency in recurrent pneumonia

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Vancomycin	15–20 mg/kg 8–12-hourly (AUC 400–600)	MRSA pneumonia	Nephrotoxicity
Linezolid	600 mg bd × 7–21 days	MRSA (preferred in PVL — toxin suppression)	Thrombocytopenia
Clindamycin (add-on)	600–900 mg 8-hourly	PVL toxin suppression	C. difficile
Treat underlying cause	—	Recurrent same-site → obstruction; different sites → aspiration/immune	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Recurrent pneumonia in the same lobe → endobronchial obstruction until proved otherwise.

SECTION 6

Obstructive Airway Diseases

Paper II / III

Asthma including ABPA, allergen immunotherapy and immunomodulation; COPD and small airway diseases; special aspects of management — long-term oxygen therapy, inhalation therapy and pulmonary rehabilitation; emphysema interventions and α1-antitrypsin deficiency (NMC syllabus II-B6, I-D4).

6.1 Asthma — Pathogenesis, Phenotypes & Diagnosis 115
6.2 Management of Chronic Asthma — GINA Tracks, MART & Control Assessment 117
6.3 Acute Severe Asthma — Assessment, ICU Management & Mechanical Ventilation 118
6.4 Difficult-to-Treat & Severe Asthma — Phenotyping and Biologics 120
6.5 Allergic Bronchopulmonary Aspergillosis 122
6.6 Occupational Asthma, Allergen Immunotherapy & Inducible Laryngeal Obstruction 124
6.7 COPD — Definition, Pathogenesis, Risk Factors & Assessment 125
6.8 Pharmacotherapy of Stable COPD 127
6.9 Acute Exacerbation of COPD & Role of NIV 129
6.10 Systemic Manifestations, Comorbidities, Hyperinflation & Cor Pulmonale 131
6.11 Asthma–COPD Overlap & Small Airway Diseases (Bronchiolitis) 132
6.12 Oxygen Therapy, LTOT, Oxygen Toxicity & Hyperbaric Oxygen 133
6.13 Pulmonary Rehabilitation & Airway Clearance Techniques 135
6.14 Interventions for Emphysema — LVRS, Bronchoscopic Lung Volume Reduction & Bullous Disease 136
6.15 Alpha-1 Antitrypsin Deficiency 137

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6.1 Asthma — Pathogenesis, Phenotypes & Diagnosis

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B6 Asthma
- ✓ Describe the immunopathogenesis of asthma (type-2 high and low) and airway remodelling
- ✓ Diagnose asthma using GINA criteria — variable symptoms and confirmed variable airflow limitation
- ✓ Explain the relationship between obesity and asthma

IN BRIEF Asthma is a heterogeneous disease with chronic airway inflammation, defined by variable respiratory symptoms (wheeze, breathlessness, chest tightness, cough) together with variable expiratory airflow limitation. India has ~35 million asthmatics with high underdiagnosis and low ICS use.

Pathogenesis

- **Type-2 (T2-high, ~50–70%):** allergen/irritant → epithelial alarmins (TSLP, IL-33, IL-25) → Th2 and ILC2 → IL-4 (IgE switching), IL-5 (eosinophils), IL-13 (mucus, AHR, FeNO) → mast cell activation (histamine, LTC4/D4, PGD2) → early and late-phase responses.
- **T2-low:** neutrophilic (Th17, smoking, obesity), paucigranulocytic (smooth muscle dysfunction).
- **Remodelling:** epithelial damage, goblet cell hyperplasia, subepithelial fibrosis, smooth muscle hypertrophy/hyperplasia, angiogenesis — fixed obstruction.
- **AHR:** exaggerated narrowing to stimuli (methacholine, cold air, exercise).
- **Risk factors:** atopy, family history, viral infections (RSV, rhinovirus), allergens (house dust mite, cockroach), occupational agents, smoking, air pollution, obesity, maternal smoking, prematurity.

Asthma phenotypes

Phenotype	Features
Allergic	Childhood onset, atopy, eosinophilic, good ICS response
Non-allergic	Adult, often neutrophilic/paucigranulocytic, poorer ICS response
Adult (late) onset	Women, non-allergic, may be eosinophilic (AERD, CRSwNP)
Asthma with persistent airflow limitation	Long-standing, remodelling
Asthma with obesity	Prominent symptoms, little eosinophilia
Exercise-induced bronchoconstriction, AERD, occupational, cough-variant	Specific triggers

Obesity and asthma

- Obesity increases incidence (dose-response, women > men), severity and poor control.
- **Mechanisms:** mechanical (reduced FRC and ERV — airways at low volume, increased closure), adipokines (↑ leptin, ↓ adiponectin — systemic inflammation), T2-low neutrophilic inflammation, comorbidities (OSA, GERD, deconditioning, depression), reduced ICS response, vitamin D.
- **Weight loss** (≥5–10%, bariatric surgery) improves control; exercise, treat OSA/GERD.

Diagnosis (GINA 2025)

Confirming variable expiratory airflow limitation

Test	Positive criterion (adults)
Bronchodilator responsiveness	FEV1 or FVC increase >12% and >200 mL (or >10% predicted, ATS/ERS 2022)
PEF variability	Average daily diurnal variability >10% (2 weeks, twice daily)
Treatment trial	FEV1 ↑ >12% and >200 mL after 4 weeks of ICS
Exercise challenge	FEV1 fall >10% and >200 mL
Bronchial challenge	Methacholine PD20 ≤200 µg; mannitol fall ≥15%
Variation between visits	FEV1 variation >12% and >200 mL

- Supportive: FeNO >50 ppb, blood eosinophils ≥300, atopy (SPT/sIgE).
- **Differential diagnosis:** COPD, bronchiectasis, VCD/inducible laryngeal obstruction, heart failure, central airway obstruction/tumour, ABPA, EGPA, hyperventilation, GERD, chronic cough syndromes.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Confirm diagnosis before starting controller if possible — but never delay ICS in a symptomatic patient; document variability later.

LANDMARK TRIALS & GUIDELINES

• Global Initiative for Asthma (GINA) report 2025; Asthma Insights and Management (INDIA) / Global Burden of Disease India 2019.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Obesity as a risk factor related to asthma. *[DNB Dec 2022 P1]*

6.2 Management of Chronic Asthma — GINA Tracks, MART & Control Assessment

‹ NMC curriculum

Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B6 Asthma management
- ✓ Classify asthma control and future risk
- ✓ Treat asthma using GINA 2025 Track 1 (ICS-formoterol reliever) and Track 2 (SABA reliever) steps
- ✓ Explain why ICS should be started from diagnosis and never SABA alone

IN BRIEF GINA no longer recommends SABA-only treatment. The preferred approach (Track 1) uses as-needed low-dose ICS-formoterol as reliever (steps 1–2, "AIR") and as maintenance-and-reliever therapy (MART, steps 3–5), which reduces severe exacerbations compared with SABA-based regimens.

Assessment of symptom control (GINA — past 4 weeks)

Question	Well controlled	Partly controlled	Uncontrolled
Daytime symptoms >twice/week	None of the 4	1–2 of the 4	3–4 of the 4
Any night waking due to asthma			
SABA reliever >twice/week			
Any activity limitation			

- **Risk factors for exacerbations:** uncontrolled symptoms, high SABA use (≥3 canisters/year; >1/month increases death risk), inadequate ICS, poor technique/adherence, FEV1 <60%, ≥1 severe exacerbation last year, ever intubated, high FeNO/ eosinophils, smoking, obesity, pregnancy, psychosocial problems.
- Tools: ACT (≥20 controlled), ACQ.

GINA 2025 step-wise treatment (adults & adolescents)

Step	Track 1 (preferred) — reliever: as-needed low-dose ICS-formoterol	Track 2 — reliever: SABA (or ICS-SABA)
1	As-needed low-dose ICS-formoterol	Take ICS whenever SABA taken
2	As-needed low-dose ICS-formoterol	Daily low-dose ICS
3	Low-dose ICS-formoterol MART	Low-dose ICS-LABA
4	Medium-dose ICS-formoterol MART	Medium/high-dose ICS-LABA
5	Add-on LAMA; phenotype; anti-IgE, anti-IL5/5R, anti-IL4Rα, anti-TSLP; consider high-dose ICS-formoterol MART	Same add-ons

- **Other controllers:** LTRA (with boxed neuropsychiatric warning), azithromycin (step 5, non-eosinophilic), low-dose OCS only as last resort, sublingual HDM immunotherapy (steps 2–4 with HDM sensitisation, FEV1 >70%).

INVESTIGATIONS & TREATMENT AT A GLANCE

6.1 Asthma — Pathogenesis, Phenotypes & Diagnosis

‹ Back to topic (p 115)
Next topic 6.2 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Spirometry bronchodilator response	< 10% of predicted FEV1	≥ 10% predicted (or ≥ 12% & 200 mL)	Variable airflow — asthma
PEF diurnal variability	< 10%	> 10% (average over 1–2 weeks)	Asthma
FeNO	< 25 ppb	> 50 ppb	Type-2 inflammation, ICS-responsive
Blood eosinophils	< 150/µL	≥ 300/µL	Eosinophilic phenotype
Methacholine PC20	> 16 mg/mL	< 4 mg/mL	Airway hyperresponsiveness
Total & specific IgE / skin-prick	IgE < 100 IU/mL	Raised / sensitised	Allergic asthma

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Budesonide–formoterol (as needed)	200/6 µg 1 puff PRN	Mild asthma (GINA Track 1, steps 1–2)	Not SABA-only therapy
Salbutamol	100–200 µg PRN	Reliever (Track 2)	Overuse > 2 canisters/year = risk
Montelukast	10 mg at night	Add-on, allergic rhinitis	Neuropsychiatric warning

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Never treat adult asthma with SABA alone — every patient needs ICS-containing therapy.

- **MART doses:** budesonide-formoterol 200/6 one inhalation BD (or OD) + one as needed; max 12 inhalations/day (8 in step 3 is typical). Beclomethasone-formoterol 100/6 alternative.
- **Step down** after 3 months of good control (reduce ICS 25–50%); never stop ICS completely.

Why ICS from the time of diagnosis

- Asthma is an inflammatory disease even when symptoms are mild — **30–40% of asthma deaths and severe exacerbations occur in "mild" asthma**.
- SABA alone increases eosinophilic inflammation, β-receptor down-regulation, AHR and exacerbation and death risk (dose-dependent — SABINA).
- Early ICS reduces severe exacerbations (START trial — budesonide reduced severe events by 44% in recent-onset mild asthma), hospitalisations and mortality, and may slow FEV1 decline/remodelling.
- **SYGMA 1/2, Novel START, PRACTICAL:** as-needed budesonide-formoterol reduced severe exacerbations by ~60% vs SABA alone and was non-inferior to daily ICS with much lower steroid exposure.

Non-pharmacological

- Inhaler technique and adherence checks, written action plan, self-monitoring (PEF), smoking cessation, avoid triggers (HDM measures, occupational exposure), vaccination (influenza, COVID-19, pneumococcal), weight loss, breathing exercises, treat rhinitis/GERD/OSA.

✦ RECENT ADVANCES & GUIDELINE UPDATES

▸ GINA 2024–25: anti-inflammatory reliever (AIR) concept; tezepelumab for severe asthma regardless of phenotype; albuterol-budesonide (ICS-SABA) as reliever in Track 2 (MANDALA).

★ CLINICAL PEARLS & EXAM FOCUS

▸ "No SABA-only treatment for any adult or adolescent" — the most important GINA change since 2019.

LANDMARK TRIALS & GUIDELINES

- O'Byrne PM et al, SYGMA-1 NEJM 2018; Beasley R et al, Novel START NEJM 2019; Pauwels RA et al, START Lancet 2003.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Classification of control of asthma. *[DNB Dec 2022 P2]*
2. Why inhaled corticosteroids are recommended to be commenced from the time of diagnosis of bronchial asthma? *[DNB Jun 2025 P2]*

6.3 Acute Severe Asthma — Assessment, ICU Management & Mechanical Ventilation

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B6; II-B10 Emergencies
- ✓ Assess severity of an acute asthma exacerbation
- ✓ Manage acute severe and life-threatening asthma in the emergency department and ICU
- ✓ Ventilate a patient with severe asthma avoiding dynamic hyperinflation

IN BRIEF Severe asthma exacerbation needs immediate oxygen, repeated SABA–ipratropium, systemic steroids and IV magnesium; PaCO2 rising towards normal or above is a danger sign. Mechanical ventilation aims to minimise dynamic hyperinflation through low minute ventilation and long expiratory time, accepting hypercapnia.

INVESTIGATIONS & TREATMENT AT A GLANCE

6.2 Management of Chronic Asthma — GINA Tracks, MART & Control Assessment

‹ Back to topic (p 117)
Next topic 6.3 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
ACT (Asthma Control Test)	≥ 20	< 20 (≤ 15 poorly controlled)	Control
GINA symptom control (4 questions)	0 = well controlled	3–4 = uncontrolled	Control
Spirometry (FEV1)	> 80% predicted	< 60%	Future-risk factor
Exacerbations in past year	0	≥ 1 severe	Risk — step up
Inhaler technique / adherence	Correct	Errors / missed doses	Check before step-up

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Step 3 MART	Budesonide/formoterol 200/6 µg 1 puff bd + PRN	Track 1 step 3	Max 12 puffs/day
Step 4 MART	200/6 µg 2 puffs bd + PRN	Track 1 step 4	—
Medium/high ICS–LABA (Track 2)	Fluticasone/salmeterol 250/50 µg 1 puff bd	Alternative track	Thrush, dysphonia
Tiotropium (step 5 add-on)	2.5 µg × 2 puffs daily (SMI)	Step 5	Dry mouth
Step-down	Reduce ICS by 25–50% after 3 months control	Stable asthma	Do not stop ICS completely

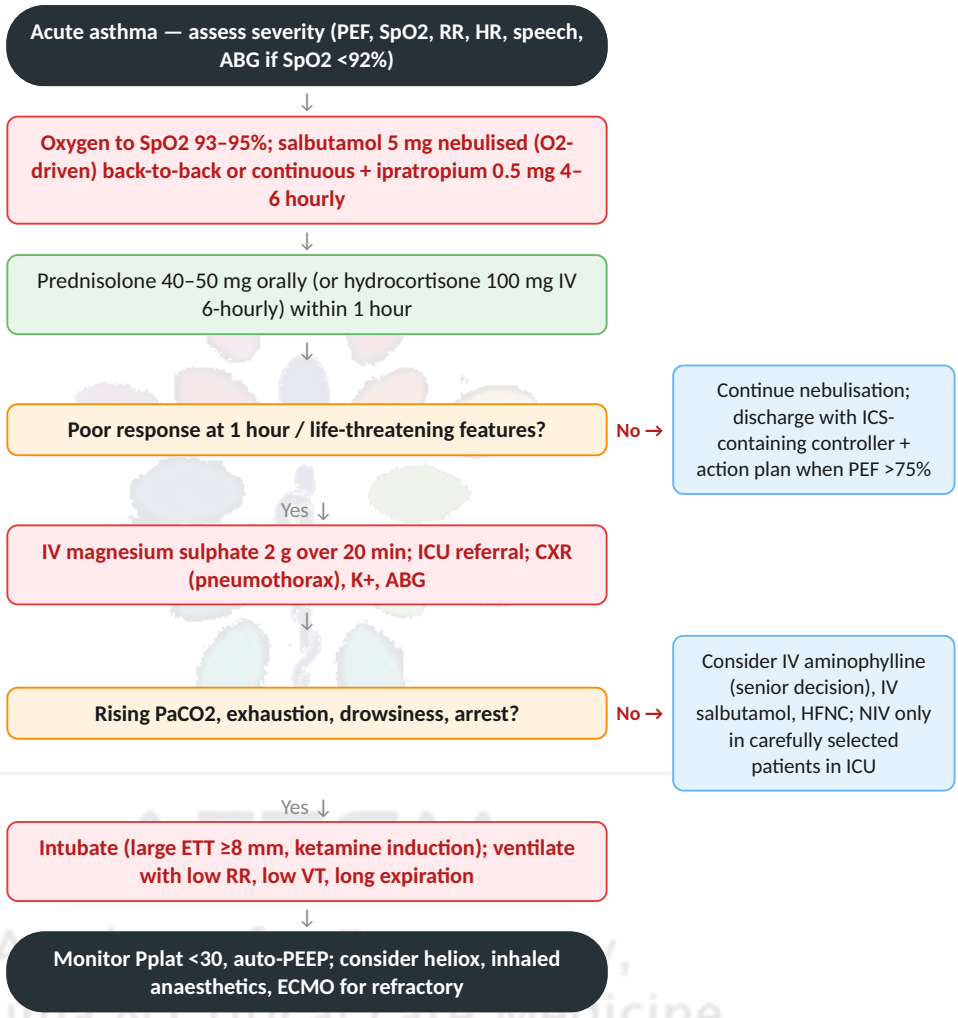
★ PRESCRIBING & INTERPRETATION PEARLS

- Review at 1–3 months after any change; step down after 3 months of good control.

Severity assessment of acute asthma (BTS/GINA)

Severity	Features
Moderate	PEF >50–75% best/predicted; talks in phrases; no features of severe
Acute severe (any one)	PEF 33–50%; RR ≥25; HR ≥110; cannot complete sentences in one breath
Life-threatening (any one)	PEF <33%; SpO2 <92%; PaO2 <8 kPa (60 mmHg); normal PaCO2 (4.6–6 kPa); silent chest, cyanosis, poor effort; arrhythmia, hypotension; exhaustion, altered consciousness
Near-fatal	Raised PaCO2 and/or need for mechanical ventilation with raised inflation pressures

Algorithm 6.1 · Management of acute severe asthma



Mechanical ventilation in acute asthma

- **Goal:** avoid dynamic hyperinflation (barotrauma, hypotension) — controlled hypoventilation (permissive hypercapnia, pH >7.15–7.20).
- **Settings:** volume control, VT 6–8 mL/kg PBW, RR 10–14, high inspiratory flow (60–100 L/min, square wave) to shorten Ti, I:E ≥1:3–1:5, applied PEEP low/zero (or ≤80% of measured auto-PEEP in spontaneously breathing), FiO2 to SpO2 >92%.
- **Monitor:** Pplat <30 cmH2O, auto-PEEP <10–15 (end-expiratory hold), flow waveform returning to zero.
- **Hypotension after intubation:** disconnect ventilator for 30–60 s (apnoea test), fluids; exclude tension pneumothorax.
- Deep sedation (propofol, ketamine — bronchodilator), avoid prolonged NMB with steroids (myopathy); extubate early.

★ **CLINICAL PEARLS & EXAM FOCUS**

▸ A "normal" PaCO2 in acute severe asthma is a life-threatening sign — the patient is tiring.

LANDMARK TRIALS & GUIDELINES

- BTS/SIGN British guideline on the management of asthma (2019, BTS/NICE/SIGN 2024); Kew KM et al, Cochrane IV magnesium 2014.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Management of acute severe asthma in ICU. *[DNB Dec 2022 P2]*
2. Severity assessment of an acute exacerbation of asthma. *[DNB Dec 2021 P3]*
3. Role of mechanical ventilation during an acute asthma exacerbation. *[DNB Dec 2021 P3]*

6.4 Difficult-to-Treat & Severe Asthma — Phenotyping and Biologics

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B6 Immunomodulation in asthma
- ✓ Distinguish uncontrolled, difficult-to-treat and severe asthma
- ✓ Phenotype severe asthma and select biologic therapy
- ✓ Describe anti-IL-5 agents and other monoclonal antibodies

IN BRIEF Difficult-to-treat asthma is uncontrolled despite GINA step 4–5 or needs high-dose treatment; most cases are due to modifiable factors (technique, adherence, comorbidities). Severe asthma is the subset remaining uncontrolled despite optimisation — ~3–5% of asthmatics — and is phenotyped for biologics.

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INVESTIGATIONS & TREATMENT AT A GLANCE

6.3 Acute Severe Asthma — Assessment, ICU Management & Mechanical Ventilation

‹ Back to topic (p 118)
Next topic 6.4 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
PEF / FEV1	> 75% best/predicted	33–50% severe; < 33% life-threatening	Severity
SpO_2 / PaO_2	≥ 94%	SpO_2 < 92% / PaO_2 < 60 mmHg	Life-threatening
$PaCO_2$	Low (< 35) in acute asthma	Normal (40) or rising	Impending respiratory failure
Serum potassium	3.5–5.0	< 3.5	High-dose β-agonist effect — replace
Lactate	< 2 mmol/L	> 4	Salbutamol-induced (type B) lactic acidosis
CXR	Normal/hyperinflated	Pneumothorax, pneumonia, pneumomediastinum	Complications

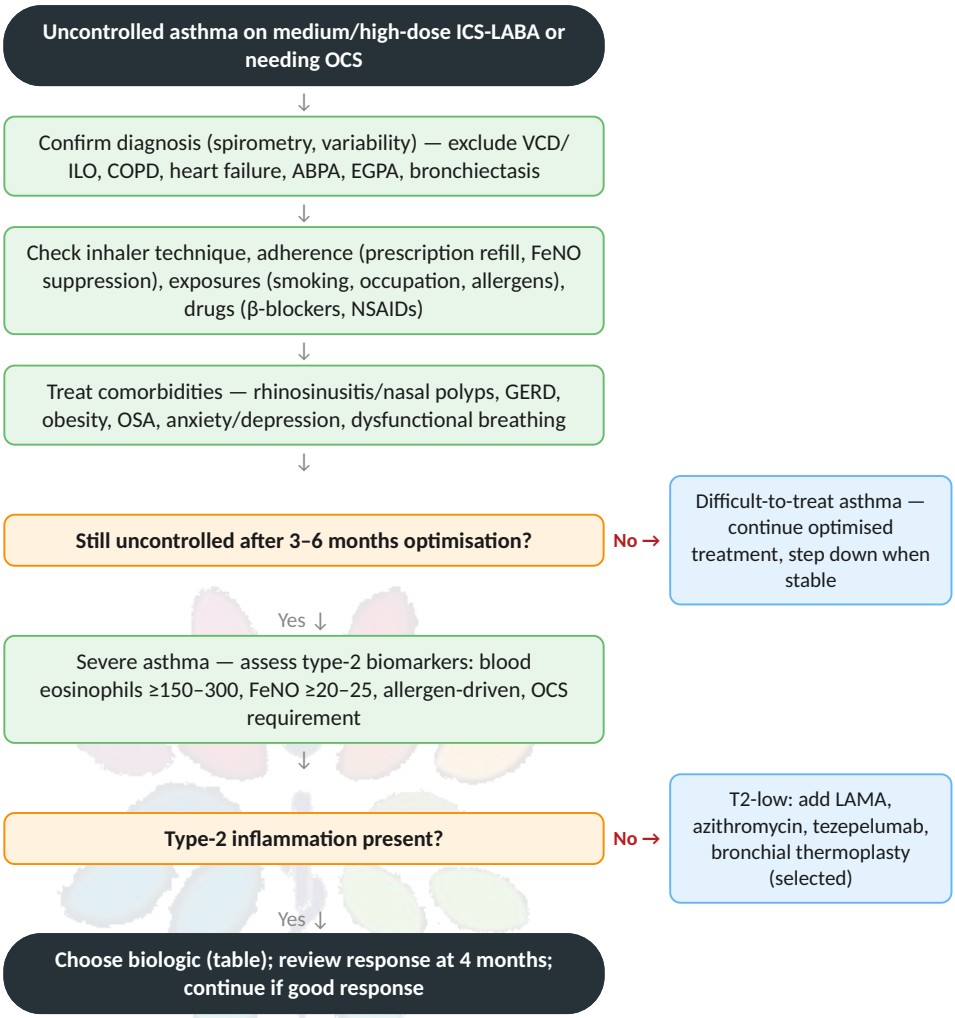
Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Salbutamol nebulised	5 mg every 20 min × 3, then hourly; continuous 10–15 mg/h	Acute severe asthma	Tachycardia, hypokalaemia, lactic acidosis
Ipratropium	0.5 mg nebulised every 20 min × 3, then 4–6-hourly	Severe attack	—
Prednisolone / hydrocortisone	Pred 40–50 mg × 5–7 days; hydrocortisone 100 mg IV 6-hourly	All acute asthma	Hyperglycaemia
Magnesium sulphate	2 g IV over 20 min	Life-threatening / poor response	Hypotension
Ketamine (intubation / refractory)	1–2 mg/kg IV induction	Intubation for asthma	Secretions; bronchodilator properties

★ PRESCRIBING & INTERPRETATION PEARLS

- Ventilating asthma: low RR (8–10), Vt 6–8 mL/kg, long expiratory time, permissive hypercapnia (pH > 7.2).

Algorithm 6.2 · Approach to difficult-to-treat asthma (GINA severe asthma guide)



Phenotyping

- Clinical: age at onset, atopy, nasal polyps (CRSwNP), AERD, obesity, OCS dependence.
- Biomarkers: blood eosinophils, FeNO, total/specific IgE, sputum eosinophils (>3%).
- Phenotype-guided therapy improves outcomes and reduces OCS use and its toxicity.

Biologics for severe asthma

Biologic	Target	Eligibility	Dose	Additional indications
Omalizumab	Free IgE	Allergic (sensitisation to perennial allergen), IgE 30–1500 IU/mL	SC every 2–4 weeks (dose by IgE and weight)	CRSwNP, chronic urticaria
Mepolizumab	IL-5	Eosinophils ≥150–300/µL	100 mg SC 4-weekly	EGPA (300 mg), HES, CRSwNP, COPD (eos ≥300)
Reslizumab	IL-5	Eosinophils ≥400	3 mg/kg IV 4-weekly	—
Benralizumab	IL-5Rα (ADCC depletes eosinophils)	Eosinophils ≥150–300	30 mg SC 4-weekly ×3, then 8-weekly	EGPA (MANDARA)
Dupilumab	IL-4Rα (IL-4 + IL-13)	Eos ≥150 or FeNO ≥25, OCS-dependent	200–300 mg SC 2-weekly	Atopic dermatitis, CRSwNP, COPD (BOREAS/NOTUS); avoid if eos >1500
Tezepelumab	TSLP	Severe asthma regardless of phenotype	210 mg SC 4-weekly	Effective even in T2-low

Anti-IL-5 agents in detail

- IL-5 is the key cytokine for eosinophil maturation, survival and recruitment.
- **Efficacy:** ~50% reduction in exacerbations; OCS-sparing (SIRIUS, ZONDA); improve FEV1 modestly; best in high eosinophils, frequent exacerbations, nasal polyps, adult onset.
- **Safety:** injection reactions, headache, helminth infection (screen/treat strongyloides), rare anaphylaxis; herpes zoster (mepolizumab — vaccinate).
- **Response assessment** at 4 months: exacerbations, OCS dose, symptoms, lung function; switch if poor response.

Bronchial thermoplasty

- Radiofrequency energy to airway smooth muscle via bronchoscope in 3 sessions; modest reduction in exacerbations (AIR2); reserved for selected T2-low severe asthma in centres (not first-line).

✦ RECENT ADVANCES & GUIDELINE UPDATES

▸ GINA 2025: remission on treatment as a goal in severe asthma; depemokimab (twice-yearly anti-IL-5, SWIFT trials 2024) under review.

★ CLINICAL PEARLS & EXAM FOCUS

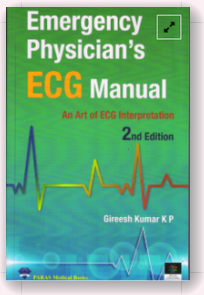
▸ Before calling asthma "severe", check technique, adherence and comorbidities — >50% of referrals turn out to be difficult-to-treat, not severe.

LANDMARK TRIALS & GUIDELINES

- Chung KF et al, ERS/ATS severe asthma guideline 2014 (updated 2020); Menzies-Gow A et al, NAVIGATOR (tezepelumab) NEJM 2021.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. The importance of phenotyping in the management of chronic severe asthma. *[DNB Oct 2023 P2]*
2. Difficult-to-treat asthma. *[BFUHS 2020 P2]*
3. Use of anti-interleukin-5 agents for treatment of severe asthma. *[BFUHS 2023 P2]*
4. Monoclonal antibody in bronchial asthma. *[Manipal 2014 P4]*



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6.5 Allergic Bronchopulmonary Aspergillosis

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B6 Asthma including ABPA
- ✓ Describe the pathogenesis and clinical features of ABPA
- ✓ Apply ISHAM-AWG (2024) diagnostic criteria and clinical staging
- ✓ Treat ABPA with glucocorticoids and antifungals and monitor response

INVESTIGATIONS & TREATMENT AT A GLANCE

6.4 Difficult-to-Treat & Severe Asthma — Phenotyping and Biologics

‹ Back to topic (p 120)
Next topic 6.5 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Blood eosinophils	< 150/µL	≥ 150 (anti-IL-5) / ≥ 300	Biologic selection
FeNO	< 25 ppb	≥ 25 ppb (dupilumab predictor)	Type-2 high
Total IgE	30–1500 IU/mL for omalizumab dosing	—	Omalizumab eligibility
Specific IgE / skin test (perennial allergen)	—	Sensitised	Allergic phenotype
HRCT	—	Bronchiectasis, mucus plugs, ABPA	Comorbidity
Adherence (prescription refill)	> 80%	< 80%	Difficult vs severe asthma

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Omalizumab	75–600 mg SC every 2–4 weeks (IgE/weight table)	Severe allergic asthma	Anaphylaxis (0.1–0.2%)
Mepolizumab	100 mg SC every 4 weeks	Eosinophilic asthma ≥ 150/µL	Herpes zoster (rare)
Benralizumab	30 mg SC every 4 weeks × 3, then every 8 weeks	Eosinophilic asthma	Headache
Dupilumab	600 mg load, then 300 mg SC every 2 weeks	Type-2 asthma, OCS-dependent	Hypereosinophilia, conjunctivitis
Tezepelumab	210 mg SC every 4 weeks	Severe asthma regardless of phenotype	Pharyngitis

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Assess response at 4 months; switch biologic if no meaningful response.

IN BRIEF ABPA is a complex hypersensitivity (types I, III, IV) to Aspergillus fumigatus colonising the airways of asthmatics and CF patients. India has one of the highest burdens — 5–20% of asthmatics in chest clinics. Early diagnosis prevents bronchiectasis and fibrosis.

Pathogenesis

- Inhaled conidia germinate in mucus of susceptible hosts (HLA-DR2/DR5, CFTR heterozygosity, IL-10, SP-A2 polymorphisms) → Th2 response (IL-4, IL-5, IL-13), high IgE, eosinophilia → mucus plugging (eosinophils + fungal hyphae), proximal bronchiectasis, fibrosis.

Clinical and radiological features

- Poorly controlled asthma, expectoration of brownish-black mucus plugs (31–69%), haemoptysis, fever, wheeze; recurrent "pneumonia".
- **CXR:** fleeting opacities, finger-in-glove (mucoid impaction), tram-track, toothpaste shadows, collapse.
- **HRCT:** central (proximal) bronchiectasis (affecting inner two-thirds of lung), mucus plugging, **high-attenuation mucus (HAM — denser than paraspinal muscle; pathognomonic)**, centrilobular nodules, tree-in-bud, mosaic attenuation, fibrosis.

ISHAM-AWG revised diagnostic criteria (2024)

Component	Criteria
Predisposing condition / compatible clinico-radiology	Asthma, CF, bronchiectasis, COPD, or compatible clinical-radiological presentation
Essential (both)	A. fumigatus-specific IgE ≥0.35 kUA/L; serum total IgE ≥500 IU/mL (if <500 but all other criteria met — may still diagnose)
Other (≥2)	A. fumigatus-specific IgG/precipitins; blood eosinophils ≥500/µL (steroid-naive; prior count acceptable); HRCT — bronchiectasis, mucus plugging or HAM (HAM alone confirms in sensitised patient)

- Older Rosenberg–Patterson criteria (8 major) of historical interest; ABPM (allergic bronchopulmonary mycosis) when caused by other fungi (Bipolaris, Schizophyllum).

Clinical staging (ISHAM-AWG/Agarwal)

Stage	Definition
0 — Asymptomatic	Meets criteria, no prior diagnosis, controlled asthma
1 — Acute	1a with mucoid impaction, 1b without
2 — Response	Clinical and radiological improvement, IgE fall ≥25% at 8 weeks
3 — Exacerbation	Worsening with IgE rise ≥50% and/or new radiological changes
4 — Remission	Sustained response ≥6 months off therapy
5a — Treatment-dependent	≥2 exacerbations within 6 months of stopping, or worsening on tapering
5b — Glucocorticoid-dependent asthma	Asthma needs OCS while ABPA is controlled
6 — Advanced	Extensive bronchiectasis, fibrosis, type II respiratory failure, cor pulmonale

Treatment

- **Acute stage (ABPA with symptoms): prednisolone 0.5 mg/kg/day ×2–4 weeks, tapered and stopped over 3–4 months** (Agarwal medium dose regimen equivalent to higher doses with fewer AEs), **or itraconazole 200 mg BD ×4 months** (fewer AEs, slower response; preferred in diabetes/osteoporosis); combination for recurrent exacerbations.
- **Monitoring:** symptoms, CXR, serum IgE every 6–8 weeks (target ≥25–35% fall; new baseline), spirometry.
- **Treatment-dependent:** lowest-dose steroid, itraconazole/voriconazole long term, biologics (omalizumab, mepolizumab, benralizumab, dupilumab — OCS-sparing), nebulised amphotericin (prevents exacerbations).
- **Avoid ICS monotherapy** for ABPA itself; continue asthma care.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Screen every asthmatic in India with A. fumigatus sIgE — ABPA is common and under-diagnosed (often mis-treated as TB).

LANDMARK TRIALS & GUIDELINES

- Agarwal R et al, revised ISHAM-AWG criteria for ABPA/ABPM, Eur Respir J 2024; Agarwal R et al, prednisolone vs itraconazole trials, Eur Respir J 2016/2018.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Criteria for diagnosis and clinical staging of ABPA. *[DNB Oct 2024 P2]*
2. Allergic bronchopulmonary aspergillosis. *[DNB Oct 2023 P2]*
3. Diagnosis and management of allergic bronchopulmonary aspergillosis. *[DNB Jun 2021 P2]*
4. Describe diagnosis and management of allergic bronchopulmonary aspergillosis. *[WBUHS 2020 P2]*
5. Enumerate eosinophilic lung diseases. Discuss pathogenesis, clinical features and management of ABPA. *[VNSGU 2025 P3]*

6.6 Occupational Asthma, Allergen Immunotherapy & Inducible Laryngeal Obstruction

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B6; II-B3 Occupational disease
- ✓ Define and classify occupational asthma and its diagnostic work-up
- ✓ Describe specific allergen immunotherapy (SCIT/SLIT)
- ✓ Recognise vocal cord dysfunction (inducible laryngeal obstruction)

IN BRIEF Work-related asthma comprises occupational asthma (caused by work — sensitiser-induced or irritant-induced) and work-exacerbated asthma. It accounts for 15–20% of adult-onset asthma and is curable if exposure stops early.

Classification of work-related asthma

Type	Mechanism	Agents / features
Sensitiser-induced (immunological, with latency)	HMW (IgE): flour (baker's), latex, animal proteins, enzymes; LMW (IgE/non-IgE): isocyanates, wood dust (plicatic acid — red cedar), acid anhydrides, platinum salts, persulphates (hairdressers)	Latency months–years; specific inhalation challenge confirms
Irritant-induced (RADS)	Single high-level exposure: chlorine, ammonia, smoke	Onset within 24 h; no latency
Low-dose irritant-induced	Repeated moderate exposures	Contentious
Work-exacerbated asthma	Pre-existing asthma worsened by work (dust, cold, exertion)	—

Diagnosis

- History linking symptoms to work (better on weekends/holidays), latency, exposure details (job, MSDS).
- **Serial PEF** ≥4 times/day for 3–4 weeks at work and away — OASYS-2 score; serial spirometry; **non-specific BHR** changes at work vs off-work; SPT/sIgE to HMW agents; **specific inhalation challenge (SIC)** — gold standard; induced sputum eosinophils at work.
- **Newer methods:** FeNO changes, exhaled breath condensate, component-resolved diagnostics, workplace challenges.
- **Treatment: complete removal from exposure** (best outcome if early — <1 year of symptoms), standard asthma therapy, compensation and workplace surveillance; respiratory protective equipment is inferior.

Allergen immunotherapy

- Repeated administration of allergen to induce tolerance (IgG4 blocking antibodies, Tregs, IL-10, ↓ Th2).
- **SCIT** (subcutaneous — build-up and maintenance, 3–5 years) and **SLIT** (sublingual tablets/drops — HDM SLIT tablet recommended by GINA steps 2–4 with FEV1 >70%).

INVESTIGATIONS & TREATMENT AT A GLANCE

6.5 Allergic Bronchopulmonary Aspergillosis

‹ Back to topic (p 122)
Next topic 6.6 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Total IgE	< 500 IU/mL	≥ 500 IU/mL (ISHAM-AWG 2024)	ABPA essential criterion
Aspergillus-specific IgE	< 0.35 kUA/L	≥ 0.35 kUA/L	Sensitisation (essential)
Aspergillus IgG / precipitins	Negative	Positive	Supportive
Eosinophils	< 500/µL	> 500/µL	Supportive
HRCT	—	Central bronchiectasis, high-attenuation mucus (HAM)	HAM = ABPA, predicts relapse
IgE monitoring	—	Fall ≥ 20–25% = response; rise ≥ 50% = exacerbation	Follow-up

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Prednisolone	0.5 mg/kg/day × 2 weeks, taper over 3–4 months	Acute ABPA	Steroid toxicity
Itraconazole	200 mg bd × 4 months	Steroid-sparing / alternative	TDM; heart failure; drug interactions
Omalizumab / dupilumab	Per dosing	Steroid-dependent ABPA	Specialist

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Treat ABPA when symptomatic or IgE high with new infiltrates — not asymptomatic serology alone.

- Indications: allergic rhinitis ± asthma with confirmed sensitisation, controlled asthma; contraindicated in uncontrolled/severe asthma (FEV1 <70%), β-blockers, malignancy, autoimmune disease, pregnancy (initiation).
- Risk: systemic reactions/anaphylaxis (SCIT — observe 30 min, adrenaline ready).

Vocal cord dysfunction / inducible laryngeal obstruction (ILO)

- Paradoxical adduction of vocal cords on inspiration → inspiratory stridor, throat tightness, voice change; triggered by exercise, irritants, stress, GERD; often misdiagnosed as refractory asthma (coexists in 20–30%).
- **Diagnosis:** laryngoscopy during symptoms/provocation (continuous laryngoscopy during exercise — CLE); flow–volume loop truncated inspiratory limb; normal SpO2.
- **Treatment:** speech therapy (breathing techniques), treat triggers (GERD, rhinitis), psychological support; heliox/reassurance acutely; avoid escalating asthma therapy.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Occupational asthma: diagnose with serial PEF and remove early — the longer the exposure after symptoms, the more likely persistent asthma.

LANDMARK TRIALS & GUIDELINES

- Tarlo SM et al, ACCP consensus on work-related asthma, Chest 2008; ERS task force on specific inhalation challenge 2014.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Write a note on various causes, newer diagnostic methods and treatment modalities of occupational asthma. *[DNB May 2024 P3]*
2. Describe the definition and classification of occupational asthma. *[BFUHS 2022 P3]*
3. Vocal cord dysfunction. *[NEET-SS/DNB 2024]*

6.7 COPD — Definition, Pathogenesis, Risk Factors & Assessment

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B6 COPD
- ✓ Define COPD (GOLD 2025) and describe its aetiotypes and pathophysiology
- ✓ Assess COPD by spirometric grade, symptoms and exacerbation history (ABE)
- ✓ Recognise COPD in non-smokers and sex differences

IN BRIEF COPD is a heterogeneous lung condition with chronic respiratory symptoms due to airway and/or alveolar abnormalities causing persistent, often progressive, airflow obstruction (post-bronchodilator FEV1/FVC <0.7). India has ~55 million COPD patients, with biomass smoke a major non-smoking cause.

GOLD aetiotypes

Aetiotype	Description
COPD-G	Genetically determined (α1-ATD)
COPD-D	Abnormal lung development (prematurity, low birth weight)
COPD-C	Cigarette smoking (incl. second-hand, waterpipe, cannabis)
COPD-P	Pollution (biomass, ambient, occupational)
COPD-I	Infection (childhood infections, TB-associated, HIV)
COPD-A	COPD with asthma
COPD-U	Unknown cause

INVESTIGATIONS & TREATMENT AT A GLANCE

6.6 Occupational Asthma, Allergen Immunotherapy & Inducible Laryngeal Obstruction

‹ Back to topic (p 124)
Next topic 6.7 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Serial PEF at work vs away	Variability < 20%	Work-related pattern (OASYS score > 2.5)	Occupational asthma
Specific inhalation challenge	No fall	FEV1 fall ≥ 15%	Gold standard
Specific IgE (HMW agents, e.g. flour)	Negative	Positive	Sensitiser
Laryngoscopy during symptoms	Normal	Paradoxical vocal-cord adduction in inspiration	Inducible laryngeal obstruction
Flow-volume loop (during symptoms)	Normal inspiratory limb	Truncated inspiratory loop	ILO

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Removal from exposure	Early	Sensitiser-induced asthma	Prognosis best if early
Allergen immunotherapy (SCIT/SLIT)	HDM SLIT tablet daily × 3 years	Allergic rhinitis/asthma with HDM	Uncontrolled asthma (FEV1 < 70%) — avoid
Speech & language therapy	Breathing retraining	ILO	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Unlike asthma, ILO causes inspiratory stridor and throat tightness, and SpO_2 stays normal.

Risk factors for COPD in non-smokers

- **Household air pollution** from biomass (wood, dung, crop residue) — women in rural India; ambient PM2.5; occupational dusts, gases and fumes (mining, construction, agriculture).
- Post-TB obstructive disease (TOPD), childhood respiratory infections, chronic asthma, poor lung growth, low socioeconomic status, α1-ATD, second-hand smoke.
- Biomass COPD: more airway-predominant (small airway disease, bronchial anthracofibrosis), less emphysema, more PH.

Pathophysiology

- Chronic inflammation (neutrophils, macrophages, CD8 T cells, IL-8, LTB4, TNF), oxidative stress, protease imbalance, accelerated ageing → small airway disease (loss of terminal bronchioles — up to 40% before spirometric COPD), emphysema → airflow limitation, air trapping, hyperinflation, gas exchange abnormalities, mucus hypersecretion, PH, systemic effects.

Spirometric grading (post-bronchodilator FEV1/FVC <0.70)

GOLD grade	FEV1 % predicted
1 — Mild	≥80%
2 — Moderate	50–79%
3 — Severe	30–49%
4 — Very severe	<30%

Combined assessment — GOLD ABE

Group	Exacerbation history (past year)	Symptoms
A	0–1 moderate (no hospitalisation)	mMRC 0–1, CAT <10
B	0–1 moderate	mMRC ≥2, CAT ≥10
E	≥2 moderate or ≥1 leading to hospitalisation	Any symptoms

- Other assessments: blood eosinophils, comorbidities, CT (emphysema, bronchiectasis, lung cancer screening), α1-AT level (once in all), BODE index (BMI, Obstruction, Dyspnoea, Exercise — prognosis).

Sex differences in COPD

- Women: greater susceptibility to smoke and biomass per pack-year, more airway-predominant disease, smaller airways, more dyspnoea, anxiety/depression, osteoporosis; more underdiagnosed; higher rates of exacerbation hospitalisation; men: more emphysema, cardiovascular comorbidity. Rising female COPD incidence.

Pre-COPD and PRISm

- Pre-COPD: symptoms/structural/physiological abnormalities with FEV1/FVC ≥0.7 — may progress. PRISm: preserved ratio impaired spirometry (see 2.2).

✦ RECENT ADVANCES & GUIDELINE UPDATES

▸ GOLD 2025: PRISm and young COPD recognised; blood eosinophils guide ICS; dupilumab and ensifentrine added; CT for selected patients.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Group E replaces old C and D — exacerbation history is the strongest predictor of future exacerbations.

LANDMARK TRIALS & GUIDELINES

- GOLD 2025 report; Salvi S et al, India burden (Lancet Glob Health 2018 — GBD India respiratory diseases).

INVESTIGATIONS & TREATMENT AT A GLANCE

6.7 COPD — Definition, Pathogenesis, Risk Factors & Assessment

‹ Back to topic (p 125)
Next topic 6.8 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Post-BD FEV1/FVC	≥ 0.70 (> LLN)	< 0.70	COPD (GOLD)
FEV1 % predicted (GOLD grade)	≥ 80% GOLD 1	50–79% GOLD 2; 30–49% GOLD 3; < 30% GOLD 4	Severity
mMRC / CAT	mMRC 0–1; CAT < 10	mMRC ≥ 2; CAT ≥ 10	Symptom burden (GOLD A/B/E)
Blood eosinophils	< 100/µL	≥ 300/µL	ICS benefit
α1-antitrypsin	1.0–2.0 g/L	< 0.57 g/L	AATD — screen all COPD once (WHO)
SpO_2 / ABG	SpO_2 > 92%	PaO_2 ≤ 55 mmHg	LTOT need
CT chest	—	Emphysema, bronchiectasis, nodules	Phenotype, cancer screen

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Smoking cessation	Varenicline 0.5 mg → 1 mg bd × 12 weeks	All smokers	—
Biomass exposure reduction	Clean fuel (LPG)	Rural Indian women	—
Vaccines	Influenza, pneumococcal, RSV, Tdap	All COPD	—

★ **PRESCRIBING & INTERPRETATION PEARLS**

- GOLD 2025 groups: A (low symptoms, no exacerbation), B (high symptoms), E (≥ 2 moderate or ≥ 1 hospitalised exacerbation).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Assessment of COPD severity. *[DNB Dec 2022 P2]*
2. Risk factors for COPD in non-smokers. *[BFUHS 2019 P3]*
3. Differences between men and women with COPD. *[DNB Oct 2023 P4]*

6.8 Pharmacotherapy of Stable COPD

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B6 COPD management
- ✓ Initiate pharmacotherapy by GOLD group and follow up by dominant trait (dyspnoea vs exacerbations)
- ✓ Use blood eosinophils to guide ICS and recognise ICS adverse effects
- ✓ Describe triple therapy, PDE4 inhibitors, macrolides and biologics in COPD

IN BRIEF Long-acting bronchodilators are the foundation; dual LABA+LAMA is preferred for most symptomatic patients. ICS is added only for exacerbation-prone patients with blood eosinophils ≥100–300 cells/µL, and single-inhaler triple therapy reduces exacerbations and mortality in selected patients.

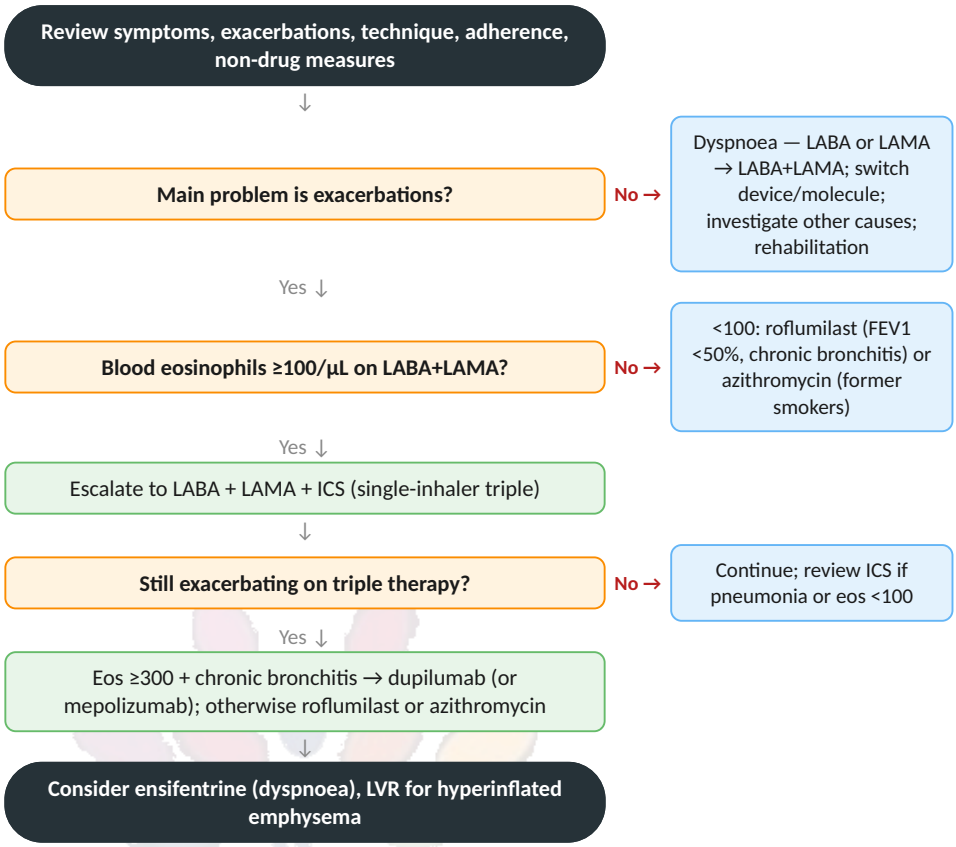
Initial pharmacological treatment (GOLD 2025)

Group	Initial therapy
A	A bronchodilator (long-acting preferred unless very occasional symptoms)
B	LABA + LAMA (single inhaler preferred)
E	LABA + LAMA; consider LABA + LAMA + ICS if blood eosinophils ≥300

- **ICS + LABA not encouraged** in COPD; if asthma features → ICS-containing regimen.

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Algorithm 6.3 · Follow-up pharmacotherapy (GOLD 2025)



Significance of blood eosinophils

- Continuous relationship: <100/µL — little ICS benefit; 100–300 — consider; ≥300 — ICS most likely beneficial.
- Higher eosinophils predict exacerbation risk, ICS response, biologic (dupilumab, mepolizumab) response; low eosinophils + ICS → more pneumonia. Also predicts response to oral steroids in exacerbations (eosinophil-guided prednisolone — STARR2).

Adverse effects of ICS in COPD

- **Pneumonia** (fluticasone > budesonide; risk factors: current smoking, age ≥55, prior exacerbations/pneumonia, BMI <25, low eosinophils, severe FEV1), oral candidiasis, dysphonia, skin bruising, diabetes control, cataract, fractures/osteoporosis, mycobacterial (TB, NTM) infection.
- **ICS withdrawal** reasonable if pneumonia, inappropriate indication, or eos <100 (risk of exacerbation if eos ≥300).

Triple therapy

- Single-inhaler triple (FF/UMEC/VI, BUD/GLY/FORM, BDP/FF/G): **IMPACT** and **ETHOS** — reduced moderate/severe exacerbations and all-cause mortality vs dual bronchodilator, in symptomatic patients with exacerbation history.

Other agents

- **Roflumilast** (PDE4 inhibitor, 500 µg OD): FEV1 <50% + chronic bronchitis + exacerbations; side effects — diarrhoea, weight loss, insomnia.
- **Azithromycin** 250 mg daily/500 mg thrice weekly: reduces exacerbations (more in former smokers).
- **Mucolytics/antioxidants:** N-acetylcysteine (high dose 600 mg BD), carbocisteine, erdosteine — reduce exacerbations in non-ICS patients.
- **Dupilumab** (BOREAS, NOTUS): eosinophils ≥300 with chronic bronchitis — 30% fewer exacerbations, better FEV1. **Mepolizumab** (MATINEE 2025) eos ≥300.
- **Ensifentrine** (PDE3/4 inhibitor, nebulised) — bronchodilator and anti-inflammatory (ENHANCE trials).

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- Vaccines (influenza, pneumococcal, RSV, Tdap, zoster, COVID-19), smoking cessation, rehabilitation, oxygen when indicated.

Emerging pharmacotherapies

- Biologics (anti-IL-33 — itepekimab, tozorakimab; anti-TSLP — tezepelumab), anti-inflammatory PDE4B inhibitors, CFTR potentiators for mucus, α1-AT augmentation, stem cell therapy (experimental).

★ CLINICAL PEARLS & EXAM FOCUS

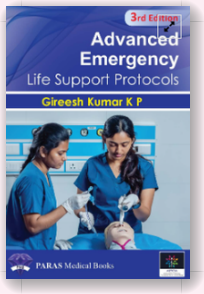
▸ Triple therapy is for exacerbators with eosinophils ≥100 — not for every COPD patient.

LANDMARK TRIALS & GUIDELINES

- Lipson DA et al, IMPACT NEJM 2018; Rabe KF et al, ETHOS NEJM 2020; Bhatt SP et al, BOREAS NEJM 2023 and NOTUS NEJM 2024.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Algorithm for the initiation of pharmacological management of COPD. *[DNB Jun 2025 P2]*
2. Adverse effects of inhaled corticosteroids in COPD. *[DNB Jun 2025 P2]*
3. Significance of blood eosinophil in COPD management. *[DNB Jun 2025 P4]*
4. Role of inhaled corticosteroids for treating patients having COPD. *[BFUHS 2022 P2]*
5. Inhaled corticosteroids for patients with stable COPD. *[BFUHS 2019 P2]*
6. Current status of fixed triple drug inhaler combinations in obstructive pulmonary disease. *[BFUHS 2020 P4]*
7. Pharmacological agents useful in preventing COPD exacerbations. *[BFUHS 2020 P3]*
8. Emerging pharmacotherapies for COPD. *[Manipal 2014 P4]*



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6.9 Acute Exacerbation of COPD & Role of NIV

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B6; II-B10 Respiratory failure in obstructive airway disease
- ✓ Define and grade AECOPD (Rome proposal) and identify causes
- ✓ Manage AECOPD with bronchodilators, steroids, antibiotics and controlled oxygen
- ✓ Indicate and apply NIV in AECOPD

IN BRIEF An exacerbation is an event with worsening dyspnoea and/or cough and sputum over ≤14 days, often with tachypnoea/tachycardia, caused by airway infection, pollution or other insult. NIV in hypercapnic acidotic exacerbation halves mortality and intubation.

Rome severity classification (GOLD 2023+)

Severity	Criteria
Mild	VAS dyspnoea <5, RR <24, HR <95, SpO2 ≥92% (resting) and change ≤3%, CRP <10
Moderate	≥3 of: VAS ≥5, RR ≥24, HR ≥95, SpO2 <92% or change >3%, CRP ≥10; ABG may show hypoxaemia/hypercapnia without acidosis

INVESTIGATIONS & TREATMENT AT A GLANCE

6.8 Pharmacotherapy of Stable COPD

‹ Back to topic (p 127)
Next topic 6.9 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Exacerbation history	0–1 moderate	≥ 2 moderate or ≥ 1 hospitalised	Group E — escalate
Blood eosinophils	< 100	≥ 300	Add ICS in E / escalation
Inhaler technique	Correct	Errors	Fix before escalation
QTc / hearing (azithromycin)	Normal	Prolonged / loss	Avoid azithro

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
LAMA (tiotropium)	18 µg daily (DPI) / 2.5 µg × 2 SMI	GOLD A (or LABA)	Urinary retention
LABA + LAMA	Indacaterol/glycopyrronium 110/50 µg daily	GOLD B, E	Tachycardia
LABA + LAMA + ICS (single inhaler)	Fluticasone furoate/ umeclidinium/vilanterol 100/62.5/25 µg daily	E with eos ≥ 300	Pneumonia
Roflumilast	250 µg × 4 weeks then 500 µg daily	FEV1 < 50%, chronic bronchitis, exacerbations	Weight loss, diarrhoea
Azithromycin	250 mg daily	Exacerbations in former smokers	QTc, hearing
Dupilumab / ensifentrine	Dupilumab 300 mg SC 2-weekly; ensifentrine 3 mg nebulised bd	COPD with eos ≥ 300 on triple / add-on bronchodilator	Specialist

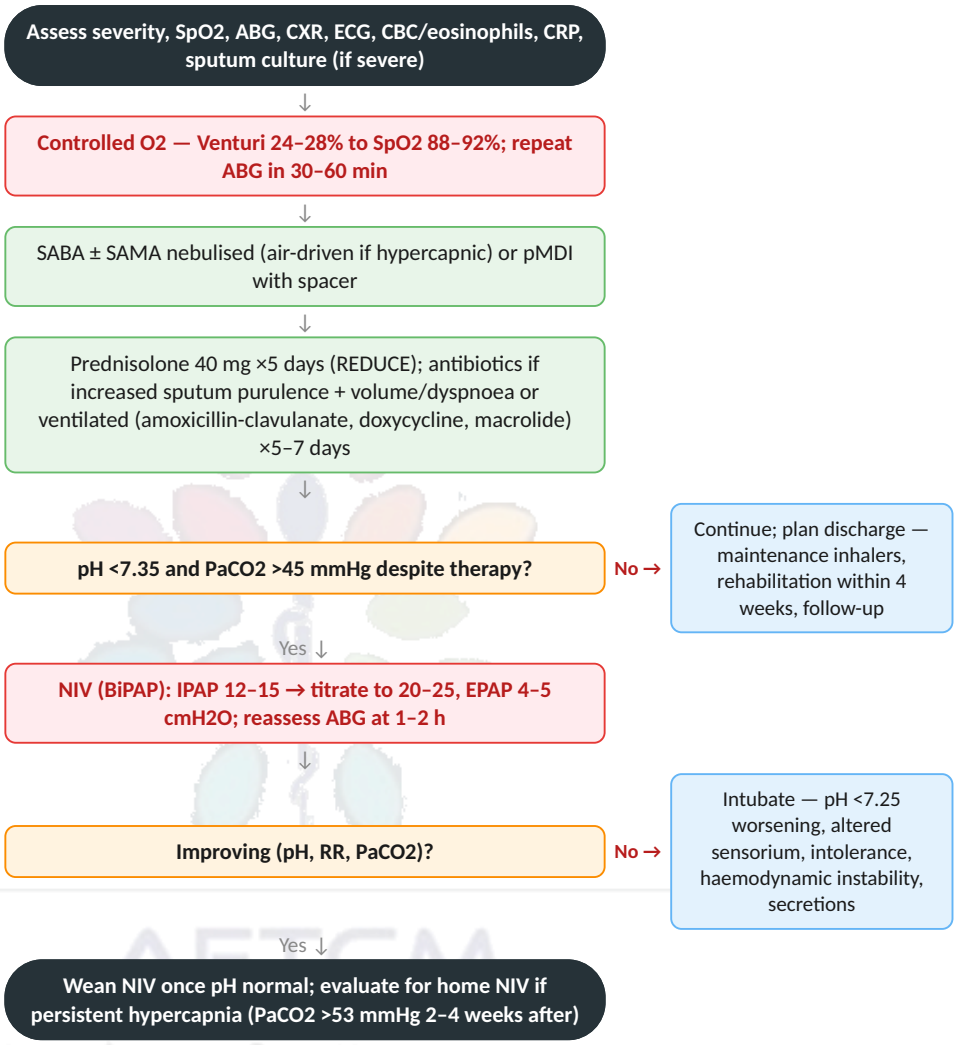
★ **PRESCRIBING & INTERPRETATION PEARLS**

- Withdraw ICS if repeated pneumonia or eosinophils < 100/µL.

Severity	Criteria
Severe	Hypercapnia and acidosis (PaCO2 >45, pH <7.35)

- **Causes:** viral (rhinovirus, influenza, RSV) and bacterial (H. influenzae, S. pneumoniae, M. catarrhalis, Pseudomonas in severe), air pollution, non-adherence; **mimics** — PE, pneumonia, pneumothorax, HF, arrhythmia, ACS.

Algorithm 6.4 · Management of AECOPD



NIV in AECOPD — evidence and indications

- **Indications:** respiratory acidosis pH ≤7.35 with PaCO2 ≥45 mmHg; severe dyspnoea with fatigue/increased work of breathing; persistent hypoxaemia despite O2.
- **Benefits:** reduces intubation (~65%), mortality (~46%), hospital stay, VAP; best for pH 7.25–7.35; trial possible down to 7.15 in ICU with readiness to intubate.
- **Contraindications:** cardiac/respiratory arrest, inability to protect airway, copious secretions, severe agitation, facial trauma/surgery, upper GI bleed, recent upper airway/oesophageal surgery, undrained pneumothorax, haemodynamic instability.
- **Home NIV** after hospitalisation with persistent hypercapnia (HOT-HMV — reduced readmission/death); high-intensity NIV in stable hypercapnic COPD (Köhnlein 2014).

Prevention of readmission

- Discharge bundle: inhaler technique, optimised maintenance therapy, rehabilitation, smoking cessation, vaccination, action plan, follow-up within 1 month, treat comorbidities.

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★ **CLINICAL PEARLS & EXAM FOCUS**

▸ Five days of prednisolone is enough (REDUCE) — and eosinophil-guided steroids can avoid them altogether when eos <2%.

LANDMARK TRIALS & GUIDELINES

- Leuppi JD et al, REDUCE JAMA 2013; Murphy PB et al, HOT-HMV JAMA 2017; BTS/ICS guideline on acute hypercapnic respiratory failure 2016; ERS/ATS NIV guideline 2017.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Management of acute exacerbation of COPD. *[BFUHS 2020 P2]*
2. Role of NIV in acute exacerbation of COPD. *[DNB Oct 2024 P3]*
3. Non-invasive mechanical ventilation indications for patient of acute exacerbation of COPD. *[DNB Dec 2021 P3]*

6.10 Systemic Manifestations, Comorbidities, Hyperinflation & Cor Pulmonale

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B6; II-B5 Cor pulmonale
- ✓ Describe systemic manifestations and complications of COPD
- ✓ Explain the clinical significance of static and dynamic hyperinflation
- ✓ Describe aetiopathogenesis, features and complications of cor pulmonale

IN BRIEF COPD is a systemic disease: systemic inflammation, hypoxaemia, inactivity and steroids produce skeletal muscle dysfunction, cachexia, osteoporosis, cardiovascular disease, anxiety and depression. Hyperinflation, not FEV1, best explains exertional dyspnoea.

Systemic manifestations and comorbidities of COPD

System	Manifestation
Musculoskeletal	Skeletal muscle dysfunction (type I fibre loss, mitochondrial dysfunction), sarcopenia, cachexia (FFMI <15/16), osteoporosis (fragility fractures)
Cardiovascular	IHD, HF, arrhythmias (AF, MAT), hypertension, PH/cor pulmonale
Metabolic	Diabetes, metabolic syndrome, anaemia (or polycythaemia)
Neuropsychiatric	Anxiety, depression, cognitive impairment
Others	Lung cancer (2–5× risk), GERD, OSA (overlap), bronchiectasis, CKD

Complications of COPD

Category	Complications
Acute	Exacerbations, pneumonia, pneumothorax (bullae), acute-on-chronic respiratory failure, arrhythmias, PE
Chronic	Chronic hypercapnic failure, polycythaemia, PH and cor pulmonale, lung cancer, malnutrition, frailty, depression

Osteoporosis and skeletal muscle dysfunction

- Prevalence of osteoporosis 30–60%; risk from inactivity, low BMI, vitamin D deficiency, systemic inflammation, smoking, OCS/ICS. Screen with DXA, FRAX; treat with calcium/vitamin D, bisphosphonates, minimise OCS.
- Muscle dysfunction (quadriceps weakness in 30%) predicts mortality independent of FEV1; best treated with pulmonary rehabilitation (resistance + endurance), nutritional supplementation, NMES in severe.

INVESTIGATIONS & TREATMENT AT A GLANCE

6.9 Acute Exacerbation of COPD & Role of NIV

‹ Back to topic (p 129)
Next topic 6.10 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
ABG	pH 7.35–7.45	pH < 7.35 with $PaCO_2$ > 45	Indication for NIV
ABG after 1–2 h NIV	pH improving	No improvement	Consider intubation
CXR	—	Pneumonia, pneumothorax, oedema	Alternative diagnoses
Sputum culture	—	Pathogen (if purulent)	Antibiotic choice
ECG / troponin / BNP	Normal	Arrhythmia, MI, heart failure	Mimics/comorbidities
CRP	< 10 mg/L	> 40 mg/L	Supports antibiotics

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Salbutamol + ipratropium	2.5–5 mg + 0.5 mg nebulised 4–6-hourly	Exacerbation	Drive with air if hypercapnic
Prednisolone	40 mg daily × 5 days (REDUCE)	Exacerbation	Hyperglycaemia
Antibiotics	Amoxicillin–clavulanate 625 mg tds or doxycycline 100 mg bd × 5 days	Increased sputum purulence	—
Controlled oxygen	Target SpO_2 88–92% (Venturi 24–28%)	Hypoxaemia	Over-oxygenation → hypercapnia
NIV (BiPAP)	IPAP 15–20 (up to 25), EPAP 4–5 cmH_2O	pH < 7.35 and $PaCO_2$ > 45	Contraindications: vomiting, unable to protect airway

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Do not discontinue NIV abruptly — wean over 24–48 h once pH normal.

Hyperinflation

- **Static:** loss of elastic recoil raises FRC/RV (RV/TLC >40%). **Dynamic:** expiratory time insufficient (tachypnoea of exercise/exacerbation) → end-expiratory lung volume rises (fall in IC measured during exercise), intrinsic PEEP.
- **Consequences:** inspiratory muscles at mechanical disadvantage (flattened diaphragm, loss of zone of apposition), increased elastic load and intrinsic PEEP threshold load, reduced IC → limits VT expansion (neuromechanical dissociation, dyspnoea), cardiac effects (reduced venous return, small heart), exercise limitation.
- **Clinical markers:** IC/TLC <25% predicts mortality; lung-volume reduction with bronchodilators ("deflation") improves dyspnoea even with small FEV1 change.

Cor pulmonale

- RV hypertrophy/dilatation and failure due to PH from lung disease (group 3), excluding left heart and congenital causes.
- **Aetiopathogenesis:** alveolar hypoxia → HPV and vascular remodelling, hypercapnia/acidosis, polycythaemia (viscosity), loss of vascular bed (emphysema), inflammation; causes — COPD (commonest), ILD, OSA/OHS, kyphoscoliosis, NM disease, post-TB, high altitude.
- **Clinical:** dyspnoea, fatigue, raised JVP (prominent v waves of TR), parasternal heave, loud P2, TR murmur, hepatomegaly, ascites, oedema (also from renal Na retention due to hypercapnia), cyanosis.
- **Investigations:** ECG (RVH), CXR (enlarged PA), echo (TAPSE, RVSP), NT-proBNP, RHC if needed.
- **Complications:** RV failure, arrhythmias, sudden death, hepatic congestion, polycythaemia/thrombosis.
- **Management:** LTOT (only proven survival benefit), treat underlying disease, diuretics carefully (avoid alkalosis), NIV for hypercapnia, venesection if Hct >55%; PAH drugs not recommended in COPD-PH (except severe PH in specialised centres; inhaled treprostinil in ILD-PH).

★ CLINICAL PEARLS & EXAM FOCUS

▸ Hyperinflation — not FEV1 — explains why bronchodilators reduce dyspnoea; measure IC.

LANDMARK TRIALS & GUIDELINES

- Agustí A et al, ECLIPSE cohort on systemic inflammation (PLoS One 2012); Casanova C et al, IC/TLC prognosis AJRCCM 2005.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Systemic manifestations of COPD. *[DNB Jun 2025 P2]*
2. Clinical significance of hyperinflation in COPD. *[DNB Jun 2025 P2]*
3. Osteoporosis and skeletal muscle dysfunction in COPD. *[DNB Oct 2023 P2]*
4. Complications of COPD. *[DNB Oct 2023 P4]*
5. Aetiopathogenesis, clinical features and complications of cor pulmonale. *[DNB Oct 2023 P2]*

6.11 Asthma–COPD Overlap & Small Airway Diseases (Bronchiolitis)

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ **NMC syllabus area:** II-B6 Diseases of small airways
✓ Recognise asthma–COPD overlap and its management principles
✓ Classify bronchiolar diseases and describe constrictive and diffuse panbronchiolitis

IN BRIEF Asthma and COPD can coexist; GINA/GOLD advise treating such patients as asthma (ICS-containing therapy, no LABA alone). Bronchiolitis — inflammation and/or fibrosis of airways <2 mm — has cellular, constrictive and panbronchiolitic forms.

INVESTIGATIONS & TREATMENT AT A GLANCE

6.10 Systemic Manifestations, Comorbidities, Hyperinflation & Cor Pulmonale

‹ Back to topic (p 131)
Next topic 6.11 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Echocardiography	PASP < 35 mmHg	TR velocity > 2.8 m/s; RV dilation	Pulmonary hypertension/cor pulmonale
Haematocrit	< 52% (men), < 48% (women)	≥ 55%	Secondary polycythaemia
BMI / FFMI	BMI 20–25; FFMI > 15 (women)/17 (men)	BMI < 21; low FFMI	Cachexia — poor prognosis
DEXA	T > −1	≤ −2.5	Osteoporosis
PHQ-9 / GAD-7	< 5	≥ 10	Depression/anxiety
BODE index	0–2	7–10	Mortality prediction

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
LTOT	≥ 15 h/day, SpO_2 90–92%	PaO_2 ≤ 55 or 56–59 with cor pulmonale/Hct > 55%	Smoking near oxygen
Furosemide	20–40 mg daily	Peripheral oedema of cor pulmonale	Hypokalaemia, alkalosis
Venesection	300–500 mL to Hct < 55%	Symptomatic polycythaemia	Only after LTOT
Alendronate + calcium/ vitamin D	70 mg weekly	Osteoporosis	Oesophagitis
Nutritional supplementation	Oral supplements	Malnourished COPD	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Cardioselective β-blockers are safe and beneficial in COPD with cardiac disease.

Asthma–COPD overlap

- Persistent airflow limitation with features of both: history of asthma/atopy or high BDR, eosinophilia/FeNO, in a smoker or biomass-exposed patient aged >40.
- Higher exacerbation risk, poorer quality of life. **Treatment:** ICS-containing regimen (ICS-LABA ± LAMA), never LABA/LAMA without ICS; treat as asthma for safety plus COPD measures (rehab, smoking cessation).

Classification of small airway (bronchiolar) disease

Type	Causes	Imaging
Cellular (infectious)	Viral (RSV, adenovirus), Mycoplasma, TB, NTM, aspiration	Centrilobular nodules, tree-in-bud
Respiratory bronchiolitis	Smoking (RB-ILD)	Centrilobular GGO, upper lobes
Follicular bronchiolitis	RA, Sjögren, CVID, HIV	Centrilobular nodules, cysts
Hypersensitivity / eosinophilic	HP, asthma	Centrilobular GGO, air trapping
Constrictive (obliterative)	Post-transplant (BOS), post-infectious (Swyer-James), toxic fumes (NO2 — silo filler's, diacetyl — popcorn lung), RA, drugs (penicillamine), Sauropus androgynus	Mosaic attenuation, expiratory air trapping, normal inspiratory CT
Diffuse panbronchiolitis	East Asian; HLA-B54; chronic sinusitis	Diffuse centrilobular nodules, bronchiectasis
DIPNECH	Neuroendocrine cell hyperplasia, women	Nodules, mosaic attenuation

- **Diffuse panbronchiolitis:** responds dramatically to low-dose erythromycin/macrolides — survival improved from 63% to >90%.
- **Bronchiolitis obliterans syndrome** (post-lung/HSCT): FEV1 decline ≥20%; azithromycin, FAM, ruxolitinib (GVHD), re-transplant.

★ **CLINICAL PEARLS & EXAM FOCUS**

▸ Normal inspiratory CT with mosaic attenuation on expiration = constrictive bronchiolitis.

LANDMARK TRIALS & GUIDELINES

- GINA/GOLD joint chapter on asthma–COPD overlap (2017, updated in GINA 2025).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Asthma–COPD overlap syndrome. *[DNB Oct 2024 P3]*

6.12 Oxygen Therapy, LTOT, Oxygen Toxicity & Hyperbaric Oxygen

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B6 Long-term oxygen therapy; Misc. Oxygen therapy
- ✓ State indications and prescription of LTOT, nocturnal and ambulatory oxygen
- ✓ Describe pulmonary oxygen toxicity and its prevention
- ✓ Describe indications of hyperbaric oxygen therapy

IN BRIEF LTOT (≥15 h/day) improves survival in COPD with severe resting hypoxaemia (NOTT, MRC). Oxygen is a drug — hyperoxia causes absorption atelectasis, hypercapnia in at-risk patients and oxidative lung injury; prescribe to target saturations.

INVESTIGATIONS & TREATMENT AT A GLANCE

6.11 Asthma–COPD Overlap & Small Airway Diseases (Bronchiolitis)

‹ Back to topic (p 132)
Next topic 6.12 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Post-BD FEV1/FVC	—	< 0.70 with BDR ≥ 400 mL	Asthma–COPD overlap features
Blood eosinophils / FeNO	Low	≥ 300 / > 25–50 ppb	ICS indication
Expiratory HRCT	No air trapping	Mosaic attenuation, air trapping	Bronchiolitis
Spirometry in bronchiolitis obliterans	Normal	Fixed obstruction (FEV1 ↓ ≥ 20% post-transplant)	BOS
Lung biopsy	—	Constrictive / follicular / respiratory bronchiolitis	Subtype

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
ICS–LABA	Budesonide/formoterol 200/6 2 puffs bd	ACO	Thrush
LAMA add-on	Tiotropium	ACO, persistent obstruction	—
Azithromycin	250 mg TIW	BOS after transplant/HSCT; DPB	QTc
FAM (fluticasone, azithromycin, montelukast)	Standard doses	Post-HSCT BOS	—
Smoking cessation	—	Respiratory bronchiolitis	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Diffuse panbronchiolitis responds dramatically to low-dose macrolides.

Indications for LTOT (GOLD/BTS)

Condition	Criteria (stable, on optimal treatment, measured twice ≥3 weeks apart)
Severe resting hypoxaemia	PaO2 ≤55 mmHg (7.3 kPa) or SaO2 ≤88%
Moderate hypoxaemia with complications	PaO2 56–59 mmHg (7.3–8 kPa) with PH/cor pulmonale, peripheral oedema, or polycythaemia (Hct >55%)

- **Prescription:** flow to achieve PaO2 ≥60 mmHg / SpO2 ≥90% at rest; ≥15 h/day (NOTT: continuous better than nocturnal 12 h; MRC: 15 h better than none); add 1 L/min during sleep and exercise; concentrator for home; smoking is a contraindication (fire risk).
- **Nocturnal oxygen:** isolated nocturnal desaturation in COPD — no mortality benefit (INOX trial); treat OSA/OHS with PAP rather than O2.
- **Ambulatory oxygen:** LOTT — no benefit in moderate resting or exertional desaturation; may be used for symptoms if demonstrable improvement in exercise capacity/dyspnoea (ILD — AmbOx showed QoL benefit).

Oxygen toxicity

- **Mechanism:** reactive oxygen species (superoxide, hydrogen peroxide, hydroxyl radical) overwhelm antioxidants (SOD, catalase, glutathione) → endothelial and type I cell injury → exudative phase (oedema, haemorrhage, hyaline membranes) then proliferative fibrosis.
- **Pulmonary (Lorrain-Smith effect):** FiO2 >0.6 for >24–48 h → tracheobronchitis, reduced VC, ARDS-like injury, fibrosis; absorption atelectasis (nitrogen washout); hypercapnia in COPD; bronchopulmonary dysplasia and retinopathy of prematurity in neonates.
- **CNS (Paul Bert effect):** hyperbaric O2 >2 ATA — seizures.
- **Other:** bleomycin-treated patients sensitive to high FiO2; paraquat poisoning (avoid O2 unless hypoxaemic).
- **Prevention:** lowest FiO2 to achieve target (SpO2 92–96%, 88–92% in hypercapnic risk); avoid hyperoxia in ICU (ICU-ROX, HOT-ICU — conservative targets acceptable); PEEP to reduce FiO2 needs.

Hyperbaric oxygen therapy

- 100% O2 at 2–3 ATA → dissolved O2 increases to ~6 mL/dL; reduces bubble size (Boyle), enhances wound healing, kills anaerobes.
- **Indications (UHMS):** decompression sickness, arterial gas embolism, severe CO poisoning (LOC, pregnancy, COHb >25%), gas gangrene, necrotising soft-tissue infections, radiation necrosis, crush injury, refractory osteomyelitis, diabetic foot ulcers, sudden sensorineural hearing loss, intracranial abscess.
- **Complications:** barotrauma (ear, sinus, pneumothorax — untreated pneumothorax is absolute contraindication), O2 toxicity seizures, myopia, claustrophobia.

★ CLINICAL PEARLS & EXAM FOCUS

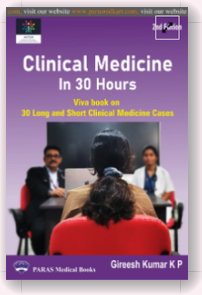
▸ LTOT saves lives only in severe resting hypoxaemia — it does not help nocturnal-only or moderate exertional desaturation.

LANDMARK TRIALS & GUIDELINES

- NOTT (Ann Intern Med 1980); MRC Working Party (Lancet 1981); LOTT (NEJM 2016); INOX (NEJM 2020).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Nocturnal and long-term oxygen therapy. *[DNB Oct 2023 P2]*
2. Describe oxygen therapy and mention oxygen toxicity. *[WBUHS 2020 P2]*
3. Oxygen toxicity. *[DNB Dec 2025 P4]*
4. Pulmonary oxygen toxicity. *[DNB Jun 2021 P3]*
5. Hyperbaric oxygen therapy. *[BFUHS 2019 P3]*



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6.13 Pulmonary Rehabilitation & Airway Clearance Techniques

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B6 Pulmonary rehabilitation; Preventive pulmonology
- ✓ Describe components, indications and evidence for pulmonary rehabilitation
- ✓ Prescribe exercise training and airway clearance techniques

IN BRIEF Pulmonary rehabilitation is a comprehensive, individualised intervention — exercise training, education and behaviour change — that improves dyspnoea, exercise capacity and quality of life in chronic respiratory disease, and reduces readmissions when started within 4 weeks of an exacerbation.

Components of pulmonary rehabilitation

Component	Details
Patient assessment	6MWT/shuttle walk, mMRC/CAT, SGRQ/CRQ, muscle strength, nutrition, anxiety/depression
Exercise training	Endurance (cycling/walking 60–80% peak work rate, 20–60 min, 3–5×/week, 6–12 weeks), interval training, resistance (upper and lower limb), inspiratory muscle training (selected)
Education & self-management	Disease knowledge, inhaler technique, action plans, energy conservation, breathing control (pursed-lip, diaphragmatic)
Psychosocial	Anxiety, depression, CBT, support groups
Nutrition	Supplementation for low BMI/FFMI, weight loss for obese
Others	Oxygen during exercise (if desaturating), NIV-assisted exercise in severe, NMES, smoking cessation, vocational counselling

Indications and evidence

- COPD (all symptomatic; GOLD B–E; post-exacerbation), ILD, bronchiectasis, PH (stable), post-COVID, pre/post lung surgery and transplant, NM disease, asthma.
- **Evidence (COPD):** Cochrane — large, clinically important improvements in dyspnoea, exercise capacity (6MWD +44 m), QoL (CRQ above MCID); post-exacerbation rehab reduces readmissions and mortality (Puhan); benefits decline over 6–12 months — maintenance needed.
- **Home-based/tele-rehabilitation** effective (equivalent outcomes); yoga-based rehab studied in India.
- **Barriers:** access, transport, awareness, reimbursement.

Airway clearance techniques

Airway clearance

Technique	Description
ACBT (active cycle of breathing)	Breathing control, thoracic expansion, forced expiration technique (huff)

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INVESTIGATIONS & TREATMENT AT A GLANCE

6.12 Oxygen Therapy, LTOT, Oxygen Toxicity & Hyperbaric Oxygen

‹ Back to topic (p 133)
Next topic 6.13 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
PaO_2 (stable, at rest)	> 60 mmHg	≤ 55 mmHg (or 56–59 with cor pulmonale/Hct > 55%)	LTOT criterion
SpO_2 on exertion	≥ 90%	< 88%	Ambulatory O_2
Nocturnal oximetry	< 30% time below 90%	> 30% time < 90%	Nocturnal O_2 / sleep study
$PaCO_2$ on O_2 titration	Rise < 7.5 mmHg	Rise > 7.5 mmHg	Risk of hypercapnia
COHb (CO poisoning)	< 3%	> 25%	Hyperbaric indication

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
LTOT	1–3 L/min ≥ 15 h/day to SpO_2 90–92%	Chronic hypoxaemia	Fire risk with smoking
Venturi mask	24–28% (4–6 L/min)	Hypercapnic COPD	—
HBOT	2.4–3.0 ATA, 90 min	CO poisoning, decompression sickness, gas embolism, necrotising infection	Barotrauma, seizures
Avoid hyperoxia	Target SpO_2 94–98% (88–92% at risk)	All acute patients	Oxygen toxicity (FiO_2 > 0.6 > 24 h)

★ **PRESCRIBING & INTERPRETATION PEARLS**

- LTOT prolongs survival only in resting hypoxaemia (NOTT, MRC); not in moderate desaturation (LOTT).

Technique	Description
Autogenic drainage	Breathing at different lung volumes to mobilise secretions
Postural drainage ± percussion/vibration	Segment-specific positioning (see 1.3)
Oscillating PEP (flutter, Acapella)	Positive pressure + oscillation splints airways, loosens mucus
High-frequency chest wall oscillation (vest)	CF, NM disease
Mechanical insufflation–exsufflation (cough assist)	NM weakness (peak cough flow <270 L/min)
Hypertonic saline / mannitol	Hydrate airway surface

★ **CLINICAL PEARLS & EXAM FOCUS**

- Start rehabilitation within 4 weeks of discharge after an exacerbation — but not during the admission's first days (early in-hospital rehab did not reduce mortality).

LANDMARK TRIALS & GUIDELINES

- Spruit MA et al, ATS/ERS statement on pulmonary rehabilitation 2013; McCarthy B et al, Cochrane 2015; Rochester CL et al, ATS guideline on PR in COPD 2023.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Role of rehabilitation in chronic obstructive pulmonary disease. *[DNB Oct 2024 P2]*
2. Evidence for pulmonary rehabilitation in COPD. *[DNB Jun 2021 P2]*
3. Role of pulmonary rehabilitation in COPD. *[BFUHS 2022 P4]*
4. Pulmonary rehabilitation in COPD. *[BFUHS 2019 P4]*
5. Discuss in detail about pulmonary rehabilitation in respiratory diseases. *[VNSGU 2025 P4]*
6. Airway clearance techniques. *[NEET-SS/DNB 2024]*

6.14 Interventions for Emphysema — LVRS, Bronchoscopic Lung Volume Reduction & Bullous Disease

‹ NMC curriculum
Previous-year Qs ›

◉ **CBME COMPETENCIES — the postgraduate should be able to:**

- ✓ **NMC syllabus area:** II-B4 Bullous lung disease; II-B6
- ✓ Select patients for lung volume reduction surgery and bronchoscopic LVR
- ✓ Describe the management of giant bullae

IN BRIEF In severe hyperinflated emphysema, removing the worst-functioning lung improves elastic recoil, diaphragm mechanics and dyspnoea. LVRS benefits upper-lobe-predominant emphysema with low exercise capacity (NETT); endobronchial valves achieve similar results in patients without collateral ventilation.

LVRS (NETT) selection

Favourable	Exclusion / high risk
Upper-lobe-predominant heterogeneous emphysema + low exercise capacity — survival benefit	FEV1 ≤20% with either DLCO ≤20% or homogeneous emphysema (high mortality)
FEV1 15–45%, TLC >100%, RV >150%	PaCO2 >60, severe PH, BMI extremes, recent smoking
Completed rehabilitation, non-smoker ≥4 months	Major comorbidity

INVESTIGATIONS & TREATMENT AT A GLANCE

6.13 Pulmonary Rehabilitation & Airway Clearance Techniques

‹ Back to topic (p 135)
Next topic 6.14 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
6MWT	—	Distance and desaturation	Baseline/outcome (MCID ~ 30 m)
Incremental shuttle walk test	—	Level reached	Exercise prescription
CAT / CRQ / SGRQ	—	Improvement ≥ 2 (CAT), ≥ 4 (SGRQ)	MCID
Sit-to-stand (1 min)	—	Repetitions	Simple functional test
Contraindication screen (ECG, BP)	—	Unstable angina, severe AS, uncontrolled arrhythmia	Exclude

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Pulmonary rehabilitation	6–12 weeks, 2–3 supervised sessions/week; endurance + resistance training	COPD, ILD, bronchiectasis, PH, post-COVID	Start within 4 weeks after exacerbation
Airway clearance (ACBT, oscillating PEP)	Twice daily	Bronchiectasis, CF	—
Supplemental O_2 during training	Keep SpO_2 ≥ 88%	Exertional desaturation	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Rehabilitation started within 3 weeks of discharge reduces readmissions.

Bronchoscopic lung volume reduction (BLVR)

Technique	Mechanism	Key requirement / trial
Endobronchial valves (Zephyr, Spiration)	One-way valves → lobar atelectasis	Intact fissures/no collateral ventilation (Chartis); LIBERATE, EMPROVE — FEV1 ↑ ~15–20%; risk pneumothorax 20–30%
Endobronchial coils	Compress parenchyma	Homogeneous; RENEW — modest; withdrawn
Thermal vapour ablation	Inflammatory fibrosis	STEP-UP — segmental
Biological sealants (lung sealant)	Polymer foam	Investigational

Bullous lung disease

- **Bulla:** air space >1 cm within lung with thin wall; **giant bulla** occupies ≥1/3 hemithorax; vanishing lung syndrome (young male smokers, upper lobes); causes — emphysema, α1-ATD, Marfan, HIV, cannabis, IV drug use.
- **Complications:** pneumothorax, infection (air–fluid level), haemoptysis, cancer, compression of normal lung.
- **Bullectomy indications:** giant bulla compressing relatively normal lung with dyspnoea, recurrent pneumothorax, infection, haemoptysis; best results with large localised bullae and preserved remaining lung (CT shows compressed vessels).
- Differentiate bulla from pneumothorax (pleural line convex vs concave edge; CT double-wall sign) — do not insert ICD into a bulla.

Lung volume reduction surgery — summary of NETT

- 1,218 patients; overall no survival difference; upper-lobe/low exercise subgroup — better survival and exercise capacity; high-risk subgroup — higher mortality.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Before valves — check fissure integrity on CT and absence of collateral ventilation by Chartis.

LANDMARK TRIALS & GUIDELINES

- Fishman A et al, NETT NEJM 2003; Criner GJ et al, LIBERATE AJRCCM 2018; GOLD 2025 interventional therapy algorithm.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Bronchoscopic lung volume reduction (bLVR). *[DNB Dec 2022 P4]*
2. Lung volume reduction surgery. *[WBUHS 2020 P2]*

6.15 Alpha-1 Antitrypsin Deficiency

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

✓ **NMC syllabus area:** II-B6 COPD; I-B10 Defence
✓ Describe the genetics, pathogenesis and clinical features of α1-ATD
✓ Describe diagnosis and augmentation therapy

IN BRIEF α1-ATD is an autosomal codominant disorder (SERPINA1) causing early panacinar emphysema (loss of antiprotease) and liver disease (polymer retention). WHO recommends testing every COPD patient once, especially in endemic regions; it is under-recognised in India.

Genetics and pathogenesis

- Alleles: M (normal), Z (Glu342Lys — polymerises in hepatocytes; levels 10–15%), S (60%), null (no protein). PiZZ — severe deficiency (<11 µM / <57 mg/dL).

INVESTIGATIONS & TREATMENT AT A GLANCE

6.14 Interventions for Emphysema — LVRS, Bronchoscopic Lung Volume Reduction & Bullous Disease

‹ Back to topic (p 136)
Next topic 6.15 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
CT emphysema quantification	—	Heterogeneous upper-lobe emphysema, intact fissures (> 80–90%)	LVRS/valve candidacy
Collateral ventilation (Chartis)	—	CV negative	Endobronchial valve eligibility
FEV1 / DLCO	FEV1 > 20%; DLCO > 20%	Both < 20%	High LVRS mortality (NETT)
RV / TLC	RV < 150%	RV > 175% predicted	Hyperinflation — candidate
6MWT	> 140 m	< 140 m	Exclusion

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Endobronchial valves (Zephyr/Spiration)	Bronchoscopic	Severe heterogeneous emphysema, CV-negative	Pneumothorax (~ 25%)
LVRS	Surgical	Upper-lobe emphysema, low exercise capacity (NETT)	Mortality, air leak
Bullectomy	Surgical	Giant bulla > ⅓ hemithorax	—
Lung transplantation	Referral	BODE 7–10, FEV1 < 20%	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Pneumothorax after valve placement needs immediate recognition — most occur within 3 days.

- **Lung:** unopposed neutrophil elastase → panacinar emphysema, lower lobe, onset 30–40s (earlier in smokers), bronchiectasis.
- **Liver:** Z polymers in ER → neonatal cholestasis, cirrhosis, HCC. Others: panniculitis, c-ANCA vasculitis.

Diagnosis

Test	Comment
Serum α1-AT level (nephelometry)	Low; acute phase reactant (may be falsely normal in inflammation — check CRP)
Phenotyping (isoelectric focusing)	Identifies Pi type
Genotyping	Z, S alleles; sequencing for rare variants
Liver biopsy	PAS-positive diastase-resistant globules

- **Who to test:** all COPD (WHO), early-onset emphysema (<45 years), basal emphysema, non-smoker COPD, family history, unexplained liver disease, necrotising panniculitis, c-ANCA vasculitis, bronchiectasis without cause.

Treatment

- Smoking cessation (most important), standard COPD therapy, vaccines, rehabilitation.
- **Augmentation therapy:** IV pooled human α1-AT 60 mg/kg weekly for PiZZ with FEV1 30–65% (and some outside); slows CT lung density decline (RAPID trial).
- LVRS less effective (basal); lung and liver transplantation; investigational — siRNA (fazirsiran) for liver, recombinant AAT, gene therapy.

★ CLINICAL PEARLS & EXAM FOCUS

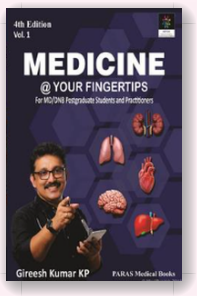
▸ Basal panacinar emphysema in a young non-smoker = α1-ATD until proven otherwise.

LANDMARK TRIALS & GUIDELINES

- Chapman KR et al, RAPID trial, Lancet 2015; ATS/ERS statement on α1-ATD 2003.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Alpha 1 antitrypsin deficiency. *[DNB Dec 2021 P1]*



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INVESTIGATIONS & TREATMENT AT A GLANCE

6.15 Alpha-1 Antitrypsin Deficiency

‹ Back to topic (p 137)
Next topic 7.1 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Serum AAT	1.0–2.0 g/L (20–50 µmol/L)	< 0.57 g/L (< 11 µmol/L)	Severe deficiency
Phenotype / genotype	PiMM	PiZZ, PiSZ, null	Risk stratification
CT chest	—	Panlobular basal emphysema	AATD pattern
LFT / liver ultrasound / elastography	Normal	Cirrhosis	Liver disease (PiZZ)
CT lung density (PD15)	—	Annual decline	Augmentation response

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
AAT augmentation (IV)	60 mg/kg weekly	PiZZ/null with FEV1 30–65% (and non-smoker)	IgA deficiency, cost
Smoking cessation	—	Mandatory	—
Standard COPD therapy	Per GOLD	All	—
Family screening	Serum AAT ± genotype	First-degree relatives	—

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Screen every COPD patient once for AATD (WHO, ATS/ERS).

SECTION 7

Interstitial Lung Diseases & Immunological Disorders

Paper II / IV

Immune defence and reactions of the interstitium; idiopathic interstitial pneumonias; hypersensitivity pneumonitis; sarcoidosis; connective tissue disease, vasculitis and diffuse alveolar haemorrhage; eosinophilic pneumonias and tropical eosinophilia; drug- and radiation-induced lung disease; cystic lung diseases (PLCH, LAM); PAP and other rare diseases (NMC syllabus II-B3, II-B4, I-D6).

7.1 Classification & Approach to Interstitial Lung Disease; HRCT Patterns 139
7.2 Idiopathic Pulmonary Fibrosis — Diagnosis, Severity, Antifibrotics & Acute Exacerbation 141
7.3 Other Idiopathic Interstitial Pneumonias — NSIP, COP, RB-ILD, DIP, AIP, PPFE; Progressive Pulmonary Fibrosis 144
7.4 Hypersensitivity Pneumonitis 145
7.5 Sarcoidosis 147
7.6 Connective Tissue Disease–Associated ILD & IPAF 149
7.7 Pulmonary Vasculitis & Diffuse Alveolar Haemorrhage 150
7.8 Eosinophilic Lung Diseases — AEP, CEP, Tropical Pulmonary Eosinophilia & HES 152
7.9 Drug-Induced & Transfusion-Related Lung Disease; Radiation Injury 153
7.10 Cystic Lung Diseases — LAM, PLCH, BHD & LIP 155
7.11 Pulmonary Alveolar Proteinosis, Alveolar Microlithiasis & Amyloidosis 156

7.1 Classification & Approach to Interstitial Lung Disease; HRCT Patterns

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B3 ILD of other aetiologies
- ✓ Classify ILDs and approach a patient with diffuse parenchymal lung disease
- ✓ Describe the role of HRCT and recognise principal HRCT patterns
- ✓ Describe the multidisciplinary discussion and emerging molecular markers

IN BRIEF ILDs (diffuse parenchymal lung diseases) are >200 disorders affecting the interstitium. Diagnosis rests on a multidisciplinary discussion (MDD) integrating clinical exposures, autoimmune serology, HRCT pattern and, when needed, BAL and lung biopsy.

Classification of ILD (ATS/ERS)

Group	Examples
Known cause/association	CTD-ILD (SSc, RA, myositis, SLE, Sjögren), HP, drug-induced, radiation, pneumoconiosis, smoking-related
Idiopathic interstitial pneumonias (IIPs)	Major: IPF, iNSIP, RB-ILD, DIP, COP, AIP; rare: LIP, PPFE; unclassifiable
Granulomatous	Sarcoidosis, HP, berylliosis, GLILD
Other forms	LAM, PLCH, PAP, eosinophilic pneumonias, amyloidosis, lymphangitic carcinomatosis

Clinical approach

- **History:** onset/tempo, exposures (birds, moulds, occupational dusts, drugs, radiation), smoking, CTD symptoms (Raynaud, arthritis, rash, dysphagia, sicca, myalgia), family history (telomere genes), GERD.
- **Examination:** clubbing (IPF, asbestosis), velcro crackles (fibrosis), inspiratory squeaks (HP), CTD signs (mechanic's hands, Gottron, sclerodactyly, telangiectasia).
- **Investigations:** PFT (restriction, low DLCO), 6MWT (desaturation), autoimmune panel (ANA, RF, anti-CCP, ENA incl. Scl-70, centromere, Ro/La, myositis panel — Jo-1 and other anti-synthetase, MDA-5; ANCA, CK), HP antigen IgG, BNP/echo, HRCT (prone and expiratory images).
- **BAL:** lymphocytosis (HP, sarcoid), eosinophils, DAH, PAP, infection. **Biopsy:** transbronchial cryobiopsy or VATS when HRCT is not diagnostic and result will change management.

HRCT patterns

Pattern	Features	Typical diseases
UIP	Basal, subpleural reticulation with honeycombing ± traction bronchiectasis; heterogeneous	IPF, RA-ILD, asbestosis, chronic HP
NSIP	Bilateral basal GGO + fine reticulation, traction bronchiectasis, **subpleural sparing**; homogeneous	Idiopathic NSIP, SSc, myositis, drugs
OP	Patchy peribronchovascular/subpleural consolidation, migratory; reversed halo (atoll)	COP, CTD, drugs, post-infection
Fibrotic HP	Upper/mid zone fibrosis, centrilobular nodules, mosaic attenuation, **three-density sign**, air trapping	Bird/mould exposure
Smoking-related	Centrilobular GGO nodules (RB-ILD), diffuse GGO (DIP), emphysema + fibrosis (CPFE)	Smokers
Cystic	Thin-walled cysts	LAM, PLCH (with nodules, upper lobe), BHD, LIP
Perilymphatic nodules	Nodules along fissures/bronchovascular bundles	Sarcoid, silicosis, lymphangitic carcinoma
Crazy paving	GGO + septal thickening	PAP, PJP, DAH, ARDS, lipoid pneumonia

Precision medicine and molecular markers in ILD

- **Genetic:** MUC5B promoter variant (rs35705950 — strongest IPF risk, better survival), telomere-related genes (TERT, TERC, RTEL1, PARN — familial pulmonary fibrosis, short telomeres, poor steroid tolerance), surfactant genes (SFTPC, SFTPA2), HLA in CTD.
- **Biomarkers:** KL-6 and SP-D (epithelial injury, disease activity — used in Japan), MMP-7, CCL18 (progression), CA19-9/CA-125 (progression), periostin; genomic classifier on transbronchial biopsy (Envisia — UIP detection).
- **Radiomics/AI:** quantitative CT (CALIPER, deep learning) predicts progression; transcriptomics, proteomics; aims — earlier diagnosis, prognosis, drug selection.

★ **CLINICAL PEARLS & EXAM FOCUS**

▸ Subpleural sparing = NSIP; three-density (mosaic) sign = HP; upper-lobe cysts with nodules in a smoker = PLCH.

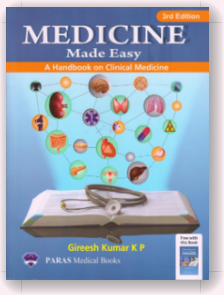
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LANDMARK TRIALS & GUIDELINES

- Travis WD et al, ATS/ERS classification of IIPs update 2013; Raghu G et al, ATS/ERS/JRS/ALAT IPF & PPF guideline 2022.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Classification of interstitial lung diseases. *[NEET-SS/DNB 2024]*
2. Diagnostic classification of interstitial lung disease in clinical practice. *[DNB Oct 2023 P4]*
3. Role of thoracic HRCT scanning in evaluating interstitial lung disease. *[BFUHS 2019 P2]*
4. Describe the newer classification of interstitial lung diseases. *[WBUHS 2020 P2]*
5. Molecular markers and the promise of precision medicine for interstitial lung disease. *[DNB Oct 2023 P4]*



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7.2 Idiopathic Pulmonary Fibrosis — Diagnosis, Severity, Antifibrotics & Acute Exacerbation

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B3 ILD
- ✓ Describe pathogenesis, clinical features and HRCT criteria of IPF (2022 guideline)
- ✓ Assess disease severity and prognosis (GAP)
- ✓ Treat IPF with antifibrotics and manage acute exacerbation

IN BRIEF IPF is a chronic progressive fibrosing ILD of unknown cause in older adults (usually >60, smokers, men) with UIP pattern on HRCT/histology. Median survival without treatment is 3–5 years; antifibrotics slow FVC decline by ~50%.

Pathogenesis

- Repetitive micro-injury to ageing alveolar epithelium (smoking, GERD micro-aspiration, pollutants, viruses) in genetically susceptible (MUC5B, telomeres) → aberrant epithelial repair, fibroblast foci, myofibroblast activation (TGF-β, PDGF, FGF), excessive ECM deposition, honeycombing.

Clinical features

- Insidious exertional dyspnoea, dry cough, bibasal fine end-inspiratory velcro crackles (>80%), clubbing (25–50%); late — cyanosis, cor pulmonale.
- Comorbidities: GERD, PH, emphysema (CPFE), lung cancer (5×), OSA, CAD, depression.

HRCT categories for IPF (ATS/ERS/JRS/ALAT 2022)

Category	Distribution	Features
UIP	Subpleural, basal predominant (often heterogeneous)	Honeycombing ± peripheral traction bronchiectasis/ bronchiolectasis
Probable UIP	Subpleural, basal	Reticulation with peripheral traction bronchiectasis, **no honeycombing**
Indeterminate	Variable	Fibrosis with features not suggestive of any specific aetiology

INVESTIGATIONS & TREATMENT AT A GLANCE

7.1 Classification & Approach to Interstitial Lung Disease; HRCT Patterns

‹ Back to topic (p 139)
Next topic 7.2 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
HRCT pattern	Normal	UIP, probable UIP, NSIP, OP, HP patterns	Guides diagnosis (MDD)
Spirometry/TLC	> 80%	TLC < 80%, FVC reduced, normal ratio	Restriction
DLCO	> 80%	< 80% (often lower than FVC)	Gas-exchange involvement
ANA, RF, anti-CCP, myositis panel, ANCA, anti-Scl-70	Negative	Positive	CTD-ILD / IPAF
BAL lymphocytes	< 15%	> 25–30%	HP, sarcoid, NSIP, OP
6MWT SpO_2	No desaturation	Fall ≥ 4% / < 88%	Severity, prognosis
Serum KL-6 (if available)	< 500 U/mL	> 1000 U/mL	ILD activity marker

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Avoid exposures	—	HP antigens, drugs	—
Supplemental O_2	SpO_2 ≥ 88–90% on exertion	Exertional hypoxaemia	—
Pulmonary rehabilitation	8–12 weeks	All fibrotic ILD	—
Vaccination	Influenza, pneumococcal, RSV, COVID	All ILD	Before immunosuppression

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Multidisciplinary discussion (clinician, radiologist, pathologist) is the reference standard for ILD diagnosis.

Category	Distribution	Features
Alternative diagnosis	Upper/mid zone, peribronchovascular, subpleural sparing	Cysts, mosaicism/three-density, nodules, consolidation, predominant GGO, pleural plaques

- **Diagnosis:** UIP or probable UIP on HRCT in appropriate clinical context (no identifiable cause) = IPF without biopsy (MDD); indeterminate → BAL ± cryobiopsy/surgical biopsy.
- **Histology (UIP):** patchy dense fibrosis with architectural distortion, honeycombing, fibroblast foci, temporal heterogeneity, subpleural/paraseptal.

Severity assessment and prognosis

- **Physiology:** FVC (mild >75%, moderate 50–75%, severe <50%), DLCO (<40% severe), 6MWD and desaturation, PH on echo.
- **GAP index** (Gender, Age, Physiology — FVC, DLCO): stages I–III with 1-year mortality 6%, 16%, 39%.
- **Markers of progression:** FVC decline ≥10% (or ≥5%) in 6–12 months, DLCO decline ≥15%, increasing fibrosis extent on HRCT, worsening dyspnoea, hospitalisation.

Antifibrotic therapy

Antifibrotics

Drug	Mechanism	Dose	Adverse effects	Evidence
Nintedanib	Tyrosine kinase inhibitor (VEGFR, FGFR, PDGFR)	150 mg BD (100 mg BD if intolerant)	Diarrhoea (60%), hepatotoxicity, bleeding risk, weight loss	INPULSIS (IPF), INBUILD (PPF), SENSCIS (SSc-ILD)
Pirfenidone	Anti-fibrotic, anti-inflammatory (↓ TGF-β)	801 mg TDS (267 mg tablets, titrated over 2 weeks)	Nausea, anorexia, photosensitive rash, hepatotoxicity	CAPACITY, ASCEND
Nerandomilast	Preferential PDE4B inhibitor	18 mg BD	Diarrhoea	FIBRONEER-IPF / -ILD (2025) — slowed FVC decline

- Start at diagnosis (mild disease benefits equally); monitor LFT monthly for 3 months; take with food; sun protection (pirfenidone).
- **No role:** prednisolone + azathioprine + NAC (PANTHER — harm), anticoagulation, sildenafil alone, ambrisentan (harm), routine anti-reflux therapy.

Non-pharmacological and supportive

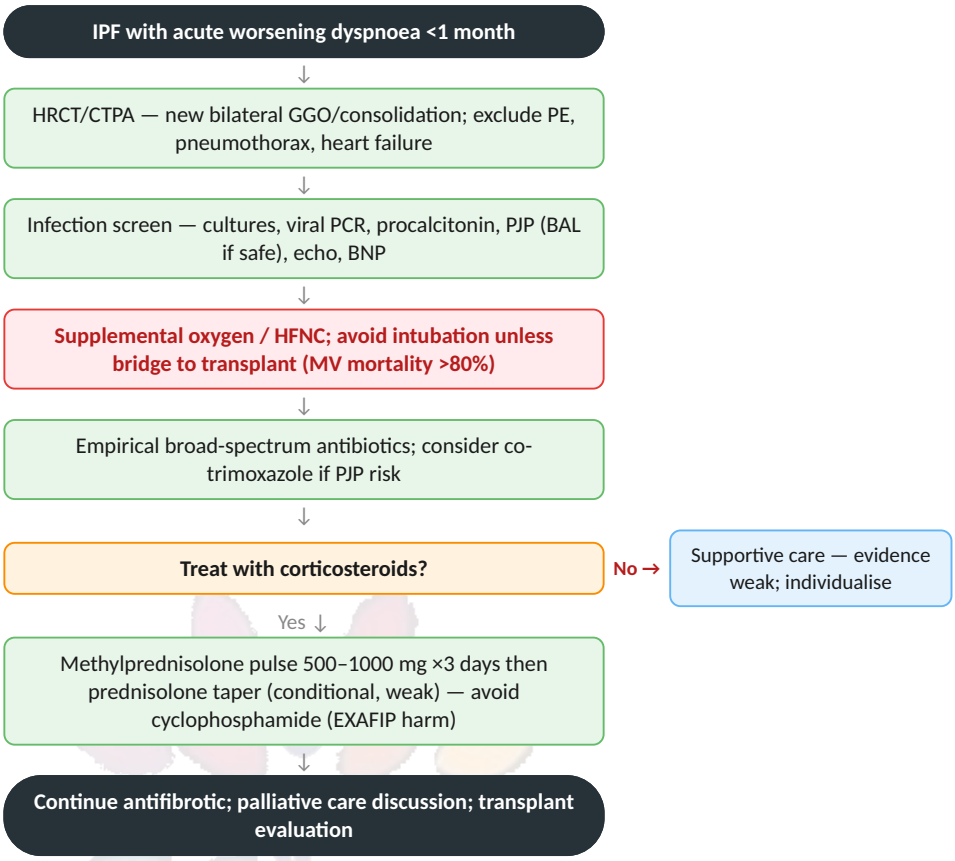
- Pulmonary rehabilitation, oxygen (rest/ambulatory for symptoms), vaccines, smoking cessation, treat GERD if symptomatic, lung transplantation referral early (FVC <80%, DLCO <40%, progression), palliative care (opioids for dyspnoea/cough), clinical trials.

Acute exacerbation of IPF (AE-IPF)

- **Definition (2016):** acute (<1 month) worsening of dyspnoea in a patient with IPF + new bilateral GGO/consolidation superimposed on UIP, not fully explained by cardiac failure or fluid overload; triggered (infection, aspiration, surgery, BAL/biopsy, drugs) or idiopathic. Incidence 5–15%/year; in-hospital mortality ~50%.

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Algorithm 7.1 · Management of acute exacerbation of IPF



✦ RECENT ADVANCES & GUIDELINE UPDATES

- 2025: nerandomilast (FIBRONEER) — first new IPF drug in a decade; inhaled treprostinil in IPF (TETON trials) improved FVC.

★ CLINICAL PEARLS & EXAM FOCUS

- "Probable UIP" (no honeycombing) is enough to diagnose IPF in the right clinical context — no biopsy needed.

LANDMARK TRIALS & GUIDELINES

- Raghu G et al, IPF/PPF guideline AJRCCM 2022; Richeldi L et al, INPULSIS NEJM 2014; King TE et al, ASCEND NEJM 2014; Collard HR et al, AE-IPF definition AJRCCM 2016.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Clinical features and HRCT criteria of idiopathic pulmonary fibrosis (IPF). *[DNB Oct 2024 P2]*
2. Assessment of disease severity in idiopathic pulmonary fibrosis. *[DNB Dec 2022 P2]*
3. Antifibrotics in treatment of IPF. *[DNB Jun 2025 P2]*
4. Acute exacerbation of idiopathic pulmonary fibrosis (AE-IPF). *[DNB Jun 2025 P3]*
5. Management of acute exacerbation in a patient with idiopathic pulmonary fibrosis. *[DNB Dec 2022 P2]*
6. Nintedanib and IPF. *[BFUHS 2020 P2]*
7. Newer anti-fibrotics for interstitial lung diseases. *[BFUHS 2020 P4]*
8. Current status of anti-fibrotic drugs in treating interstitial lung disease. *[BFUHS 2023 P2]*

INVESTIGATIONS & TREATMENT AT A GLANCE

7.2 Idiopathic Pulmonary Fibrosis — Diagnosis, Severity, Antifibrotics & Acute Exacerbation

‹ Back to topic (p 141)
Next topic 7.3 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
HRCT (UIP)	—	Subpleural basal reticulation, honeycombing, traction bronchiectasis	Definite UIP — no biopsy needed if clinical context fits
FVC % predicted	> 80%	Fall ≥ 10% in 6–12 months	Progression
DLCO	> 80%	< 40%	Advanced disease
GAP index	Stage I (0–3)	Stage III (6–8)	Mortality
Autoimmune serology	Negative	Positive	Exclude CTD-ILD
Echo / BNP	Normal	PH	Poor prognosis
Acute exacerbation work-up	—	New bilateral GGO on HRCT; exclude infection, PE, HF	AE-IPF

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Pirfenidone	267 mg tds × 1 wk → 534 mg tds × 1 wk → 801 mg tds	IPF (FVC 50–80% in trials, now broader)	Photosensitivity, GI, LFTs
Nintedanib	150 mg bd (100 mg bd if intolerant)	IPF, progressive pulmonary fibrosis, SSc-ILD	Diarrhoea, LFTs, bleeding
Nerandomilast	18 mg bd	IPF / PPF (FIBRONEER, 2025)	Diarrhoea
Methylprednisolone (AE-IPF)	500–1000 mg IV × 3 days (conditional)	Acute exacerbation	Weak evidence
Lung transplant referral	—	DLCO < 40%, FVC fall ≥ 10%, hospitalisation, PH	Early referral

★ PRESCRIBING & INTERPRETATION PEARLS

- Do not use prednisolone + azathioprine + NAC in IPF (PANTHER — harm).

7.3 Other Idiopathic Interstitial Pneumonias — NSIP, COP, RB-ILD, DIP, AIP, PPFE; Progressive Pulmonary Fibrosis

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B3 ILD
- ✓ Classify idiopathic interstitial pneumonias with clinical, radiological and histological features
- ✓ Define progressive pulmonary fibrosis and its treatment
- ✓ Recognise smoking-related ILDs

IN BRIEF The 2013 ATS/ERS classification groups IIPs into chronic fibrosing (IPF, NSIP), smoking-related (RB-ILD, DIP), acute/subacute (COP, AIP) and rare (LIP, PPFE). Any fibrosing ILD that progresses despite treatment is "progressive pulmonary fibrosis" (PPF) and benefits from nintedanib.

Classification of idiopathic interstitial pneumonias

Category	Clinical-radiological-pathological diagnosis	Pattern	Prognosis / treatment
Chronic fibrosing	IPF	UIP	Poor; antifibrotics
Chronic fibrosing	Idiopathic NSIP	NSIP	Good if cellular; steroids ± MMF; often IPAF/CTD later
Smoking-related	RB-ILD	Respiratory bronchiolitis	Excellent with smoking cessation
Smoking-related	DIP	DIP (pigmented macrophages filling alveoli)	Good; quit smoking ± steroids
Acute/subacute	COP	Organising pneumonia (Masson bodies)	Good; steroids 6 months (relapse common)
Acute/subacute	AIP (Hamman–Rich)	Diffuse alveolar damage	ARDS-like; mortality >50%
Rare	Idiopathic LIP	LIP	Steroids; exclude Sjögren, HIV, lymphoma
Rare	Idiopathic PPFE	Pleuroparenchymal fibroelastosis	Upper lobe, flat chest, pneumothorax; progressive

Cryptogenic organising pneumonia

- Subacute flu-like illness, cough, dyspnoea, fever, weight loss over weeks; often treated as non-resolving pneumonia.
- HRCT: patchy peripheral/peribronchial consolidation, migratory, reversed halo; BAL lymphocytosis with low CD4/CD8; biopsy — intra-alveolar buds of granulation tissue (Masson bodies).
- Secondary OP: infection, CTD, drugs (amiodarone, nitrofurantoin, checkpoint inhibitors), radiation (breast — outside field), aspiration, transplant.
- **Treatment:** prednisolone 0.75–1 mg/kg tapered over 6–12 months; relapse 30–50% on tapering; macrolides in mild.

Smoking-related ILD

- **RB-ILD:** pigmented macrophages in respiratory bronchioles; centrilobular GGO nodules, bronchial wall thickening; mild restriction; quit smoking.
- **DIP:** diffuse GGO with small cysts, lower zones; steroids if no improvement after cessation.
- **CPFE** (combined pulmonary fibrosis and emphysema): upper-lobe emphysema + lower-lobe fibrosis; pseudo-normal spirometry, severely reduced DLCO, high PH and lung cancer risk.
- **Smoking-related interstitial fibrosis (SRIF)**, PLCH, acute eosinophilic pneumonia (new smokers), ILA.

Progressive pulmonary fibrosis (PPF, 2022)

PPF definition — in a patient with ILD other than IPF with radiological fibrosis, ≥2 of 3 within the past year (no alternative explanation)

Domain	Criterion
Symptoms	Worsening respiratory symptoms
Physiology	Absolute FVC decline ≥5% predicted, or DLCO decline ≥10% (within 1 year)
Radiology	Increased extent/severity of traction bronchiectasis, new GGO with traction bronchiectasis, new fine reticulation, increased reticulation or honeycombing, volume loss

- **Treatment:** nintedanib (INBUILD — 57% slower FVC decline across fibrotic HP, CTD-ILD, iNSIP, unclassifiable, pneumoconioses); continue disease-specific therapy (immunosuppression for CTD-ILD/HP); pirfenidone conditional (RELIEF, uILD).

★ **CLINICAL PEARLS & EXAM FOCUS**

- Masson bodies = organising pneumonia; DAD with hyaline membranes = AIP/ARDS; temporal heterogeneity with fibroblast foci = UIP.

LANDMARK TRIALS & GUIDELINES

- Travis WD et al, AJRCCM 2013; Flaherty KR et al, INBUILD NEJM 2019.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Classification of idiopathic interstitial pneumonias. *[DNB Dec 2022 P2]*
2. Cryptogenic organizing pneumonia. *[DNB Jun 2021 P2]*
3. Respiratory bronchiolitis-ILD. *[DNB Dec 2025 P2]*
4. Smoking related interstitial pulmonary fibrosis. *[DNB Dec 2022 P1]*
5. Non-specific interstitial pneumonia. *[Manipal 2014 P4]*

7.4 Hypersensitivity Pneumonitis

‹ NMC curriculum
Previous-year Qs ›

◉ **CBME COMPETENCIES — the postgraduate should be able to:**

- ✓ **NMC syllabus area:** II-B3 Hypersensitivity pneumonitis
- ✓ Describe the aetiology and immunopathogenesis of HP
- ✓ Classify HP as non-fibrotic or fibrotic and apply ATS/JRS/ALAT 2020 diagnostic criteria
- ✓ Treat HP and describe its prognosis

IN BRIEF HP is an immune-mediated ILD in susceptible individuals after repeated inhalation of organic antigens (birds, moulds, bacteria) or chemicals. The key to treatment is antigen identification and avoidance; fibrotic HP behaves like IPF.

Common causes of HP

Disease	Antigen	Source
Farmer's lung	Saccharopolyspora rectivirgula, Thermoactinomyces vulgaris	Mouldy hay, grain, straw
Bird fancier's / pigeon breeder's lung	Avian serum proteins, droppings, feathers	Pigeons, parrots, feather pillows/duvets
Humidifier/hot-tub lung	Thermophilic actinomycetes, MAC	Contaminated humidifiers, AC, hot tubs
Bagassosis	Thermoactinomyces sacchari	Mouldy sugarcane bagasse
Mushroom worker's lung	Thermophilic actinomycetes	Mushroom compost
Machine operator's lung	Mycobacterium immunogenum	Metalworking fluids

INVESTIGATIONS & TREATMENT AT A GLANCE

7.3 Other Idiopathic Interstitial Pneumonias — NSIP, COP, RB- ILD, DIP, AIP, PPFE; Progressive Pulmonary Fibrosis

‹ Back to topic (p 144)
Next topic 7.4 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
HRCT NSIP	—	Basal GGO, reticulation, subpleural sparing	NSIP pattern
HRCT COP	—	Patchy migratory peribronchovascular consolidation, reversed halo	OP
BAL	—	Lymphocytosis (NSIP/COP); pigmented macrophages (RB-ILD/DIP)	Pattern support
Smoking history	—	Current smoker	RB-ILD, DIP
Autoimmune screen	Negative	Positive	Secondary NSIP/OP

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Prednisolone (COP)	0.75 mg/kg/day, taper over 6 months	COP	Relapse on taper (common)
Prednisolone ± mycophenolate (NSIP)	Pred 0.5 mg/kg; MMF 1–1.5 g bd	Fibrotic/cellular NSIP	—
Smoking cessation	—	RB-ILD, DIP	—
Supportive / ICU care	Lung-protective ventilation	AIP (Hamman–Rich)	High mortality

★ PRESCRIBING & INTERPRETATION PEARLS

- Reversed halo (atoll) sign suggests organising pneumonia — but exclude mucormycosis in immunocompromised.

Disease	Antigen	Source
Chemical worker's lung	Isocyanates (TDI, MDI)	Paints, polyurethane
Summer-type HP	Trichosporon	Japanese homes

Immunopathogenesis

- Type III (immune complexes, precipitins) and type IV (Th1, granulomas, CD8 lymphocytosis) hypersensitivity; genetic susceptibility (MUC5B in fibrotic HP, HLA-DR); smokers are relatively protected (nicotine suppresses).

Farmer's lung (classic acute HP)

- 4–8 hours after exposure to mouldy hay: fever, chills, cough, dyspnoea, myalgia; crackles; resolves in 24–48 h if exposure ceases; recurrent exposure → chronic disease.

ATS/JRS/ALAT 2020 HRCT patterns

Category	Non-fibrotic HP	Fibrotic HP
Typical	Diffuse GGO/mosaic, centrilobular ill-defined nodules, air trapping (three-density sign)	Fibrosis (reticulation, traction bronchiectasis) mid-upper or random + small airway features
Compatible	Uniform GGO, consolidation, cysts	Variable fibrosis (UIP-like), basal predominance
Indeterminate	—	Neither

- **Diagnostic confidence** integrates exposure identified (or not), HRCT category, BAL lymphocytosis (>20–30%), and histology (bronchiolocentric cellular interstitial pneumonia, poorly formed granulomas).
- Serum specific IgG to antigens (supportive only).

Management and prognosis

- **Antigen avoidance** — the single most important measure (home visit, remove birds/feathers, remediation of mould); occupational redeployment.
- **Non-fibrotic HP:** prednisolone 0.5 mg/kg tapered over 3–6 months if symptomatic/impaired.
- **Fibrotic HP:** mycophenolate or azathioprine (steroid-sparing); nintedanib if progressive (INBUILD); transplant.
- **Prognosis:** non-fibrotic good with avoidance; fibrotic HP median survival ~7 years (worse with UIP-like pattern, unidentified antigen, low BAL lymphocytes, honeycombing).

★ CLINICAL PEARLS & EXAM FOCUS

▸ If no antigen is found in suspected HP — look harder (feather pillow, hidden mould, home humidifier); unidentified antigen worsens prognosis.

LANDMARK TRIALS & GUIDELINES

- Raghu G et al, ATS/JRS/ALAT HP guideline AJRCCM 2020; Fernández Pérez ER et al, CHEST HP guideline 2021.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Hypersensitivity pneumonitis. *[DNB Jun 2025 P2]*
2. Prognosis and treatment of hypersensitivity pneumonitis. *[DNB Dec 2021 P2]*
3. Approach to hypersensitivity pneumonitis. *[BFUHS 2022 P4]*
4. Discuss management of hypersensitivity pneumonitis. *[WBUHS 2020 P2]*
5. Farmer's lung. *[VNSGU 2025 P3]*

INVESTIGATIONS & TREATMENT AT A GLANCE

7.4 Hypersensitivity Pneumonitis

‹ Back to topic (p 145)
Next topic 7.5 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
HRCT	—	Centrilobular nodules, GGO, mosaic attenuation, three-density sign	HP (non-fibrotic/fibrotic)
BAL lymphocytes	< 15%	> 30% (> 40% strongly suggestive)	HP
Serum IgG to antigens (precipitins)	Negative	Positive	Exposure
Exposure history	—	Birds, feathers, moulds, humidifiers	Most important clue
Transbronchial cryobiopsy	—	Poorly formed granulomas, bronchiolocentric inflammation	Confirmation

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Antigen avoidance	Complete	All HP	Most important intervention
Prednisolone	0.5 mg/kg/day × 4–8 weeks, taper	Non-fibrotic HP with significant impairment	Steroid toxicity
Mycophenolate / azathioprine	MMF 1–1.5 g bd	Steroid-sparing, fibrotic HP	—
Nintedanib	150 mg bd	Progressive fibrotic HP (INBUILD)	—

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Antigen not identified in up to half of fibrotic HP — worse prognosis.

7.5 Sarcoidosis

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B3 Sarcoidosis; I-F3
- ✓ Describe the immunopathogenesis and clinical manifestations of sarcoidosis
- ✓ Diagnose sarcoidosis (ATS 2020) and stage chest radiographs (Scadding)
- ✓ Decide when and how to treat, including glucocorticoid alternatives

IN BRIEF Sarcoidosis is a multisystem granulomatous disease of unknown cause with non-necrotising granulomas, affecting the lungs and intrathoracic nodes in >90%. Diagnosis requires a compatible presentation, non-necrotising granulomas and exclusion of other causes — in India, above all TB.

Immunopathogenesis

- Unknown antigen (mycobacterial catalase-peroxidase katG, propionibacteria, inorganic particles) in genetically susceptible (HLA-DRB1, BTNL2) → Th1/Th17 response (IFN-γ, TNF-α, IL-12) → granulomas; macrophage 1α-hydroxylase → hypercalcaemia; CD4 lymphocytosis in BAL (CD4/CD8 >3.5).

Clinical manifestations

- **Pulmonary:** asymptomatic (30–50% — incidental bilateral hilar lymphadenopathy), cough, dyspnoea, chest discomfort; fibrosis (stage IV), PH (SAPH), bronchostenosis, aspergilloma in cavities.
- **Acute syndromes: Löfgren's** (BHL + erythema nodosum + ankle periarthritis + fever — good prognosis), **Heerfordt's** (uveoparotid fever: parotid enlargement, anterior uveitis, facial palsy, fever).
- **Extrapulmonary:** skin (lupus pernio, plaques, scar sarcoid), eye (uveitis — screen all), cardiac (heart block, VT, sudden death — screen with ECG ± Holter/echo, CMR/FDG-PET), neurosarcoidosis (CN VII, hypothalamic), liver/spleen, hypercalcaemia/hypercalciuria (nephrocalcinosis), lymph nodes, bone (lacy lytic lesions), parotid, constitutional fatigue.

Erythema nodosum

- Septal panniculitis — tender red nodules on shins; causes: streptococcal infection, primary TB, sarcoidosis (Löfgren), drugs (OCPs, sulfonamides), IBD, pregnancy, Behçet, leprosy (ENL), fungal (coccidioides), idiopathic. Self-limiting 3–6 weeks; NSAIDs, treat cause.

Scadding chest radiographic stages

Stage	Findings	Spontaneous remission
0	Normal CXR (extrathoracic disease)	—
I	Bilateral hilar lymphadenopathy only	55–90%
II	BHL + parenchymal infiltrates	40–70%
III	Parenchymal infiltrates without BHL	10–20%
IV	Fibrosis (upper lobe, volume loss, honeycombing)	0%

Diagnosis (ATS 2020)

- **Compatible presentation + non-necrotising granuloma + exclusion of alternatives** (TB, fungi, lymphoma, HP, berylliosis, drug-induced, GLILD).
- **Tissue:** EBUS-TBNA of nodes (yield ~85%) — preferred; TBLB/cryobiopsy, skin lesions, salivary gland. Biopsy may be omitted in Löfgren's, Heerfordt's, asymptomatic BHL (close follow-up).
- **Baseline screening:** CBC, calcium (serum and 24-h urine), creatinine, LFT, ECG, eye examination, PFT, 25-OH and 1,25-(OH)2 vitamin D; ACE (non-specific, activity marker), sIL-2R; FDG-PET for occult/cardiac activity.

- In India: send node aspirate for Xpert/culture; IGRA/TST negative supports.

Treatment — when and how

- **Indications:** to reduce risk of mortality/permanent organ damage or improve QoL — symptomatic pulmonary disease with impairment (FVC/DLCO decline), progressive stage II–IV, cardiac, neuro, ocular (not responding to topical), hypercalcaemia, renal, disfiguring skin, severe fatigue. Asymptomatic stage I — observe.

Treatment of pulmonary sarcoidosis (ERS 2021)

Line	Drugs
First	Prednisolone 20–40 mg/day (0.3–0.6 mg/kg) ×2–6 weeks, taper to 5–10 mg/day maintenance; total 6–12 months
Second (steroid-sparing or failure)	Methotrexate 10–20 mg weekly (preferred); azathioprine, mycophenolate, leflunomide, hydroxychloroquine (skin, hypercalcaemia)
Third (refractory)	Infliximab (3–5 mg/kg; also cardiac, neuro, lupus pernio), adalimumab; JAK inhibitors (emerging)
Supportive	Antifibrotic (nintedanib) for progressive fibrotic stage IV, LTOT, PH therapy, transplant

- **Fatigue:** exercise, rehab, methylphenidate (selected). Relapse in 30–60% after tapering.

★ **CLINICAL PEARLS & EXAM FOCUS**

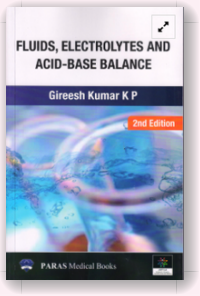
▸ Löfgren's syndrome needs no biopsy and usually no steroids — NSAIDs; resolves in 1–2 years.

LANDMARK TRIALS & GUIDELINES

- Crouser ED et al, ATS sarcoidosis diagnosis guideline AJRCCM 2020; Baughman RP et al, ERS sarcoidosis treatment guideline Eur Respir J 2021.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Approach to the initial treatment of pulmonary sarcoidosis in adults. *[DNB Jun 2025 P2]*
2. Chest radiographic stages of pulmonary sarcoidosis and their implications for treatment. *[DNB Dec 2022 P2]*
3. Indications and treatment of pulmonary sarcoidosis with alternatives to glucocorticoids. *[DNB Dec 2022 P2]*
4. Treatment of sarcoidosis. *[DNB Dec 2021 P2]*
5. Diagnosis of sarcoidosis. *[DNB Jun 2021 P2]*
6. Treatment of symptomatic pulmonary sarcoidosis. *[DNB Jun 2021 P2]*
7. Erythema nodosum. *[DNB Jun 2021 P1]*



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INVESTIGATIONS & TREATMENT AT A GLANCE

7.5 Sarcoidosis

‹ Back to topic (p 147)
Next topic 7.6 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Serum ACE	8–65 U/L	Raised (in ~ 60%)	Supportive, not diagnostic
Serum calcium / 24-h urine calcium	Ca 8.5–10.5 mg/dL; urine < 300 mg/24 h	Hypercalcaemia / hypercalciuria	Renal risk — treat
CXR Scadding stage	0	I BHL; II BHL + infiltrates; III infiltrates; IV fibrosis	Staging
EBUS-TBNA of nodes	—	Non-caseating granulomas	Diagnosis (with TB/fungal exclusion)
BAL CD4:CD8	1–2	> 3.5	Supports sarcoid
ECG / Holter / cardiac MRI	Normal	AV block, LGE	Cardiac sarcoid
Eye examination	Normal	Uveitis	Screen all

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Prednisolone	20–40 mg/day × 1–3 months, taper to 5–10 mg; total ≥ 12 months	Symptomatic pulmonary/organ sarcoid	Exclude TB first in India
Methotrexate	10–15 mg weekly + folic acid	Steroid-sparing	LFTs, pneumonitis
Infliximab	3–5 mg/kg IV at 0, 2, 6 weeks, then every 4–8 weeks	Refractory/neurosarcoid	TB reactivation — screen
Hydroxychloroquine	200–400 mg daily	Skin, hypercalcaemia	Retinal screening

★ PRESCRIBING & INTERPRETATION PEARLS

- Löfgren's syndrome (EN + BHL + arthritis) usually remits — NSAIDs only.

7.6 Connective Tissue Disease–Associated ILD & IPAF

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B3 Connective tissue diseases involving the respiratory system
- ✓ Describe pulmonary manifestations of SSc, RA, SLE and myositis
- ✓ Identify determinants of progression and treat SSc-ILD and RA-ILD
- ✓ Define interstitial pneumonia with autoimmune features

IN BRIEF CTDs affect every lung compartment — interstitium, airways, pleura, vessels, muscles. ILD is the leading cause of death in systemic sclerosis; RA-ILD (UIP pattern) behaves like IPF; anti-synthetase and anti-MDA5 myositis cause rapidly progressive ILD.

Pulmonary manifestations of CTDs

CTD	Commonest manifestations	Other
Systemic sclerosis	NSIP (anti-Scl-70/topoisomerase), PAH (limited cutaneous, anticentromere)	Aspiration (oesophageal dysmotility), lung cancer
Rheumatoid arthritis	UIP > NSIP; pleural effusion (low glucose, high LDH, RA cells)	Bronchiectasis, follicular/constrictive bronchiolitis, rheumatoid nodules, Caplan's, MTX pneumonitis, infections
SLE	Pleuritis (commonest), acute lupus pneumonitis, DAH	Shrinking lung syndrome, PAH, PE (APLA), ILD rare
Myositis (PM/DM, anti-synthetase)	NSIP/OP; rapidly progressive ILD (anti-MDA5)	Respiratory muscle weakness, aspiration
Sjögren	LIP, follicular bronchiolitis, NSIP	Cysts, lymphoma (MALT), amyloid
MCTD	NSIP, PAH	—
Ankylosing spondylitis	Apical fibrobullous disease	Chest wall restriction

Systemic sclerosis — clinical manifestations

- Raynaud's (>95%), skin thickening (limited — distal; diffuse — proximal/trunk), sclerodactyly, digital ulcers, telangiectasia, calcinosis; GI dysmotility, GERD; scleroderma renal crisis (ACE inhibitors); cardiac fibrosis; PAH; ILD.
- **Antibodies:** anti-Scl-70 (diffuse, ILD), anticentromere (limited, PAH), anti-RNA polymerase III (renal crisis, cancer).

Determinants of progression in SSc-ILD

Risk factor	Detail
Disease features	Diffuse cutaneous SSc, early disease (<5 years), anti-Scl-70
Physiology	FVC <70%, DLCO <55%, decline FVC ≥10% (or 5–9% + DLCO ≥15%) in 1 year
Imaging	Extent >20% on HRCT (Goh staging — extensive vs limited)
Biomarkers	High CRP, KL-6, CCL18; male sex, African ancestry

Treatment of SSc-ILD (ACR 2023 / EULAR 2023)

- **First-line:** mycophenolate mofetil 2–3 g/day (SLS II); tocilizumab (early, elevated CRP — focuSSced); rituximab (RECITAL, DESIRES); cyclophosphamide (SLS I) alternative.
- **Progressive/fibrotic:** add **nintedanib** (SENSCIS — slowed FVC decline 44%).
- Avoid high-dose steroids (>15 mg prednisolone — renal crisis); HSCT for selected rapidly progressive diffuse SSc; transplant.

RA-ILD

- Risk: male, older, smoker, high RF/anti-CCP, MUC5B. UIP behaves like IPF — nintedanib if progressive; NSIP/OP — steroids, MMF, rituximab, abatacept; methotrexate does not appear to cause RA-ILD (may protect) but MTX pneumonitis (acute) exists.

Interstitial pneumonia with autoimmune features (IPAF)

- ILD + no defined CTD + features from ≥2 domains: **clinical** (mechanic's hands, Raynaud, digital ulcer, Gottron, inflammatory arthritis, distal fingertip fissuring), **serological** (ANA ≥1:320, RF ≥2× ULN, anti-CCP, anti-Scl-70, Ro, La, RNP, Sm, Jo-1 and others, MDA-5), **morphological** (NSIP, OP, NSIP+OP, LIP on HRCT/histology; multicompartment involvement).
- Treated like CTD-ILD with immunosuppression (MMF, azathioprine), antifibrotic if progressive; monitor for evolution into a defined CTD.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Rapidly progressive ILD with skin ulcers, palmar papules and normal CK = anti-MDA5 dermatomyositis — urgent triple immunosuppression.

LANDMARK TRIALS & GUIDELINES

- Distler O et al, SENSCIS NEJM 2019; Tashkin DP et al, SLS II Lancet Respir Med 2016; Fischer A et al, ERS/ATS IPAF statement 2015; ACR/CHEST CTD-ILD treatment guideline 2023.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Clinical manifestations of systemic sclerosis. *[DNB Oct 2024 P2]*
2. Determinants of disease progression in systemic sclerosis-associated interstitial lung disease. *[DNB Dec 2022 P2]*
3. Treatment of interstitial lung disease in systemic sclerosis. *[DNB Dec 2022 P2]*
4. Pleuropulmonary complications of rheumatoid arthritis. *[DNB Dec 2021 P2]*
5. Briefly describe management of rheumatoid lung. *[DNB May 2024 P3]*
6. Respiratory system involvement in SLE. *[DNB Jun 2021 P2]*
7. Management of interstitial pneumonia with autoimmune features. *[BFUHS 2022 P2]*

7.7 Pulmonary Vasculitis & Diffuse Alveolar Haemorrhage

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B3 Pulmonary vasculitides; II-B4 Pulmonary haemorrhagic syndromes
- ✓ Classify vasculitides and describe pulmonary features of GPA, EGPA and MPA
- ✓ Manage ANCA-associated vasculitis including EGPA
- ✓ Recognise and manage diffuse alveolar haemorrhage

IN BRIEF ANCA-associated vasculitides (GPA, MPA, EGPA) affect small vessels of lungs and kidneys. Diffuse alveolar haemorrhage — haemoptysis, anaemia and diffuse infiltrates — is a medical emergency often due to capillaritis.

ANCA-associated vasculitis

Feature	GPA (Wegener's)	MPA	EGPA (Churg–Strauss)
ANCA	c-ANCA/PR3 (80–90%)	p-ANCA/MPO (60–70%)	MPO-ANCA (30–40%)
Upper airway	Sinusitis, saddle nose, subglottic stenosis, otitis	Rare	Nasal polyps, allergic rhinitis
Lung	Nodules (often cavitating), masses, DAH, tracheobronchial stenosis	DAH, fibrosis (UIP)	Asthma, migratory infiltrates, eosinophilic pneumonia
Kidney	Pauci-immune necrotising GN (70–80%)	GN (90%)	Less common

INVESTIGATIONS & TREATMENT AT A GLANCE

7.6 Connective Tissue Disease–Associated ILD & IPAF

‹ Back to topic (p 149)
Next topic 7.7 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
ANA / ENA (Scl-70, Jo-1, SSA)	Negative	Positive	CTD-ILD
Anti-MDA5	Negative	Positive	Rapidly progressive ILD — urgent
CK / aldolase	CK < 200 U/L	Raised	Myositis
Nailfold capillaroscopy	Normal	Dilated loops, dropout	SSc
HRCT	—	NSIP (SSc), UIP (RA), OP (myositis)	Pattern
FVC / DLCO	> 80%	FVC < 70% or decline ≥ 10%	Treat/escalate

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Mycophenolate mofetil	1–1.5 g bd	SSc-ILD (SLS-II), myositis ILD	Cytopenia, teratogenic
Tocilizumab	162 mg SC weekly	Early SSc-ILD (FVC preservation)	Infection
Nintedanib	150 mg bd	Progressive SSc/RA-ILD	Diarrhoea
Cyclophosphamide	500–750 mg/m² IV monthly × 6	Rapidly progressive CTD-ILD	Haemorrhagic cystitis, infertility
Rituximab	1 g IV × 2 (2 weeks apart)	Refractory CTD-ILD (RECITAL)	Hypogammaglobulinaemia, HBV

★ **PRESCRIBING & INTERPRETATION PEARLS**

- High-dose steroids (> 15 mg/day) can precipitate scleroderma renal crisis.

Feature	GPA (Wegener's)	MPA	EGPA (Churg–Strauss)
Other	Granulomatous inflammation, orbital mass	—	Eosinophilia >10%, neuropathy (mononeuritis), cardiac (major cause of death), skin

Management of GPA/MPA

- **Induction (severe):** glucocorticoids (methylprednisolone pulse then prednisolone with reduced-dose taper — PEXIVAS) + **rituximab** 375 mg/m² weekly ×4 (or 1 g ×2) or cyclophosphamide; avacopan (C5aR antagonist — ADVOCATE) as steroid-sparing; plasma exchange in selected severe renal disease/DAH (evidence limited).
- **Maintenance:** rituximab 500 mg 6-monthly (MAINRITSAN) ×18–24 months, or azathioprine; co-trimoxazole prophylaxis (PJP, reduces GPA relapse).
- **Non-severe:** methotrexate or MMF + steroids.

Management of EGPA

- **ACR/ACR-EULAR criteria:** asthma, eosinophilia >10%, neuropathy, migratory infiltrates, sinus disease, extravascular eosinophils.
- **Five-factor score (FFS):** proteinuria >1 g, creatinine >1.58, GI, cardiac, CNS involvement (and age >65) → poor prognosis.
- **Non-severe:** glucocorticoids + **mepolizumab 300 mg** (MIRRA) or **benralizumab** (MANDARA — non-inferior); azathioprine/MTX.
- **Severe (organ/life-threatening — cardiac, GN, neuropathy, DAH):** high-dose steroids + cyclophosphamide or rituximab; then maintenance.

Diffuse alveolar haemorrhage

Causes of DAH

Category	Causes
With capillaritis	GPA, MPA, anti-GBM (Goodpasture), SLE, APS, IgA vasculitis, cryoglobulinaemia, Behçet, isolated pauci-immune capillaritis
Bland haemorrhage	Coagulopathy, anticoagulants, mitral stenosis/LV failure, idiopathic pulmonary haemosiderosis, drugs (amiodarone, nitrofurantoin, propylthiouracil, cocaine, bevacizumab)
Diffuse alveolar damage	ARDS, HSCT (IPS), radiation, infection (leptospirosis, dengue, CMV)

- **Suspect DAH:** haemoptysis (absent in 1/3), falling haemoglobin, diffuse GGO/consolidation, hypoxaemia; rising DLCO.
- **Confirm:** BAL — progressively bloodier aliquots; haemosiderin-laden macrophages >20%; investigate cause: ANCA, anti-GBM, ANA, dsDNA, complement, APLA, urinalysis (GN), creatinine, coagulation, echo; renal/lung biopsy.
- **Management:** supportive (O2, NIV/MV with PEEP, correct coagulopathy, platelets), stop offending drug; immune-mediated — pulse methylprednisolone 500–1000 mg ×3 days + rituximab or cyclophosphamide; plasma exchange for anti-GBM (essential) and selected AAV; recombinant factor VIIa/tranexamic acid (local) for refractory; ECMO.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Haemoptysis + haematuria + AKI = pulmonary–renal syndrome — send ANCA and anti-GBM today, start pulse steroids before results.

LANDMARK TRIALS & GUIDELINES

- Walsh M et al, PEXIVAS NEJM 2020; Jayne DRW et al, ADVOCATE NEJM 2021; Wechsler ME et al, MANDARA NEJM 2024.

INVESTIGATIONS & TREATMENT AT A GLANCE

7.7 Pulmonary Vasculitis & Diffuse Alveolar Haemorrhage

‹ Back to topic (p 150)
Next topic 7.8 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
ANCA (PR3/MPO)	Negative	PR3 (GPA), MPO (MPA, EGPA)	Vasculitis
Anti-GBM	Negative	Positive	Goodpasture
BAL sequential aliquots	Clearing	Progressively haemorrhagic; > 20% haemosiderin-laden macrophages	Diffuse alveolar haemorrhage
DLCO	Normal	Raised (> 130%) acutely	Fresh alveolar blood
Urinalysis	No blood/protein	Red cell casts, proteinuria	Pulmonary–renal syndrome
Haemoglobin trend	Stable	Falling	DAH

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Methylprednisolone	500–1000 mg IV daily × 3 days, then prednisolone 1 mg/kg	DAH, severe vasculitis	Infection
Rituximab	375 mg/m² weekly × 4 or 1 g × 2	AAV induction	HBV reactivation
Cyclophosphamide	15 mg/kg IV pulses (adjusted)	AAV induction (alternative)	—
Plasma exchange	7 exchanges over 14 days	Anti-GBM disease; selected AAV with DAH	—
Mepolizumab	300 mg SC every 4 weeks	EGPA	—

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Haemoptysis is absent in a third of DAH — falling Hb with new infiltrates is enough to suspect it.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Granulomatosis with polyangiitis. *[DNB Dec 2021 P2]*
2. Describe Wegener's granulomatosis. *[WBUHS 2020 P2]*
3. Management of EGPA. *[DNB Dec 2025 P3]*
4. ANCA-associated pulmonary vasculitis. *[Manipal 2014 P4]*
5. Enumerate various causes of diffuse alveolar hemorrhage; how to suspect the diagnosis and its management. *[DNB May 2024 P3]*
6. Diffuse alveolar haemorrhage. *[DNB Oct 2023 P2]*
7. Therapy of immune mediated alveolar haemorrhage (DAH). *[DNB Dec 2021 P2]*

7.8 Eosinophilic Lung Diseases — AEP, CEP, Tropical Pulmonary Eosinophilia & HES

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B3 Eosinophilic pneumonias and tropical eosinophilia
- ✓ Classify pulmonary eosinophilias
- ✓ Diagnose and treat acute and chronic eosinophilic pneumonia and TPE
- ✓ Differentiate hypereosinophilic syndromes

IN BRIEF Eosinophilic lung diseases are defined by peripheral and/or BAL eosinophilia (BAL >25% is strongly suggestive) with pulmonary infiltrates. In India, parasitic (TPE, Löffler), ABPA and drug causes must be excluded before idiopathic diagnoses.

Classification of eosinophilic lung disease

Category	Diseases
Idiopathic	Simple pulmonary eosinophilia (Löffler, idiopathic), acute eosinophilic pneumonia, chronic eosinophilic pneumonia, idiopathic HES
Known cause	Parasitic (TPE, Ascaris, hookworm, Strongyloides, Toxocara, Paragonimus), ABPA/ABPM, drugs (NSAIDs, nitrofurantoin, daptomycin, minocycline, sulfa, ACE inhibitors), toxins, radiation
Systemic	EGPA, HES (myeloproliferative, lymphocytic), eosinophilic leukaemia
Others	Asthma, bronchocentric granulomatosis, eosinophilic bronchitis, malignancy

Acute vs chronic eosinophilic pneumonia

Feature	Acute eosinophilic pneumonia	Chronic eosinophilic pneumonia
Patient	Young, recent onset of smoking/vaping, dust (WTC), drugs	Middle-aged women, asthma (50%), atopy
Onset	<1 month (often days); acute respiratory failure	Weeks–months; fever, weight loss, cough
Blood eosinophils	Normal initially (rises later)	High (>1000/µL)
BAL eosinophils	>25%	>25% (often >40%)
Imaging	Bilateral GGO, septal thickening, effusions (ARDS-like)	Peripheral upper-lobe consolidation — "photographic negative of pulmonary oedema"
Treatment	Steroids (methylprednisolone 60–125 mg 6-hourly → oral 2 weeks); stop smoking; no relapse	Prednisolone 0.5 mg/kg; dramatic response in 48 h; relapse 50% on taper; ICS, anti-IL-5 for relapses

Tropical pulmonary eosinophilia

- Hypersensitivity to microfilariae of **Wuchereria bancrofti / Brugia malayi** trapped in lung; Indian subcontinent, SE Asia; young men.
- **Features:** nocturnal paroxysmal cough and wheeze, weight loss, fever; eosinophils >3000/µL (often >10,000), very high IgE (>1000), high anti-filarial antibodies; microfilariae absent in blood; CXR — diffuse reticulonodular mottling; PFT mixed with low DLCO.

- **Treatment: diethylcarbamazine (DEC) 6 mg/kg/day ×3 weeks** (rapid response; 20% relapse — repeat); steroids for severe/fibrosis; untreated → interstitial fibrosis.

Hypereosinophilic syndromes — differential diagnosis

Eosinophilia >1500/µL with organ damage

Type	Features / tests
Myeloproliferative (M-HES)	FIP1L1–PDGFRA fusion (males, splenomegaly, raised B12, tryptase) — imatinib-responsive; CEL
Lymphocytic (L-HES)	Clonal aberrant T cells (CD3−CD4+), skin, IL-5 driven — steroids
Familial, idiopathic	Diagnosis of exclusion
Secondary (reactive)	Parasites (Strongyloides — always test), drugs (DRESS), allergic, EGPA, malignancy (Hodgkin, T-cell lymphoma, solid tumours), adrenal insufficiency

- Organ damage: heart (endomyocardial fibrosis — Löffler endocarditis, thrombi), lung (infiltrates, effusions), skin, CNS, thrombosis. **Treat:** steroids, imatinib (PDGFRA), mepolizumab (approved for HES), hydroxyurea, interferon-α.

★ **CLINICAL PEARLS & EXAM FOCUS**

▸ Very high IgE + eosinophils >3000 + nocturnal wheeze in an Indian young adult = TPE — DEC gives dramatic relief.

LANDMARK TRIALS & GUIDELINES

- Cottin V. Eosinophilic lung diseases, Clin Chest Med 2016; Ong RK, Doyle RL. Tropical pulmonary eosinophilia, Chest 1998.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Clinical features, diagnosis and treatment of acute eosinophilic pneumonia. *[DNB Oct 2024 P2]*
2. Chronic eosinophilic pneumonia. *[DNB Dec 2021 P2]*
3. Tropical pulmonary eosinophilia. *[DNB Dec 2021 P2]*
4. Classification of pulmonary eosinophilias. *[DNB Jun 2021 P2]*
5. Differential diagnosis of hypereosinophilic syndromes (HES). *[DNB Dec 2022 P2]*
6. Eosinophilic lung disease. *[BFUHS 2022 P4]*

7.9 Drug-Induced & Transfusion-Related Lung Disease; Radiation Injury

‹ NMC curriculum
Previous-year Qs ›

◉ **CBME COMPETENCIES — the postgraduate should be able to:**

- ✓ **NMC syllabus area:** II-B3 Reactions of the interstitium to drugs; II-B4 Drug-induced pulmonary disease
- ✓ Recognise patterns and common culprits of drug-induced lung disease
- ✓ Describe methotrexate pneumonitis, amiodarone, bleomycin and checkpoint inhibitor pneumonitis, bevacizumab complications
- ✓ Differentiate TRALI from TACO; describe radiation pneumonitis

IN BRIEF Hundreds of drugs injure the lung (pneumotox.com). Patterns include HP-like, NSIP, OP, eosinophilic pneumonia, DAD, DAH, pleural disease and PAH. Diagnosis is by temporal association and exclusion; treatment is withdrawal ± steroids.

Drug-induced lung disease patterns

Pattern	Drugs
Hypersensitivity / NSIP	Methotrexate, nitrofurantoin (acute), amiodarone
Organising pneumonia	Amiodarone, bleomycin, checkpoint inhibitors, nitrofurantoin, statins
Eosinophilic pneumonia	NSAIDs, nitrofurantoin, daptomycin, minocycline, sulfa
Diffuse alveolar damage / ARDS	Bleomycin, busulfan, cyclophosphamide, gemcitabine, opioids/aspirin overdose

INVESTIGATIONS & TREATMENT AT A GLANCE

7.8 Eosinophilic Lung Diseases — AEP, CEP, Tropical Pulmonary Eosinophilia & HES

‹ Back to topic (p 152)
Next topic 7.9 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Blood eosinophils	< 500/µL	> 1500/µL (CEP/TPE)	Eosinophilic lung disease
BAL eosinophils	< 1%	> 25%	Eosinophilic pneumonia
Total IgE	< 100 IU/mL	> 1000 IU/mL	TPE, ABPA
Filarial antibodies	Low	High	Tropical pulmonary eosinophilia
Stool ova (Strongyloides) / serology	Negative	Positive	Must exclude before steroids
ANCA (MPO)	Negative	Positive (~ 40% of EGPA)	EGPA

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Prednisolone	AEP 40–60 mg/day × 2 weeks; CEP 0.5 mg/kg/day, taper over 6 months	AEP / CEP	Relapse in CEP common
Diethylcarbamazine	6 mg/kg/day × 21 days	TPE	Repeat course if relapse
Ivermectin	200 µg/kg single dose (× 2 days)	Strongyloides before steroids	—
Mepolizumab	300 mg SC 4-weekly	EGPA, HES	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Acute eosinophilic pneumonia — think new smoking or vaping; peripheral eosinophils may be normal initially.

Pattern	Drugs
Alveolar haemorrhage	Anticoagulants, PTU, bevacizumab, cocaine
Pulmonary fibrosis (chronic)	Bleomycin, busulfan, nitrofurantoin (chronic), amiodarone
PAH	Dexfenfluramine, dasatinib, methamphetamine, interferon
Pleural	Bromocriptine, ergots, drug-induced lupus (procainamide, hydralazine, INH)

Methotrexate pneumonitis

- Idiosyncratic hypersensitivity (not dose-related), 0.3–7%, usually within first year; subacute cough, fever, dyspnoea, crackles; risk — age, diabetes, hypoalbuminaemia, pre-existing lung disease, RA lung.
- HRCT: diffuse GGO/HP-like; BAL lymphocytosis; exclude infection (PJP!).
- **Management:** stop MTX permanently, prednisolone 1 mg/kg tapering; mortality up to 15%.

Amiodarone lung toxicity

- 1–5%; dose >400 mg/day, duration >2 months, elderly, prior lung disease; high-FiO2 surgery.
- HRCT: peripheral consolidation with **high attenuation** (iodine) in lung, liver, spleen; BAL foamy macrophages; low DLCO.
- Stop drug, steroids (prolonged due to long half-life).

Bevacizumab-induced complications

- Life-threatening haemoptysis (squamous histology, central cavitating tumours — contraindicated), PE/VTE, BPF and tracheo-oesophageal fistula, impaired wound healing, hypertension, proteinuria, GI perforation.

Checkpoint inhibitor pneumonitis

- 3–5% (PD-1 > PD-L1; higher with combination, NSCLC, radiotherapy); median onset 2–3 months; OP, NSIP, HP, DAD patterns.
- **Grading (CTCAE):** G1 radiological only — hold/observe; G2 symptomatic — hold, prednisolone 1–2 mg/kg; G3–4 — permanently discontinue, methylprednisolone 2–4 mg/kg; refractory → infliximab, MMF, IVIG.

Transfusion-induced lung injury

TRALI vs TACO

Feature	TRALI	TACO
Mechanism	Donor anti-HLA/HNA antibodies → neutrophil activation (two-hit)	Volume overload (hydrostatic)
Onset	Within 6 h	Within 12 h
BP	Hypotension	Hypertension
JVP, BNP	Normal, BNP low	Raised, BNP high (>1.5× pre)
Fever	Common	Uncommon
Response to diuretics	No (may worsen)	Yes
Echo	Normal LV function	LV dysfunction/overload

- Management: stop transfusion, supportive (O2, MV with lung protection) for TRALI; diuretics for TACO; report to haemovigilance (HvPI); prevention — male-only plasma, HLA-antibody screening of female donors.

Radiation lung injury

- **Radiation pneumonitis:** 1–6 months after RT (V20 >35%, mean lung dose >20 Gy, concurrent chemotherapy, ILD); GGO/consolidation conforming to the radiation port (straight edges) — prednisolone 1 mg/kg tapered over weeks.
- **Radiation fibrosis:** 6–24 months — volume loss, traction bronchiectasis within field; no specific therapy.
- OP outside field (breast RT), radiation recall (with chemotherapy).

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★ CLINICAL PEARLS & EXAM FOCUS

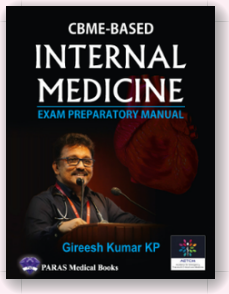
▸ Infiltrate with straight borders not respecting anatomy = radiation field.

LANDMARK TRIALS & GUIDELINES

• Pneumotox online (www.pneumotox.com); Brahmer JR et al, ASCO irAE guideline 2021; Semple JW et al, TRALI redefinition (Transfusion 2019).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Methotrexate-induced pneumonitis. *[DNB Dec 2021 P2]*
2. Bevacizumab induced lung complications. *[DNB Dec 2021 P2]*
3. Blood transfusion induced lung injuries. *[DNB Oct 2023 P2]*



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7.10 Cystic Lung Diseases — LAM, PLCH, BHD & LIP

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B4 Other pulmonary diseases of unknown etiology including PLCH, LAM
- ✓ Approach diffuse cystic lung disease by HRCT features
- ✓ Describe pulmonary and extrapulmonary manifestations of LAM, PLCH and BHD
- ✓ Treat LAM with sirolimus and PLCH with smoking cessation

IN BRIEF Diffuse cystic lung diseases (DCLDs) feature multiple thin-walled (<2 mm) air-filled cysts. HRCT distribution and associated findings, plus extrapulmonary clues and a few blood tests (VEGF-D), allow non-invasive diagnosis in most cases.

HRCT differentiation of cystic lung diseases

Disease	Cyst features	Associated findings
LAM	Round, uniform, thin-walled, diffuse (including bases and costophrenic angles)	Chylous effusion, angiomyolipomas, lymphangioleiomyomas
PLCH	Bizarre shapes (bilobed, cloverleaf), upper/mid zones, spare costophrenic angles	Nodules (some cavitating), smokers
BHD	Few, lentiform, basal, subpleural/paramediastinal, abutting vessels	Pneumothorax, skin fibrofolliculomas, renal tumours
LIP / Sjögren	Few, perivascular, lower lobes	GGO, nodules, Sjögren features
Amyloid/LCDD	Cysts with nodules (calcified)	Sjögren, plasma cell disorder
Others	PJP pneumatoceles, emphysema (no walls), honeycombing (subpleural stacked)	Context

INVESTIGATIONS & TREATMENT AT A GLANCE

7.9 Drug-Induced & Transfusion-Related Lung Disease; Radiation Injury

‹ Back to topic (p 153)
Next topic 7.10 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Drug history (pneumotox.com)	—	Amiodarone, methotrexate, nitrofurantoin, bleomycin, checkpoint inhibitors	Drug-induced ILD
DLCO	Stable	Fall > 15–20%	Early drug toxicity
HRCT	—	OP, NSIP, HP, DAD patterns; high-attenuation (amiodarone)	Pattern
BNP / echo	Normal	Raised	TACO vs TRALI
Timing after transfusion	—	Within 6 h, non-cardiogenic	TRALI
Radiation field correlation	—	Straight-edged infiltrate within field (6–12 weeks)	Radiation pneumonitis

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Stop culprit drug	—	Drug-induced ILD	—
Prednisolone	0.5–1 mg/kg/day, taper over weeks–months	Moderate–severe drug or radiation pneumonitis	—
Checkpoint inhibitor pneumonitis	Grade 2: pred 1 mg/kg; grade ≥ 3: methylpred 2 mg/kg; infliximab/MMF if refractory	ICI pneumonitis	Hold ICI
Supportive (TRALI)	O_2, ventilation; diuretics avoided	TRALI	Report to blood bank

★ PRESCRIBING & INTERPRETATION PEARLS

- TACO responds to diuretics; TRALI does not.

Pulmonary and extrapulmonary manifestations

Disease	Pulmonary	Extrapulmonary
LAM (women of child-bearing age; sporadic or TSC)	Progressive dyspnoea, recurrent pneumothorax (60–70%), chylothorax, chyloptysis, haemoptysis, obstruction with low DLCO	Renal angiomyolipomas, abdominal lymphangioleiomyomas, chylous ascites; TSC — skin, CNS, seizures
PLCH (young smokers)	Cough, dyspnoea, pneumothorax, PH	Diabetes insipidus, bone lytic lesions, skin; BRAF V600E in ~50%
BHD (FLCN mutation, AD)	Recurrent pneumothorax	Fibrofolliculomas, trichodiscomas, renal cell carcinoma (chromophobe/oncocytic)

Diagnosis

- **LAM:** HRCT + **serum VEGF-D ≥800 pg/mL** (diagnostic in compatible CT), or TSC, AML, chylous collections, or biopsy (HMB-45+, SMA+, ER/PR+ LAM cells).
- **PLCH:** HRCT in smoker; BAL CD1a >5%; biopsy (Langerhans cells CD1a/langerin+, Birbeck granules on EM).
- **BHD:** FLCN genetic testing; skin biopsy.

Treatment

- **LAM: sirolimus** (mTOR inhibitor; MILES trial — stabilised FVC/FEV1, improved QoL; trough 5–15 ng/mL) for declining lung function, FEV1 <70%, chylous effusions; avoid oestrogens; pleurodesis after first pneumothorax; lung transplant.
- **PLCH: smoking cessation** (resolution/stabilisation in many); steroids limited; cladribine or BRAF/MEK inhibitors for progressive/multisystem disease; PH therapy cautiously; transplant (recurrence possible).
- **BHD:** pleurodesis for pneumothorax, renal surveillance MRI.

★ **CLINICAL PEARLS & EXAM FOCUS**

▸ Young woman with recurrent pneumothorax + chylothorax + renal AML = LAM; check VEGF-D.

LANDMARK TRIALS & GUIDELINES

- McCormack FX et al, MILES trial NEJM 2011; ATS/JRS LAM guideline AJRCCM 2016 and 2017; Gupta N et al, ATS DCLD statement 2023.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Pulmonary and extrapulmonary manifestations of cystic interstitial lung diseases. *[DNB Jun 2025 P2]*
2. HRCT chest findings in cystic interstitial lung diseases. *[DNB Jun 2025 P2]*

7.11 Pulmonary Alveolar Proteinosis, Alveolar Microlithiasis & Amyloidosis

‹ NMC curriculum
Previous-year Qs ›

◉ **CBME COMPETENCIES — the postgraduate should be able to:**

- ✓ **NMC syllabus area:** II-B4 PAP, alveolar microlithiasis, uncommon diseases
- ✓ Describe the pathogenesis, diagnosis and treatment of PAP including whole lung lavage
- ✓ Recognise pulmonary alveolar microlithiasis and pulmonary amyloidosis

IN BRIEF PAP is accumulation of surfactant in alveoli due to impaired macrophage clearance — 90% autoimmune (anti-GM-CSF antibodies). Whole lung lavage remains the standard therapy; inhaled GM-CSF is an effective newer option.

INVESTIGATIONS & TREATMENT AT A GLANCE

7.10 Cystic Lung Diseases — LAM, PLCH, BHD & LIP

‹ Back to topic (p 155)
Next topic 7.11 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
HRCT	—	Thin-walled diffuse round cysts (LAM); bizarre upper-lobe cysts + nodules (PLCH)	Cyst pattern
Serum VEGF-D	< 600 pg/mL	≥ 800 pg/mL	Diagnostic of LAM (with cysts)
FLCN gene testing	—	Mutation	Birt–Hogg–Dubé
Smoking history	—	Heavy smoker	PLCH
Anti-SSA / immunoglobulins	—	Positive / raised	LIP (Sjögren)
Renal imaging	—	Angiomyolipoma (LAM/TSC), renal tumours (BHD)	Associations

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Sirolimus	2 mg daily (trough 5–15 ng/mL)	LAM with declining FEV1 (MILES)	Mouth ulcers, hyperlipidaemia
Smoking cessation	—	PLCH	Mainstay
Pleurodesis	After first pneumothorax	LAM, BHD	Transplant considerations
Avoid oestrogens	—	LAM	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Recurrent pneumothorax in a young woman → think LAM; check VEGF-D.

Classification of PAP

Type	Cause
Autoimmune (90%)	Anti-GM-CSF autoantibodies
Secondary	Haematological malignancy (MDS, CML), silica/dust inhalation (acute silicoproteinosis), immunodeficiency, infections
Congenital/hereditary	CSF2RA/B mutations (GM-CSF receptor), surfactant protein (SFTPB, SFTPC, ABCA3) mutations

Clinical and diagnostic features

- Insidious dyspnoea, cough, "clinical–radiological dissociation" (extensive CT changes, mild symptoms); secondary infections (Nocardia, NTM, fungi).
- **HRCT:** geographic GGO with smooth septal thickening — **crazy paving**.
- **BAL:** milky, opaque fluid; PAS-positive acellular material, foamy macrophages; serum anti-GM-CSF antibody (diagnostic for autoimmune PAP); serum KL-6, LDH elevated.

Whole lung lavage

- Under general anaesthesia with double-lumen endotracheal tube; one lung ventilated while the other is filled with warm saline (1 L aliquots, up to 15–50 L total) and drained with chest percussion until effluent clears; the other lung lavaged days later.
- **Indications:** dyspnoea limiting activity, PaO2 <65 mmHg, A–a gradient >40, shunt >10–12%.
- **Complications:** hypoxaemia, hydropneumothorax, fluid spill to ventilated lung, infection, hypothermia.
- Response durable in ~2/3; repeat lavages often needed.
- **Inhaled GM-CSF (molgramostim, sargramostim):** improves A–a gradient and DLCO (IMPALA, PAGE, IMPALA-2 trials); rituximab, plasmapheresis for refractory; lung transplant (recurrence).

Pulmonary alveolar microlithiasis

- Autosomal recessive SLC34A2 (type IIb sodium-phosphate cotransporter) mutation → calcium phosphate microliths in alveoli; India and Turkey have many cases.
- **Clinical–radiological dissociation:** "sandstorm" lung on CXR, black pleura sign (subpleural cysts), crazy paving with calcification on CT; progressive respiratory failure over decades.
- No effective treatment (etidronate ineffective); lung transplantation.

Pulmonary amyloidosis

- Forms: tracheobronchial (submucosal plaques — wheeze, stenosis), nodular (single/multiple, calcify), diffuse alveolar septal (AL systemic — poor prognosis), pleural effusion, mediastinal nodes.
- Diagnosis: Congo red apple-green birefringence; typing by mass spectrometry; treat underlying plasma cell dyscrasia; bronchoscopic resection/radiotherapy for airway disease.

★ CLINICAL PEARLS & EXAM FOCUS

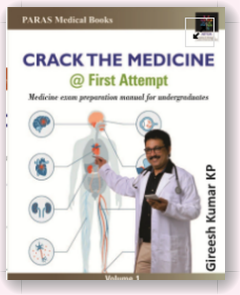
▸ Crazy paving: PAP, PJP, DAH, ARDS, lipoid pneumonia, mucinous adenocarcinoma — BAL usually distinguishes them.

LANDMARK TRIALS & GUIDELINES

- Trapnell BC et al, IMPALA molgramostim NEJM 2020; Trapnell BC et al, IMPALA-2 NEJM 2025.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Pulmonary alveolar proteinosis (PAP). *[DNB Oct 2023 P2]*
2. Describe whole lung lavage in pulmonary alveolar proteinosis. *[WBUHS 2020 P2]*
3. Whole lung lavage. *[BFUHS 2020 P4]*



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INVESTIGATIONS & TREATMENT AT A GLANCE

7.11 Pulmonary Alveolar Proteinosis, Alveolar Microlithiasis & Amyloidosis

‹ Back to topic (p 156)
Next topic 8.1 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
HRCT	—	Crazy-paving (PAP); "sandstorm" calcified micronodules (PAM)	Pattern
BAL appearance (PAP)	Clear	Milky, PAS-positive material	Diagnostic
Anti-GM-CSF antibody	Negative	Positive	Autoimmune PAP
Serum LDH / KL-6	Normal	Raised	PAP activity
Congo red / fat-pad biopsy	Negative	Apple-green birefringence	Amyloidosis
Serum free light chains	κ:λ 0.26–1.65	Abnormal ratio	AL amyloid

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Whole-lung lavage	10–20 L saline per lung under GA	PAP with hypoxaemia	Specialist centre
Inhaled GM-CSF (molgramostim)	300 µg nebulised daily (IMPALA-2)	Autoimmune PAP	—
Supportive / transplant	—	PAM	No proven therapy
Chemotherapy (bortezomib-based)	Haematology protocol	Systemic AL amyloidosis	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Crazy paving is not specific to PAP — also oedema, haemorrhage, PCP, mucinous adenocarcinoma.

SECTION 8

Occupational & Environmental Lung Disease, Smoking & Inhalational Injury

Paper II / III

Occupational lung diseases and pneumoconioses; air-pollution induced disease and global warming; health hazards of smoking and smoking cessation; toxic lung, inhalational injury, chemical/biological agents and radiation (NMC syllabus II-B3, II-B4, I-E4, Preventive pulmonology).

8.1 Approach to Occupational Lung Disease; Silicosis 159
8.2 Coal Workers' Pneumoconiosis, Asbestos-Related Diseases, Byssinosis & Other Dusts 161
8.3 Air Pollution, Ozone & Climate Change — Respiratory and Extrapulmonary Effects 162
8.4 Health Hazards of Smoking & Smoking Cessation 164
8.5 Toxic Inhalational Injury, Chemical & Biological Agents, Radiation Sickness & Strangulation 165

8.1 Approach to Occupational Lung Disease; Silicosis

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B4 Occupational lung diseases and pneumoconiosis
- ✓ Take an occupational history and classify occupational lung diseases
- ✓ Describe the pathogenesis, radiology (ILO classification) and diseases associated with silica exposure
- ✓ Describe compensation and prevention under Indian law

IN BRIEF Pneumoconiosis is the non-neoplastic reaction of the lung to inhaled mineral dust. Silicosis — from crystalline silica in mining, stone crushing, sandblasting, quarrying, slate-pencil and agate industries — is the commonest in India and a notifiable, compensable but incurable disease.

Classification of occupational lung diseases

Category	Examples
Pneumoconioses	Silicosis, coal workers' pneumoconiosis, asbestosis, berylliosis, siderosis, stannosis, baritosis, talcosis, hard-metal lung disease
Airway diseases	Occupational asthma, RADS, byssinosis, COPD from dusts/fumes, constrictive bronchiolitis (diacetyl)
Hypersensitivity pneumonitis	Farmer's lung, bagassosis, mushroom worker's
Infections	TB (silica, healthcare), anthrax (wool sorters), Q fever, psittacosis, brucellosis
Malignancy	Mesothelioma, lung cancer (asbestos, silica, radon, arsenic, chromium, nickel, diesel exhaust)
Acute toxic injury	Chlorine, ammonia, phosgene, NO2 (silo filler), metal fume fever, polymer fume fever

Occupational history

- All jobs held (chronological), tasks, materials, duration and intensity of exposure, protective equipment, ventilation, similar illness in co-workers, symptoms in relation to work, hobbies (birds), home environment (biomass).

Silicosis — pathogenesis

- Respirable crystalline silica (quartz, cristobalite, tridymite; <5 µm) → engulfed by alveolar macrophages → lysosomal membrane damage, NLRP3 inflammasome activation, IL-1β, ROS → macrophage death and re-ingestion cycle → fibroblast activation (TGF-β, PDGF) → **silicotic nodule** (concentric whorled hyalinised collagen, birefringent particles under polarised light) in perilymphatic regions; nodes with eggshell calcification.

Clinical forms of silicosis

Form	Exposure	Features
Chronic simple	>10 years, low–moderate	Small rounded upper-zone nodules; often asymptomatic
Progressive massive fibrosis (complicated)	Continued exposure	Conglomerate masses >1 cm, upper zones, migrate to hila ("angel wings"), emphysema, respiratory failure
Accelerated	5–10 years, high	Rapid progression, autoimmunity
Acute silicoproteinosis	Weeks–5 years, very high (sandblasting, tunnelling)	PAP-like, crazy paving, rapid fatal failure

Radiological features

- Small rounded opacities (p <1.5 mm, q 1.5–3 mm, r 3–10 mm) in upper/posterior zones, perilymphatic on HRCT (subpleural, centrilobular); hilar/mediastinal nodes with **eggshell calcification** (5–10%); PMF — bilateral symmetrical upper-zone masses with peripheral emphysema, may cavitate (TB!).
- **ILO International Classification of Radiographs of Pneumoconioses (2022 revision, digital images):** profusion 12-point scale (0/– to 3/+), shape/size of small opacities (rounded p, q, r; irregular s, t, u), zones affected, large opacities (A, B, C), pleural abnormalities.

Diseases associated with silica exposure

Category	Associations
Infections	**Tuberculosis** (3–30× risk; silicotuberculosis), NTM, nocardia, fungi
Malignancy	Lung cancer (IARC group 1)
Airway	COPD, chronic bronchitis
Autoimmune	Systemic sclerosis (Erasmus syndrome), RA (Caplan's), SLE, ANCA vasculitis
Renal	Silica nephropathy, CKD
Others	Cor pulmonale, pneumothorax, broncholithiasis

Management and prevention

- No proven therapy reverses silicosis; stop exposure, treat TB (screen annually; TPT for TBI), vaccines, rehabilitation, LTOT, treat airflow obstruction; whole lung lavage (acute), nintedanib in progressive fibrotic pneumoconiosis (INBUILD included), lung transplantation; tetrandrine (China), pirfenidone under study.
- **Prevention:** substitution (non-silica abrasives), wet drilling, local exhaust ventilation, enclosures, respirators (N95/P100), PEL (0.05 mg/m^3 respirable silica), medical surveillance (pre-placement and periodic CXR, spirometry).
- **Indian legal framework:** notifiable under Factories Act 1948 and Mines Act 1952; compensation under Employees' Compensation Act 1923 / ESI Act; Rajasthan Pneumoconiosis Policy (2019) — compensation ₹3 lakh (diagnosis) + ₹2 lakh (death), certified by Pneumoconiosis Board; Occupational Safety, Health and Working Conditions Code 2020; NIOH Ahmedabad.

★ **CLINICAL PEARLS & EXAM FOCUS**

▸ Upper-zone nodules + eggshell nodes in a stone-crusher = silicosis — and screen him for TB every year.

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LANDMARK TRIALS & GUIDELINES

- ILO Guidelines for the use of the ILO International Classification of Radiographs of Pneumoconioses (revised 2022); Leung CC et al, Silicosis, Lancet 2012.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Radiological features of silicosis. *[DNB Jun 2021 P1]*
2. Describe pathogenesis of silicosis and diseases associated with exposure to silica dust. *[BFUHS 2022 P3]*
3. Silicosis. *[Manipal 2014 P4]*
4. Describe pulmonary diseases caused by inhaled inorganic dust. *[WBUHS 2020 P2]*

8.2 Coal Workers' Pneumoconiosis, Asbestos-Related Diseases, Byssinosis & Other Dusts

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B4 Occupational lung diseases
- ✓ Describe CWP and its prevention
- ✓ Describe asbestos-related pleural and parenchymal diseases with radiological features
- ✓ Recognise byssinosis, berylliosis and metal-induced lung diseases

IN BRIEF Coal dust causes simple CWP, PMF, COPD and Caplan's syndrome. Asbestos causes a spectrum of pleural disease (plaques, effusion, diffuse thickening, round atelectasis, mesothelioma) and parenchymal disease (asbestosis, lung cancer) after long latency.

Coal workers' pneumoconiosis

- Coal macule (dust-laden macrophages around respiratory bronchioles with focal emphysema) → simple CWP (small rounded opacities) → PMF; also chronic bronchitis/COPD, Caplan's (rheumatoid nodules), TB risk (lower than silica).
- **Prevention — primary:** dust suppression (water sprays, ventilation, continuous mining with dust extraction), lower exposure limits, respirators, job rotation; **secondary:** periodic CXR and spirometry (surveillance programme), removal from exposure of workers with early disease, smoking cessation; **tertiary:** compensation, rehabilitation, TB screening.

Asbestos-related diseases

Disease	Latency	Features
Pleural plaques	20–30 years	Commonest; parietal pleura (posterolateral, diaphragmatic), calcified "holly-leaf"/ candle-wax; marker of exposure; benign
Benign asbestos pleural effusion	10–20 years	Earliest; exudative, eosinophilic/bloody; diagnosis of exclusion
Diffuse pleural thickening	15–30 years	Visceral pleura, costophrenic angle obliteration, restriction
Round atelectasis	—	Peripheral mass with comet-tail of vessels, adjacent pleural thickening
Asbestosis	20–30 years, dose-dependent	Basal subpleural fibrosis (UIP-like), subpleural curvilinear lines, parenchymal bands, plaques; asbestos bodies (ferruginous bodies) in BAL/biopsy
Lung cancer	20–40 years	Synergy with smoking (multiplicative — 50–90×)
Mesothelioma	30–50 years	Pleura/peritoneum; crocidolite (blue) > amosite > chrysotile

- **Asbestos types:** serpentine (chrysotile — white, curly) and amphiboles (crocidolite, amosite, tremolite — straight needle-like, most carcinogenic). India still uses chrysotile (ban on mining but not use).

Byssinosis

- Cotton, flax, hemp, sisal dust (endotoxin) — textile mill workers (Mumbai, Ahmedabad, Coimbatore).

INVESTIGATIONS & TREATMENT AT A GLANCE

8.1 Approach to Occupational Lung Disease; Silicosis

‹ Back to topic (p 159)
Next topic 8.2 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
CXR ILO classification	0/0	Profusion ≥ 1/0; small rounded opacities p, q, r (upper zones)	Silicosis
HRCT	—	Centrilobular/perilymphatic nodules, eggshell calcified nodes, PMF	Silicosis/PMF
Spirometry/DLCO	Normal	Restrictive/mixed; ↓ DLCO	Functional impairment
Sputum Xpert / culture	Negative	Positive	Silicotuberculosis — screen all
IGRA/TST	Negative	Positive	TPT indicated (WHO strong)
Occupational history	—	Stone crushing, quarrying, sandblasting, agate	Exposure

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Removal from exposure	—	All	Compensation (ESI Act, Workmen's Compensation)
TPT	3HP / 6H	Silicosis with TB infection	—
ATT (silicotuberculosis)	Standard regimen; some extend to 8–9 months	Active TB	—
Whole-lung lavage	—	Acute silicoproteinosis	Limited evidence
Lung transplantation	—	End-stage silicosis	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Silicosis is a notifiable occupational disease; TB risk is increased about 30-fold.

- **"Monday morning fever/chest tightness"** — symptoms on first day after break, improving through week; acute cross-shift FEV1 decline; chronic → COPD.
- **Schilling grades:** 0, ½ (occasional Monday tightness), 1 (every Monday), 2 (Monday + other days), 3 (permanent with impairment).
- Prevention: dust control, steaming of cotton, surveillance; bronchodilators, removal from exposure.

Other occupational lung diseases

Agent	Disease	Clue
Beryllium	Chronic beryllium disease	Sarcoid-like; BeLPT positive; aerospace, electronics
Hard metal (cobalt, tungsten carbide)	Giant cell interstitial pneumonia	Bizarre multinucleated giant cells in BAL
Iron	Siderosis (welders)	Benign; dense micronodules
Tin, barium	Stannosis, baritosis	Very dense opacities, benign
Talc	Talcosis	Cosmetic, IV drug users (talc granulomatosis)
Synthetic stone (engineered quartz)	Accelerated silicosis	Kitchen countertop workers — emerging epidemic
Indium	PAP/fibrosis	Electronics

★ CLINICAL PEARLS & EXAM FOCUS

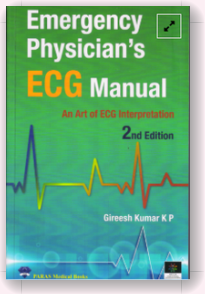
▸ Pleural plaques = exposure, not disease; asbestosis requires parenchymal fibrosis + exposure history ± asbestos bodies.

LANDMARK TRIALS & GUIDELINES

- ATS official statement: diagnosis and initial management of non-malignant diseases related to asbestos (2004); Helsinki criteria (2014 update).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Radiographic features of asbestosis in respiratory system. *[DNB Oct 2023 P2]*
2. Asbestos related pleural disorders. *[BFUHS 2023 P2]*
3. Primary and secondary prevention of coal workers' pneumoconiosis. *[BFUHS 2020 P3]*



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8.3 Air Pollution, Ozone & Climate Change — Respiratory and Extrapulmonary Effects

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B4 Air-pollution induced diseases; I-E4; Misc. Global warming
- ✓ Describe major air pollutants, Indian NAAQS and AQI
- ✓ Describe pulmonary and extrapulmonary effects of ambient and household air pollution
- ✓ Describe health impacts of global warming relevant to respiratory medicine

INVESTIGATIONS & TREATMENT AT A GLANCE

8.2 Coal Workers' Pneumoconiosis, Asbestos-Related Diseases, Byssinosis & Other Dusts

‹ Back to topic (p 161)
Next topic 8.3 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
CXR / HRCT (asbestos)	—	Pleural plaques (calcified, diaphragmatic), lower-zone fibrosis, round atelectasis	Asbestos-related disease
Spirometry / DLCO	Normal	Restriction, ↓ DLCO	Asbestosis
Pleural fluid / biopsy	—	Exudate; malignant cells	Benign asbestos effusion vs mesothelioma
Asbestos bodies (BAL)	< 1/mL	> 1/mL	Exposure marker
Serial PEF / spirometry (byssinosis)	—	Monday-morning FEV1 fall	Byssinosis (cotton dust)
CXR (coal workers)	—	Small rounded opacities; PMF; Caplan nodules (RA)	CWP

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Removal from exposure	—	All pneumoconioses	Compensation
Smoking cessation	—	Synergistic lung cancer risk with asbestos	—
Bronchodilators	Per COPD	Byssinosis, airflow obstruction	—
Mesothelioma pathway	Oncology referral	Malignant effusion	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Asbestos + smoking: multiplicative lung cancer risk (~ 50-fold).

IN BRIEF Air pollution is India's leading environmental risk factor (~1.7 million deaths/year, GBD 2019). PM2.5 penetrates alveoli and enters the circulation, causing respiratory, cardiovascular, metabolic and neurological disease; household biomass smoke is a major cause of COPD in non-smoking women.

Indian National Ambient Air Quality Standards (CPCB 2009)

Pollutant	24-hour	Annual
PM2.5	60 µg/m³	40 µg/m³ (WHO 2021 annual guideline: 5)
PM10	100 µg/m³	60 µg/m³ (WHO: 15)
NO2	80 µg/m³	40 µg/m³
SO2	80 µg/m³	50 µg/m³
Ozone (O3)	180 (1-h); 100 (8-h) µg/m³	—
CO	4 mg/m³ (1-h); 2 mg/m³ (8-h)	—
Lead, NH3, benzene, benzo(a)pyrene, arsenic, nickel	Specified	Specified

- **AQI (India, 2014):** Good 0–50, Satisfactory 51–100, Moderate 101–200, Poor 201–300, Very poor 301–400, Severe 401–500 — linked to health advisories and GRAP (Graded Response Action Plan, Delhi NCR).
- **National Clean Air Programme (NCAP, 2019):** 40% reduction in PM10 by 2025–26 in 131 non-attainment cities; Pradhan Mantri Ujjwala Yojana (LPG for poor households).

Respiratory effects of ambient air pollution

Effect	Details
Acute	Exacerbations of asthma and COPD, increased respiratory infections (children — ALRI), hospital admissions, mortality spikes
Chronic	Reduced lung growth in children, accelerated FEV1 decline, incident asthma and COPD, lung cancer (PM2.5 — IARC group 1; EGFR-mutant adenocarcinoma in never-smokers), ILD progression, TB risk
Ozone	Oxidant — airway inflammation, neutrophilia, decreased FEV1 (esp. exercising outdoors, summer afternoons), AHR, asthma exacerbations; tropospheric O3 from NOx + VOCs in sunlight

Extrapulmonary effects of air pollution

- **Cardiovascular:** MI, stroke, arrhythmia, HF, hypertension, atherosclerosis (systemic inflammation, endothelial dysfunction, autonomic imbalance, thrombosis).
- **Metabolic:** diabetes, insulin resistance; **renal:** CKD; **neurological:** dementia, cognitive decline, stroke, depression, autism risk; **pregnancy:** low birth weight, preterm birth, stillbirth; **others:** conjunctivitis, skin ageing, bone loss, cancers (bladder, breast).

Household air pollution

- Solid fuel combustion (wood, dung, crop residue, coal) in poorly ventilated kitchens — PM2.5 levels often >500 µg/m³; COPD (women), ALRI in children, TB, cataract, lung cancer, low birth weight.
- Interventions: clean fuels (LPG — PMUY), improved cookstoves, ventilation, chimneys.

Global warming and respiratory health

- Heat waves (dehydration, worsened COPD), increased ozone and wildfire smoke, longer and more intense pollen seasons (allergic rhinitis/asthma), thunderstorm asthma, dust storms, changes in vector-borne and fungal (e.g., Coccidioides range) infections, floods and mould exposure, climate migration, air conditioning disparities.
- Mitigation: low-carbon inhalers (DPIs over pMDIs with HFC propellants), sustainable healthcare.

Individual protection advice

- Check AQI, avoid outdoor exercise on poor-AQI days and near traffic, N95 masks for vulnerable when AQI poor, indoor air purifiers (HEPA), keep rescue medications, action plans for asthma/COPD.

★ CLINICAL PEARLS & EXAM FOCUS

▸ WHO 2021 lowered the annual PM2.5 guideline to 5 µg/m³ — Indian standard (40) is eight times higher.

LANDMARK TRIALS & GUIDELINES

- WHO Global Air Quality Guidelines 2021; India State-Level Disease Burden Initiative Air Pollution Collaborators, Lancet Planet Health 2021.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Describe the lung problems of poor ambient air quality; national standards of ambient air quality. *[DNB May 2024 P3]*
2. Respiratory effects of ambient air pollution. *[BFUHS 2020 P3]*
3. Extrapulmonary effects of air pollution. *[BFUHS 2020 P4]*
4. Ozone effect. *[VNSGU 2025 P3]*

8.4 Health Hazards of Smoking & Smoking Cessation

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B4 Health hazards of smoking; Preventive pulmonology
- ✓ Describe health hazards of tobacco (smoked and smokeless) and e-cigarettes
- ✓ Describe pharmacological and non-pharmacological approaches to cessation (5As, 5Rs)
- ✓ Describe COTPA and quit-clinic activities

IN BRIEF India has ~267 million tobacco users (GATS-2, 2016–17) — 29% of adults, with smokeless tobacco commoner than smoking. Tobacco kills ~1.35 million Indians a year. Combining behavioural support with pharmacotherapy (varenicline or combination NRT) gives the highest quit rates.

Health hazards of tobacco

System	Effects
Respiratory	COPD, lung cancer (85%), asthma worsening, ILDs (RB-ILD, DIP, PLCH, CPFE, AEP), TB (2–3×), pneumonia, spontaneous pneumothorax
Cardiovascular	IHD, stroke, PAD (Buerger's), aortic aneurysm, sudden death
Cancers	Oral (smokeless — India's commonest male cancer), larynx, oesophagus, pancreas, bladder, kidney, cervix, AML
Others	Peptic ulcer, osteoporosis, cataract, macular degeneration, infertility, erectile dysfunction, diabetes, poor wound healing
Pregnancy	Low birth weight, preterm, stillbirth, SIDS, placental abruption
Second-hand smoke	Lung cancer, IHD, childhood asthma, otitis media, SIDS

- **E-cigarettes/vapes:** nicotine addiction, EVALI (vitamin E acetate), gateway in youth; banned in India (Prohibition of Electronic Cigarettes Act, 2019).

Non-pharmacological approaches

- **5 As** (willing to quit): Ask, Advise, Assess, Assist, Arrange follow-up. **5 Rs** (unwilling): Relevance, Risks, Rewards, Roadblocks, Repetition. **Very Brief Advice** (Ask–Advise–Act) — 30 seconds.

INVESTIGATIONS & TREATMENT AT A GLANCE

8.3 Air Pollution, Ozone & Climate Change — Respiratory and Extrapulmonary Effects

‹ Back to topic (p 162)
Next topic 8.4 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
AQI (India)	0–50 good	> 200 poor; > 300 very poor; > 400 severe	Advise vulnerable groups
PM2.5 (24-h)	WHO guideline 15 µg/m³; NAAQS India 60	> 60 µg/m³	Health risk
COHb (household biomass)	< 3%	Raised	Indoor air pollution exposure
Spirometry in exposed	Normal	Obstruction	Pollution-related COPD
PEF diary on high-pollution days	Stable	Fall > 20%	Exacerbation trigger

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
N95 mask	During poor AQI	Vulnerable patients outdoors	Fit; not for long continuous use in severe lung disease
Indoor air purifier (HEPA)	Room-size matched	High-pollution periods	—
Action plan step-up	Increase reliever / MART	Asthma/COPD on bad AQI days	—
Clean cooking fuel (LPG)	—	Biomass users	PMUY scheme

★ PRESCRIBING & INTERPRETATION PEARLS

- Advise patients to check AQI before outdoor exercise — avoid outdoor exertion when AQI > 200.

- Behavioural counselling (motivational interviewing, CBT), quit-line (India **1800-112-356**), mCessation ("mTobaccoCessation" SMS — missed call 011-22901701), group therapy, self-help materials, yoga, physical activity.
- Fagerström Test for Nicotine Dependence (FTND); exhaled CO monitoring.

Pharmacotherapy

Drug	Dose	Notes
Nicotine replacement (patch 21/14/7 mg, gum 2/4 mg, lozenge, inhaler, spray)	Patch 21 mg/day if >10 cigarettes; taper over 8–12 weeks	Combination NRT (patch + short-acting) ≈ varenicline efficacy
Varenicline (α4β2 partial agonist)	0.5 mg OD ×3 days → 0.5 mg BD ×4 days → 1 mg BD ×12 weeks (quit day at 1–2 weeks)	Most effective single agent (EAGLES); nausea, vivid dreams; neuropsychiatric safety confirmed
Bupropion SR (NDRI)	150 mg OD ×3 days → BD ×7–12 weeks	Contraindicated in seizures, eating disorders, MAOI
Cytisine	25-day course	Cheap, effective (ORCA-2)
Second-line	Nortriptyline, clonidine	—

COTPA (Cigarettes and Other Tobacco Products Act, 2003)

- Section 4 — ban on smoking in public places (2008 rules); Section 5 — ban on advertising, sponsorship; Section 6a — no sale to minors <18; 6b — no sale within 100 yards of educational institutions; Section 7 — pictorial health warnings (85% of pack surface); Section 7–9 — packaging/labelling; penalties.
- Other measures: National Tobacco Control Programme (2007–08), tobacco cessation centres, WHO FCTC ratified 2004, taxation (GST + cess), ban on gutka/pan masala with tobacco (FSSA 2011), COTPA amendments on OTT content warnings.

Quit-smoking clinic activities

- Registration, assessment (tobacco history, FTND, CO), motivational counselling, quit plan and date, pharmacotherapy, follow-up at 1, 2, 4, 6, 12 weeks and 6 months (verify abstinence with CO <10 ppm), relapse prevention, records/reporting under NTCP, training of HCWs, community outreach, integration with TB/NCD clinics.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Varenicline or combination NRT + counselling doubles to triples quit rates; offer cessation at every visit.

LANDMARK TRIALS & GUIDELINES

- Anthenelli RM et al, EAGLES Lancet 2016; Global Adult Tobacco Survey-2 India 2016–17; WHO MPOWER.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Describe the pharmacological and non-pharmacological approach for smoking cessation. *[DNB May 2024 P2]*
2. Smoking cessation therapy. *[DNB Jun 2021 P1]*
3. Cigarettes and other tobacco products act (COTPA). *[BFUHS 2020 P3]*
4. Quit smoking clinic activities. *[BFUHS 2019 P3]*

8.5 Toxic Inhalational Injury, Chemical & Biological Agents, Radiation Sickness & Strangulation

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B4 Toxic lung and other inhalational injuries
- ✓ Classify toxic gases by solubility and manage inhalation injury including smoke and CO
- ✓ Classify chemical warfare agents and features of biological weapons
- ✓ Describe acute radiation syndrome and the effect of strangulation on respiration

INVESTIGATIONS & TREATMENT AT A GLANCE

8.4 Health Hazards of Smoking & Smoking Cessation

‹ Back to topic (p 164)
Next topic 8.5 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Pack-years	—	≥ 20 pack-years	Risk stratification; LDCT eligibility
Fagerström test	0–3 low	≥ 7 high dependence	Pharmacotherapy intensity
Exhaled CO	< 7 ppm	> 10 ppm	Verify abstinence
Urinary cotinine	< 50 ng/mL	> 100 ng/mL	Tobacco exposure
Spirometry	Normal	FEV1/FVC < 0.70	COPD case-finding

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Varenicline	0.5 mg daily × 3 d, 0.5 mg bd × 4 d, then 1 mg bd × 12 weeks	Most effective monotherapy	Nausea; renal dosing
Nicotine replacement (patch + gum)	Patch 21 mg/24 h (> 10 cig/day) + gum 2–4 mg PRN	All smokers	Skin reaction; combination better
Bupropion SR	150 mg daily × 3 days, then 150 mg bd × 7–12 weeks	Alternative	Seizure threshold
Cytisine	25-day tapering course (1.5 mg tablets)	Low-cost alternative	Nausea
Behavioural support (5A/5R)	Quitline 1800-11-2356	All	—

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Combining varenicline with NRT or counselling gives the highest quit rates.

IN BRIEF The site of injury from inhaled toxins depends on water solubility — highly soluble gases injure the upper airway; poorly soluble gases reach the alveoli causing delayed pulmonary oedema. Mass casualty preparedness covers chemical, biological, radiological and nuclear (CBRN) events.

Irritant gases by water solubility

Solubility	Gases	Site / features
High	Ammonia, sulphur dioxide, hydrogen chloride, formaldehyde	Eyes, nose, throat, larynx — immediate warning symptoms
Intermediate	Chlorine	Upper and lower airways; RADS
Low	Phosgene, nitrogen dioxide (silo filler's), ozone, cadmium fumes	Alveoli — delayed (6–24 h) non-cardiogenic pulmonary oedema; bronchiolitis obliterans later

Smoke inhalation injury

- Thermal upper airway injury (oedema — early intubation if stridor, hoarseness, facial burns with singed hair), chemical injury (aldehydes, acrolein), systemic toxicity (CO, cyanide).
- **CO poisoning:** headache, confusion, cherry-red (rare), SpO2 falsely normal — co-oximetry (COHb >3% non-smoker, >10% smoker); 100% O2 by NRBM (half-life 74 min vs 300 min on air); HBO for LOC, neurological signs, pregnancy, COHb >25%, cardiac ischaemia.
- **Cyanide:** lactic acidosis >8 mmol/L — hydroxocobalamin 5 g IV.
- Bronchoscopy for grading, nebulised heparin/NAC (adjunct), lung-protective ventilation.

Chemical warfare agents

Class	Examples	Mechanism / features	Antidote / management
Nerve agents	Sarin, soman, tabun, VX (organophosphates)	AChE inhibition — SLUDGE, bronchorrhoea, bronchospasm, paralysis	Atropine, pralidoxime, diazepam
Vesicants (blister)	Sulphur mustard, lewisite	Skin blisters, airway necrosis, pseudomembranes, late bronchiectasis/BO	Decontamination, BAL for lewisite
Pulmonary (choking)	Chlorine, phosgene, diphosgene	Pulmonary oedema	Supportive, rest
Blood agents	Hydrogen cyanide, cyanogen chloride	Cellular hypoxia	Hydroxocobalamin, nitrites/ thiosulphate
Incapacitating / riot control	BZ, CS, capsaicin (pepper spray)	Lacrimation, cough	Fresh air, irrigation

Biological agents as bioweapons — key features

- Easy to produce and disseminate (aerosol, 1–5 µm), high morbidity/mortality, person-to-person transmission (some), stable in environment, cause panic, need special public health preparedness; delayed recognition (incubation).
- **CDC Category A:** anthrax (inhalational — widened mediastinum, haemorrhagic effusions; ciprofloxacin/doxycycline + raxibacumab), plague (pneumonic — streptomycin/gentamicin, doxycycline), tularaemia, smallpox, botulinum toxin, viral haemorrhagic fevers. **Category B/C:** brucella, Q fever, ricin, Nipah, hantavirus.

Acute radiation syndrome (ARS)

- Whole-body dose >1 Gy: **prodromal** (nausea, vomiting — time to emesis indicates dose), **latent**, **manifest illness** — haematopoietic (1–8 Gy; marrow failure, infection, bleeding), gastrointestinal (6–30 Gy), neurovascular (>20–30 Gy; fatal), **recovery or death**. Cutaneous radiation syndrome.
- **Management:** decontamination, triage by lymphocyte kinetics (absolute count at 48 h), antiemetics, G-CSF/GM-CSF, antibiotics/ antifungals, transfusion (irradiated), HSCT; potassium iodide for radioiodine (thyroid), Prussian blue (caesium), DTPA (plutonium).

Strangulation and its effect on respiration

- Mechanisms of death/injury: venous occlusion (congestion, petechiae), carotid occlusion (cerebral hypoxia), airway compression (asphyxia), vagal inhibition (carotid sinus — cardiac arrest).
- **Respiratory effects:** laryngeal/tracheal injury (hyoid/thyroid cartilage fractures, oedema, stridor), negative-pressure pulmonary oedema after release of obstruction, aspiration pneumonia, ARDS, hypoxic brain injury → central hypoventilation; delayed airway obstruction (observe 24 h).
- Management: airway assessment (fibreoptic laryngoscopy), CT neck angiography (carotid dissection), oxygen, NIV/MV for oedema, medicolegal documentation.

Other inhalational injuries

- Metal fume fever (zinc oxide — welders; flu-like), polymer fume fever (PTFE), mercury vapour pneumonitis, paraquat (fibrosis — avoid O2), hydrocarbon aspiration (kerosene in children — chemical pneumonitis), near-drowning (surfactant washout, ARDS).

★ CLINICAL PEARLS & EXAM FOCUS

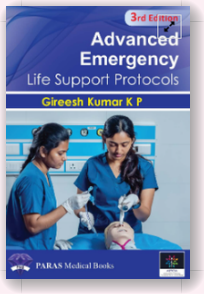
▸ Poorly soluble gas (phosgene, NO2) — the patient feels fine initially; observe for 24 h for delayed pulmonary oedema.

LANDMARK TRIALS & GUIDELINES

- Gorguner M, Akgun M. Acute inhalation injury, Eurasian J Med 2010; CDC bioterrorism agent categories; IAEA medical management of radiation emergencies.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. What are the chemical warfare agents (CWAs) and describe their types. *[BFUHS 2022 P3]*
2. What are the key features of biological agents which can be used as a bioweapon? *[BFUHS 2022 P3]*
3. Clinical manifestation and management of acute radiation sickness. *[BFUHS 2022 P3]*
4. Strangulation and its effect on respiration. *[DNB Oct 2023 P2]*



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INVESTIGATIONS & TREATMENT AT A GLANCE

8.5 Toxic Inhalational Injury, Chemical & Biological Agents, Radiation Sickness & Strangulation

‹ Back to topic (p 165)
Next topic 9.1 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
COHb	< 3%	> 10–15%	CO poisoning (smoke)
Lactate	< 2 mmol/L	> 8 mmol/L	Cyanide toxicity likely (smoke inhalation)
Bronchoscopy	Normal	Soot, mucosal oedema, ulceration	Inhalation injury grading
Methaemoglobin	< 1.5%	> 20%	Oxidant gases/nitrates
ABG / P/F ratio	> 300	< 200	ARDS
Cholinesterase	Normal	Low	Organophosphate / nerve agent

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
100% oxygen	15 L/min non-rebreather	Smoke inhalation, CO	—
Hydroxocobalamin	5 g IV over 15 min (repeat once)	Suspected cyanide (lactate > 8)	Red urine/skin; interferes with lab tests
Atropine + pralidoxime	Atropine 1–2 mg IV doubling every 5 min; pralidoxime 30 mg/kg then 8 mg/kg/h	Organophosphate/nerve agent	Dry secretions end-point
Nebulised heparin + NAC	Heparin 5000–10 000 U + NAC 3 mL 20% 4-hourly	Smoke inhalation (some centres)	Limited evidence
Early intubation	—	Stridor, facial burns with airway oedema	Airway swells rapidly

★ PRESCRIBING & INTERPRETATION PEARLS

- Chlorine/ammonia: mostly upper airway injury; phosgene: delayed pulmonary oedema up to 24 h — observe.

SECTION 9

Pulmonary Circulation — Hypertension, Embolism, Oedema & Haemoptysis

Paper II / III

Pulmonary hypertension and cor pulmonale; pulmonary oedema; pulmonary thromboembolic disease and infarction; cardiac problems in pulmonary patients and pulmonary complications of cardiac disease; haemoptysis and vascular malformations (NMC syllabus II-B5).

9.1 Pulmonary Hypertension — Definition, Classification & Diagnosis 168
9.2 Pulmonary Arterial Hypertension — Risk Stratification & Treatment; CTD-PAH and Drug-Induced PAH 170
9.3 CTEPH, Portopulmonary Hypertension & Hepatopulmonary Syndrome 172
9.4 Pulmonary Embolism — Diagnosis & Risk Stratification 173
9.5 Management of PE & VTE; Prophylaxis, HIT, DOACs & Fat Embolism 175
9.6 Pulmonary Oedema; Cardiac–Pulmonary Interactions 177
9.7 Haemoptysis — Causes, Massive Haemoptysis & Bronchial Artery Embolisation 178

9.1 Pulmonary Hypertension — Definition, Classification & Diagnosis

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B5 Pulmonary hypertension
- ✓ Define PH haemodynamically (ESC/ERS 2022) and classify into five groups
- ✓ Describe the diagnostic algorithm including echocardiographic probability and right heart catheterisation

IN BRIEF PH is now defined as mean pulmonary arterial pressure >20 mmHg at rest on right heart catheterisation. Pre-capillary PH (PAWP ≤15, PVR >2 WU) includes PAH (group 1) and lung/CTEPH causes; post-capillary PH is due to left heart disease, the commonest cause overall.

Haemodynamic definitions (ESC/ERS 2022)

Definition	Criteria
PH	mPAP >20 mmHg
Pre-capillary PH	mPAP >20, PAWP ≤15, PVR >2 WU
Isolated post-capillary PH	mPAP >20, PAWP >15, PVR ≤2 WU
Combined pre- and post-capillary PH	mPAP >20, PAWP >15, PVR >2 WU
Exercise PH	mPAP/CO slope >3 mmHg/L/min between rest and exercise

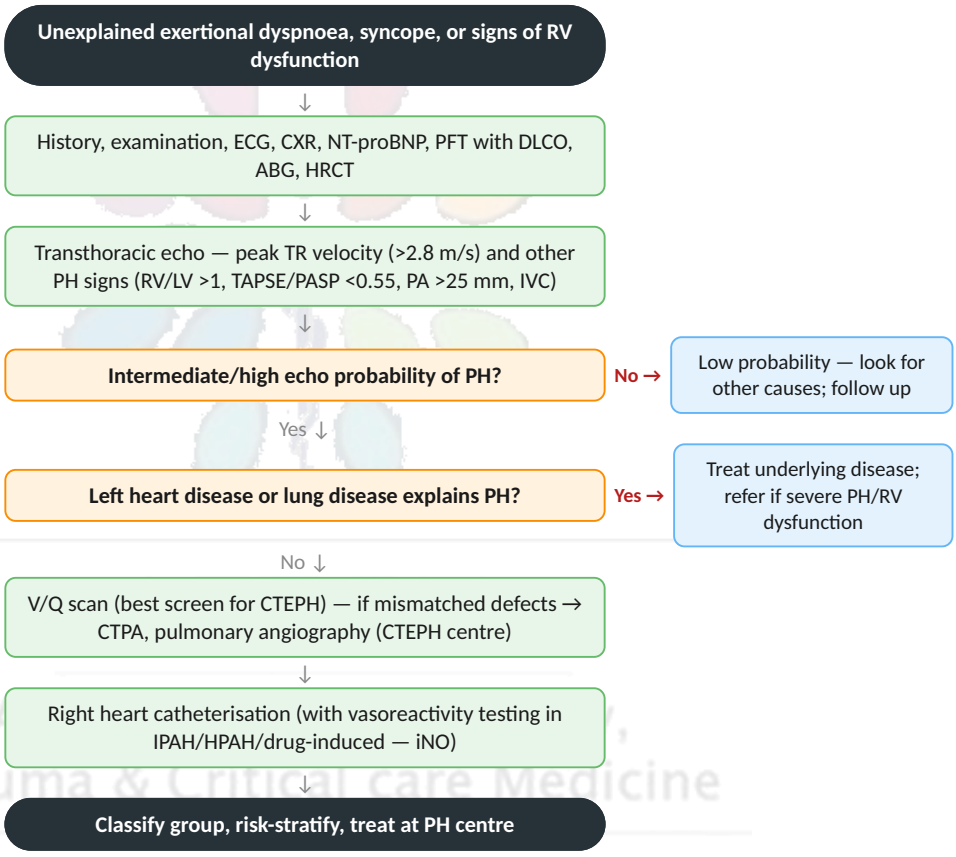
Clinical classification (Nice 2018 / ESC 2022)

Group	Categories
1 — PAH	Idiopathic, heritable (BMPR2), drug/toxin-induced, associated (CTD, HIV, portal hypertension, CHD, schistosomiasis), vasoreactive, PVOD/PCH
2 — Left heart disease	HFpEF, HFrEF, valvular, congenital
3 — Lung disease/hypoxia	COPD, ILD, OSA/OHS, high altitude, developmental
4 — Pulmonary artery obstruction	CTEPH, other obstructions (tumour, arteritis)
5 — Unclear/multifactorial	Haematological (chronic haemolytic anaemia, MPN), systemic (sarcoidosis, PLCH), metabolic, chronic renal failure, fibrosing mediastinitis

Symptoms and signs

- Exertional dyspnoea, fatigue, syncope (advanced), chest pain, haemoptysis; signs — loud P2, RV heave, TR murmur, raised JVP, oedema, hepatomegaly; Ortner's (hoarseness — PA compressing recurrent laryngeal nerve).

Algorithm 9.1 · Diagnostic approach to suspected PH (ESC/ERS 2022)



Echocardiography in respiratory medicine

- Estimates PASP ($4v^2$ + RAP), RV size/function (TAPSE <17 mm, FAC, S'), pericardial effusion (poor prognosis in PAH), IVC; differentiates group 2 (LA enlargement, LV dysfunction, E/e'); bubble study for shunt (PFO, PAVM, HPS).
- Uses: PH screening (SSc annually, portal hypertension pre-LT, HHT), unexplained dyspnoea, cor pulmonale, acute PE risk stratification (RV/LV >1, McConnell's sign, 60/60 sign), pericardial disease, guiding fluids in ICU, ECMO.

★ CLINICAL PEARLS & EXAM FOCUS

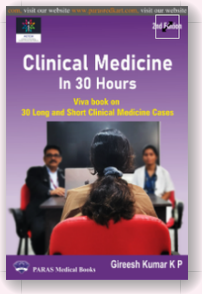
▸ The new PH threshold is mPAP >20 mmHg (not ≥25) and PVR >2 WU (not ≥3).

LANDMARK TRIALS & GUIDELINES

- Humbert M et al, 2022 ESC/ERS guidelines for PH, Eur Heart J/Eur Respir J 2022.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Classification, symptoms and management of pulmonary hypertension. *[DNB Oct 2023 P2]*
2. Discuss the causes, classification system and management of pulmonary hypertension. *[VNSGU 2025 P2]*



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9.2 Pulmonary Arterial Hypertension — Risk Stratification & Treatment; CTD-PAH and Drug-Induced PAH

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B5 Pulmonary hypertension
- ✓ Risk-stratify PAH and outline treatment of group 1 PAH
- ✓ Describe pharmacotherapy of PAH by pathway
- ✓ Approach PAH in CTD and drug/toxin-induced PAH; nitric oxide in pulmonary diseases

IN BRIEF PAH is a progressive vasculopathy (endothelial dysfunction, smooth-muscle proliferation, in-situ thrombosis, plexiform lesions). Treatment targets three pathways — endothelin, nitric oxide, prostacyclin — plus activin signalling (sotatercept), guided by multiparametric risk.

Risk stratification (ESC 2022 four-strata at follow-up)

Variable	Low	Intermediate-low	Intermediate-high	High
WHO FC	I–II	—	III	IV
6MWD	>440 m	320–440	165–319	<165 m
NT-proBNP (ng/L)	<300	300–649	650–1100	>1100

PAH-specific drugs

Pathway	Class / drugs	Notes
Endothelin	ERAs — ambrisentan 5–10 mg, bosentan 125 mg BD, macitentan 10 mg	Teratogenic, hepatotoxicity (bosentan), oedema, anaemia
Nitric oxide	PDE5i — sildenafil 20 mg TDS, tadalafil 40 mg OD; sGC stimulator — riociguat (also CTEPH)	Never combine PDE5i with riociguat or nitrates
Prostacyclin	Epoprostenol IV (most effective in severe), treprostinil (SC/IV/inhaled), iloprost (inhaled), selexipag (oral IP agonist), beraprost	Line infections, jaw pain, flushing
Activin signalling	Sotatercept (activin ligand trap, SC 3-weekly)	STELLAR — improved 6MWD, PVR; telangiectasia, bleeding, ↑ Hb

INVESTIGATIONS & TREATMENT AT A GLANCE

9.1 Pulmonary Hypertension — Definition, Classification & Diagnosis

‹ Back to topic (p 168)
Next topic 9.2 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Echo TR velocity	≤ 2.8 m/s	> 3.4 m/s high probability	PH screening
RHC mPAP	≤ 20 mmHg	> 20 mmHg	PH definition (ESC/ERS 2022)
PAWP	≤ 15 mmHg	> 15	Post-capillary (group 2)
PVR	≤ 2 WU	> 2 WU	Pre-capillary
NT-proBNP	< 300 ng/L	> 1100 ng/L	High risk
V/Q scan	Normal perfusion	Mismatched segmental defects	CTEPH — screen all PH
HRCT, PFT, sleep study, HIV, LFT, ANA	—	Abnormal	Group 3/5 and associated causes

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Oxygen	Keep SpO_2 > 90%	Hypoxaemic PH	—
Diuretics (furosemide)	20–80 mg daily	RV failure	Hypokalaemia
Treat underlying disease	—	Groups 2, 3	PAH drugs not routinely for groups 2/3

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Diagnostic RHC is required before PAH-specific therapy.

Pathway	Class / drugs	Notes
Vasoreactive	High-dose CCB (nifedipine, diltiazem, amlodipine)	Only if positive vasoreactivity test (fall in mPAP ≥10 to ≤40 with normal CO) — ~10% IPAH

Treatment algorithm (group 1)

- **General:** supervised exercise, vaccines, contraception (avoid pregnancy), psychosocial support, diuretics, O2 (keep PaO2 ≥60), iron repletion, anticoagulation not routinely (IPAH individual).
- **Initial:** low/intermediate risk without cardiopulmonary comorbidity → **upfront dual oral combination ERA + PDE5i** (AMBITION); high risk → triple including IV/SC prostacyclin.
- **Follow-up:** reassess 3–4 months; intermediate-low → add selexipag or switch PDE5i to riociguat; intermediate-high/high → add IV/SC prostacyclin, refer for transplant; add sotatercept.
- Interventional: balloon atrial septostomy (bridge), lung transplantation.

PAH in connective tissue disease

- SSc (8–12% — commonest CTD-PAH; limited cutaneous, anticentromere), SLE, MCTD, Sjögren.
- **Screening in SSc:** annual echo, DLCO, NT-proBNP; DETECT algorithm (FVC/DLCO >1.6, telangiectasia, anticentromere, NT-proBNP, urate, right-axis deviation); RHC if positive.
- Differentiate group 1 from group 3 (ILD) and group 2 (cardiac involvement) and PVOD; treat with combination (ERA + PDE5i); immunosuppression helps SLE/MCTD-PAH but not SSc-PAH; worse prognosis than IPAH.

Drug- and toxin-induced PAH

- **Definite:** aminorex, fenfluramine, dexfenfluramine, benfluorex, methamphetamine, dasatinib, toxic rapeseed oil. **Likely:** amphetamines, cocaine, interferon α/β, alkylating agents (mitomycin C, cyclophosphamide → PVOD), bosutinib, DAAs for HCV, leflunomide, L-tryptophan, St John's wort.
- Stop drug; dasatinib PAH may reverse partially; treat as PAH.

Nitric oxide in pulmonary diseases

- Endogenous NO (eNOS) → sGC → cGMP → vasodilatation, anti-proliferative; iNOS in airway inflammation (FeNO).
- **Inhaled NO (5–40 ppm):** selective pulmonary vasodilator — vasoreactivity testing, PPHN in neonates, post-cardiac surgery PH, RV failure, rescue in refractory ARDS hypoxaemia (improves oxygenation transiently, no mortality benefit, AKI risk), HAPE; methaemoglobinaemia and NO2 toxicity; rebound PH on abrupt withdrawal.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Vasoreactivity testing is only for IPAH, HPAH and drug-induced PAH — never start CCBs without it.

LANDMARK TRIALS & GUIDELINES

- Galiè N et al, AMBITION NEJM 2015; Hoeper MM et al, STELLAR NEJM 2023; Sitbon O et al, GRIPHON (selexipag) NEJM 2015.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Describe the outline of management of Group 1 pulmonary arterial hypertension. *[DNB May 2024 P4]*
2. Pharmacotherapy in pulmonary arterial hypertension. *[DNB Oct 2024 P2]*
3. Pulmonary arterial hypertension secondary to drugs and toxins. *[DNB Oct 2023 P4]*
4. Approach to PAH in a patient with connective tissue disease. *[BFUHS 2019 P4]*
5. Nitric oxide in pulmonary diseases. *[Manipal 2014 P4]*

INVESTIGATIONS & TREATMENT AT A GLANCE

9.2 Pulmonary Arterial Hypertension — Risk Stratification & Treatment; CTD-PAH and Drug-Induced PAH

‹ Back to topic (p 170)
Next topic 9.3 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Risk score (ESC/ERS 4-strata)	Low	Intermediate-low/high, high	Treatment escalation
6MWD	> 440 m	< 165 m	Risk
WHO FC	I–II	IV	Risk
NT-proBNP	< 300 ng/L	> 1100	Risk
Cardiac index / RAP	CI ≥ 2.5; RAP < 8	CI < 2.0; RAP > 14	Risk
LFT (ERA), Hb	Normal	ALT > 3× ULN; anaemia	ERA monitoring

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Ambrisentan + tadalafil	Ambrisentan 5–10 mg daily + tadalafil 40 mg daily	Initial dual therapy (low/ intermediate risk)	Teratogenic (ERA); oedema; hypotension
Macitentan	10 mg daily	PAH	Anaemia, teratogenic
Sildenafil	20 mg tds	PAH	Never with nitrates
Selexipag	200 µg bd titrated weekly to max 1600 µg bd	Add-on intermediate risk	Headache, diarrhoea
IV epoprostenol / SC treprostinil	Titrate from 2 ng/kg/min	High risk	Line infection, rebound if interrupted
Sotatercept	0.3 → 0.7 mg/kg SC every 3 weeks	Add-on (STELLAR)	Telangiectasia, ↑ Hb, ↓ platelets

★ **PRESCRIBING & INTERPRETATION PEARLS**

- CCBs only for vasoreactive idiopathic/heritable/drug-induced PAH.

9.3 CTEPH, Portopulmonary Hypertension & Hepatopulmonary Syndrome

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B5 Pulmonary hypertension
- ✓ Diagnose and treat CTEPH
- ✓ Differentiate portopulmonary hypertension from hepatopulmonary syndrome and manage both

IN BRIEF Liver disease produces two opposite pulmonary vascular disorders: vasoconstriction/remodelling (portopulmonary hypertension) and vasodilatation with intrapulmonary shunting (hepatopulmonary syndrome). CTEPH is the only potentially curable form of PH.

Chronic thromboembolic pulmonary hypertension

- 2–4% after acute PE; organised fibrotic thrombi + small-vessel arteriopathy; risk — recurrent VTE, large PE, splenectomy, APS, VA shunts, thyroid replacement, malignancy, non-O blood group.
- **Diagnosis:** after ≥3 months of effective anticoagulation — V/Q scan (mismatched defects; normal V/Q excludes), CTPA (webs, bands, eccentric thrombi, mosaic perfusion, bronchial collaterals), pulmonary angiography, RHC.
- **Treatment (CTEPH team):** lifelong anticoagulation (VKA preferred in APS); **pulmonary endarterectomy** (surgical, deep hypothermic circulatory arrest — potentially curative); **balloon pulmonary angioplasty** for inoperable distal disease; **riociguat** (CHEST-1) or macitentan for inoperable/residual PH; multimodal.

Portopulmonary hypertension vs hepatopulmonary syndrome

Feature	Portopulmonary hypertension (PoPH)	Hepatopulmonary syndrome (HPS)
Pathology	Vasoconstriction, remodelling, plexiform lesions	Intrapulmonary vascular dilatation (↑ NO, endothelin-B), AV shunts
Prevalence in cirrhosis	2–6%	10–30%
Symptoms	Exertional dyspnoea, syncope, RV failure	Dyspnoea, **platypnoea–orthodeoxia**, cyanosis, clubbing, spider naevi
Oxygenation	Mild hypoxaemia	A–a gradient ≥15 (≥20 if >64 years), PaO2 <80
Diagnosis	Echo → RHC: mPAP >20, PVR >2 WU, PAWP ≤15	**Contrast (bubble) echo — bubbles in LA after 3–6 cardiac cycles**; Tc-MAA shunt fraction >6%
Liver transplant	Contraindicated if mPAP ≥45–50 despite therapy; may proceed if mPAP <35 and PVR <5 with treatment	**Definitive cure**; MELD exception points if PaO2 <60
Treatment	PAH therapies (macitentan — PORTICO, PDE5i, prostanoids); avoid β-blockers; ± TIPS caution	Oxygen, liver transplantation (resolution in >80% within 1 year); no effective drugs

HPS — definitive management

- Liver transplantation is the only effective treatment; severity (PaO2 <50 very severe) predicts post-transplant mortality and delayed resolution; post-transplant hypoxaemia managed with O2, Trendelenburg, iNO, ECMO.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Bubbles appearing in the left heart after 3 cardiac cycles = intrapulmonary shunt (HPS/PAVM); within 3 cycles = intracardiac shunt.

LANDMARK TRIALS & GUIDELINES

- Delcroix M et al, ERS CTEPH statement 2021; Krowka MJ et al, ILTS practice guidelines on PoPH and HPS, Transplantation 2016.

INVESTIGATIONS & TREATMENT AT A GLANCE

9.3 CTEPH, Portopulmonary Hypertension & Hepatopulmonary Syndrome

‹ Back to topic (p 172)
Next topic 9.4 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
V/Q scan	Normal	Mismatched defects	CTEPH (more sensitive than CTPA)
CTPA / pulmonary angiography	—	Webs, bands, chronic thrombi	Operability
Contrast echo (bubble)	No late bubbles	Late bubbles (3–6 cycles)	Hepatopulmonary syndrome
A–a gradient (HPS)	< 15 mmHg	≥ 15 (≥ 20 if > 64 y)	HPS criterion
RHC in portopulmonary HTN	mPAP ≤ 20	mPAP > 20, PVR > 2 WU, PAWP ≤ 15	PoPH (MELD exception if mPAP < 35 on treatment)

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Pulmonary endarterectomy	Surgical	Operable CTEPH	Expert centre
Riociguat	1 mg tds → titrate to 2.5 mg tds	Inoperable/residual CTEPH	Hypotension; no PDE5i
Balloon pulmonary angioplasty	Interventional	Inoperable distal CTEPH	Reperfusion injury
Lifelong anticoagulation	Warfarin INR 2–3 (DOAC data limited)	CTEPH	Bleeding
Liver transplant	—	HPS (PaO_2 < 60)	Reverses HPS

★ PRESCRIBING & INTERPRETATION PEARLS

- Avoid β-blockers in portopulmonary hypertension — worsen cardiac output.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Hepatopulmonary syndrome diagnosis and definitive management. *[DNB Oct 2024 P2]*
2. Portopulmonary hypertension. *[DNB Dec 2021 P2]*

9.4 Pulmonary Embolism — Diagnosis & Risk Stratification

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B5 Pulmonary thromboembolic disease
- ✓ Apply clinical probability scores (Wells, revised Geneva), PERC, D-dimer and imaging
- ✓ Risk-stratify acute PE (ESC 2019) using haemodynamics, PESI and RV assessment

IN BRIEF PE presents variably — from incidental to shock. Diagnosis follows a probability-based strategy: low/intermediate probability → age-adjusted D-dimer; high probability or positive D-dimer → CTPA. Risk stratification determines treatment intensity.

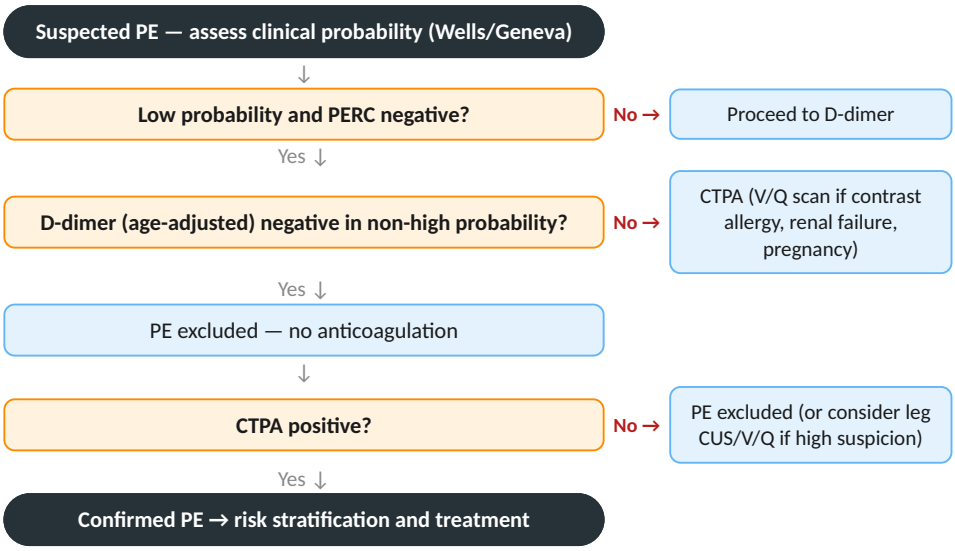
Wells score for PE

Item	Points
Clinical signs of DVT	3
PE is the most likely diagnosis	3
Heart rate >100	1.5
Immobilisation ≥3 days or surgery within 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (treatment within 6 months/ palliative)	1

- Two-level: ≤4 PE unlikely; >4 PE likely. Three-level: <2 low, 2–6 intermediate, >6 high.
- **PERC rule** (low-probability patients): all 8 negative (age <50, HR <100, SpO2 ≥95%, no haemoptysis, no oestrogen, no prior VTE, no unilateral leg swelling, no recent surgery/trauma) → no further testing.
- **YEARS algorithm:** 3 items (DVT signs, haemoptysis, PE most likely) + D-dimer thresholds (<1000 if 0 items, <500 if ≥1) — reduces CTPA use; adapted for pregnancy.
- **Age-adjusted D-dimer:** age × 10 µg/L for >50 years.

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Algorithm 9.2 · Diagnosis of suspected PE (haemodynamically stable)



- **Unstable (shock):** bedside echo showing RV dysfunction is sufficient to justify reperfusion if CTPA not feasible.

Imaging and other tests

- **CTPA:** filling defects, RV/LV >1; **V/Q scan:** preferred in pregnancy, renal failure, contrast allergy; **CUS of legs:** proximal DVT confirms VTE; **echo:** RV dilatation, McConnell sign, 60/60 sign, thrombus in transit.
- **ECG/CXR:** sinus tachycardia, S1Q3T3, RBBB; CXR — Westermark (oligaemia), Hampton's hump (infarct), Fleischner (enlarged PA), Palla's sign.
- **ABG:** hypoxaemia, hypocapnia, increased A–a gradient.

ESC 2019 risk stratification

Risk	Haemodynamic instability	PESI III–V / sPESI ≥1	RV dysfunction (echo/CT)	Troponin elevated
High	+	(+)	+	(+)
Intermediate-high	–	+	+	+
Intermediate-low	–	+	One or none positive	—
Low	–	–	– (assessed optional)	–

- **Haemodynamic instability:** cardiac arrest, obstructive shock (SBP <90 or vasopressors with hypoperfusion), persistent hypotension (SBP <90 or drop ≥40 for >15 min).

★ CLINICAL PEARLS & EXAM FOCUS

▸ Never order D-dimer in high-probability patients — go straight to CTPA.

LANDMARK TRIALS & GUIDELINES

- Konstantinides SV et al, 2019 ESC/ERS acute PE guideline, Eur Heart J 2020; van der Hulle T et al, YEARS Lancet 2017.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Diagnosis of pulmonary embolism. *[DNB Jun 2021 P2; BFUHS 2019 P3]*
2. Diagnosis of pulmonary thromboembolism. *[NEET-SS/DNB 2024]*
3. Wells clinical prediction score model. *[DNB Dec 2021 P2]*

INVESTIGATIONS & TREATMENT AT A GLANCE

9.4 Pulmonary Embolism — Diagnosis & Risk Stratification

‹ Back to topic (p 173)
Next topic 9.5 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Wells score	≤ 4 (PE unlikely)	> 4 (PE likely)	Pre-test probability
D-dimer	< 0.5 µg/mL (age-adjusted: age × 10 µg/L if > 50 y)	Raised	Rule-out only if low probability
CTPA	No filling defect	Filling defect	Diagnosis
RV/LV ratio (CT/echo)	< 0.9	> 1.0	RV dysfunction
Troponin / NT-proBNP	Normal	Raised	Intermediate-high risk
sPESI	0	≥ 1	Not low risk
Compression USG legs	Compressible	Non-compressible vein	DVT (supports PE)

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
LMWH (empirical)	Enoxaparin 1 mg/kg SC 12-hourly	Start while awaiting imaging if high probability	Renal dosing
UFH	80 U/kg bolus, 18 U/kg/h (aPTT 1.5–2.5×)	High-risk PE / possible thrombolysis	HIT
Thrombolysis (alteplase)	100 mg over 2 h (or 0.6 mg/kg over 15 min in arrest)	High-risk (shock) PE	Absolute contraindications
Oxygen	Target SpO_2 ≥ 90%	Hypoxaemia	—

★ **PRESCRIBING & INTERPRETATION PEARLS**

- PERC rule (all 8 negative) excludes PE without D-dimer in low-risk patients.

9.5 Management of PE & VTE; Prophylaxis, HIT, DOACs & Fat Embolism

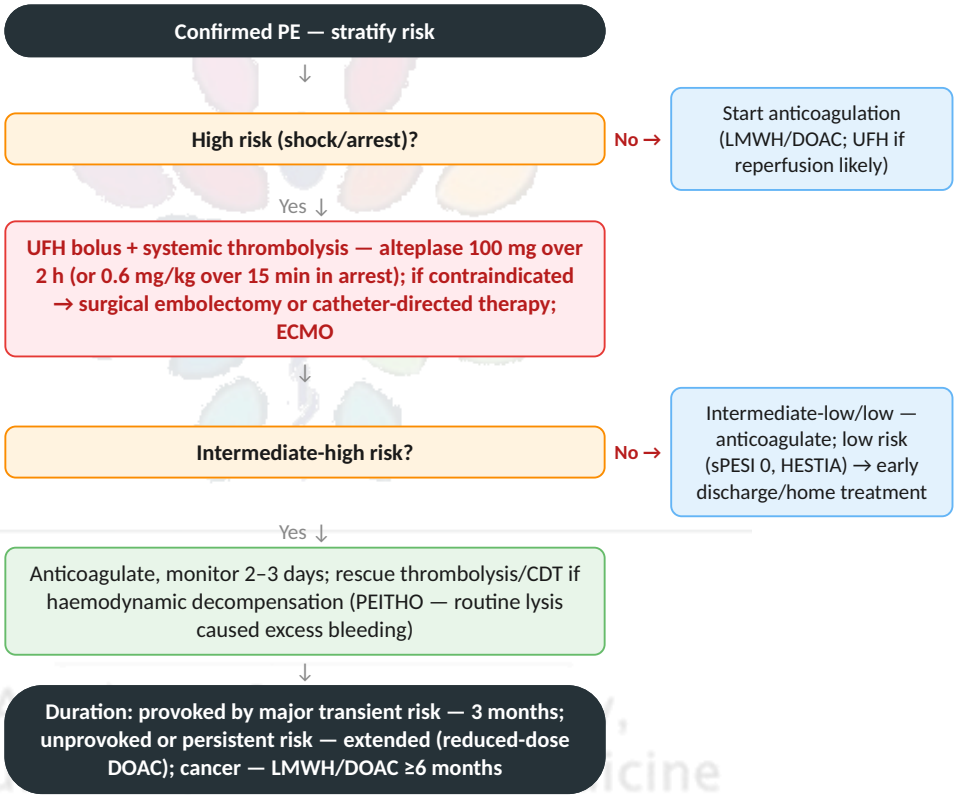
‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B5 Pulmonary thromboembolism; II-B10
- ✓ Treat PE by risk category including thrombolysis and catheter-directed therapy
- ✓ Choose anticoagulant and duration; use DOACs appropriately
- ✓ Prevent VTE in hospitalised patients; manage HIT; recognise fat embolism syndrome

IN BRIEF Anticoagulation is the cornerstone; systemic thrombolysis is reserved for high-risk (shock) PE and for intermediate-high-risk patients who deteriorate. DOACs are preferred over VKAs in most patients (not in APS, severe renal failure, pregnancy).

Algorithm 9.3 · Management of acute PE by risk (ESC 2019)



Anticoagulants for VTE

Drug	Dose	Notes
LMWH (enoxaparin)	1 mg/kg BD or 1.5 mg/kg OD SC	Pregnancy, cancer; renal dose adjustment (CrCl <30: 1 mg/kg OD)
UFH	80 U/kg bolus, 18 U/kg/h (aPTT 1.5–2.5×)	Shock, thrombolysis planned, severe renal failure, high bleeding risk (reversible)
Fondaparinux	5–10 mg SC OD by weight	HIT history
Apixaban	10 mg BD ×7 days → 5 mg BD (2.5 mg BD extended)	No initial parenteral needed (AMPLIFY)
Rivaroxaban	15 mg BD ×21 days → 20 mg OD (10 mg extended)	With food (EINSTEIN-PE)

Drug	Dose	Notes
Dabigatran / edoxaban	After 5 days LMWH	RE-COVER, Hokusai
Warfarin	INR 2–3, overlap ≥5 days with LMWH	APS, mechanical valves, low cost

- **DOAC avoid:** APS (triple positive), CrCl <15–30, pregnancy/lactation, strong P-gp/CYP3A4 interactions (rifampicin!), extremes of weight (caution). Reversal: idarucizumab (dabigatran), andexanet alfa / PCC (Xa inhibitors).

VTE prophylaxis

- Risk assessment: **Padua score** ≥4 (medical), **Caprini** (surgical), IMPROVE bleeding score.
- Pharmacological: enoxaparin 40 mg OD, UFH 5000 U BD–TDS, fondaparinux 2.5 mg; mechanical (IPC) if bleeding risk; extended prophylaxis after major cancer/orthopaedic surgery. Early mobilisation.

Heparin-induced thrombocytopenia

- Immune (IgG against PF4–heparin) → platelet activation → thrombocytopenia (fall >50%) 5–10 days after heparin with paradoxical thrombosis (venous > arterial).
- **4T score** (thrombocytopenia, timing, thrombosis, other causes) — ≤3 low probability; intermediate/high → stop all heparin (including flushes), send PF4 ELISA ± serotonin release assay, start **non-heparin anticoagulant** (argatroban, bivalirudin, fondaparinux, DOAC); no platelet transfusion; warfarin only after platelet recovery >150,000 (overlap).

Fat embolism syndrome

- 24–72 h after long-bone/pelvic fractures (also liposuction, pancreatitis, sickle crisis); fat globules + inflammatory free fatty acids.
- **Gurd's criteria:** major — respiratory insufficiency, cerebral involvement, petechial rash (axilla, conjunctiva); minor — tachycardia, fever, retinal fat, jaundice, renal changes, anaemia, thrombocytopenia, high ESR, fat macroglobulinaemia. MRI brain "starfield" pattern.
- Treatment: supportive (O2, lung-protective ventilation), early fracture fixation (prevention); steroids controversial.

★ CLINICAL PEARLS & EXAM FOCUS

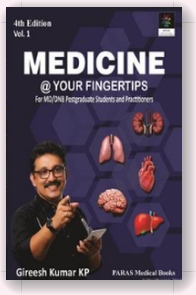
▸ In PE with shock — thrombolyse; in intermediate-high risk — anticoagulate and watch closely, lyse only if they deteriorate.

LANDMARK TRIALS & GUIDELINES

- Meyer G et al, PEITHO NEJM 2014; Agnelli G et al, AMPLIFY NEJM 2013; Cuker A et al, ASH HIT guideline 2018.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Management of pulmonary embolism. *[DNB Oct 2024 P2; DNB Jun 2021 P2]*
2. Prevention of venous thromboembolism. *[DNB Dec 2021 P3]*
3. Heparin-induced thrombocytopenia. *[DNB Dec 2021 P3]*
4. Newer anticoagulants in pulmonary embolism. *[BFUHS 2019 P4]*
5. Write in brief about venous thromboembolism. *[BFUHS 2019 P1]*
6. Fat embolism. *[BFUHS 2019 P2]*



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INVESTIGATIONS & TREATMENT AT A GLANCE

9.5 Management of PE & VTE; Prophylaxis, HIT, DOACs & Fat Embolism

‹ Back to topic (p 175)
Next topic 9.6 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Platelets (heparin)	Stable	Fall > 50% day 5–10	HIT — 4T score, PF4 antibody
INR (warfarin)	2–3	> 3 bleeding risk; < 2 thrombosis	Monitoring
Anti-Xa (LMWH)	0.6–1.0 IU/mL (peak, bd dosing)	—	Obesity, CKD, pregnancy
Creatinine clearance	> 50 mL/min	< 30	DOAC choice/dose
Thrombophilia screen	—	APS, protein C/S deficiency	Unprovoked VTE (selected)

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Apixaban	10 mg bd × 7 days, then 5 mg bd	VTE treatment	Not in APS (triple +); CrCl < 25
Rivaroxaban	15 mg bd × 21 days, then 20 mg daily with food	VTE	Avoid in APS
Warfarin	Overlap with LMWH ≥ 5 days till INR ≥ 2 × 24 h	VTE, APS, valves	Interactions; rifampicin ↓ effect
Enoxaparin prophylaxis	40 mg SC daily	Medical inpatients at risk	Renal adjustment
Argatroban / fondaparinux	Argatroban 2 µg/kg/min; fondaparinux 7.5 mg daily	HIT	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Duration: provoked 3 months; unprovoked — consider extended; cancer — 6 months+ (DOAC or LMWH).

9.6 Pulmonary Oedema; Cardiac–Pulmonary Interactions

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B5 Pulmonary oedema; Cardiac problems in pulmonary patients
- ✓ Differentiate cardiogenic from non-cardiogenic pulmonary oedema
- ✓ Manage acute cardiogenic pulmonary oedema; recognise re-expansion and negative-pressure oedema
- ✓ Recognise radiological signs (Kerley lines) and pulmonary complications of cardiac disease; SVC syndrome

IN BRIEF Pulmonary oedema results from increased hydrostatic pressure (cardiogenic) or increased permeability (non-cardiogenic/ARDS). Distinction relies on clinical context, BNP, echo and imaging; management of cardiogenic oedema centres on oxygen/CPAP, nitrates and diuretics.

Cardiogenic vs non-cardiogenic pulmonary oedema

Feature	Cardiogenic	Non-cardiogenic (ARDS)
History	Cardiac disease, MI, arrhythmia, fluid overload	Sepsis, aspiration, pancreatitis, trauma, transfusion
Examination	S3, raised JVP, cool peripheries, cardiomegaly	Warm peripheries (sepsis), no S3, normal JVP
CXR	Cardiomegaly, upper lobe diversion, Kerley B lines, peribronchial cuffing, effusions, bat-wing perihilar	Normal heart size, peripheral patchy infiltrates, air bronchograms, few effusions/septal lines
BNP	>500 pg/mL	<100 pg/mL
Echo	LV dysfunction, valve disease, high filling pressures (E/e' >14)	Normal LV
PAWP (if measured)	>18 mmHg	≤18 mmHg
Oedema fluid/plasma protein	<0.65	>0.7

Lymphatic (Kerley) lines

- **Kerley B:** short (1–2 cm) horizontal lines at lung bases perpendicular to pleura — thickened interlobular septa (interstitial oedema, lymphangitis carcinomatosa, pneumoconiosis, sarcoid). **Kerley A:** longer (2–6 cm) oblique lines towards hilum in upper zones. **Kerley C:** reticular network.

Management of acute cardiogenic pulmonary oedema

- Sit upright, O2 to SpO2 >90%, **CPAP/NIV** (reduces intubation), IV nitroglycerin (if SBP >110), IV furosemide 40–80 mg (or 1–2.5× home dose), treat precipitant (ACS — PCI; arrhythmia; hypertensive crisis — nitrates high dose); morphine not routinely; inotropes/mechanical support in cardiogenic shock; ultrafiltration in refractory.

Special types of pulmonary oedema

- **Re-expansion pulmonary oedema:** after rapid re-expansion of a lung collapsed >3 days (large pneumothorax/effusion); ipsilateral oedema within 1–24 h; mortality up to 20%; prevention — remove ≤1.5 L effusion at a time, stop if chest discomfort/cough, avoid suction initially on ICD; treatment supportive.
- **Negative-pressure pulmonary oedema:** after upper airway obstruction (laryngospasm, strangulation, epiglottitis) in young muscular patients — CPAP.
- **Neurogenic** (SAH, seizures, head injury — sympathetic surge), **HAPE**, **opioid/salicylate**, **immersion (swimming) oedema**, **TRALI/TACO**, **post-pneumonectomy**.

Cardiac problems in pulmonary patients

- RV dysfunction/cor pulmonale, arrhythmias (MAT in COPD, AF from β-agonists/theophylline, hypoxia), IHD (shared risk factors; β1-selective blockers safe in COPD), Takotsubo in severe asthma/COPD exacerbations.

Pulmonary complications of cardiac disease

- Pulmonary oedema, pleural effusion (HF — bilateral, right > left; transudate; NT-proBNP in fluid), PH group 2, haemoptysis (mitral stenosis — pulmonary apoplexy, haemosiderosis, ossific nodules), Dressler's syndrome (post-MI/post-cardiotomy pleuropericarditis — NSAIDs/colchicine), cardiac cachexia, central sleep apnoea, amiodarone lung, phrenic palsy after cardiac surgery, chylothorax after surgery.

Jugular venous pulse and superior vena cava syndrome

- JVP reflects RA pressure; raised in RV failure, TR (large v waves), constriction (Kussmaul's), tamponade (absent y descent); **non-pulsatile raised JVP** = SVC obstruction.
- **SVC syndrome:** malignancy (60–85%: SCLC, NSCLC, lymphoma, germ cell), thrombosis (catheters, pacemaker leads), fibrosing mediastinitis (histoplasma, TB); facial plethora, neck/arm swelling, dilated chest-wall collaterals, Pemberton's sign, headache, stridor (emergency), cerebral oedema.
- **Management:** CECT; obtain tissue before steroids/RT (lymphoma); **endovascular stenting** for rapid relief (severe/stridor); chemotherapy (SCLC, lymphoma), RT; anticoagulation for thrombosis; elevate head, diuretics/steroids short-term.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Re-expansion oedema — "slow and small": remove ≤1–1.5 L pleural fluid at once.

LANDMARK TRIALS & GUIDELINES

- McDonagh TA et al, 2021 ESC heart failure guidelines (2023 update); Yu DS et al, SVC syndrome review, Lancet Oncol (Wilson LD NEJM 2007).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Management of pulmonary oedema. *[DNB Oct 2024 P2]*
2. Differentiate between cardiogenic and non-cardiogenic pulmonary oedema. *[BFUHS 2019 P1]*
3. Re-expansion pulmonary oedema. *[DNB Oct 2023 P2]*
4. 2D ECHO in respiratory medicine. *[DNB Oct 2023 P4]*
5. Lymphatic (Kerley) lines. *[NEET-SS/DNB 2024]*
6. Jugular pulse and the superior vena cava syndrome. *[NEET-SS/DNB 2024]*

9.7 Haemoptysis — Causes, Massive Haemoptysis & Bronchial Artery Embolisation

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B10 Emergency problems; I-B13
- ✓ Enumerate causes of haemoptysis and approach diagnosis
- ✓ Define massive (life-threatening) haemoptysis and manage it in a stepwise manner
- ✓ Manage recurrent haemoptysis

IN BRIEF Haemoptysis is expectoration of blood from below the vocal cords. Life-threatening haemoptysis kills by asphyxiation, not exsanguination. In India, TB and post-TB sequelae (bronchiectasis, aspergilloma, Rasmussen aneurysm) are the commonest causes; bronchial artery embolisation is the first-line definitive therapy.

Causes of haemoptysis

Category	Causes
Airway	Bronchiectasis, bronchitis, bronchogenic carcinoma, carcinoid, foreign body, Dieulafoy's lesion, broncholithiasis, trauma
Parenchymal	TB (active and post-TB), lung abscess, necrotising pneumonia, aspergilloma/CPA, mucormycosis, paragonimiasis, hydatid

INVESTIGATIONS & TREATMENT AT A GLANCE

9.6 Pulmonary Oedema; Cardiac–Pulmonary Interactions

‹ Back to topic (p 177)
Next topic 9.7 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
BNP / NT-proBNP	BNP < 100; NT-proBNP < 300 pg/mL	Raised	Cardiogenic oedema
Lung ultrasound	A-lines	Bilateral B-lines (≥ 3 per zone)	Interstitial oedema
Echo (LVEF, E/e')	LVEF ≥ 50%; E/e' < 14	Low EF; E/e' > 14	Cardiac cause
Pleural/oedema fluid:serum protein	< 0.65 (hydrostatic)	> 0.7 (permeability)	Cardiogenic vs ARDS
Troponin / ECG	Normal	ACS	Precipitant

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Furosemide	40–80 mg IV (or 1–2.5 × home dose)	Cardiogenic pulmonary oedema	Hypokalaemia
Nitroglycerin	10–200 µg/min IV infusion	Hypertensive pulmonary oedema	Hypotension; avoid with PDE5i
CPAP / NIV	CPAP 5–10 cmH_2O	Acute cardiogenic pulmonary oedema	Hypotension
Non-cardiogenic oedema	Treat cause; lung-protective ventilation	ARDS, neurogenic, HAPE	—

★ **PRESCRIBING & INTERPRETATION PEARLS**

- CPAP reduces intubation in cardiogenic oedema — start early.

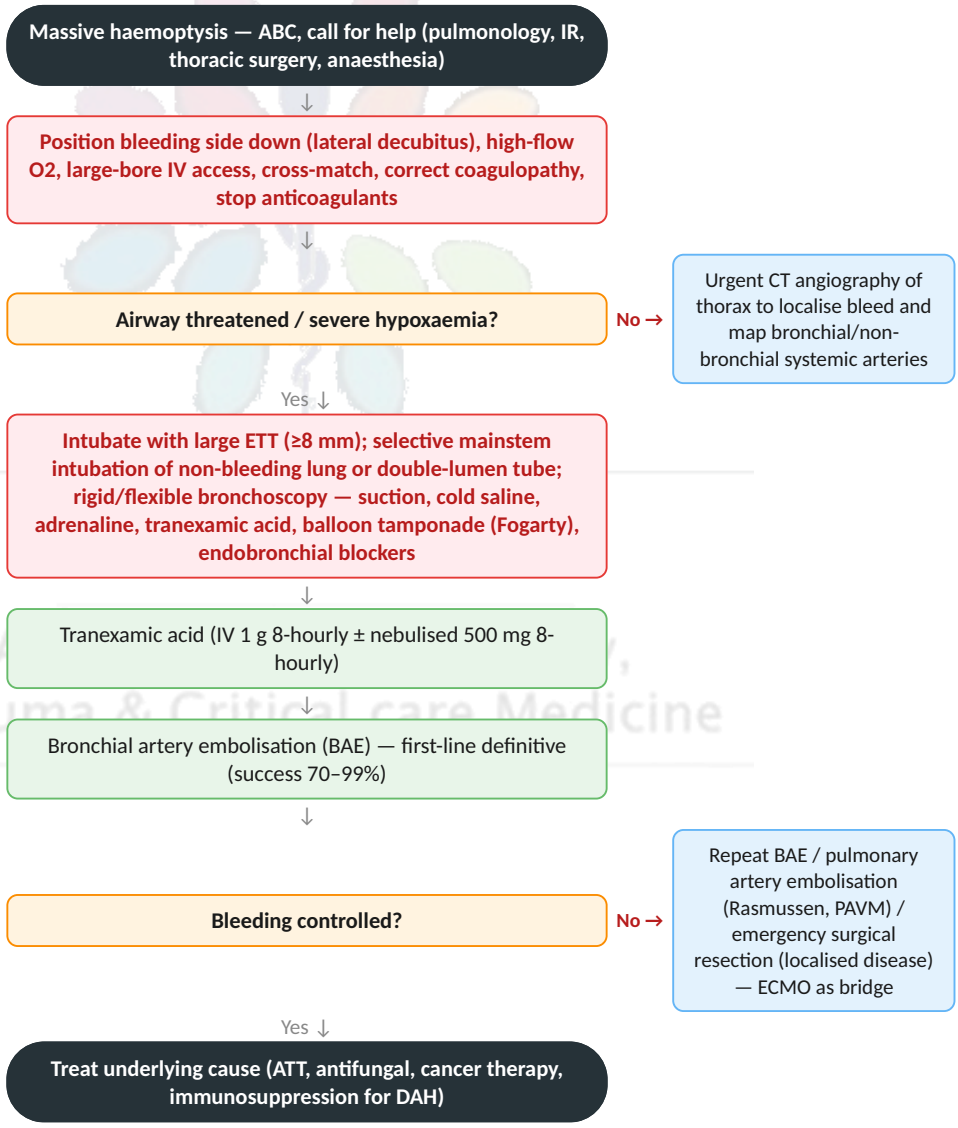
Category	Causes
Vascular	PE/infarction, PAVM, mitral stenosis, LV failure, pulmonary artery aneurysm (Rasmussen, Behçet), aortobronchial fistula
Diffuse alveolar haemorrhage	Vasculitis (GPA, MPA, anti-GBM), SLE, coagulopathy, anticoagulants, leptospirosis
Iatrogenic	Biopsy (TBLB, TTNA), PA catheter rupture, bevacizumab
Pseudo-haemoptysis	Upper airway/nasopharyngeal bleed, haematemesis, Serratia (red pigment)

- **Definition of massive/life-threatening:** variably 100–600 mL/24 h; functionally — any amount causing airway obstruction, hypoxaemia or haemodynamic instability, or >100 mL/h.

Haemoptysis vs haematemesis

Haemoptysis	Haematemesis
Coughed up, frothy, bright red, alkaline	Vomited, dark/coffee-ground, acidic, food particles
Respiratory history, prodrome of cough	GI/liver disease, nausea
Macrophages with haemosiderin	Melaena may follow

Algorithm 9.4 · Management of massive haemoptysis



Bronchial artery embolisation

- Selective angiography identifies hypertrophied, tortuous bronchial and non-bronchial systemic collaterals (intercostal, internal mammary, phrenic); embolise with PVA particles (>300–500 µm) or gelfoam, coils for aneurysms.
- **Complications:** chest pain, dysphagia, transient fever; **spinal cord ischaemia** (anterior spinal artery — transverse myelitis, rare), bronchial necrosis.
- **Recurrence:** 10–55% (aspergilloma, CPA, TB — highest); repeat BAE, definitive surgery.

Recurrent haemoptysis

- Identify and treat cause (sputum AFB/NAAT, Aspergillus IgG, CT angiography, bronchoscopy); repeat BAE; surgical resection for localised disease (aspergilloma, destroyed lobe, bronchiectasis) if adequate reserve; antifungal therapy for CPA; long-term tranexamic acid (selected); bronchiectasis care; radiotherapy for tumour/aspergilloma (selected).

★ CLINICAL PEARLS & EXAM FOCUS

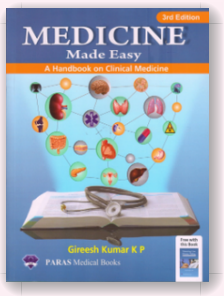
▸ In massive haemoptysis, the bleeding lung goes down — "good lung up".

LANDMARK TRIALS & GUIDELINES

- Davidson K, Shojaee S. Managing massive hemoptysis, Chest 2020; Wand O et al, nebulised tranexamic acid RCT, Chest 2018.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Enumerate the different causes of haemoptysis and management of massive haemoptysis. *[WBUHS 2020 P2]*
2. Discuss causes and management of massive hemoptysis. *[WBUHS 2022 P1]*
3. Describe the causes of haemoptysis; discuss management of massive haemoptysis. *[VNSGU 2025 P2]*
4. Management of recurrent haemoptysis. *[DNB Jun 2021 P2]*
5. Enumerate the causes, diagnostic approach and management of haemoptysis. *[Manipal 2014 P1]*



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INVESTIGATIONS & TREATMENT AT A GLANCE

9.7 Haemoptysis — Causes, Massive Haemoptysis & Bronchial Artery Embolisation

‹ Back to topic (p 178)
Next topic 10.1 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Volume of haemoptysis	—	> 100–600 mL/24 h or any amount with instability	Massive / life-threatening
CT angiography	—	Bleeding site, bronchial artery hypertrophy, Rasmussen aneurysm	Localisation, BAE planning
Coagulation profile / platelets	INR < 1.2; platelets > 150	Coagulopathy	Correct
Sputum Xpert / AFB	Negative	Positive	Active TB
Aspergillus IgG	Negative	Positive	Aspergilloma/CPA
Bronchoscopy	—	Active bleeding site, endobronchial tumour	Localisation, therapy

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Tranexamic acid	1 g IV 8-hourly or 500 mg nebulised 8-hourly	Haemoptysis	Thrombosis
Bronchial artery embolisation	—	Massive/recurrent haemoptysis	Spinal artery ischaemia
Endobronchial (cold saline, adrenaline 1:20 000, balloon tamponade)	—	Bleeding during bronchoscopy	—
Surgical resection	Lobectomy	Localised disease, failed BAE (aspergilloma)	High risk in emergency
Position	Bleeding side down; selective intubation of non-bleeding lung	Airway protection	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Death in massive haemoptysis is from asphyxia — protect the airway first.

SECTION 10

Lung Cancer & Other Thoracic Tumours

Paper II / IV

Neoplastic and non-neoplastic lesions of the lung — epidemiology, natural history, pathology, staging and principles of medical, surgical and radiation treatment; solitary pulmonary nodule; carcinoid and metastatic tumours; interventional bronchoscopy in central airway obstruction (NMC syllabus II-B7, I-D7, I-E2).

10.1 Epidemiology, Risk Factors, Genetic Susceptibility & Lung Cancer Screening 181
10.2 WHO Classification, Pathology, Tumour Markers & Liquid Biopsy 183
10.3 Clinical Features, Paraneoplastic Syndromes & Electrolyte Disorders 184
10.4 Diagnosis & Staging — TNM 9th Edition, EBUS & Mediastinal Staging 186
10.5 Treatment of NSCLC — Surgery, SBRT, Chemoradiation, Targeted Therapy & Immunotherapy 187
10.6 Small Cell Lung Cancer 189
10.7 Solitary Pulmonary Nodule & Incidental Nodules 190
10.8 Carcinoid Tumours, Metastatic Lung Disease & Other Tumours 192
10.9 Interventional Bronchoscopy — Central Airway Obstruction, Heat Ablation & Palliation 193

10.1 Epidemiology, Risk Factors, Genetic Susceptibility & Lung Cancer Screening

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B7 Epidemiology; I-E2
- ✓ Describe the epidemiology and risk factors of lung cancer in India
- ✓ Describe genetic susceptibility and molecular carcinogenesis
- ✓ Select patients for low-dose CT screening and implement Lung-RADS; discuss vaccines in lung cancer

IN BRIEF Lung cancer is the leading cause of cancer death worldwide (~1.8 million deaths/year) and among Indian men. Indian patients present younger, at advanced stage, with more adenocarcinoma and more never-smokers (biomass, air pollution) than in the West; misdiagnosis as TB delays treatment.

Risk factors

- **Tobacco smoking** (80–90%; dose–response, bidi more carcinogenic), second-hand smoke; **occupational:** asbestos, radon (uranium miners — 2nd commonest cause globally), arsenic, chromium, nickel, beryllium, cadmium, silica, diesel exhaust, polycyclic aromatic hydrocarbons; **environmental:** PM2.5 air pollution, household biomass/coal smoke, radon; **lung disease:** COPD, IPF, prior TB (scar carcinoma), HIV; **family history**; prior radiotherapy.

Genetic susceptibility

- **Inherited:** CHRNA3/5 nicotinic receptor locus (15q25), TERT, HLA region, CYP1A1 and GSTM1-null polymorphisms (carcinogen metabolism), germline EGFR T790M (familial), Li–Fraumeni (TP53), BRCA2; family history doubles risk.
- **Somatic drivers:** EGFR (India 23–30% of adenocarcinomas, never-smokers, women), ALK (5%), ROS1, KRAS (G12C in smokers), BRAF, MET, RET, HER2, NTRK; tumour suppressors TP53, RB1 (SCLC), STK11, KEAP1.
- **Field cancerisation** and multistep carcinogenesis (hyperplasia → metaplasia → dysplasia → CIS → invasive).

Lung cancer screening

Screening trials and eligibility

Item	Details
NLST (2011)	LDCT vs CXR: 20% reduction in lung cancer mortality
NELSON (2020)	LDCT: 24% (men) – 33% (women) mortality reduction
USPSTF 2021	Age 50–80, ≥20 pack-years, current smoker or quit within 15 years — annual LDCT
ACS 2023	50–80, ≥20 pack-years regardless of years since quitting

- **Implementation:** shared decision-making, smoking cessation integration, structured reporting (**Lung-RADS v2022:** 1 negative, 2 benign, 3 probably benign — 6-month LDCT, 4A/4B/4X suspicious — 3-month LDCT/PET/biopsy), MDT, quality-assured LDCT (<1.5 mSv).
- **Harms:** false positives (~25% NLST), overdiagnosis, radiation, invasive procedures, incidental findings, anxiety; in India TB granulomas increase false positives — screening not yet programmatic (studies ongoing in high-risk, incl. never-smoker women).
- **Patient selection models:** PLCOm2012 (≥1.5% 6-year risk) outperforms age/pack-year criteria.

Vaccines in lung cancer

- Therapeutic cancer vaccines aim to stimulate immunity against tumour antigens: **CIMAvax-EGF** (Cuba — anti-EGF antibodies, prolonged survival in NSCLC maintenance), **racotumomab** (anti-idiotype), tecemotide (MUC1 — START trial negative), belagenpumatucel, MAGE-A3 (MAGRIT negative); mRNA personalised neoantigen vaccines (BNT116) in trials.
- **Preventive:** HPV/HBV analogies do not apply; smoking cessation remains the main prevention.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Never-smoker woman with "non-resolving TB" and pleural effusion — think EGFR-mutant adenocarcinoma.

LANDMARK TRIALS & GUIDELINES

- National Lung Screening Trial Research Team NEJM 2011; de Koning HJ et al, NELSON NEJM 2020; Noronha V et al, Indian lung cancer profile (JCO Glob Oncol).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Genetic susceptibility to lung cancer. *[DNB Jun 2021 P1]*
2. Patient selection and implementation in lung cancer screening. *[DNB Oct 2023 P4]*
3. Role of vaccines in lung cancer. *[BFUHS 2019 P3]*

INVESTIGATIONS & TREATMENT AT A GLANCE

10.1 Epidemiology, Risk Factors, Genetic Susceptibility & Lung Cancer Screening

‹ Back to topic (p 181)
Next topic 10.2 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
LDCT screening eligibility	—	Age 50–80, ≥ 20 pack-years, quit < 15 y (USPSTF 2021)	Annual LDCT
Lung-RADS category	1–2 (annual LDCT)	3 (6-month LDCT); 4A/4B/4X (further work-up)	Nodule management
Pack-years	—	≥ 20	Risk
Radon / occupational exposure	—	Asbestos, silica, arsenic, radon	Risk factors
Family history / genetic	—	First-degree relative	Increased risk

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Smoking cessation	Varenicline / NRT	All smokers	Single most effective prevention
LDCT screening	Annual	High-risk group	False positives, radiation, overdiagnosis
Shared decision-making	—	Before screening	—

★ **PRESCRIBING & INTERPRETATION PEARLS**

- NLST: 20% lung-cancer mortality reduction; NELSON: 24% in men.

10.2 WHO Classification, Pathology, Tumour Markers & Liquid Biopsy

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B7; I-D7 Tumours of the lung
- ✓ Describe WHO 2021 classification of lung tumours and IHC-based subtyping
- ✓ Describe tumour markers and liquid biopsy in lung cancer

IN BRIEF Accurate histological subtyping (adenocarcinoma vs squamous vs neuroendocrine) and molecular profiling on small biopsies/cytology determine therapy. Liquid biopsy (ctDNA) complements tissue genotyping and monitors resistance.

WHO 2021 classification (major types)

Category	Types / features
Adenocarcinoma (~50–60%)	Precursors — AAH, AIS; minimally invasive (≤5 mm invasion); invasive non-mucinous (lepidic, acinar, papillary, micropapillary, solid — grading 2020 IASLC), invasive mucinous; TTF-1/Napsin A+
Squamous cell carcinoma (~20–25%)	Central, keratinisation, cavitation; p40/p63, CK5/6+; hypercalcaemia (PTHrP)
Neuroendocrine	Typical carcinoid (<2 mitoses/2 mm^2, no necrosis), atypical carcinoid (2–10, punctate necrosis), large cell NEC, small cell carcinoma (>10 mitoses, Ki-67 >50%)
Large cell carcinoma	Diagnosis of exclusion on resection (null IHC)
Others	Adenosquamous, sarcomatoid (pleomorphic, carcinosarcoma, pulmonary blastoma), salivary-type (adenoid cystic, mucoepidermoid), NUT carcinoma, SMARCA4-deficient tumours
Benign	Hamartoma (popcorn calcification, fat), papillomas, sclerosing pneumocytoma

Tumour markers in lung cancer

Serum tumour markers

Marker	Association	Use
CEA	Adenocarcinoma	Prognosis, monitoring; raised in smokers, GI cancers
CYFRA 21-1 (cytokeratin 19 fragment)	Squamous cell carcinoma	Prognosis, monitoring response
NSE, ProGRP	SCLC, neuroendocrine	Diagnosis support, monitoring (ProGRP more specific)
SCC antigen	Squamous	Limited
CA-125, CA 19-9	Adenocarcinoma, mesothelioma (pleural)	Limited

- Not recommended for screening or diagnosis alone; useful for monitoring and prognosis; pleural fluid CEA >5 ng/mL supports malignant effusion.

Liquid biopsy

- Analysis of tumour-derived material in blood/body fluids: **ctDNA** (cell-free DNA fragments), circulating tumour cells, exosomes, microRNAs.
- **Techniques:** ddPCR, BEAMing, NGS panels (Guardant360, FoundationOne Liquid).
- **Uses:** genotyping at diagnosis when tissue insufficient/unsafe (EGFR, ALK fusions), detecting resistance mutations (EGFR T790M, C797S, MET amplification) on progression, monitoring response, minimal residual disease after surgery, early detection (multi-cancer tests — research); pleural fluid supernatant and CSF liquid biopsy.
- **Limitations:** lower sensitivity (false negatives in low-shedding tumours — "plasma first, tissue if negative"), clonal haematopoiesis (CHIP) false positives, cost.

Molecular testing (IASLC/CAP)

- All non-squamous NSCLC (and squamous in never-smokers/young): broad NGS for EGFR, ALK, ROS1, BRAF, KRAS, MET ex14, RET, NTRK, HER2, NRG1; PD-L1 IHC (TPS) for all.

★ CLINICAL PEARLS & EXAM FOCUS

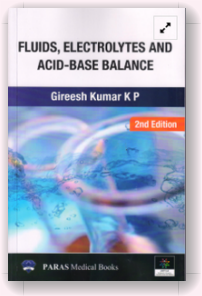
▸ TTF-1+ = adenocarcinoma (or SCLC); p40+ = squamous — get IHC on every small biopsy before writing "NSCLC-NOS".

LANDMARK TRIALS & GUIDELINES

- WHO Classification of Thoracic Tumours, 5th edition (2021); Rolfo C et al, IASLC liquid biopsy statement, J Thorac Oncol 2021.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Tumour markers in lung cancer. *[DNB Oct 2023 P2]*
2. Liquid biopsy tests for lung cancer patients. *[DNB Dec 2022 P4]*



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10.3 Clinical Features, Paraneoplastic Syndromes & Electrolyte Disorders

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B7; II-B11 Extrapulmonary manifestations
- ✓ Describe the local, regional, metastatic and paraneoplastic manifestations of lung cancer
- ✓ Differentiate paraneoplastic syndromes of SCLC and squamous carcinoma
- ✓ Manage hyponatraemia and hypercalcaemia of malignancy

IN BRIEF Most lung cancers present with symptoms of advanced disease. Paraneoplastic syndromes — endocrine, neurological, haematological, musculoskeletal — occur in ~10% and may precede the diagnosis; SCLC produces ectopic ADH/ACTH and autoimmune neurological syndromes, squamous cell carcinoma produces PTHrP.

Clinical manifestations

Category	Features
Local (central tumours)	Cough, haemoptysis, wheeze, stridor, post-obstructive pneumonia, collapse
Regional spread	Hoarseness (left recurrent laryngeal), SVC syndrome, Pancoast (C8–T2 pain, Horner's, hand muscle wasting), phrenic palsy, dysphagia, pleural/pericardial effusion
Metastatic	Brain (headache, seizures), bone (pain, fractures, hypercalcaemia), liver, adrenal, lymph nodes, skin
Constitutional	Weight loss, anorexia, fatigue, fever

INVESTIGATIONS & TREATMENT AT A GLANCE

10.2 WHO Classification, Pathology, Tumour Markers & Liquid Biopsy

‹ Back to topic (p 183)
Next topic 10.3 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Histology + IHC (TTF-1, Napsin A, p40)	—	TTF-1/Napsin A + → adenocarcinoma; p40 + → squamous	Subtype
EGFR mutation	Wild type	Exon 19 del / L858R	EGFR TKI (osimertinib)
ALK (IHC/FISH)	Negative	Rearranged	ALK TKI (alectinib, lorlatinib)
ROS1, BRAF V600E, KRAS G12C, MET ex14, RET, NTRK, HER2	Negative	Positive	Targeted therapy
PD-L1 (22C3 TPS)	< 1%	≥ 50% high	Immunotherapy selection
Liquid biopsy (ctDNA)	—	Driver mutation; T790M on progression	When tissue insufficient
CEA / CYFRA 21-1 / NSE / ProGRP	CEA < 5 ng/mL	Raised	Monitoring (not diagnosis); NSE/ProGRP in SCLC

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Osimertinib	80 mg daily	EGFR-mutant NSCLC (adjuvant & advanced)	ILD, QTc, cardiomyopathy
Alectinib	600 mg bd with food	ALK+ NSCLC	Myalgia, CK rise, bradycardia
Pembrolizumab	200 mg IV 3-weekly or 400 mg 6-weekly	PD-L1 ≥ 50% NSCLC	Immune-related AEs
Sotorasib	960 mg daily	KRAS G12C (second line)	Hepatotoxicity

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Test all non-squamous NSCLC (and young/never-smoker squamous) for driver mutations before first-line therapy.

Paraneoplastic syndromes by histology

Syndrome	Histology	Mechanism / features
SIADH (hyponatraemia)	SCLC (10–15%)	Ectopic ADH (also ANP)
Ectopic Cushing's	SCLC, carcinoid	ACTH — hypokalaemia, alkalosis, hyperglycaemia, weakness (rapid, without cushingoid habitus)
Hypercalcaemia	**Squamous** cell carcinoma	PTHrP; bone metastases
Lambert–Eaton myasthenic syndrome	SCLC	VGCC antibodies — proximal weakness improving with use, autonomic, areflexia
Paraneoplastic encephalomyelitis, sensory neuronopathy, cerebellar degeneration	SCLC	Anti-Hu (ANNA-1), anti-Yo, anti-Ri, anti-CRMP5, SOX2
Hypertrophic osteoarthropathy, clubbing	Adenocarcinoma, squamous	VEGF/PDGF
Gynaecomastia	Large cell	hCG
Hypercoagulability (Trousseau)	Adenocarcinoma	Migratory thrombophlebitis, NBTE, DIC
Dermatomyositis, acanthosis nigricans	Any	—
Haematological	Any	Eosinophilia, leucocytosis (G-CSF), thrombocytosis, anaemia

Paraneoplastic syndromes of squamous cell carcinoma

- Hypercalcaemia (PTHrP — commonest), hypertrophic pulmonary osteoarthropathy, clubbing, leucocytosis/eosinophilia (G-CSF/GM-CSF), hypercoagulability, dermatomyositis, Bazex syndrome (acrokeratosis paraneoplastica — upper aerodigestive SCC).

Electrolyte imbalance in SCLC

- **Hyponatraemia:** SIADH (euvolaemic, Uosm >100, UNa >30, low urate) ± ANP; also chemotherapy (cisplatin — salt wasting, cyclophosphamide, vincristine), vomiting, adrenal metastases. **Treatment:** fluid restriction, hypertonic saline for severe symptoms (rise ≤8–10 mmol/L/24 h), tolvaptan (avoid long-term — hepatotoxicity), urea, demeclocycline; chemotherapy treats the cause.
- **Hypokalaemia with metabolic alkalosis:** ectopic ACTH (also hyperglycaemia, hypertension) — check cortisol, ACTH; metyrapone, ketoconazole, osilodrostat, bilateral adrenalectomy.
- **Hypomagnesaemia, hypocalcaemia:** cisplatin; tumour lysis syndrome (hyperkalaemia, hyperphosphataemia, hypocalcaemia, hyperuricaemia) with chemotherapy in bulky SCLC — hydration, rasburicase.
- Hypercalcaemia is rare in SCLC (bone metastases).

Hypercalcaemia of malignancy — management

- IV normal saline 200–300 mL/h, zoledronic acid 4 mg (renal adjust) or denosumab 120 mg (renal failure), calcitonin for rapid short-term effect, steroids (lymphoma/granulomatous), dialysis if severe with renal failure.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Hyponatraemia, Cushing's and Lambert–Eaton → SCLC; hypercalcaemia → squamous.

LANDMARK TRIALS & GUIDELINES

- Kanaji N et al, Paraneoplastic syndromes associated with lung cancer, World J Clin Oncol 2014.

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INVESTIGATIONS & TREATMENT AT A GLANCE

10.3 Clinical Features, Paraneoplastic Syndromes & Electrolyte Disorders

‹ Back to topic (p 184)
Next topic 10.4 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Serum calcium (corrected)	8.5–10.5 mg/dL	> 12 (moderate), > 14 (severe)	PTHrP — squamous cell
Serum sodium / urine osmolality	Na 135–145	Na < 130 with urine osm > 100	SIADH — SCLC
Morning cortisol / ACTH	Normal	High ACTH, hypokalaemic alkalosis	Ectopic ACTH — SCLC
Anti-VGCC antibodies	Negative	Positive	Lambert–Eaton (SCLC)
Anti-Hu antibodies	Negative	Positive	Paraneoplastic encephalomyelitis
Clubbing / HPOA X-ray	—	Periosteal reaction of long bones	HPOA — NSCLC

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Zoledronic acid	4 mg IV over 15 min (renal-adjust)	Hypercalcaemia of malignancy	Hydrate first; osteonecrosis
0.9% saline	200–300 mL/h	Hypercalcaemia	Fluid overload
Fluid restriction / tolvaptan	< 1 L/day; tolvaptan 15 mg daily	SIADH	Overcorrection (> 10 mmol/L/24 h); hepatotoxicity
3% saline	100–150 mL bolus	Symptomatic severe hyponatraemia	ODS risk
Amifampridine (3,4-DAP)	10–20 mg 3–4 times daily	LEMS	Seizures

★ PRESCRIBING & INTERPRETATION PEARLS

- Correct sodium by no more than 8–10 mmol/L in 24 h.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Electrolyte imbalance in small cell lung carcinoma. *[DNB Oct 2024 P2]*
2. Paraneoplastic syndrome in squamous cell lung carcinoma. *[DNB Oct 2024 P2]*
3. Paraneoplastic syndromes. *[DNB Jun 2021 P2]*

10.4 Diagnosis & Staging — TNM 9th Edition, EBUS & Mediastinal Staging

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B7 Staging
- ✓ Plan the diagnostic and staging work-up to obtain tissue and stage in one step
- ✓ Describe TNM 9th edition (2024) descriptors and stage groups
- ✓ Describe the role of EBUS-TBNA in mediastinal staging

IN BRIEF The goal is to diagnose and stage with the least invasive, highest-yield procedure — sampling the site that would confer the highest stage. PET-CT and EBUS-TBNA have largely replaced mediastinoscopy for mediastinal staging.

Diagnostic and staging work-up

- CECT chest + upper abdomen → **PET-CT** (for potentially curable disease) → **brain MRI** (stage II and above, or symptoms; all stage III–IV) → tissue: EBUS-TBNA (central tumours/nodes), radial EBUS/navigational bronchoscopy (peripheral), CT-guided TTNA (peripheral — pneumothorax 15–25%), pleural fluid cytology/thoracoscopy, biopsy of metastasis (liver, adrenal, node, skin), sputum cytology (central squamous).
- Molecular profiling and PD-L1 on all diagnostic samples.

TNM 9th edition (IASLC, 2024) — key descriptors

Descriptor	Definition
T1 (a/b/c)	≤3 cm (≤1, >1–2, >2–3 cm), surrounded by lung/visceral pleura
T2 (a/b)	>3–5 cm (a >3–4, b >4–5), or main bronchus involvement (not carina), visceral pleural invasion, atelectasis/obstructive pneumonitis extending to hilum
T3	>5–7 cm, or chest wall, parietal pleura, phrenic nerve, parietal pericardium invasion, separate nodule same lobe
T4	>7 cm, or diaphragm, mediastinum, heart, great vessels, trachea, carina, recurrent laryngeal nerve, oesophagus, vertebral body; separate nodule different ipsilateral lobe
N1	Ipsilateral peribronchial/hilar/intrapulmonary nodes
N2a / N2b (new)	Ipsilateral mediastinal/subcarinal — **single station (N2a)** vs **multiple stations (N2b)**
N3	Contralateral mediastinal/hilar, any scalene or supraclavicular
M1a	Separate nodule in contralateral lobe, pleural/pericardial nodules or malignant effusion
M1b	Single extrathoracic metastasis in a single organ
M1c1 / M1c2 (new)	Multiple extrathoracic metastases in **one organ system (M1c1)** vs **multiple organ systems (M1c2)**

Simplified stage groups

Stage	Typical TNM
IA	T1 N0 M0
IB / IIA	T2a N0 / T2b N0
IIB	T1–2 N1; T3 N0; **T1 N2a (9th edition)**
IIIA	T1–2 N2b, T2 N2a, T3 N1, T4 N0–1

Stage	Typical TNM
IIIB / IIIC	N3 or T3–4 N2; T3–4 N3
IVA	M1a, M1b
IVB	M1c (M1c1 and M1c2)

Other staging systems

- **SCLC:** VALSG — limited stage (confined to one hemithorax, encompassable in one radiation field) vs extensive stage; TNM also applied.
- **Mesothelioma:** IASLC/AJCC TNM; **Carcinoid:** TNM.

Endobronchial ultrasound for mediastinal lymphadenopathy

- **Convex-probe EBUS (CP-EBUS):** real-time TBNA of stations 2R/L, 4R/L, 7, 10, 11, 12 (± EUS-B for 5, 8, 9, left adrenal); 22G/19G needles; rapid on-site evaluation (ROSE).
- **Accuracy in staging:** sensitivity ~90%, NPV ~90%; combined EBUS + EUS > mediastinoscopy (ASTER trial); negative EBUS with high suspicion (PET-positive N2) → confirm with mediastinoscopy.
- **Other indications:** sarcoidosis (granulomas — yield 80–90% with EBUS vs TBLB), TB lymphadenitis (Xpert on aspirate), lymphoma (core/cryo-biopsy), central lesions adjacent to airway, restaging after neoadjuvant therapy, PE detection.
- **Radial EBUS** (rEBUS) — localises peripheral nodules for biopsy.
- Complications: very low (<1%) — bleeding, infection (mediastinitis), pneumothorax.

★ **CLINICAL PEARLS & EXAM FOCUS**

▸ Biopsy the lesion that gives the highest stage first — e.g., EBUS of a PET-avid N3 node diagnoses and stages simultaneously.

LANDMARK TRIALS & GUIDELINES

- Rami-Porta R et al, IASLC proposals for TNM 9th edition, J Thorac Oncol 2023–24; Annema JT et al, ASTER JAMA 2010; Silvestri GA et al, ACCP staging guideline 2013.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Staging of lung cancer. *[BFUHS 2020 P4]*
2. Discuss classification systems for staging of lung cancer. *[VNSGU 2025 P4]*
3. Endobronchial ultrasound for evaluating mediastinal lymphadenopathy. *[BFUHS 2019 P2]*

10.5 Treatment of NSCLC — Surgery, SBRT, Chemoradiation, Targeted Therapy & Immunotherapy

‹ NMC curriculum
Previous-year Qs ›

◉ **CBME COMPETENCIES — the postgraduate should be able to:**

- ✓ **NMC syllabus area:** II-B7 Principles of treatment; I-G3
- ✓ Outline stage-wise treatment of NSCLC
- ✓ Describe targeted therapies including oral EGFR TKIs and immunotherapy
- ✓ Describe stereotactic body radiotherapy and prognostic factors in NSCLC

IN BRIEF Early-stage NSCLC is treated with curative surgery (or SBRT), increasingly with perioperative immunotherapy or targeted therapy; stage III with concurrent chemoradiation followed by consolidation; and stage IV according to driver mutation and PD-L1 status — transforming survival in oncogene-addicted disease.

INVESTIGATIONS & TREATMENT AT A GLANCE

10.4 Diagnosis & Staging — TNM 9th Edition, EBUS & Mediastinal Staging

‹ Back to topic (p 186)
Next topic 10.5 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
CECT chest + upper abdomen	—	T and N staging, adrenal/liver mets	Staging
PET-CT	SUVmax < 2.5	FDG-avid nodes/metastases	Mediastinal/distant staging
EBUS-TBNA	—	Malignant nodes (N2/N3)	Mediastinal staging (sensitivity ~ 90%)
MRI brain	Normal	Metastases	Stage ≥ II / symptomatic
Pleural fluid cytology	Negative	Malignant cells	M1a
TNM 9th edition	—	N2a single-station, N2b multi-station; M1c1 single-organ, M1c2 multi-organ	Stage groups

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
EBUS-TBNA	22G needle, ≥ 3 passes per station; ROSE	Staging and diagnosis	—
Mediastinoscopy	Surgical	Negative EBUS with high suspicion	—
Pleural fluid drainage before staging	—	Large effusion	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Stage before treatment — N2 disease changes surgery to multimodality therapy.

Stage-wise treatment of NSCLC (2024–25)

Stage	Treatment
I–II (operable)	Lobectomy (segmentectomy for ≤2 cm peripheral — JCOG0802, CALGB 140503) + systematic nodal dissection; adjuvant chemotherapy stage II–IIIA; adjuvant osimertinib for EGFR+ (ADAURA), alectinib for ALK+ (ALINA); perioperative chemo-immunotherapy (KEYNOTE-671, CheckMate 77T) or neoadjuvant (CheckMate 816)
I (inoperable)	SBRT (local control >90%)
III resectable	Neoadjuvant/perioperative chemo-immunotherapy → surgery
III unresectable	Concurrent platinum chemoradiation → durvalumab 12 months (PACIFIC); EGFR+ → osimertinib after CRT (LAURA)
IV with driver	Targeted therapy (table)
IV without driver	PD-L1 ≥50%: pembrolizumab/atezolizumab/cemiplimab monotherapy; any PD-L1: platinum doublet + pembrolizumab (pemetrexed for non-squamous; paclitaxel for squamous); nivolumab + ipilimumab ± chemo
Oligometastatic	Systemic therapy + local consolidative therapy (SBRT/surgery)

Targeted therapies for oncogene-driven NSCLC

Alteration	First-line drugs
EGFR (exon 19 del, L858R)	Osimertinib (FLAURA) ± chemotherapy (FLAURA2); amivantamab + lazertinib (MARIPOSA); gefitinib/erlotinib/afatinib (older, cheaper — India)
EGFR exon 20 insertion	Amivantamab + chemotherapy (PAPILLON)
ALK	Alectinib, lorlatinib (CROWN — superior PFS), brigatinib
ROS1	Crizotinib, entrectinib, repotrectinib
BRAF V600E	Dabrafenib + trametinib
KRAS G12C	Sotorasib, adagrasib (second-line)
MET exon 14 skipping	Capmatinib, tepotinib
RET	Selpercatinib, pralsetinib
NTRK	Larotrectinib, entrectinib
HER2 mutation	Trastuzumab deruxtecan

Oral TKIs for lung adenocarcinoma

- **EGFR TKIs:** 1st gen (gefitinib 250 mg, erlotinib 150 mg — reversible), 2nd gen (afatinib, dacomitinib — irreversible pan-HER), 3rd gen (**osimertinib 80 mg** — mutant-selective, covers T790M, CNS penetration).
- **Response:** ORR 60–80%, PFS 10–19 months (osimertinib 18.9 months, OS 38.6 months in FLAURA).
- **Adverse effects:** acneiform rash, diarrhoea, paronychia, stomatitis, ILD (1–4% — stop), QT (osimertinib), cardiomyopathy.
- **Resistance:** T790M (1st/2nd gen — switch to osimertinib), C797S, MET/HER2 amplification, small-cell transformation — rebiopsy/liquid biopsy.

Immunotherapy in lung cancer

- Checkpoint inhibitors block PD-1 (pembrolizumab, nivolumab, cemiplimab), PD-L1 (atezolizumab, durvalumab) or CTLA-4 (ipilimumab, tremelimumab), restoring T-cell antitumour activity.
- **Biomarkers:** PD-L1 TPS (≥50% — monotherapy; KEYNOTE-024 5-year OS 32%), TMB; poor response in EGFR/ALK+ tumours.
- **Immune-related adverse events:** pneumonitis, colitis, hepatitis, endocrinopathies (thyroiditis, hypophysitis, adrenal insufficiency, diabetes), dermatitis, myocarditis — steroids per grade.

Stereotactic body radiotherapy (SBRT/SABR)

- High-dose ablative radiation in few fractions (e.g., 54 Gy in 3 fractions peripheral; 60 Gy in 8 central) with precise image guidance and motion management.

- **Indications:** medically inoperable or refusing surgery stage I NSCLC (≤5 cm, N0); oligometastases; recurrence; selected lung metastases.
- **Outcomes:** local control >90%; comparable to surgery in small trials (STARS); toxicity — radiation pneumonitis, chest wall pain/rib fracture, oesophagitis, central airway toxicity ("no-fly zone" — ultracentral tumours).

Prognostic factors in NSCLC

- **Tumour:** stage (most important), histology and grade, driver mutations (EGFR/ALK better), PD-L1, visceral pleural/lymphovascular invasion, STAS, completeness of resection (R0).
- **Patient:** performance status (ECOG), weight loss >10%, age, sex (women better), comorbidities, smoking status; **laboratory:** high LDH, low albumin, anaemia, high NLR, hypercalcaemia.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Test every non-squamous NSCLC for drivers before starting immunotherapy — immunotherapy works poorly and is more toxic (pneumonitis/hepatitis) before osimertinib.

LANDMARK TRIALS & GUIDELINES

- Soria JC et al, FLAURA NEJM 2018; Antonia SJ et al, PACIFIC NEJM 2017; Wu YL et al, ADAURA NEJM 2020; Reck M et al, KEYNOTE-024 NEJM 2016.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Factors affecting prognosis of non-small cell lung carcinoma (NSCLC). *[DNB Dec 2022 P2]*
2. Oral tyrosine kinase inhibitors for lung adenocarcinoma. *[BFUHS 2019 P2]*
3. Immunotherapy in lung cancer. *[BFUHS 2020 P2]*
4. Stereotactic body radiotherapy (SBRT). *[DNB Dec 2025 P2]*

10.6 Small Cell Lung Cancer

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B7 Treatment
- ✓ Describe the biology, staging and clinical behaviour of SCLC
- ✓ Treat limited- and extensive-stage SCLC

IN BRIEF SCLC (~13–15%) is a high-grade neuroendocrine tumour of heavy smokers (TP53 and RB1 inactivation), with rapid doubling, early metastasis and initial chemosensitivity but early relapse. Median survival: limited stage 15–30 months; extensive stage ~12 months with chemo-immunotherapy.

Treatment of SCLC

Stage	Treatment
Very limited (T1–2 N0)	Lobectomy + adjuvant chemotherapy (rare)
Limited stage	Concurrent cisplatin/carboplatin + etoposide with thoracic RT (45 Gy BD over 3 weeks — Turrisi, or 60–70 Gy OD) started early; **consolidation durvalumab** (ADRIATIC, 2024 — OS benefit); PCI (25 Gy) or MRI surveillance in responders
Extensive stage	Carboplatin + etoposide + atezolizumab (IMpower133) or durvalumab (CASPIAN) ×4 then maintenance immunotherapy; thoracic RT for residual disease (selected); PCI or MRI surveillance
Relapse	Platinum-sensitive (>6 months): rechallenge platinum-etoposide; topotecan, lurbinectedin, irinotecan; **tarlatamab** (DLL3 × CD3 bispecific T-cell engager — DeLLphi-301/304)
Palliative	RT for brain/bone metastases, SVC stenting, management of SIADH/Cushing's

INVESTIGATIONS & TREATMENT AT A GLANCE

10.5 Treatment of NSCLC — Surgery, SBRT, Chemoradiation, Targeted Therapy & Immunotherapy

‹ Back to topic (p 187)
Next topic 10.6 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
FEV1 / DLCO (pre-op)	> 80% predicted	ppoFEV1 or ppoDLCO < 60% → exercise test; < 30% high risk	Operability
VO_2max (CPET)	> 20 mL/kg/min	< 10 mL/kg/min	High risk
Performance status (ECOG)	0–1	≥ 3	Systemic therapy fitness
PD-L1 / driver mutations	—	Positive	Treatment selection
CBC, LFT, creatinine, TSH (ICI)	Normal	Abnormal	Toxicity monitoring

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Lobectomy / segmentectomy	Surgical	Stage I–II (segmentectomy for ≤ 2 cm peripheral)	—
SBRT	48–60 Gy in 3–8 fractions	Inoperable stage I	Central tumours caution
Chemoradiation + durvalumab	Cisplatin-based CRT; durvalumab 10 mg/kg 2-weekly × 12 months	Unresectable stage III (PACIFIC)	Pneumonitis
Neoadjuvant nivolumab + chemo	Nivolumab 360 mg + platinum doublet × 3	Resectable stage II–IIIA (CheckMate 816)	—
Carboplatin + pemetrexed + pembrolizumab	Carboplatin AUC 5, pemetrexed 500 mg/m², pembro 200 mg 3-weekly	Advanced non-squamous NSCLC	Folate + B12 with pemetrexed

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Pemetrexed needs folic acid 400 µg daily and vitamin B12 1 mg IM every 9 weeks.

Key issues

- Brain metastases common (50% at 2 years); PCI reduces symptomatic brain metastases and improved survival in ES-SCLC (Slotman) but Japanese trial showed no OS benefit with MRI surveillance — either acceptable.
- Molecular subtypes (SCLC-A ASCL1, -N NEUROD1, -P POU2F3, -I inflamed) may guide future therapy.
- Paraneoplastic syndromes (see 10.3) may improve with tumour response.
- **Transformation** of EGFR-mutant adenocarcinoma to SCLC on TKI.

★ CLINICAL PEARLS & EXAM FOCUS

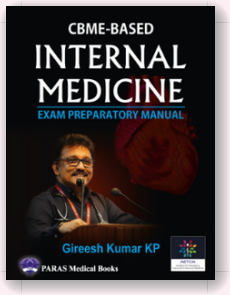
▸ SCLC is a systemic disease at diagnosis — even "limited stage" needs chemotherapy; surgery only for rare T1–2 N0 nodules.

LANDMARK TRIALS & GUIDELINES

- Horn L et al, IMpower133 NEJM 2018; Paz-Ares L et al, CASPIAN Lancet 2019; Cheng Y et al, ADRIATIC NEJM 2024; Ahn MJ et al, DeLLphi-301 NEJM 2023.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Treatment of small cell lung carcinoma (SCLC). *[DNB Dec 2022 P2]*
2. Recent TNM staging of carcinoma lung; diagnosis and management of small cell carcinoma; paraneoplastic syndromes with SCLC. *[VNSGU 2025 P3]*



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10.7 Solitary Pulmonary Nodule & Incidental Nodules

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B7 Solitary pulmonary nodule
- ✓ Define a solitary pulmonary nodule and list its differential diagnosis
- ✓ Estimate malignancy probability and apply Fleischner 2017 and BTS guidance

IN BRIEF An SPN is a round opacity ≤3 cm surrounded by lung, without atelectasis or adenopathy (>3 cm = mass). Management balances malignancy probability (clinical risk + size + morphology + growth) against risks of surveillance, biopsy or resection.

Differential diagnosis of SPN

Category	Causes
Malignant	Primary lung cancer (adenocarcinoma), carcinoid, solitary metastasis (colon, kidney, breast, sarcoma, melanoma), lymphoma
Infectious granuloma	Tuberculoma (commonest in India), histoplasmoma, cryptococcoma, NTM, round pneumonia, abscess, hydatid, dirofilaria
Benign neoplasm	Hamartoma (popcorn calcification, fat), chondroma, lipoma, sclerosing pneumocytoma

INVESTIGATIONS & TREATMENT AT A GLANCE

10.6 Small Cell Lung Cancer

‹ Back to topic (p 189)
Next topic 10.7 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Histology	—	Small round blue cells, high Ki-67, neuroendocrine markers	SCLC
Staging (limited vs extensive)	—	Extensive: beyond one radiation field	Treatment plan
Sodium / cortisol	Normal	SIADH / Cushing's	Paraneoplastic
MRI brain	Normal	Metastases	—
NSE / ProGRP	—	Raised	Monitoring

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Etoposide + cisplatin/ carboplatin	Etoposide 100 mg/m² d1–3 + cisplatin 75 mg/m² d1, 3-weekly × 4–6	Limited and extensive	Myelosuppression
Thoracic RT (concurrent)	45 Gy bd in 30 fractions	Limited stage	Oesophagitis
Atezolizumab / durvalumab + chemo	Atezolizumab 1200 mg 3-weekly	Extensive stage	irAEs
Durvalumab consolidation	1500 mg 4-weekly	Limited stage after CRT (ADRIATIC)	—
Prophylactic cranial irradiation / MRI surveillance	25 Gy in 10 fractions	Responders	Neurocognitive
Lurbinectedin / tarlatamab	Lurbinectedin 3.2 mg/m² 3-weekly; tarlatamab per protocol	Relapsed SCLC	Myelosuppression; CRS

★ **PRESCRIBING & INTERPRETATION PEARLS**

- SCLC is exquisitely chemosensitive but relapses early — surgery only for T1–2 N0.

Category	Causes
Inflammatory	Rheumatoid nodule, GPA, sarcoid, organising pneumonia, IgG4
Vascular	PAVM (feeding artery), infarct, haematoma, pulmonary vein varix
Congenital / other	Bronchogenic cyst, sequestration, mucocele (bronchial atresia), round atelectasis, intrapulmonary lymph node (perifissural)

Features suggesting malignancy vs benignity

Malignant	Benign
Age >50, smoker, prior cancer, upper lobe	Age <35, non-smoker
Size >8 mm; growth (VDT 20–400 days)	Stable ≥2 years (solid), VDT <20 or >400 days
Spiculated (corona radiata), lobulated, pleural tag	Smooth, well-defined
Eccentric/stippled calcification; air bronchogram, pseudocavitation	Central, laminated, diffuse or popcorn calcification; fat (hamartoma)
Part-solid with solid component >6 mm	Perifissural triangular shape (lymph node)
PET SUVmax >2.5 (beware TB, false negatives in AIS/carcinoid)	Enhancement <15 HU

Risk calculators

- **Brock (PanCan)** model (incidental/screen-detected: age, sex, family history, emphysema, size, type, upper lobe, count, spiculation); **Herder** (adds PET); **Mayo Clinic**.

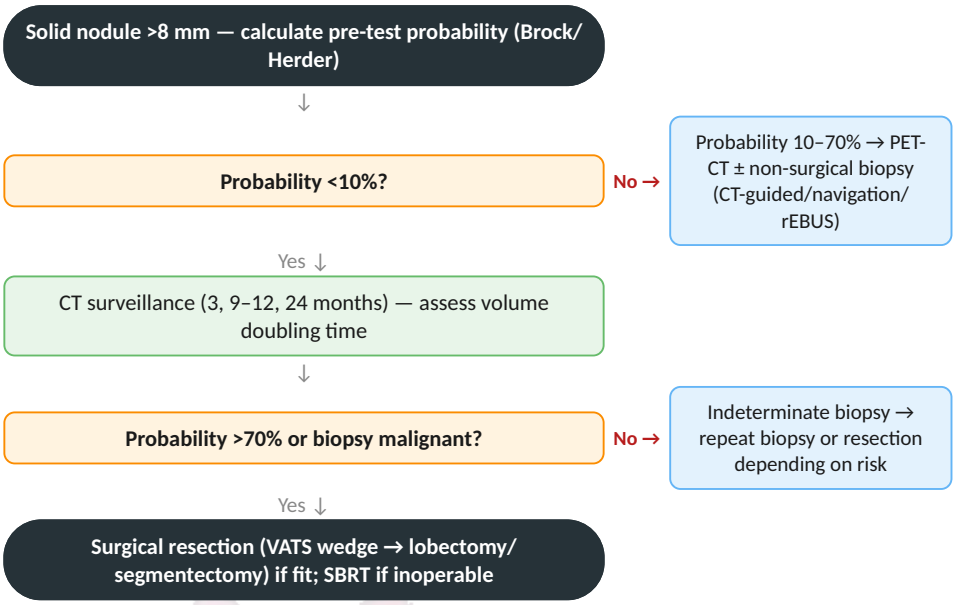
Fleischner Society 2017 — solid nodules (incidental, ≥35 years)

Size	Low risk	High risk
<6 mm (<100 mm³)	No routine follow-up	Optional CT at 12 months
6–8 mm	CT 6–12 months, then consider 18–24	CT 6–12 months, then 18–24 months
>8 mm	CT 3 months, PET-CT, or tissue sampling	Same

- **Subsolid:** pure GGN <6 mm — no follow-up; ≥6 mm — CT 6–12 months then every 2 years to 5 years; part-solid ≥6 mm — CT 3–6 months; persistent with solid component ≥6 mm → highly suspicious (PET, biopsy, resection).
- Not for screening (Lung-RADS), <35 years, cancer patients, immunosuppressed.

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Algorithm 10.1 · Management of an incidental solid nodule >8 mm



★ CLINICAL PEARLS & EXAM FOCUS

▸ In India, a tuberculoma is the commonest SPN — but in a smoker >50 years, assume cancer until proven otherwise.

LANDMARK TRIALS & GUIDELINES

• MacMahon H et al, Fleischner Society guidelines, Radiology 2017; Callister MEJ et al, BTS pulmonary nodule guideline, Thorax 2015.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Solitary lung lesion. *[DNB Jun 2025 P2]*
2. Differential diagnosis of incidental pulmonary nodule. *[DNB Dec 2022 P2]*
3. Solitary pulmonary nodule: definition, differential diagnosis and management. *[BFUHS 2020 P2]*

10.8 Carcinoid Tumours, Metastatic Lung Disease & Other Tumours

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B7 Non-neoplastic and neoplastic diseases
- ✓ Manage localised lung carcinoid and carcinoid syndrome
- ✓ Describe patterns of pulmonary metastases and lymphangitis carcinomatosa
- ✓ Recognise other primary pulmonary tumours (hamartoma, lymphoma, sarcoma)

IN BRIEF Pulmonary carcinoids are well-differentiated neuroendocrine tumours (typical/atypical) of younger, non-smoking patients, often central, causing haemoptysis and recurrent pneumonia. Surgery is curative for localised disease.

Clinical features of carcinoid

- Central (80%) — cough, haemoptysis (vascular), wheeze, recurrent post-obstructive pneumonia, collapse; peripheral — incidental nodule. Cherry-red polypoid lesion at bronchoscopy (bleeds on biopsy).
- **Carcinoid syndrome** (<5% of lung carcinoids; liver metastases): flushing (bronchial carcinoids — prolonged, with periorbital oedema), diarrhoea, wheeze, hypotension; carcinoid heart disease (left-sided possible with bronchial carcinoid). Ectopic ACTH (Cushing's), acromegaly (GHRH).

INVESTIGATIONS & TREATMENT AT A GLANCE

10.7 Solitary Pulmonary Nodule & Incidental Nodules

‹ Back to topic (p 190)
Next topic 10.8 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Nodule size (Fleischner 2017, solid)	< 6 mm (no follow-up in low risk)	6–8 mm: CT 6–12 months; > 8 mm: CT 3 months/PET/biopsy	Management
Subsolid nodule	Pure GGN < 6 mm	Part-solid ≥ 6 mm with solid component ≥ 6 mm	Higher malignancy risk
Brock (PanCan) / Herder model	< 10%	> 70%	Malignancy probability
PET-CT	SUV < 2.5	Avid	Malignancy likely (false + in TB/ infection)
Volume doubling time	> 400 days (or < 20 days) benign	20–400 days	Malignant growth

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Surveillance CT	Per Fleischner	Low-intermediate risk	Radiation, anxiety
CT-guided / navigational biopsy	—	Intermediate risk	Pneumothorax (15–25%)
Surgical resection (wedge/ segment)	—	High probability	—
SBRT	—	High risk, unfit for surgery	—

★ PRESCRIBING & INTERPRETATION PEARLS

- In India, granulomas (TB) cause PET-avid nodules — biopsy before resection where feasible.

- **Investigations:** CT, **Ga-68 DOTATATE PET-CT** (somatostatin receptor imaging; FDG for atypical), 24-h urine 5-HIAA, chromogranin A, bronchoscopic biopsy, Ki-67.

Management of localised lung carcinoid

- **Surgery:** anatomical resection (lobectomy, sleeve resection for central tumours to preserve lung, segmentectomy for small peripheral typical carcinoids) with **systematic lymph node dissection**. 5-year survival typical >90%, atypical 60–70%.
- Bronchoscopic resection (laser/cryo) for purely intraluminal typical carcinoid in poor surgical candidates or as bridge.
- Adjuvant therapy generally not indicated (consider for node-positive atypical); long-term follow-up (late recurrence).

Unresectable/metastatic carcinoid and syndrome management

- **Symptom control:** somatostatin analogues (octreotide LAR 30 mg, lanreotide 120 mg 4-weekly), short-acting octreotide for breakthrough; telotristat (refractory diarrhoea); avoid triggers (alcohol, stress); **octreotide cover before surgery/procedures** to prevent carcinoid crisis.
- **Antiproliferative:** SSAs (slow progression), everolimus (RADIANT-4), PRRT (177Lu-DOTATATE), temozolomide-based chemotherapy (atypical), liver-directed therapy (embolisation, resection), platinum-etoposide for high-grade.

Metastatic lung disease

- Haematogenous **cannon-ball metastases** (RCC, sarcoma, choriocarcinoma, colon, melanoma, testicular), miliary (thyroid, melanoma, RCC), cavitating (squamous of head/neck/cervix, sarcoma), calcified (osteosarcoma, chondrosarcoma, mucinous adenocarcinoma, thyroid), endobronchial (RCC, breast, colon, melanoma).
- **Lymphangitis carcinomatosa:** breast, stomach, lung, pancreas, prostate — smooth/nodular septal thickening, peribronchovascular thickening, effusion; severe dyspnoea; steroids palliative; treat primary.
- **Pulmonary metastasectomy** criteria: primary controlled, no extrathoracic disease, all lesions resectable with adequate reserve, long disease-free interval (sarcoma, colorectal, RCC, germ cell).

Other tumours

- **Hamartoma** (commonest benign — popcorn calcification, fat; observe), MALT lymphoma (BALT), lymphomatoid granulomatosis (EBV), primary pulmonary sarcoma, pulmonary blastoma, Kaposi sarcoma (HIV — flame-shaped peribronchovascular), inflammatory myofibroblastic tumour (ALK), epithelioid haemangioendothelioma.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Never biopsy a cherry-red endobronchial polyp without preparing for bleeding — it is a carcinoid until proven otherwise.

LANDMARK TRIALS & GUIDELINES

- ENETS/NANETS consensus on lung neuroendocrine tumours (Caplin ME, Ann Oncol 2015); ESMO lung NET guideline 2021.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Management of lung carcinoid tumours with localised disease. *[DNB Dec 2022 P2]*
2. Symptoms of carcinoid syndrome and their management in patients with unresectable disease. *[DNB Dec 2022 P2]*

10.9 Interventional Bronchoscopy — Central Airway Obstruction, Heat Ablation & Palliation

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B7 Treatment; Diagnostic tests — therapeutic bronchoscopy
- ✓ Classify central airway obstruction and choose bronchoscopic modalities
- ✓ Describe heat-ablative (laser, electrocautery, APC) and cold modalities and stents
- ✓ Describe bronchoscopic palliation in lung cancer

INVESTIGATIONS & TREATMENT AT A GLANCE

10.8 Carcinoid Tumours, Metastatic Lung Disease & Other Tumours

‹ Back to topic (p 192)
Next topic 10.9 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Chromogranin A	< 100 ng/mL	Raised	Neuroendocrine tumour
24-h urinary 5-HIAA	< 10 mg/24 h	> 25 mg/24 h	Carcinoid syndrome (rare in lung)
Ki-67	< 2% typical carcinoid	2–20% atypical	Grading
Ga-68 DOTATATE PET	—	Avid	Well-differentiated NET
Primary site work-up	—	Breast, colon, kidney, melanoma, sarcoma	Metastatic lung disease

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Surgical resection	Lobectomy / sleeve	Typical/atypical carcinoid	—
Octreotide LAR	20–30 mg IM 4-weekly	Carcinoid syndrome, unresectable NET	Gallstones
Everolimus	10 mg daily	Progressive lung NET	Stomatitis, pneumonitis
Pulmonary metastasectomy	—	Limited metastases with controlled primary	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Bronchial carcinoid presents with haemoptysis and recurrent pneumonia in young non-smokers.

IN BRIEF Central airway obstruction (CAO — trachea, main and intermediate bronchi) causes dyspnoea, stridor and post-obstructive pneumonia; malignant CAO occurs in 20–30% of lung cancers. Rigid bronchoscopy with debulking, ablation and stenting provides rapid relief, improving quality of life and enabling further oncological therapy.

Types of central airway obstruction

Type	Description	Preferred approach
Intraluminal (endobronchial)	Tumour inside lumen	Mechanical debulking, heat ablation (laser, electrocautery, APC), cryo-recanalisation
Extrinsic compression	Tumour/node outside wall	Airway stent (silicone, SEMS), balloon dilatation
Mixed	Both	Debulking + stent
Benign stenosis	Post-intubation, TB, tracheostomy, GPA	Dilatation, radial cuts, stents (silicone), surgery

Bronchoscopic heat-ablative modalities

Modality	Mechanism	Advantages	Cautions
Nd:YAG / thulium / diode laser	Thermal coagulation and vaporisation; deep penetration (Nd:YAG 5–10 mm)	Rapid, precise debulking; good haemostasis	FiO2 <0.4 (airway fire), perforation, cost
Electrocautery (snare, knife, probe)	High-frequency current — coagulation/ cutting	Cheap, versatile, polyp snaring	Airway fire, current dispersion; FiO2 <0.4
Argon plasma coagulation (APC)	Ionised argon conducts current — superficial (2–3 mm) non-contact	Haemostasis, superficial tumour, around corners	Gas embolism, fire
Microwave / radiofrequency ablation (peripheral)	Navigational ablation of peripheral nodules	Inoperable early cancer (research)	—

- **Cold modalities:** cryorecanalisation/cryodebulking (immediate), cryotherapy (delayed effect), photodynamic therapy (delayed, photosensitivity), brachytherapy (delayed), mechanical debulking with rigid barrel/microdebrider.
- **Stents:** silicone (Dumon, Y-stents — removable, need rigid), self-expanding metal stents (SEMS — covered for malignancy; avoid uncovered in benign), hybrid; complications — migration, granulation, mucus plugging, infection.

Bronchoscopic palliation for lung cancer

- Relief of malignant CAO (dyspnoea, stridor), control of haemoptysis (APC, cold saline, adrenaline, tamponade), drainage of post-obstructive pneumonia, endobronchial brachytherapy, stenting for TEF (dual oesophageal + airway stents), photodynamic therapy; allows extubation in ventilated patients and improves eligibility for chemotherapy/RT.
- **Safety:** general anaesthesia/rigid bronchoscope, jet ventilation, low FiO2 for heat therapy, readiness for bleeding control.

★ CLINICAL PEARLS & EXAM FOCUS

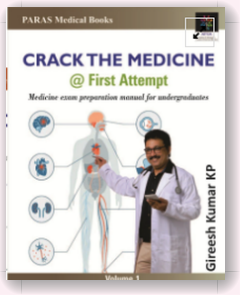
▸ Heat ablation needs FiO2 <40% — high oxygen in the airway turns the bronchoscope into a blowtorch.

LANDMARK TRIALS & GUIDELINES

- Ernst A et al, Central airway obstruction, AJRCCM 2004; Mahmood K et al, ACCP guideline on malignant CAO (2024).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Bronchoscopic management of central airway obstruction. *[DNB Jun 2021 P2]*
2. Write a detailed note on various bronchoscopic heat-ablative modalities. *[DNB May 2024 P2]*
3. Bronchoscopic palliation for lung cancer. *[BFUHS 2023 P2]*



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INVESTIGATIONS & TREATMENT AT A GLANCE

10.9 Interventional Bronchoscopy — Central Airway Obstruction, Heat Ablation & Palliation

‹ Back to topic (p 193)
Next topic 11.1 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
CT / bronchoscopy	Patent airway	> 50% luminal narrowing	Central airway obstruction (CAO)
Flow–volume loop	Normal	Fixed/variable obstruction	CAO physiology
SpO_2 / ABG	Normal	Hypoxaemia	Urgency
Lesion type	—	Intraluminal / extrinsic / mixed	Choice of technique

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Rigid bronchoscopy + debulking	Mechanical coring	Intraluminal tumour	Bleeding
Electrocautery / argon plasma coagulation / laser	APC 30–40 W	Endobronchial tumour, haemostasis	Airway fire (FiO_2 < 0.4)
Cryotherapy / cryorecanalisation	—	Tumour, foreign body, clots	—
Airway stent (silicone / SEMS)	—	Extrinsic compression, malacia	Migration, granulation, mucus
Heliox	70:30 or 80:20	Temporising in critical stenosis	Low FiO_2 limitation

★ PRESCRIBING & INTERPRETATION PEARLS

- Keep FiO_2 < 40% during laser/APC to avoid airway fire.

SECTION 11

Pleura, Mediastinum, Diaphragm & Chest Wall

Paper II

Pleural dynamics and effusions; non-neoplastic and neoplastic pleural disease; pneumothorax; pyothorax and bronchopleural fistula; fibrothorax; non-neoplastic and neoplastic disorders of the mediastinum; diseases of the diaphragm and chest wall (NMC syllabus II-B8, II-B9, II-B13).

11.1 Pleural Physiology & Approach to Pleural Effusion 196
11.2 Parapneumonic Effusion, Empyema & Bronchopleural Fistula 198
11.3 Malignant & Recurrent Pleural Effusions, Hepatic Hydrothorax & Indwelling Pleural Catheters 200
11.4 Malignant Pleural Mesothelioma 202
11.5 Chylothorax, Pseudochylothorax, Haemothorax, Fibrothorax & Trapped Lung 203
11.6 Pneumothorax — Primary, Secondary, Recurrent, Iatrogenic; Persistent Air Leak 204
11.7 Mediastinal Masses, Mediastinitis & Pneumomediastinum 206
11.8 Diseases of the Diaphragm — Paralysis, Eventration & Elevated Hemidiaphragm 207
11.9 Chest Wall Disorders — Kyphoscoliosis, Thoracoplasty, Flail Chest, Marfan Syndrome & Neuromuscular Restriction 209

11.1 Pleural Physiology & Approach to Pleural Effusion

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B9 Pleural dynamics and effusions
- ✓ Explain pleural fluid formation and absorption and mechanisms of effusion
- ✓ Differentiate transudates from exudates (Light's criteria and alternatives)
- ✓ Interpret pleural fluid analysis; manage effusions in the critically ill and at-bleeding-risk patient

IN BRIEF Pleural fluid is filtered from parietal systemic capillaries and drained by parietal lymphatic stomata. An effusion forms when formation exceeds absorption. The first step is to classify it as a transudate (systemic factors) or exudate (local pleural disease).

Mechanisms of pleural effusion

- ↑ Hydrostatic pressure (HF), ↓ oncotic pressure (hypoalbuminaemia), ↑ capillary permeability (infection, malignancy, inflammation), ↓ lymphatic drainage (malignancy, yellow nail), ↓ pleural pressure (trapped lung), passage from peritoneum (hepatic hydrothorax, peritoneal dialysis, Meigs).

Light's criteria — exudate if ≥1 present

Criterion	Exudate
Pleural fluid protein / serum protein	>0.5
Pleural fluid LDH / serum LDH	>0.6
Pleural fluid LDH	>2/3 upper limit of normal serum LDH

- Sensitivity ~98%, but misclassifies ~25% of transudates (diuretic-treated HF) as exudates → use **serum–pleural albumin gradient >1.2 g/dL** or **protein gradient >3.1 g/dL** = transudate; **pleural NT-proBNP >1500 pg/mL** = HF.
- Other exudate markers: pleural cholesterol >45–60 mg/dL.

Causes of pleural effusion

Transudates	Exudates
Heart failure (commonest overall)	Parapneumonic/empyema, tuberculosis (commonest exudate in India)
Cirrhosis (hepatic hydrothorax)	Malignancy (lung, breast, lymphoma, mesothelioma)
Nephrotic syndrome, hypoalbuminaemia	Pulmonary embolism (can be either)
Peritoneal dialysis, urinothorax	Pancreatitis, oesophageal rupture, subphrenic abscess
Hypothyroidism, constrictive pericarditis, SVC obstruction	RA, SLE, post-cardiac injury (Dressler), drugs, uraemia, asbestos, chylothorax, sarcoid, yellow nail syndrome, Meigs (benign ovarian tumour)

Pleural fluid analysis

Test	Interpretation
Appearance	Pus (empyema), milky (chylo/pseudochylothorax), bloody (Hct >50% of blood = haemothorax; malignancy, PE, trauma), anchovy paste (amoebic), black (aspergillus)
Cell count	Neutrophilic (parapneumonic, PE, pancreatitis, early TB); lymphocytic >50% (TB, malignancy, lymphoma, chylothorax, post-CABG, RA); eosinophilic >10% (air/blood in pleura, drugs, parasites, asbestos, EGPA)
Glucose <60 mg/dL	Empyema, RA (<30), TB, malignancy, lupus, oesophageal rupture
pH <7.2	Complicated parapneumonic effusion — drain; also oesophageal rupture (<6), RA, malignancy (poor prognosis)
ADA ≥40 U/L	TB (see 4.12)
Amylase	Pancreatitis, oesophageal rupture (salivary), malignancy
Triglycerides >110 mg/dL / chylomicrons	Chylothorax
Cytology	Malignancy (sensitivity ~60%; repeat once, then biopsy)
Creatinine (fluid/serum >1)	Urinothorax
β2-transferrin	Duropleural fistula

Uraemic pleuritis and Dressler's syndrome

- **Uraemic pleuritis:** fibrinous, often haemorrhagic exudate in ESRD (3%); fever, rub; dialysis intensification; fibrothorax may need decortication.
- **Dressler's (post-cardiac injury syndrome):** 1–6 weeks after MI/cardiac surgery/pacemaker; pericarditis + pleuritis + fever; left-sided exudate; NSAIDs/aspirin + colchicine, steroids if refractory.

Effusions in the critically ill and at-bleeding-risk patients

- Common (40–60% of ICU patients): HF/fluid overload, atelectasis, hypoalbuminaemia, parapneumonic, post-surgery, haemothorax.
- **Thoracentesis is safe with ultrasound guidance** even in mechanically ventilated patients and those with mild coagulopathy (INR <2–3, platelets >25–50 $\times 10^9$/L) or on antiplatelets — routine correction of coagulation is not needed (BTS 2023); hold anticoagulants (DOAC/warfarin) where possible for elective procedures; use small-bore drains.

- **Drainage benefit in ventilated patients:** improves oxygenation and may aid weaning in large effusions.

★ CLINICAL PEARLS & EXAM FOCUS

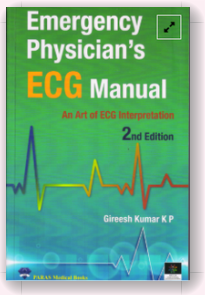
▸ Diuretic-treated HF effusion "fails" Light's criteria — check albumin gradient or NT-proBNP before calling it an exudate.

LANDMARK TRIALS & GUIDELINES

- Light RW et al, Ann Intern Med 1972; Roberts ME et al, BTS guideline for pleural disease, Thorax 2023.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Differentiating transudative from exudative pleural effusions. *[NEET-SS/DNB 2024]*
2. Pleural effusions in the critically ill and 'at-bleeding-risk' population. *[DNB Oct 2023 P4]*
3. Uraemic pleuritis. *[Manipal 2014 P2]*
4. Dressler's syndrome. *[Manipal 2014 P2]*



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11.2 Parapneumonic Effusion, Empyema & Bronchopleural Fistula

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B9 Pyothorax and bronchopleural fistula
- ✓ Classify parapneumonic effusions and decide on drainage
- ✓ Treat empyema with antibiotics, chest tube, intrapleural fibrinolytics and surgery

IN BRIEF About 40% of bacterial pneumonias have an effusion; 10% become complicated (fibrinopurulent) and some progress to empyema (pus). Early drainage of complicated effusions and intrapleural tPA–DNase reduce the need for surgery.

Stages of parapneumonic effusion

Stage	Features	Fluid	Management
Exudative (simple)	Free-flowing, sterile	pH >7.2, glucose >60, LDH <1000	Antibiotics alone
Fibrinopurulent (complicated)	Loculations, bacterial invasion	pH <7.2, glucose <60, LDH >1000, Gram stain/culture +	Chest tube drainage
Organising (empyema/ fibrothorax)	Peel, trapped lung	Pus	Drainage + fibrinolytics / VATS decortication

- **Microbiology:** community — S. milleri group (S. anginosus), S. pneumoniae, anaerobes, S. aureus; hospital — MRSA, gram-negatives; India — TB empyema, Klebsiella, melioidosis.

INVESTIGATIONS & TREATMENT AT A GLANCE

11.1 Pleural Physiology & Approach to Pleural Effusion

‹ Back to topic (p 196)
Next topic 11.2 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Light's criteria	Transudate: protein ratio ≤ 0.5; LDH ratio ≤ 0.6; LDH ≤ ⅔ ULN	Any criterion met → exudate	Classification
Serum–pleural albumin gradient	> 1.2 g/dL → transudate	≤ 1.2	Diuretic-treated heart failure
Pleural NT-proBNP	< 1500 pg/mL	> 1500 pg/mL	Cardiac effusion
Pleural ADA	< 40 U/L	> 40 U/L	TB (lymphocytic)
Pleural pH / glucose	pH 7.60–7.64; glucose = serum	pH < 7.2; glucose < 60 mg/dL	Complicated parapneumonic, RA, TB, malignancy
Pleural cytology	Negative	Malignant cells (sensitivity ~ 60%)	Malignancy
Pleural triglycerides	< 50 mg/dL	> 110 mg/dL	Chylothorax

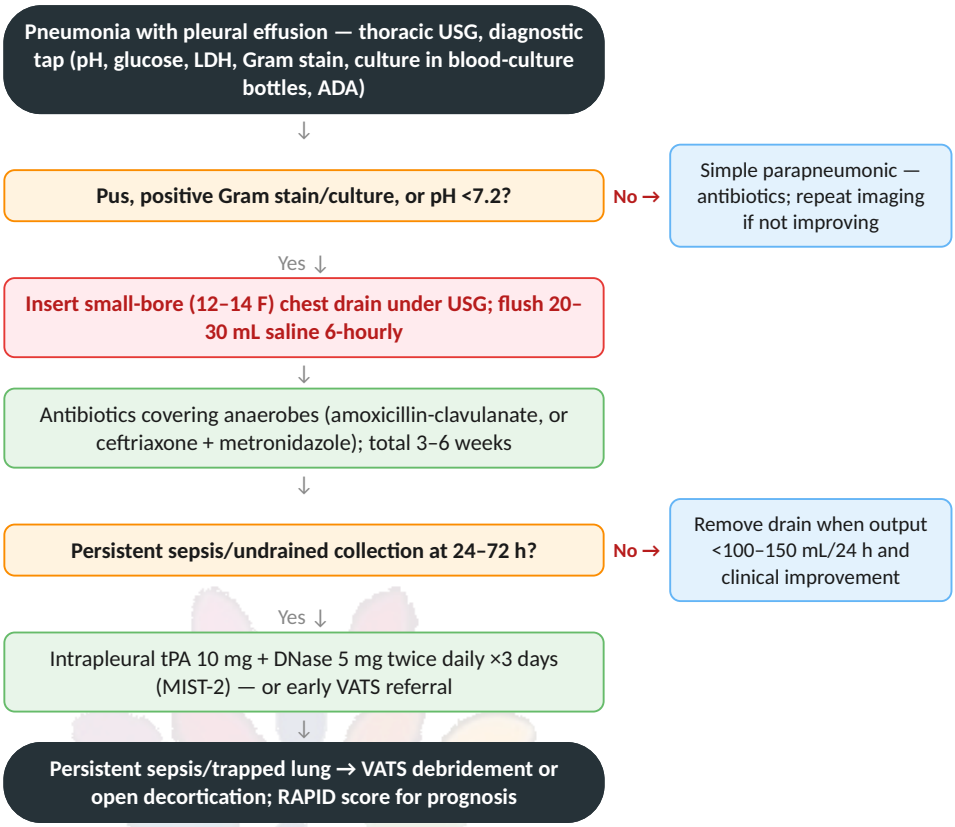
Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Diagnostic thoracentesis	50–60 mL	New unexplained effusion	USG-guided
Therapeutic thoracentesis	≤ 1.5 L at a time	Symptomatic large effusion	Re-expansion oedema
Furosemide	40 mg daily	Heart-failure effusion	—
Pleural biopsy (thoracoscopy)	—	Undiagnosed exudate	—

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Use ultrasound for every pleural procedure (BTS 2023).

Algorithm 11.1 · Management of pleural infection (BTS 2023)



Empyema — key points

- **RAPID score** (Renal urea, Age, Purulence, Infection source — hospital, Dietary albumin) predicts mortality.
- **MIST-2:** tPA + DNase combination improved radiographic drainage, reduced surgical referral and hospital stay vs either alone or placebo; streptokinase alone ineffective (MIST-1).
- Empyema necessitans (TB, actinomyces), TB empyema (chronic, calcified — ATT + drainage ± decortication).

Bronchopleural fistula

- Communication between bronchus and pleural space: post-pneumonectomy/lobectomy (1–4%; right pneumonectomy, residual tumour, radiation, diabetes), necrotising pneumonia/abscess, TB, empyema, persistent air leak after pneumothorax, ARDS on MV.
- **Features:** persistent air leak, expectoration of pleural fluid (lying on healthy side), falling air–fluid level after pneumonectomy, empyema, tension pneumothorax.
- **Diagnosis:** CT (bronchial communication), bronchoscopy (methylene blue, balloon occlusion to localise), ventilation scintigraphy.
- **Management:** drain the pleural space, antibiotics, nutrition; bronchoscopic closure (fibrin glue, cyanoacrylate, endobronchial valves, Amplatzer occluders, stents); surgical repair with muscle/omental flap, open window thoracostomy (Eloesser flap, Clagett procedure); on MV — lower VT, PEEP, independent lung ventilation.

★ CLINICAL PEARLS & EXAM FOCUS

▸ pH <7.2 = drain; tPA + DNase together (never either alone).

LANDMARK TRIALS & GUIDELINES

- Rahman NM et al, MIST-2 NEJM 2011; Roberts ME et al, BTS 2023 pleural guideline.

INVESTIGATIONS & TREATMENT AT A GLANCE

11.2 Parapneumonic Effusion, Empyema & Bronchopleural Fistula

‹ Back to topic (p 198)
Next topic 11.3 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Pleural pH	> 7.2	< 7.2	Complicated — drain
Pleural glucose	> 60 mg/dL	< 40–60 mg/dL	Complicated
Pleural LDH	< 1000 U/L	> 1000 U/L	Complicated
Pleural Gram stain / culture	Negative	Positive / frank pus	Empyema — drain
USG	Anechoic	Septations, loculations	Fibrinolytics / VATS
RAPID score	0–2 low	5–7 high	Mortality

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Co-amoxiclav / ceftriaxone + metronidazole	1.2 g IV 8-hourly / 2 g + 500 mg	Pleural infection	Total 3–6 weeks
Intercostal drain	12–14 F Seldinger	Complicated effusion/empyema	Flush 20 mL saline 6-hourly
tPA + DNase (MIST-2)	tPA 10 mg + DNase 5 mg bd × 3 days	Loculated pleural infection	Bleeding
VATS decortication	—	Failed medical therapy	—
BPF management	Drain, endobronchial valves, surgical closure	Bronchopleural fistula	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Do not use streptokinase alone (MIST-1 — no benefit).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Bacterial empyema. *[DNB Dec 2021 P2]*

11.3 Malignant & Recurrent Pleural Effusions, Hepatic Hydrothorax & Indwelling Pleural Catheters

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

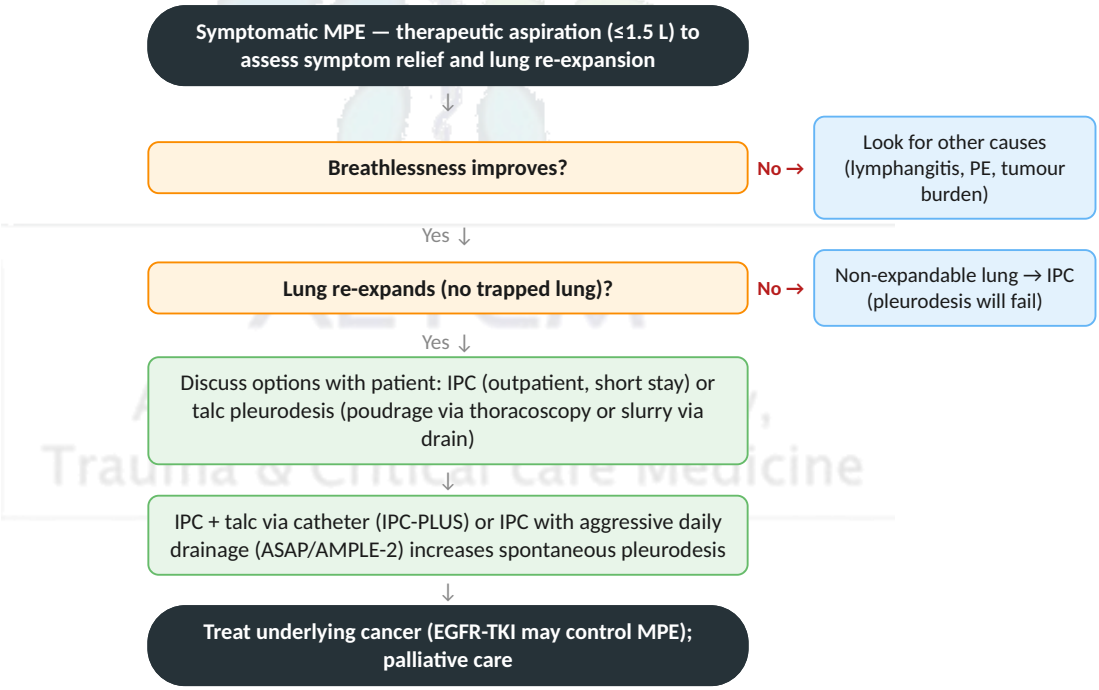
- ✓ **NMC syllabus area:** II-B9 Neoplastic pleural diseases
- ✓ Diagnose malignant pleural effusion and choose definitive management (pleurodesis vs IPC)
- ✓ Enumerate causes and management of recurrent non-malignant effusions including hepatic hydrothorax
- ✓ Describe portable/ambulatory devices for pleural effusion

IN BRIEF Malignant pleural effusion (MPE) indicates stage IV disease (median survival 3–12 months). The aim is symptom relief with minimal hospitalisation — talc pleurodesis or an indwelling pleural catheter (IPC), chosen by lung re-expansion, patient preference and prognosis (LENT score).

Malignant pleural effusion

- Causes: lung (commonest), breast, lymphoma, ovary, GI, mesothelioma; unknown primary.
- **Diagnosis:** cytology (60%; two samples ~70%), image-guided or thoracoscopic biopsy (>90%) — also for molecular profiling.
- **LENT score** (LDH, ECOG, NLR, Tumour type) predicts survival.

Algorithm 11.2 · Management of symptomatic malignant pleural effusion



Pleurodesis vs indwelling pleural catheter

Feature	Talc pleurodesis	Indwelling pleural catheter
Hospital stay	4–6 days (slurry)	Day case
Success	70–80% (TIME1; poudrage = slurry — TAPPS)	Symptom control ~90%; auto-pleurodesis 30–50%
Trapped lung	Ineffective	Effective

Feature	Talc pleurodesis	Indwelling pleural catheter
Complications	Pain, fever, ARDS (small-particle talc — use graded talc)	Infection (5%), blockage, tract metastasis, catheter care burden
Evidence	TIME2, AMPLE: IPC fewer re-interventions and hospital days	—

Recurrent non-malignant pleural effusion

- **Causes:** refractory HF, hepatic hydrothorax, nephrotic syndrome, ESRD/peritoneal dialysis, chylothorax, yellow nail syndrome, lupus/RA, post-CABG, benign asbestos effusion, trapped lung, Meigs, pancreatic pseudocyst fistula, TB (rare), urinothorax.
- **Management:** optimise the underlying condition; therapeutic thoracentesis; IPC (increasingly used in refractory HF and hepatic hydrothorax — REDUCE trial); pleurodesis (less effective in transudates); shunts.

Hepatic hydrothorax

- Transudate (>500 mL) in cirrhosis without cardiopulmonary cause, usually **right-sided (85%)** — ascitic fluid passes through **diaphragmatic defects** under negative intrathoracic pressure; may occur without clinical ascites.
- **Diagnosis:** diagnostic tap (transudate, SAAG-like; exclude SBEP — PMN >250/µL with positive culture or >500 cells), radionuclide scintigraphy (Tc-99m albumin in peritoneum → pleura).
- **Management:** sodium restriction (2 g/day), diuretics (spironolactone + furosemide), therapeutic thoracentesis for symptoms; **TIPS** for refractory (response 70–80%); **liver transplantation** definitive; VATS repair of diaphragmatic defect + pleurodesis; IPC in selected (infection risk). **Avoid chest tubes** (massive fluid/protein loss, infection, renal failure).
- **Spontaneous bacterial empyema** — antibiotics (cefotaxime), no chest drain unless frank pus.

Portable devices for pleural effusion management

- **Indwelling pleural catheters** (PleurX, Rocket) — tunnelled silicone catheters with one-way valve; drained at home into vacuum bottles 3×/week or daily.
- **Pleuroperitoneal shunts** (Denver) — manual pump; trapped lung/chylothorax.
- **Ambulatory small-bore drains with flutter valves/Heimlich valves**, digital drainage systems (air leak quantification), portable thoracic suction units; handheld USG for guidance.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Trapped lung → IPC, not talc. Hepatic hydrothorax → never a chest tube; think TIPS/transplant.

LANDMARK TRIALS & GUIDELINES

- Davies HE et al, TIME2 JAMA 2012; Thomas R et al, AMPLE JAMA 2017; Bhatnagar R et al, IPC-PLUS NEJM 2018; Bhatnagar R, TAPPS JAMA 2020.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Enumerate various causes of recurrent non-malignant pleural effusion. *[DNB May 2024 P2]*
2. Describe portable devices useful for management of pleural effusion. *[DNB May 2024 P2]*
3. Management options in malignant pleural effusion. *[BFUHS 2022 P2]*
4. Hepatic hydrothorax. *[BFUHS 2019 P2]*
5. Enumerate causes of ascites; aetiology, clinical features and management of hepatic hydrothorax. *[VNSGU 2025 P2]*

INVESTIGATIONS & TREATMENT AT A GLANCE

11.3 Malignant & Recurrent Pleural Effusions, Hepatic Hydrothorax & Indwelling Pleural Catheters

‹ Back to topic (p 200)
Next topic 11.4 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Pleural cytology	Negative	Positive	MPE
Thoracoscopic biopsy	—	Malignant histology (> 90%)	Diagnosis
LENT score	0–1 low	5–7 high (median survival ~ 1 month)	Prognosis
Lung re-expansion after drainage	Full	Trapped lung (elastance > 14.5 cmH_2O/L)	IPC rather than pleurodesis
SAAG (hepatic hydrothorax)	—	Transudate, SAAG > 1.1 in ascites	Hepatic hydrothorax

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Talc pleurodesis	4–5 g slurry or poudrage	MPE with expandable lung	Fever, ARDS (non-graded talc)
Indwelling pleural catheter	Drain 3 times weekly (or daily for pleurodesis, ASAP)	Trapped lung / patient preference	Infection, tract metastasis
Diuretics + TIPS	Spironolactone 100 mg + furosemide 40 mg; TIPS	Hepatic hydrothorax	Avoid chest drain (protein loss)
Systemic anticancer therapy	—	Chemosensitive tumours (SCLC, lymphoma, breast)	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Avoid ICD in hepatic hydrothorax — massive fluid and protein loss.

11.4 Malignant Pleural Mesothelioma

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B9 Neoplastic pleural diseases; I-D7
- ✓ Describe aetiology, pathology and diagnosis of pleural mesothelioma
- ✓ Describe treatment options including immunotherapy

IN BRIEF Pleural mesothelioma is an aggressive tumour of mesothelial cells, causally linked to asbestos (latency 30–50 years). It presents with chest pain and unilateral effusion with nodular pleural thickening; median survival is 12–18 months, improved by first-line nivolumab–ipilimumab.

Aetiology and pathology

- Asbestos (amphiboles >> chrysotile), erionite (Turkey), radiation, germline **BAP1** mutation (BAP1 tumour predisposition syndrome); SV40 controversial.
- **Histology:** epithelioid (60–70%, best prognosis), sarcomatoid (worst), biphasic; IHC — calretinin, WT-1, D2-40, CK5/6 positive; **BAP1 loss**, **MTAP loss/CDKN2A deletion (FISH)** distinguish from reactive mesothelial hyperplasia.

Clinical and diagnostic features

- Chest pain (dull, chest wall), dyspnoea, weight loss, sweats; recurrent exudative effusion, chest wall invasion along biopsy tracts.
- **CT:** circumferential nodular pleural thickening >1 cm, mediastinal pleural involvement, volume loss ("frozen hemithorax"); PET-CT, MRI (chest wall/diaphragm invasion).
- **Diagnosis:** thoracoscopic (or image-guided) pleural biopsy — cytology alone insufficient (sensitivity <30%); serum mesothelin, fibulin-3 (limited).
- Staging: IASLC TNM 8th/9th edition.

Treatment of pleural mesothelioma

- **First-line systemic: nivolumab + ipilimumab** (CheckMate 743 — OS 18.1 vs 14.1 months; greatest benefit in non-epithelioid); platinum + pemetrexed ± bevacizumab (MAPS) or + pembrolizumab (KEYNOTE-483 — 2024); second-line: rechallenge, vinorelbine, gemcitabine.
- **Surgery:** controversial — MARS-2 (2024) showed extended pleurectomy/decortication increased harm without survival benefit; EPP abandoned; surgery limited to trials/selected epithelioid.
- **Radiotherapy:** palliative (chest wall pain); prophylactic tract irradiation not routine (SMART, PIT trials).
- **Palliation:** IPC/talc for effusion, analgesia (opioids, cordotomy), multidisciplinary palliative care; **compensation** for occupational exposure.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Unilateral effusion + nodular pleural thickening involving mediastinal pleura in an ex-asbestos worker = mesothelioma until proven otherwise — get a thoracoscopic biopsy.

LANDMARK TRIALS & GUIDELINES

- Baas P et al, CheckMate 743 Lancet 2021; Lim E et al, MARS-2 Lancet Respir Med 2024; Popat S et al, ESMO mesothelioma guideline 2022.

INVESTIGATIONS & TREATMENT AT A GLANCE

11.4 Malignant Pleural Mesothelioma

‹ Back to topic (p 202)
Next topic 11.5 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Pleural biopsy (IHC: calretinin, WT1, D2-40; BAP1 loss)	—	Mesothelial origin, invasion	Mesothelioma
CT / PET	—	Nodular pleural thickening > 1 cm, mediastinal pleural involvement	Malignant thickening
Serum mesothelin	Low	Raised	Supportive (low sensitivity)
Asbestos exposure	—	Positive (latency 30–40 y)	Risk
Histological subtype	—	Epithelioid (best), biphasic, sarcomatoid (worst)	Prognosis

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Nivolumab + ipilimumab	Nivo 360 mg 3-weekly + ipi 1 mg/kg 6-weekly	First-line (non-epithelioid especially)	irAEs
Cisplatin + pemetrexed (± bevacizumab)	Cisplatin 75 mg/m² + pemetrexed 500 mg/m² 3-weekly	Epithelioid	Vitamin supplementation
IPC / talc pleurodesis	—	Effusion control	—
Pleurectomy/decortication	Surgical	Selected	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Biopsy tracts can seed tumour — prophylactic radiotherapy is no longer routine.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Treatment of pleural mesothelioma. *[DNB Oct 2024 P2]*
2. Mesothelioma. *[Manipal 2014 P4]*

11.5 Chylothorax, Pseudochylothorax, Haemothorax, Fibrothorax & Trapped Lung

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B9 Non-neoplastic pleural diseases; fibrothorax
- ✓ Differentiate chylothorax and pseudochylothorax and list causes of chylothorax
- ✓ Manage haemothorax and its complications
- ✓ Distinguish trapped lung from lung entrapment; manage fibrothorax

IN BRIEF Milky pleural fluid is either chyle (disrupted thoracic duct — triglycerides, chylomicrons) or pseudochyle (cholesterol crystals in long-standing effusions). Haemothorax needs drainage to prevent clotting, empyema and fibrothorax.

Chylothorax vs pseudochylothorax

Feature	Chylothorax	Pseudochylothorax (cholesterol effusion)
Onset	Acute/subacute	Chronic (years), thick pleura
Triglycerides	>110 mg/dL	Usually <50 mg/dL
Cholesterol	<200 mg/dL	>200 mg/dL
Chylomicrons (lipoprotein electrophoresis)	Present	Absent
Cholesterol crystals (rhomboid, polarised)	Absent	Present
Causes	See below	Chronic TB effusion, RA, therapeutic pneumothorax, chronic empyema

Causes of chylothorax

- **Traumatic:** surgery (oesophagectomy, cardiac, lung resection, neck dissection), central line, trauma.
- **Non-traumatic:** malignancy (lymphoma — commonest, metastatic), LAM, TB, sarcoidosis, SVC/subclavian thrombosis, congenital lymphatic malformations (Gorham–Stout, Noonan), yellow nail syndrome, cirrhosis/chylous ascites (transdiaphragmatic), filariasis, idiopathic.
- **Management:** treat cause, drain, **low-fat diet with MCT** or **NPO with TPN**, octreotide; thoracic duct embolisation (interventional radiology) or surgical ligation if output >1 L/day for 5–7 days; pleurodesis/IPC; sirolimus for LAM; radiotherapy for lymphoma.

Haemothorax

- Pleural Hct ≥50% of blood Hct. Causes: trauma (commonest), iatrogenic (central lines, thoracentesis, biopsy), anticoagulation, malignancy, vascular rupture (aorta, PAVM), endometriosis (catamenial), spontaneous haemopneumothorax (adhesion tear).
- **Management:** large-bore chest drain (28–32 F) in trauma; **thoracotomy** if initial drainage >1500 mL or >200 mL/h for 2–4 h, haemodynamic instability; retained haemothorax → early VATS (within 3–7 days) or intrapleural fibrinolytics.
- **Complications:** retained clotted haemothorax, empyema (5–30%), **fibrothorax/trapped lung**, re-expansion oedema, hypovolaemic shock, coagulopathy, catamenial recurrence.

Trapped lung and fibrothorax

- **Lung entrapment** — lung cannot expand due to active pleural process (malignancy, infection) — reversible with treatment. **Trapped lung** — mature fibrous visceral peel from prior inflammation (empyema, haemothorax, TB, CABG, uraemia) — fixed, non-expandable lung with chronic transudate-like effusion.

- **Diagnosis:** pleural manometry (elastance >14.5 cmH2O/L, negative opening pressure), ex-vacuo pneumothorax after drainage, failure to re-expand, CT peel; chest discomfort on drainage.
- **Fibrothorax:** dense fibrous pleural thickening restricting the hemithorax (restrictive PFT); decortication if symptomatic with underlying lung healthy and disease >6 months stable (TB must be inactive).

★ CLINICAL PEARLS & EXAM FOCUS

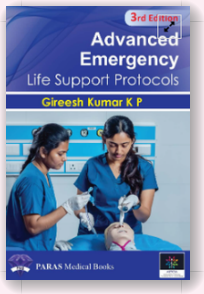
▸ Milky fluid — measure triglycerides and cholesterol: TG >110 = chyle; cholesterol >200 with crystals = pseudochyle.

LANDMARK TRIALS & GUIDELINES

- Agrawal V et al, Chylothorax review, Chest 2008; Huggins JT et al, Trapped lung review, Clin Chest Med 2013.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Difference between chylothorax and pseudochylothorax and causes of chylothorax. *[DNB Oct 2024 P2]*
2. Complications of hemothorax. *[DNB Jun 2025 P2]*
3. Hemothorax. *[Manipal 2014 P4]*



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11.6 Pneumothorax — Primary, Secondary, Recurrent, Iatrogenic; Persistent Air Leak

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B9 Pneumothorax
- ✓ Classify pneumothorax and describe pleural gas reabsorption
- ✓ Manage pneumothorax per BTS 2023 (including conservative and ambulatory approaches)
- ✓ Manage recurrent and iatrogenic pneumothorax and persistent air leak

IN BRIEF Pneumothorax is air in the pleural space — primary (no clinical lung disease; tall young smokers, subpleural blebs) or secondary (underlying lung disease — COPD, TB, PJP, cystic lung disease). The BTS 2023 guideline focuses on symptoms and patient priorities rather than size alone, endorsing conservative and ambulatory management.

Reabsorption of pleural gas

- Air is absorbed at ~1.25–2.2% of hemithorax volume per day because the sum of partial pressures in capillary blood (venous ~706 mmHg) is lower than in the pleural space (~760) — nitrogen diffuses out.
- **Supplemental high-flow oxygen** (FiO2 ~1) washes out nitrogen, widening the gradient and increasing reabsorption 3–4×; use in hospitalised patients (caution in hypercapnic COPD).

INVESTIGATIONS & TREATMENT AT A GLANCE

11.5 Chylothorax, Pseudochylothorax, Haemothorax, Fibrothorax & Trapped Lung

‹ Back to topic (p 203)
Next topic 11.6 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Pleural triglycerides	< 50 mg/dL	> 110 mg/dL; chylomicrons	Chylothorax
Pleural cholesterol	< 200 mg/dL	> 200 mg/dL, cholesterol crystals	Pseudochylothorax
Pleural:blood haematocrit	< 0.01	> 0.5	Haemothorax
Lymphangiography / MR lymphangiography	—	Leak site	Chylothorax
CT	—	Thick pleura, trapped lung	Fibrothorax

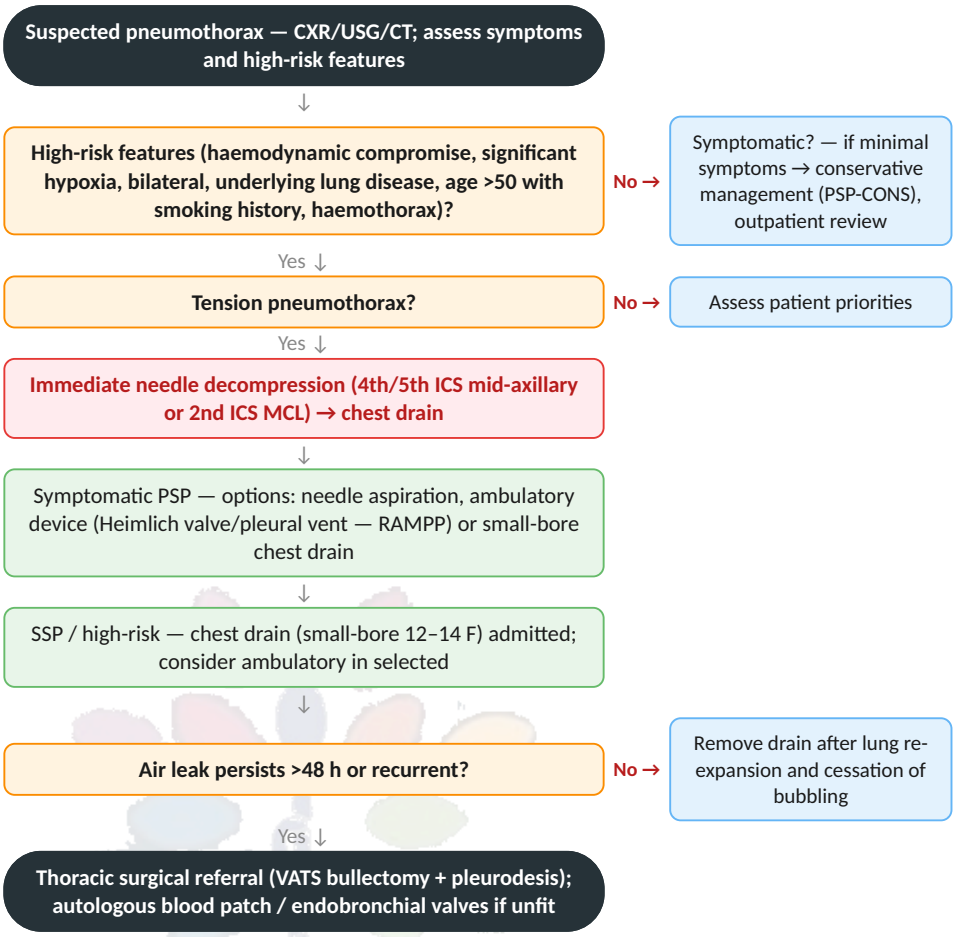
Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Low-fat MCT diet / TPN	—	Chylothorax	Malnutrition
Octreotide	100 µg SC 8-hourly	Chylothorax	—
Thoracic duct ligation / embolisation	—	Output > 1 L/day for > 5 days	—
Large-bore ICD	28–32 F	Haemothorax	VATS if > 1500 mL initial or > 200 mL/h × 2–4 h
Decortication	—	Fibrothorax/trapped lung with symptoms	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Pseudochylothorax is usually old TB or rheumatoid effusion — look for cholesterol crystals.

Algorithm 11.3 · Management of spontaneous pneumothorax (BTS 2023)



- **Conservative management:** PSP-CONS showed non-inferior re-expansion at 8 weeks with fewer complications and recurrences in selected patients with moderate–large PSP.
- **Advice:** smoking cessation (recurrence ↑), no flying until resolution (BTS: 1 week after radiological resolution), avoid diving permanently unless bilateral surgical pleurectomy.

Recurrent pneumothorax

- Recurrence: PSP ~30% (mostly within 1 year), SSP ~40–50%; risk — smoking, tall stature, women (catamenial), COPD, CF, LAM.
- **Definitive intervention indications:** second ipsilateral, first contralateral, bilateral simultaneous, persistent air leak (>3–5 days), spontaneous haemothorax, high-risk professions (pilots, divers), pregnancy (planned), SSP first episode (consider).
- **Procedures:** VATS bullectomy + mechanical pleurodesis (abrasion) or pleurectomy (recurrence <5%); medical thoracoscopic talc poudrage; chemical pleurodesis via drain (talc slurry, tetracycline) for non-surgical candidates; autologous blood patch.

Iatrogenic pneumothorax

- **Causes:** TTNA (commonest, 15–25%), central venous cannulation (subclavian), thoracentesis, TBLB/cryobiopsy, pleural biopsy, mechanical ventilation (barotrauma), CPR, pacemaker insertion, acupuncture, liver biopsy, tracheostomy.
- **Management:** small, asymptomatic — observation with oxygen; symptomatic — aspiration (often sufficient — no recurrence risk); drain if ventilated (positive pressure enlarges it) or large/persistent; prevention — USG guidance, operator experience.

Persistent air leak (PAL)

- Air leak >5 days (post-operative or spontaneous/secondary); grade by Cerfolio (forced expiratory, expiratory, continuous) or digital drainage.

- **Management:** water seal vs suction (no consistent difference), Heimlich valve/ambulatory drainage, **autologous blood patch pleurodesis** (50–100 mL patient's blood via drain, 70–90% success), chemical pleurodesis, **endobronchial valves** (localise with balloon occlusion), surgical repair (VATS staple/sealant, pleurectomy).

★ CLINICAL PEARLS & EXAM FOCUS

▸ In hospital, give high-flow oxygen to a pneumothorax patient — it speeds reabsorption 4-fold.

LANDMARK TRIALS & GUIDELINES

- Roberts ME et al, BTS guideline 2023; Brown SGA et al, PSP-CONS NEJM 2020; Hallifax RJ et al, RAMPP Lancet 2020.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Re-absorption of pleural gases in a pneumothorax. *[DNB Dec 2022 P1]*
2. Recurrent pneumothorax. *[DNB Oct 2023 P2]*
3. Management of recurrent pneumothorax. *[DNB Jun 2021 P2]*
4. Treatment of pneumothorax. *[DNB Dec 2021 P2]*
5. Etiology and management of iatrogenic pneumothorax. *[DNB Oct 2024 P2]*
6. Management of persistent air leak. *[NEET-SS/DNB 2024]*
7. Bronchopleural fistula. *[Manipal 2014 P4]*

11.7 Mediastinal Masses, Mediastinitis & Pneumomediastinum

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B8 Diseases of the mediastinum
- ✓ Approach a mediastinal mass by compartment and age
- ✓ Assess and manage thymoma and mediastinal germ cell tumours
- ✓ Describe fibrosing mediastinitis and causes of pneumomediastinum

IN BRIEF Mediastinal masses are classified by ITMIG compartment (see 1.5). Thymoma, lymphoma, germ cell tumours and thyroid goitre dominate the prevascular compartment; neurogenic tumours the paravertebral. Fibrosing mediastinitis — a sequel of granulomatous infection — compresses vital mediastinal structures.

Thymoma — clinical assessment and management

- Commonest anterior mediastinal tumour in adults (40–60 years); 30–50% have **myasthenia gravis**; other paraneoplastic syndromes — pure red cell aplasia, hypogammaglobulinaemia (Good syndrome), SLE, autoimmune encephalitis.
- **Evaluation:** CECT/MRI (encapsulated vs invasive, chemical-shift MRI to exclude thymic hyperplasia), anti-AChR antibodies, CBC, immunoglobulins; avoid needle biopsy of resectable encapsulated mass (seeding) — direct resection; biopsy if unresectable/uncertain (lymphoma).
- **WHO histology:** A, AB, B1, B2, B3, thymic carcinoma. **Masaoka–Koga staging:** I (encapsulated) to IV (pleural/pericardial dissemination, metastases); TNM 9th now used.
- **Treatment:** complete surgical resection (thymectomy — sternotomy/VATS/robotic) is the key prognostic factor; post-operative RT for incomplete resection/stage III; chemotherapy (cisplatin–doxorubicin–cyclophosphamide) for unresectable/neoadjuvant; myasthenic patients — optimise before surgery (pyridostigmine, IVIG/plasmapheresis); long-term follow-up (late recurrence).

Mediastinal germ cell tumours

Type	Markers	Treatment
Mature teratoma (benign)	Normal	Surgical excision (fat, calcification, teeth on CT)
Seminoma	β-hCG mildly ↑ (≤10%), AFP normal	Chemotherapy (BEP/EP) or RT — highly curable

INVESTIGATIONS & TREATMENT AT A GLANCE

11.6 Pneumothorax — Primary, Secondary, Recurrent, Iatrogenic; Persistent Air Leak

‹ Back to topic (p 204)
Next topic 11.7 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
CXR (inspiratory)	No visible rim	Apical rim; size ≥ 2 cm at hilum (BTS)	Large pneumothorax
Lung ultrasound	Lung sliding, B-lines	Absent sliding, lung point	Pneumothorax (lung point specific)
CT chest	—	Blebs/bullae; underlying disease	Recurrence risk, secondary cause
ABG	Normal	Hypoxaemia	Secondary pneumothorax severity

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Conservative / aspiration	16–18 G cannula, max 2.5 L	Primary, minimally symptomatic	Follow-up CXR
Ambulatory device / small-bore ICD	8–14 F	Symptomatic primary; secondary	Air leak monitoring
Supplemental O_2 (high flow)	10–15 L/min	Hospitalised (accelerates reabsorption)	Caution in CO_2 retainers
VATS bullectomy + pleurodesis	—	Recurrence, persistent air leak > 5 days, occupational (pilots/ divers)	—
Autologous blood patch	50–100 mL via ICD	Persistent air leak unfit for surgery	Infection

★ PRESCRIBING & INTERPRETATION PEARLS

- Tension pneumothorax: immediate needle decompression (4th–5th ICS mid-axillary line) then ICD.

Type	Markers	Treatment
Non-seminomatous (embryonal, yolk sac, choriocarcinoma, mixed)	AFP ↑ and/or β-hCG ↑, LDH ↑	BEP chemotherapy → resection of residual mass; poorer prognosis; Klinefelter association, haematological malignancies

- Young men with anterior mediastinal mass — always check AFP, β-hCG, LDH and testicular ultrasound.

Fibrosing mediastinitis

- Excessive fibrosis of mediastinal soft tissue: **granulomatous** (histoplasmosis — USA, **TB** — India), idiopathic, IgG4-related disease, radiation, Behçet, drugs (methysergide).
- **Clinical:** compression of SVC (SVC syndrome), pulmonary arteries/veins (PH, haemoptysis, mimics PE/MS), airways (cough, wheeze, recurrent pneumonia), oesophagus (dysphagia).
- **Evaluation:** CT (focal calcified mass — granulomatous; diffuse infiltrative — IgG4/idiopathic), CTPA, V/Q, bronchoscopy, IgG4 levels, histoplasma/TB tests, biopsy.
- **Management:** no proven medical therapy for granulomatous form (antifungals/steroids ineffective); ATT if active TB; steroids/rituximab for IgG4/idiopathic; **endovascular stenting** of SVC/PA/PV, airway stents; surgery high-risk.

Pneumomediastinum

- **Spontaneous (primary):** young men; asthma attacks, vomiting (Boerhaave must be excluded), Valsalva, cocaine/cannabis inhalation, childbirth, intense exercise; Macklin effect — alveolar rupture → air tracks along bronchovascular sheaths to mediastinum.
- **Secondary:** trauma, oesophageal rupture (Boerhaave, endoscopy), tracheobronchial injury, mechanical ventilation (barotrauma), COVID-19, ILD (dermatomyositis — anti-MDA5), dental extraction, retroperitoneal air.
- **Clinical:** retrosternal pain, dyspnoea, subcutaneous emphysema, **Hamman's sign** (mediastinal crunch), dysphonia; CXR — continuous diaphragm sign, ring around artery, thymic sail sign; CT.
- **Management:** spontaneous — benign, rest, analgesia, O2, avoid Valsalva; evaluate for oesophageal rupture (contrast swallow) if vomiting, fever, effusion; secondary — treat cause; malignant pneumomediastinum (tension) → decompression.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Young man + anterior mediastinal mass → AFP, β-hCG, LDH and testicular USG before biopsy.

LANDMARK TRIALS & GUIDELINES

- Carter BW et al, ITMIG classification (Radiographics 2017); Girard N et al, ESMO thymic epithelial tumours guideline 2015; Rossi SE et al, fibrosing mediastinitis (Radiographics 2001).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Write a note on etiology, clinical features, evaluation and management of fibrosing mediastinitis. *[DNB May 2024 P2]*
2. Spontaneous pneumomediastinum. *[DNB Dec 2021 P2]*
3. Etiology of pneumomediastinum. *[NEET-SS/DNB 2024]*
4. Clinical assessment and management of thymoma. *[DNB Dec 2021 P2]*
5. Germ cell tumours of mediastinum. *[DNB Jun 2021 P2]*

11.8 Diseases of the Diaphragm — Paralysis, Eventration & Elevated Hemidiaphragm

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B13 Diseases of the diaphragm
- ✓ List causes of unilateral elevation of the hemidiaphragm
- ✓ Differentiate eventration from paralysis and manage both

INVESTIGATIONS & TREATMENT AT A GLANCE

11.7 Mediastinal Masses, Mediastinitis & Pneumomediastinum

‹ Back to topic (p 206)
Next topic 11.8 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
CT (ITMIG compartment)	—	Prevascular / visceral / paravertebral	Differential
AFP / β-hCG / LDH	AFP < 10; β-hCG < 5	Raised	Germ cell tumour
Anti-AChR antibodies	Negative	Positive	Thymoma
CT in mediastinitis	—	Fat stranding, fluid, gas, abscess	Acute mediastinitis
Contrast swallow	Intact	Leak	Oesophageal perforation (Boerhaave)

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Thymectomy	Surgical	Thymoma	Myasthenic crisis
Antibiotics + drainage	Pip-taz 4.5 g + fluconazole; surgical drainage	Descending necrotising mediastinitis	High mortality
Pneumomediastinum (spontaneous)	Rest, analgesia, observation	Benign	Exclude perforation
Chemotherapy (BEP)	Oncology	Germ cell tumour	Bleomycin lung toxicity

★ PRESCRIBING & INTERPRETATION PEARLS

- Fibrosing mediastinitis in India is most often TB/histoplasmosis — can cause SVC and pulmonary vein obstruction.

IN BRIEF An elevated hemidiaphragm may be due to diaphragmatic weakness/paralysis or eventration, loss of lung volume above, a subphrenic process below, or a pseudo-elevation (subpulmonic effusion). Fluoroscopic or ultrasound sniff test distinguishes paralysis.

Causes of unilateral elevation of the hemidiaphragm

Category	Causes
Diaphragmatic	Phrenic nerve palsy (tumour — lung cancer, thymoma; cardiac surgery; neck surgery/trauma; neuralgic amyotrophy; idiopathic; herpes zoster), eventration, hernia, rupture (trauma)
Thoracic (above)	Lobar collapse/atelectasis, lobectomy/pneumonectomy, pulmonary hypoplasia, fibrosis (upper lobe pulls up hilum)
Abdominal (below)	Subphrenic abscess, hepatomegaly/liver mass, splenomegaly, gaseous distension (Chilaiditi), ascites (usually bilateral), interposition
Pseudo-elevation	Subpulmonic pleural effusion, diaphragmatic tumour, pleural mass
Other	Scoliosis, pain splinting (rib fracture, pleurisy, abdominal surgery)

Diaphragmatic eventration

- Congenital thinning of diaphragmatic muscle (replaced by fibroelastic tissue) — permanent elevation, usually left, partial (anteromedial right) or complete; acquired eventration from long-standing phrenic palsy (atrophy).
- **Clinical:** often asymptomatic adult; dyspnoea, orthopnoea, GI symptoms (left — gastric volvulus), recurrent infections in infants.
- **Sniff test:** eventration — reduced but **normal-direction** movement (or none); paralysis — **paradoxical upward** movement.
- **Management:** observe if asymptomatic; **diaphragmatic plication** (VATS/open) for symptomatic — improves dyspnoea, FVC, and abdominal symptoms.

Diaphragmatic paralysis — evaluation and management

- **Tests:** fluoroscopic/USG sniff test, diaphragm USG (excursion <1 cm, thickening fraction <20%), supine vs upright VC (fall >25% unilateral weakness, >50% bilateral), MIP/SNIP, Pdi, phrenic nerve conduction/EMG, CT chest/neck (nerve compression), MRI C-spine.
- **Management:** treat cause; unilateral — usually conservative (recovery in post-surgical/neuralgic over 1–2 years); plication if symptomatic; bilateral — nocturnal NIV, phrenic nerve pacing (high cervical cord injury with intact phrenic nerves), diaphragm pacing; weight loss; OSA screening.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Paradoxical upward movement on sniffing = paralysis; reduced but normal-direction movement = eventration.

LANDMARK TRIALS & GUIDELINES

- McCool FD, Tzelepis GE. Dysfunction of the diaphragm, NEJM 2012.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Diaphragmatic eventration. *[DNB Oct 2023 P1]*
2. Causes of unilateral elevation of the hemidiaphragm. *[DNB Jun 2025 P2]*

INVESTIGATIONS & TREATMENT AT A GLANCE

11.8 Diseases of the Diaphragm — Paralysis, Eventration & Elevated Hemidiaphragm

‹ Back to topic (p 207)
Next topic 11.9 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Sniff test	Downward movement	Paradoxical upward	Unilateral paralysis
Ultrasound excursion / thickening fraction	Excursion > 1 cm; TF > 20%	< 1 cm; TF < 20%	Weakness
Supine vs upright VC	Fall < 10%	Fall > 25–30%	Bilateral weakness
Phrenic conduction / EMG	Normal	Abnormal	Neuropathy
CT chest/neck	—	Mass along phrenic course	Cause (tumour)

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
NIV	Nocturnal BiPAP	Bilateral paralysis with hypercapnia	—
Plication	VATS	Symptomatic unilateral paralysis/eventration	—
Treat cause	—	Tumour, neuralgic amyotrophy	—

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Many idiopathic phrenic palsies recover over 1–2 years — wait before plication.

11.9 Chest Wall Disorders — Kyphoscoliosis, Thoracoplasty, Flail Chest, Marfan Syndrome & Neuromuscular Restriction

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B13 Disorders of chest wall
- ✓ Describe the respiratory consequences and management of kyphoscoliosis and post-thoracoplasty lung
- ✓ Describe flail chest pathophysiology and management
- ✓ Recognise clinical signs of Marfan syndrome and its pulmonary manifestations

IN BRIEF Chest wall disorders cause extraparenchymal restriction (low TLC, normal/high KCO), increased work of breathing and, when severe, nocturnal hypoventilation progressing to chronic hypercapnic respiratory failure — highly responsive to home NIV.

Kyphoscoliosis

- **Definition:** lateral spinal curvature (scoliosis) with anteroposterior angulation (kyphosis), quantified by **Cobb angle**; respiratory failure risk when Cobb >100–120°, especially onset before age 5 (impaired alveolar development).
- **Causes:** idiopathic (80% — adolescent females), congenital (vertebral anomalies), neuromuscular (polio, muscular dystrophy, SMA, cerebral palsy), connective tissue (Marfan, Ehlers–Danlos), neurofibromatosis, post-TB (Pott's — gibbus), trauma, ankylosing spondylitis.
- **Pathophysiology:** reduced chest wall compliance, respiratory muscle mechanical disadvantage, V/Q mismatch, microatelectasis, sleep hypoventilation (REM), PH and cor pulmonale.
- **Treatment:** bracing and surgical correction (spinal fusion) in growing children with progressive curves; **nocturnal NIV** for hypercapnic failure (improves survival — better than LTOT alone), LTOT, respiratory physiotherapy, vaccination, treat PH.

Thoracoplasty

- Historical collapse therapy for cavitary TB (1930s–1950s): resection of upper ribs to collapse underlying lung; also used for chronic empyema/BPF (space obliteration).
- **Late sequelae** (decades later): restrictive defect, scoliosis, chronic hypercapnic respiratory failure, PH/cor pulmonale, bronchiectasis/destroyed lung — managed with nocturnal NIV, LTOT; modern use — tailored thoracoplasty for post-pneumonectomy empyema.

Flail chest

- ≥3 adjacent ribs fractured in ≥2 places (or sternal separation) → free-floating segment with **paradoxical inward movement on inspiration**.
- **Pathophysiology:** underlying pulmonary contusion (main cause of hypoxaemia), pain-induced hypoventilation, ineffective cough, atelectasis, pneumonia.
- **Management:** O2, aggressive analgesia (epidural/paravertebral/serratus anterior block), physiotherapy, NIV/HFNC; intubation and positive pressure ventilation ("internal pneumatic stabilisation") for respiratory failure; **surgical rib fixation (SSRF)** reduces ventilator days, pneumonia and tracheostomy in selected patients; treat haemopneumothorax.

Clinical signs of Marfan syndrome

- **Revised Ghent criteria (2010):** aortic root dilatation (Z ≥2) + ectopia lentis, or FBN1 mutation, or systemic score ≥7.
- **Systemic features:** tall stature, arm span > height (ratio >1.05), arachnodactyly (**wrist (Walker–Murdoch) and thumb (Steinberg) signs**), pectus carinatum/excavatum, scoliosis, joint hypermobility, high-arched palate, dolichocephaly, enophthalmos, downslanting palpebral fissures, malar hypoplasia, retrognathia, striae, dural ectasia, protrusio acetabuli, flat feet, myopia >3 D; mitral valve prolapse, aortic regurgitation/dissection.
- **Pulmonary:** spontaneous pneumothorax (apical bullae), emphysema-like changes, bronchiectasis, OSA (craniofacial), restrictive defect from chest wall deformity; avoid scuba diving and contact sports.

Neuromuscular and obesity-related restriction

- NM diseases (ALS, DMD, myasthenia, post-polio, high cord injury): monitor FVC, MIP, SNIP, peak cough flow; NIV when FVC <50%, MIP <−60 cmH2O, PaCO2 ≥45 or nocturnal desaturation; cough assist when PCF <270 L/min.
- Obesity: see 15.7 and 13.4.

★ CLINICAL PEARLS & EXAM FOCUS

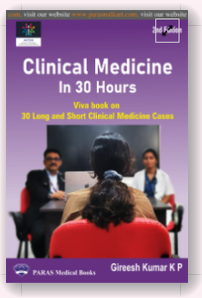
▸ Kyphoscoliosis with Cobb >100° and morning headaches = nocturnal hypoventilation — start home NIV.

LANDMARK TRIALS & GUIDELINES

- Simonds AK. Home ventilation, Eur Respir J 2003; Loeys BL et al, revised Ghent nosology J Med Genet 2010; Marasco SF et al, rib fixation RCT J Am Coll Surg 2013.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Kyphoscoliosis: definition, causes and treatment. *[DNB Dec 2021 P2]*
2. Thoracoplasty. *[DNB Dec 2021 P2]*
3. Flail chest. *[DNB Oct 2024 P1]*
4. Clinical signs of Marfan's syndrome. *[DNB Jun 2021 P2]*



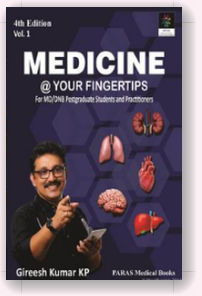
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INVESTIGATIONS & TREATMENT AT A GLANCE

11.9 Chest Wall Disorders — Kyphoscoliosis, Thoracoplasty, Flail Chest, Marfan Syndrome & Neuromuscular Restriction

‹ Back to topic (p 209)
Next topic 12.1 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Cobb angle	< 10°	> 100° → respiratory failure risk	Kyphoscoliosis
VC	> 80%	< 50% predicted (< 1–1.5 L)	Hypercapnic failure risk
Overnight oximetry / $TcCO_2$	Normal	Nocturnal hypoventilation	Start NIV
ABG (awake)	$PaCO_2$ < 45	> 45 mmHg	Chronic hypoventilation
CT chest (flail)	—	≥ 3 adjacent ribs fractured in ≥ 2 places	Flail chest

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Domiciliary NIV	IPAP 14–20, EPAP 4–5, backup rate 14–16	Chest-wall hypoventilation	—
Analgesia (flail)	Epidural / serratus anterior block; paracetamol + NSAID	Rib fractures	Avoid excess opioids
Surgical rib fixation	—	Flail chest on ventilator	—
Pectus repair (Nuss)	—	Symptomatic pectus excavatum (Haller index > 3.25)	—

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Treat nocturnal hypoventilation in kyphoscoliosis early — NIV improves survival.

SECTION 12

Critical Care Pulmonary Medicine

Paper III

Management of emergency problems in pulmonary disease; ARDS; respiratory failure in obstructive and other diseases; sepsis; respiratory and haemodynamic monitoring; non-invasive and mechanical ventilation; principles of critical care, complications and severity-of-illness scores; ethical and end-of-life issues (NMC syllabus II-B10).

12.1 Respiratory Failure — Types, Mechanisms & Approach to Acute Hypoxaemia 211
12.2 Oxygen Delivery Devices, High-Flow Nasal Cannula & ROX Index 213
12.3 Non-Invasive Ventilation 215
12.4 Mechanical Ventilation — Modes, Waveforms, Alarms & Auto-PEEP 216
12.5 ARDS — Definition, Lung-Protective Ventilation, Prone Positioning & Refractory Hypoxaemia 219
12.6 Ventilating Obstructive Airway Disease — COPD & Dynamic Hyperinflation 221
12.7 Weaning, Tracheostomy, VILI & Complications of Mechanical Ventilation 222
12.8 Sepsis & Septic Shock; SOFA Score; Common Medical Emergencies in the Respiratory ICU 224
12.9 Monitoring in Respiratory Critical Care — Pulse Oximetry, Capnography, Haemodynamics & Central Lines 225
12.10 Airway Management & the Difficult Airway 227
12.11 Ethics, End-of-Life Care & Brain Death in Critical Care 228

12.1 Respiratory Failure — Types, Mechanisms & Approach to Acute Hypoxaemia

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B10 Respiratory failure
- ✓ Define and classify respiratory failure (type I, II, acute, chronic, acute-on-chronic)
- ✓ Diagnose and manage type I respiratory failure; differentiate causes of type II failure on ABG
- ✓ Evaluate and manage the non-ventilated hospitalised adult with acute hypoxaemia

IN BRIEF Respiratory failure is failure of gas exchange — PaO2 <60 mmHg (type I, hypoxaemic) or PaCO2 >45–50 mmHg with acidaemia (type II, hypercapnic/ventilatory). Type I reflects lung parenchymal or vascular disease; type II reflects pump failure (drive, nerves, muscles, chest wall) or severe airway disease.

Types of respiratory failure

Type	Mechanism	Examples
I — Hypoxaemic	V/Q mismatch, shunt, diffusion	Pneumonia, ARDS, pulmonary oedema, PE, ILD, atelectasis

Type	Mechanism	Examples
II — Hypercapnic	Alveolar hypoventilation (↓ drive, NM weakness, chest wall, ↑ dead space/load)	COPD, asthma, opioids, GBS, MG, kyphoscoliosis, OHS
III — Perioperative	Atelectasis (↓ FRC)	Post-abdominal surgery, obesity
IV — Shock	Hypoperfusion of respiratory muscles	Septic/cardiogenic shock

Acute vs chronic respiratory failure

Feature	Acute	Chronic	Acute-on-chronic
Onset	Minutes–hours	Days–months	Exacerbation on chronic
pH (type II)	Low (acidaemic)	Near normal (compensated)	Low, with high HCO3
HCO3	Normal	High	High
Clinical	Distress, confusion	Polycythaemia, cor pulmonale, few symptoms	Mixed

Type I respiratory failure — diagnosis and management

- **Diagnosis:** ABG (PaO2 <60 on room air, normal/low PaCO2, raised A–a gradient), P/F ratio, CXR/USG/CT, ECG, echo, D-dimer/CTPA as indicated.
- **Management:** treat cause; oxygen titrated to SpO2 92–96%; HFNC (better comfort, lower intubation than conventional O2 — FLORALI); CPAP for cardiogenic oedema; awake proning (COVID); intubation with lung-protective ventilation if failing (ROX index); avoid delayed intubation.

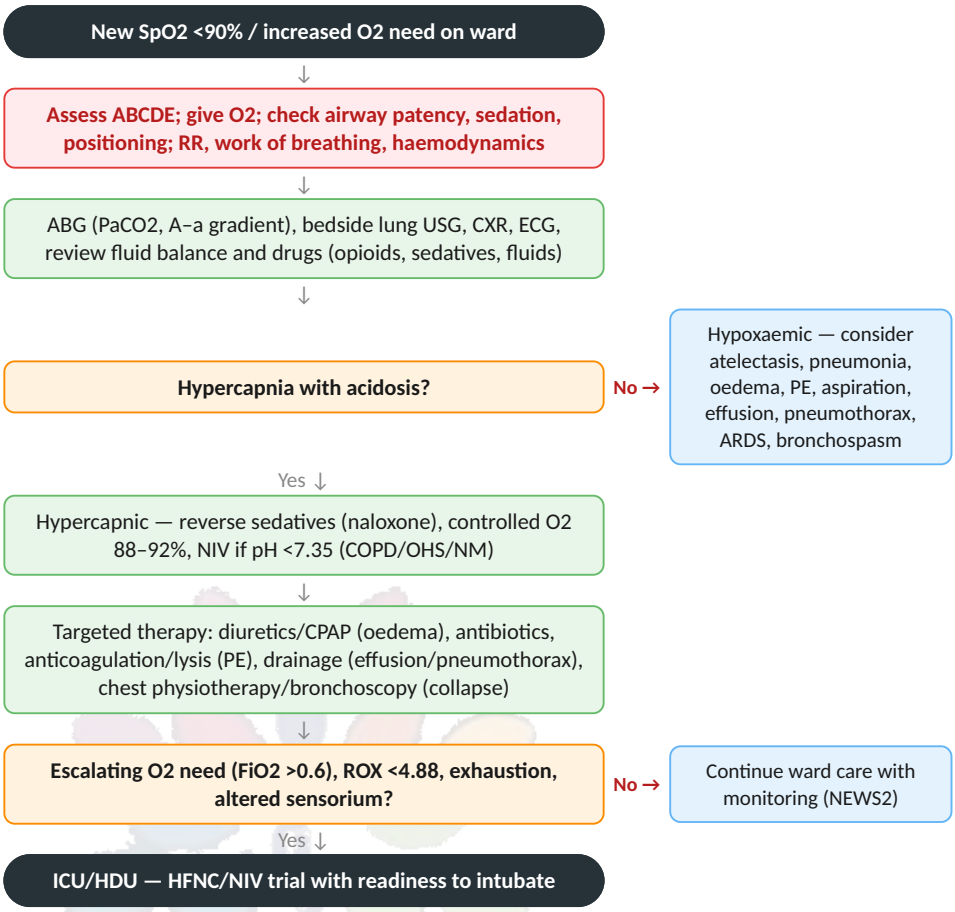
Differential diagnosis of type II respiratory failure on ABG

Hypercapnia — localise by A–a gradient and clinical clues

Category	Clues	Examples
Central (↓ drive)	Normal A–a gradient, shallow slow breathing, low P0.1, no distress	Opioids, sedatives, brainstem stroke, CCHS, hypothyroidism, metabolic alkalosis
Neuromuscular	Normal A–a, rapid shallow breathing, weak cough, paradox, low VC/MIP	GBS, MG, MND, myopathy, critical illness polyneuropathy, high cord injury, phrenic palsy
Chest wall / obesity	Normal A–a (early), restriction	Kyphoscoliosis, OHS, flail chest, thoracoplasty
Airway / lung	Raised A–a gradient, wheeze, hyperinflation	COPD, severe asthma, bronchiectasis, CF, end-stage ILD, upper airway obstruction
Increased CO2 production	Fever, sepsis, overfeeding (carbohydrate)	With limited reserve

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Algorithm 12.1 · Hospitalised non-ventilated adult with acute hypoxaemia



★ CLINICAL PEARLS & EXAM FOCUS

- Low PaCO2 with severe hypoxaemia is good; a "normalising" PaCO2 in a tachypnoeic patient means fatigue.

LANDMARK TRIALS & GUIDELINES

- Frat JP et al, FLORALI NEJM 2015; Rochwerg B et al, ERS/ATS NIV guideline Eur Respir J 2017.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Hypercapnic respiratory failure. *[DNB Oct 2023 P1]*
2. Acute vs chronic respiratory failure. *[DNB Oct 2023 P3]*
3. Diagnosis and management of type I respiratory failure. *[DNB Oct 2024 P3]*
4. Differential diagnosis in a patient presenting with type II respiratory failure on ABG. *[DNB Oct 2024 P3]*
5. Evaluation and management of the non-ventilated, hospitalised adult patient with acute hypoxaemia. *[DNB Jun 2025 P3]*
6. Define respiratory failure. Discuss different types of respiratory failure. *[WBUHS 2022 P1]*

12.2 Oxygen Delivery Devices, High-Flow Nasal Cannula & ROX Index

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B10; Therapeutic — oxygen therapy
- ✓ Classify oxygen delivery devices and their FiO2
- ✓ Describe the physiology, indications and monitoring of HFNC including ROX index

INVESTIGATIONS & TREATMENT AT A GLANCE

12.1 Respiratory Failure — Types, Mechanisms & Approach to Acute Hypoxaemia

‹ Back to topic (p 211)
Next topic 12.2 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
PaO_2	80–100 mmHg	< 60 mmHg	Type 1 respiratory failure
$PaCO_2$	35–45 mmHg	> 45 mmHg with pH < 7.35	Type 2 (acute)
P/F ratio	> 400	< 300	Hypoxaemic failure severity
A–a gradient	< 15–20 mmHg	Raised	Parenchymal cause
Lactate	< 2 mmol/L	> 4	Shock / hypoperfusion
CXR / lung ultrasound	Normal	Consolidation, B-lines, effusion, pneumothorax	Cause

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Controlled O_2	SpO_2 94–98% (88–92% if CO_2 risk)	All	—
HFNC	40–60 L/min, FiO_2 0.4–1.0	Type 1 failure	ROX < 3.85 at 12 h → failure
NIV (BiPAP)	IPAP 12–20, EPAP 5	Type 2 failure, cardiogenic oedema	Delayed intubation risk
Intubation + lung-protective ventilation	Vt 6 mL/kg PBW	Failure of non-invasive support	—

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Hypoventilation → normal A–a gradient; V/Q mismatch improves with O_2; shunt does not.

IN BRIEF Oxygen devices are low-flow (variable FiO2 depending on patient's inspiratory flow) or high-flow (meet or exceed patient demand — fixed FiO2). HFNC delivers heated, humidified gas up to 60 L/min with FiO2 up to 1.0 and provides low-level PEEP and dead-space washout.

Oxygen delivery devices

Device	Flow (L/min)	Approximate FiO2	Comments
Nasal cannula	1–6	0.24–0.44 (+0.04/L)	Comfortable; variable FiO2
Simple face mask	5–10	0.35–0.55	≥5 L/min to prevent CO2 rebreathing
Venturi (air-entrainment) mask	2–15 (by colour)	0.24, 0.28, 0.31, 0.35, 0.40, 0.60 fixed	Controlled O2 in hypercapnic COPD
Partial rebreather	10–15	0.6–0.8	Reservoir bag
Non-rebreather (reservoir) mask	10–15	0.8–0.95	Emergencies, CO poisoning
HFNC	20–60	0.21–1.0 precise	Humidified; PEEP 3–7 cmH2O at 60 L/min (mouth closed)
CPAP / NIV	—	Up to 1.0	Positive pressure

HFNC physiology and indications

- **Mechanisms:** matches inspiratory demand (accurate FiO2), washes out nasopharyngeal dead space (↓ PaCO2, ↓ WOB), modest PEEP (↑ EELV), heated humidification (mucociliary function, comfort), reduced RR.
- **Indications:** acute hypoxaemic failure (FLORALI — lower 90-day mortality in P/F ≤200), post-extubation (prevention of reintubation — low/high-risk with NIV), pre-oxygenation and apnoeic oxygenation during intubation, COVID-19, DNI patients/palliation, bronchoscopy, selected hypercapnic COPD.
- **Initial setting:** 50–60 L/min, FiO2 titrated to SpO2 92–96%.

ROX index

- **ROX = (SpO2/FiO2) / respiratory rate.**
- Measured at 2, 6 and 12 h of HFNC in pneumonia-related hypoxaemic failure: **≥4.88** at 2, 6 or 12 h predicts HFNC success (lower risk of intubation); **<2.85 at 2 h, <3.47 at 6 h, <3.85 at 12 h** predict failure → consider intubation (Roca 2019).
- Avoids delayed intubation (which increases mortality — Kang 2015).

★ CLINICAL PEARLS & EXAM FOCUS

▸ Venturi masks deliver fixed FiO2 regardless of breathing pattern — the device of choice for hypercapnic COPD.

LANDMARK TRIALS & GUIDELINES

- Roca O et al, ROX index AJRCCM 2019; Frat JP et al, FLORALI NEJM 2015.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Oxygen delivery devices. *[DNB Oct 2024 P3]*
2. What is ROX index? *[BFUHS 2022 P3]*

INVESTIGATIONS & TREATMENT AT A GLANCE

12.2 Oxygen Delivery Devices, High-Flow Nasal Cannula & ROX Index

‹ Back to topic (p 213)
Next topic 12.3 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
ROX index	≥ 4.88 at 2, 6, 12 h	< 3.85	HFNC failure — consider intubation
SpO_2/FiO_2	> 315	< 235 (≈ P/F 200)	Severity
Respiratory rate	12–20	> 30 despite HFNC	Failure
$PaCO_2$ on HFNC	Stable	Rising	Need NIV

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Nasal cannula	1–6 L/min (FiO_2 24–44%)	Mild hypoxaemia	Dryness > 4 L/min
Venturi mask	24–60%	Precise FiO_2 (COPD)	—
Non-rebreather mask	10–15 L/min (FiO_2 60–90%)	Severe hypoxaemia (short-term)	Reservoir must stay inflated
HFNC	30–60 L/min, 31–37 °C, FiO_2 titrated	Hypoxaemic failure, post-extubation	Delayed intubation

★ PRESCRIBING & INTERPRETATION PEARLS

- HFNC generates ~ 1 cmH_2O PEEP per 10 L/min with mouth closed.

12.3 Non-Invasive Ventilation

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B10 Non-invasive ventilation; Therapeutic — CPAP/BiPAP
- ✓ State indications, benefits and contraindications of NIV in acute and chronic respiratory failure
- ✓ Set up, titrate and monitor NIV; identify NIV failure

IN BRIEF NIV delivers positive pressure through a mask without an artificial airway. It is the standard of care in hypercapnic COPD exacerbation and cardiogenic pulmonary oedema, and is used in selected hypoxaemic failure, immunocompromised patients, weaning and long-term home ventilation.

Evidence for NIV (ERS/ATS 2017)

Condition	Recommendation
AECOPD with acidosis (pH ≤7.35)	Strong — reduces intubation and mortality
Cardiogenic pulmonary oedema (CPAP or bilevel)	Strong
Immunocompromised with ARF	Conditional
Post-operative ARF	Conditional
Palliation of dyspnoea (DNI)	Conditional
Prevention of post-extubation failure in high-risk	Conditional
Facilitate weaning in hypercapnic patients	Conditional
De novo hypoxaemic ARF (pneumonia, ARDS)	No recommendation — HFNC preferred; helmet NIV promising
Asthma	No recommendation

Benefits

- Unloads respiratory muscles (IPAP), counters intrinsic PEEP (EPAP), recruits alveoli, reduces preload/afterload (LV failure), avoids intubation complications (VAP, sedation), preserves speech, swallowing and cough.

Contraindications

- **Absolute:** cardiac/respiratory arrest, inability to fit mask (facial trauma/burns/surgery), upper airway obstruction (fixed), uncontrolled vomiting.
- **Relative:** inability to protect airway/clear secretions, severe agitation/coma (except hypercapnic coma — trial in ICU), haemodynamic instability, recent upper GI/oesophageal surgery, undrained pneumothorax, severe hypoxaemia, multiorgan failure.

Practical set-up

- Oronasal (full face) mask first in acute; bilevel ventilator with leak compensation; start IPAP 10–12, EPAP 4–5 cmH2O, increase IPAP by 2–5 every 10 min to 20–25 (pressure support = IPAP − EPAP); backup rate 12–16; O2 to SpO2 88–92% (hypercapnic).
- **Monitor:** comfort, synchrony, leak, RR, SpO2, ABG at 1–2 h and 4–6 h; HR, sensorium.
- **NIV failure predictors:** no improvement in pH/PaCO2/RR at 1–2 h, pH <7.25, GCS <11, high severity score, pneumonia, large VT (>9.5 mL/kg) in hypoxaemic failure (HACOR score >5).

Role of NIV in chronic respiratory failure

- **Indications (home NIV):** COPD with persistent hypercapnia (PaCO2 >52 mmHg) after exacerbation or stable — high-intensity NIV reduces mortality/readmission; **OHS** (if CPAP fails/with daytime hypercapnia); **NM disease** (ALS, DMD — improves survival and QoL); **chest wall** disease (kyphoscoliosis, thoracoplasty); central hypoventilation.
- **Criteria (NM/chest wall):** symptoms of hypoventilation + PaCO2 ≥45 mmHg, or nocturnal SpO2 ≤88% for ≥5 min, or FVC <50%/ MIP <60 cmH2O (ALS).
- Modes: spontaneous-timed bilevel, volume-assured pressure support (AVAPS), high-intensity NIV (IPAP 20–30).

★ **CLINICAL PEARLS & EXAM FOCUS**

▸ Reassess ABG at 1–2 hours of NIV — no improvement means escalate, not "wait and see".

LANDMARK TRIALS & GUIDELINES

- Rochwerg B et al, ERS/ATS NIV guideline 2017; Köhnlein T et al, Lancet Respir Med 2014; Murphy PB et al, HOT-HMV JAMA 2017.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Non-invasive ventilation in adults with acute respiratory failure: benefits and contraindications. *[DNB Jun 2025 P3]*
2. Benefits and contraindications of non-invasive ventilation in adults with acute respiratory failure. *[DNB Dec 2022 P3]*
3. Describe the indications of NIV and role of NIV in chronic respiratory failure. *[WBUHS 2020 P2]*
4. Role of NIV in acute respiratory failure. *[BFUHS 2022 P4]*

12.4 Mechanical Ventilation — Modes, Waveforms, Alarms & Auto-PEEP

‹ NMC curriculum
Previous-year Qs ›

◉ **CBME COMPETENCIES — the postgraduate should be able to:**

- ✓ **NMC syllabus area:** II-B10 Mechanical ventilation
- ✓ Describe volume and pressure modes, pressure support and newer modes
- ✓ Explain the relationship between flow, time, volume and frequency; interpret waveforms
- ✓ Respond to alarms and to sudden desaturation with raised plateau pressure

IN BRIEF Ventilator modes are defined by the control variable (volume or pressure), the breath sequence (CMV, IMV, CSV) and the targeting scheme. Understanding the equation of motion — Paw = (V/C) + (R × flow) + PEEP — lets the clinician read waveforms and troubleshoot.

Common modes

Mode	Description	Notes
Volume control (VC-CMV / A/C)	Set VT, RR, flow, PEEP; pressure varies with compliance/resistance	Guaranteed VT; monitor Ppeak/Pplat; flow asynchrony
Pressure control (PC-CMV)	Set inspiratory pressure, Ti, RR; VT varies	Decelerating flow, limits pressure; monitor VT
PRVC (volume-targeted pressure control)	Pressure adapted breath-by-breath to target VT	Adaptive
SIMV	Mandatory breaths synchronised with spontaneous breaths ± PS	Slower weaning than PSV/SBT
Pressure support (PSV, CSV)	Patient-triggered, pressure-limited, flow-cycled (25% of peak flow)	Weaning, comfort; no backup rate (apnoea alarm)
CPAP	Spontaneous breathing at set pressure	SBT
APRV	Long high CPAP with brief releases	Recruitment in ARDS; spontaneous breathing

INVESTIGATIONS & TREATMENT AT A GLANCE

12.3 Non-Invasive Ventilation

‹ Back to topic (p 215)
Next topic 12.4 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
pH / $PaCO_2$ before NIV	pH ≥ 7.35	pH 7.25–7.35 with $PaCO_2$ > 45	Ideal NIV window
ABG at 1–2 h	Improving	pH not improving or worsening	NIV failure — intubate
HACOR score	≤ 5	> 5 at 1 h	NIV failure in hypoxaemic patients
GCS	≥ 9 (≥ 10 in many protocols)	Falling	Airway risk

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
BiPAP (S/T)	IPAP 10–12 → 20–25; EPAP 4–5; backup RR 12–16	AECOPD, OHS, NMD	Leaks, aerophagia
CPAP	5–10 cmH_2O	Cardiogenic pulmonary oedema, OSA	Hypotension
Interface	Oronasal mask initially	Acute NIV	Pressure sores — dressings
Sedation (if needed)	Dexmedetomidine 0.2–0.7 µg/kg/h	Intolerance	Bradycardia

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Contraindications: cardiac/respiratory arrest, inability to protect airway, vomiting, recent upper GI surgery, undrained pneumothorax.

Mode	Description	Notes
Newer	NAVA (diaphragm EAdi-triggered), PAV+ (proportional to effort), ASV (adaptive support — closed-loop), SmartCare (automated weaning), Intellivent	Better synchrony; limited outcome data

Pressure support ventilation

- Each breath is triggered (pressure/flow), pressure-limited (set PS above PEEP), and **flow-cycled** (ends when flow falls to a set % of peak, typically 25%).
- Patient controls RR, Ti, VT; used for weaning (PS 5–8 cmH2O ≈ SBT); risks — hypoventilation in weak drive, over-assistance (central apnoea, ineffective triggering in COPD — raise cycle-off to 40–50%).

Interrelationship of flow, time, volume and frequency

- **VT = flow × Ti** (square wave) — at fixed VT, higher flow shortens Ti.
- **Total cycle time = 60/RR = Ti + Te**; I:E = Ti:Te. Increasing RR or Ti shortens Te → **dynamic hyperinflation/auto-PEEP** in obstructive disease.
- **Minute ventilation = VT × RR** determines PaCO2 ($PaCO_2 \propto VCO_2/VA$).
- In pressure control, VT depends on driving pressure × compliance and on Ti relative to time constant ($\tau = R \times C$; 3τ for ~95% filling/emptying).

Waveform interpretation

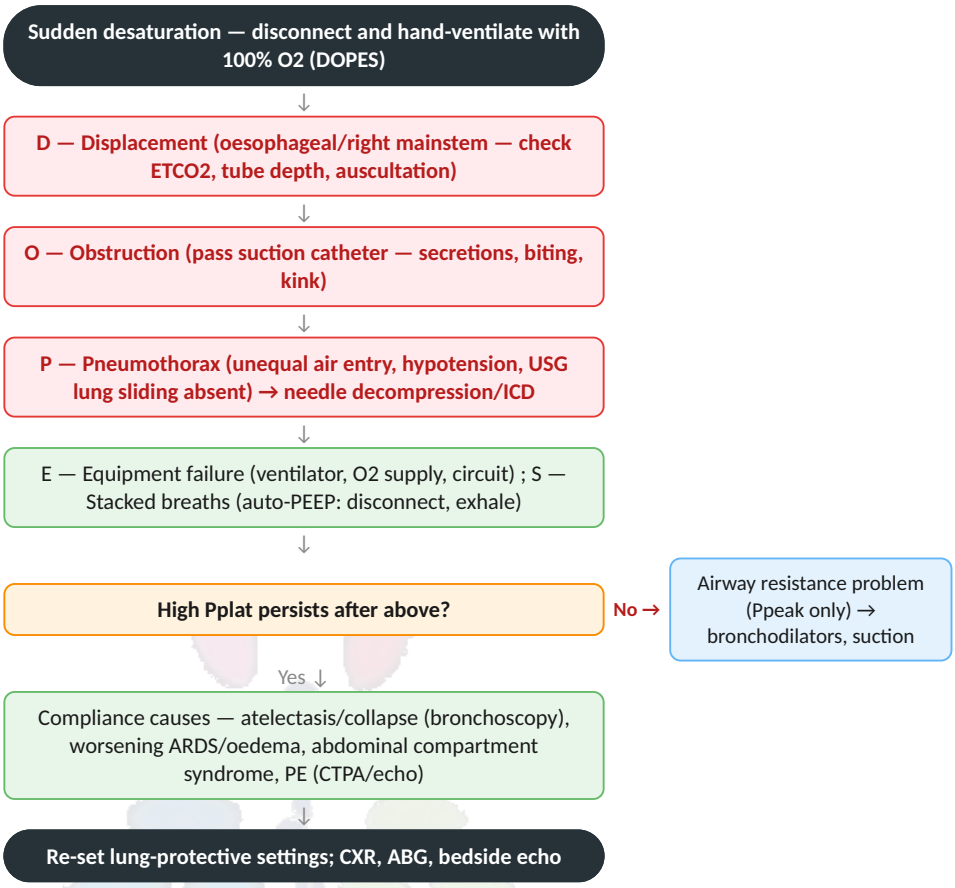
Waveform finding	Meaning
Expiratory flow not returning to zero before next breath	Auto-PEEP (air trapping)
High Ppeak, normal Pplat (large gap)	↑ Airway resistance — bronchospasm, secretions, kinked/bitten tube, small ETT
High Ppeak and high Pplat	↓ Compliance — pneumothorax, mainstem intubation, atelectasis, oedema, ARDS, abdominal distension
Scooped (concave) pressure in VC	Flow starvation — increase flow/switch to PC
Double triggering, ineffective efforts, reverse triggering	Patient–ventilator asynchrony

Alarms in pressure ventilation

- **Low VT / low minute volume:** leak (cuff, circuit, BPF), decreased compliance, increased resistance, decreased effort (PSV), disconnection.
- **High VT/MV:** increased drive (pain, anxiety, acidosis, sepsis), improved compliance (risk of volutrauma), auto-triggering (leak, cardiac oscillations).
- **High RR / apnoea:** distress vs low drive (sedation).
- **High pressure** (in PC — limited by design; inspiratory pressure overshoot), **low PEEP/leak, high/low FiO2, power/gas supply failure.**

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Algorithm 12.2 · Ventilated patient with sudden desaturation and raised plateau pressure



Auto-PEEP (intrinsic PEEP)

- Positive alveolar pressure at end-expiration due to incomplete emptying — high RR, high VT, short Te, airway obstruction (COPD, asthma), small ETT.
- **Measure:** end-expiratory hold manoeuvre (total PEEP − set PEEP); flow waveform.
- **Effects:** barotrauma, hypotension (↓ venous return), increased WOB and **ineffective triggering** (patient must overcome iPEEP before triggering), overestimated Pplat/compliance errors.
- **Management:** ↓ RR and VT, ↑ inspiratory flow (shorten Ti), bronchodilators, sedation; in spontaneously breathing — **set external PEEP at ~75–85% of iPEEP** to reduce triggering effort.

★ CLINICAL PEARLS & EXAM FOCUS

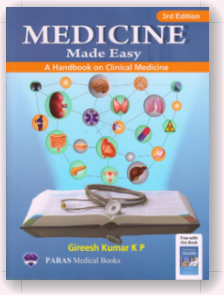
▸ Ppeak–Pplat gap = resistance; Pplat − PEEP = driving pressure (compliance) — the two numbers that diagnose most ventilator problems.

LANDMARK TRIALS & GUIDELINES

- Tobin MJ, Principles and Practice of Mechanical Ventilation (3rd ed.); Chatburn RL, ventilator mode taxonomy, Respir Care 2014.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Modes and alarms in pressure ventilation. *[DNB Oct 2023 P3]*
2. Interrelationship between flow, time, volume and respiratory frequency in mechanical ventilation. *[DNB Oct 2023 P3]*
3. Approach to a ventilated patient with sudden desaturation and raised plateau pressure. *[DNB Oct 2024 P3]*
4. Pressure support ventilation. *[BFUHS 2023 P2]*
5. Newer modes of mechanical ventilation. *[BFUHS 2020 P4]*
6. Auto-PEEP. *[BFUHS 2022 P1]*



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12.5 ARDS — Definition, Lung-Protective Ventilation, Prone Positioning & Refractory Hypoxaemia

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B10 Acute respiratory distress syndrome
- ✓ Define ARDS (Berlin and 2023 global definition) and classify severity; list differential diagnoses
- ✓ Apply lung-protective ventilation including low VT, PEEP, permissive hypercapnia and recruitment
- ✓ Manage refractory hypoxaemia — prone, NMB, iNO, ECMO/ECCO2R; role of corticosteroids

IN BRIEF ARDS is acute diffuse inflammatory lung injury with increased permeability oedema causing hypoxaemia and bilateral opacities not explained by heart failure. Mortality is 30–45%. Only lung-protective ventilation, prone positioning in severe ARDS and conservative fluids have consistent survival or outcome benefit.

Definitions

Item	Berlin (2012)	Global definition (2023)
Timing	Within 1 week of insult or new/worsening symptoms	Same
Imaging	Bilateral opacities on CXR/CT	Bilateral opacities on CXR/CT or **bilateral B-lines/ consolidation on lung ultrasound**
Origin	Not fully explained by HF/fluid overload	Same
Oxygenation (intubated)	P/F ≤300 with PEEP ≥5: mild 200–300, moderate 100–200, severe ≤100	Same, or **SpO2/FiO2 ≤315** (SpO2 ≤97%)
Non-intubated	—	P/F ≤300 or S/F ≤315 on **HFNC ≥30 L/min** or NIV/CPAP ≥5 cmH2O
Resource-limited settings	—	S/F ≤315; no PEEP or flow requirement

Causes and pathophysiology

- **Direct (pulmonary):** pneumonia (commonest), aspiration, inhalation injury, contusion, near-drowning, reperfusion. **Indirect:** sepsis (commonest extrapulmonary), pancreatitis, trauma, massive transfusion/TRALI, burns, drugs.
- **Phases:** exudative (0–7 days — DAD, hyaline membranes, oedema), proliferative (7–21 days — type II hyperplasia, fibroblasts), fibrotic.
- "**Baby lung**" concept — small aerated lung with normal specific compliance; heterogeneity; risk of VILI.

Differential diagnosis of ARDS

Mimic	Clue
Cardiogenic pulmonary oedema	Echo LV dysfunction, BNP, response to diuretics
DAH	Falling Hb, haemoptysis, BAL bloody
Acute eosinophilic pneumonia	BAL eosinophils >25%, new smoker — steroids

INVESTIGATIONS & TREATMENT AT A GLANCE

12.4 Mechanical Ventilation — Modes, Waveforms, Alarms & Auto-PEEP

‹ Back to topic (p 216)
Next topic 12.5 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Plateau pressure	< 30 cmH_2O	> 30	Over-distension
Peak – plateau	< 10 cmH_2O	> 10	High airway resistance
Auto-PEEP (expiratory hold)	0	> 5 cmH_2O	Dynamic hyperinflation
Driving pressure	< 15 cmH_2O	> 15	Mortality risk
Flow waveform	Expiratory flow returns to zero	Fails to return to baseline	Air trapping
Pressure–time scalloping	Smooth	Scooped inspiratory curve	Flow starvation

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Volume control (AC/VC)	Vt 6–8 mL/kg PBW, RR 14–18, PEEP 5, FiO_2 to SpO_2 92–96%	Initial settings	—
Propofol	5–50 µg/kg/min	Sedation	Hypotension, PRIS
Fentanyl	25–100 µg/h	Analgesia-first sedation	Accumulation
Rocuronium / cisatracurium	Roc 1.2 mg/kg intubation; cisatracurium 0.15 mg/kg then 1–3 µg/kg/min	Intubation; severe ARDS	ICU weakness

★ **PRESCRIBING & INTERPRETATION PEARLS**

- DOPE for sudden deterioration: Displacement, Obstruction, Pneumothorax, Equipment failure (+ stacking).

Mimic	Clue
AIP / AE-IPF / acute HP / COP	Background ILD, BAL, steroid response
Drug-induced pneumonitis	Exposure, eosinophils
Lymphangitic carcinomatosis, leukaemic infiltration	Malignancy history
Bilateral pneumonia (PJP, viral)	Immunosuppression, BAL

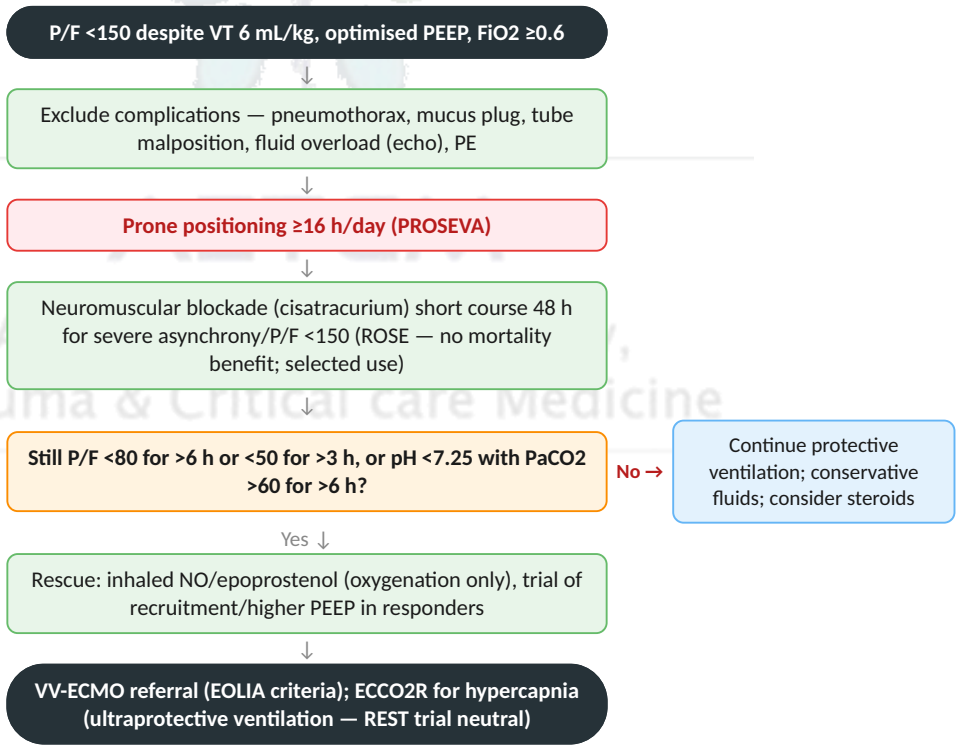
Lung-protective ventilation (ARDSNet)

- **VT 6 mL/kg predicted body weight** (4–8; PBW male = 50 + 0.91 × (height cm − 152.4); female 45.5 + …), **Pplat ≤30 cmH2O**, **driving pressure ≤15 cmH2O** (strongest mortality association), RR up to 35 to maintain pH ≥7.25–7.30.
- **PEEP:** higher PEEP for moderate–severe ARDS (meta-analysis), using ARDSNet FiO2/PEEP tables, best compliance, oesophageal pressure (EPVent-2 neutral), EIT; avoid overdistension.
- **Permissive hypercapnia:** accept PaCO2 rise (pH ≥7.20) to limit VT/pressures — contraindicated in raised ICP, severe pulmonary hypertension/RV failure, severe metabolic acidosis, sickle cell disease, uncontrolled arrhythmia.
- **Recruitment manoeuvres:** sustained inflation (30–40 cmH2O ×30–40 s) or staircase recruitment with PEEP titration; **ART trial** — staircase RM with high PEEP increased mortality — **routine prolonged high-pressure RMs not recommended**; brief RMs in selected responders.

Prone positioning

- **Physiology:** more homogeneous distribution of ventilation and transpulmonary pressure (dorsal recruitment > ventral derecruitment), better V/Q (perfusion stays dorsal), improved secretion drainage, reduced VILI, RV unloading.
- **PROSEVA:** P/F <150 with FiO2 ≥0.6, PEEP ≥5 — prone ≥16 h/day early — 28-day mortality 16% vs 33%.
- Complications: pressure sores, ETT displacement, facial oedema, line dislodgement, brachial plexus injury.

Algorithm 12.3 · Refractory hypoxaemia in ARDS



ECCO2R and ECMO

- **VV-ECMO:** EOLIA (2018) — 60-day mortality 35% vs 46% (NS but Bayesian/meta-analysis favour); CESAR — referral to ECMO centre improved survival.

- **ECCO2R (extracorporeal CO2 removal):** low-flow (0.3–1 L/min) device removing CO2 — enables ultra-low VT (3–4 mL/kg) in ARDS or avoids intubation/eases weaning in hypercapnic COPD; REST trial showed no benefit and more adverse events (bleeding, haemolysis).

Corticosteroids in ARDS

- Evidence mixed: DEXA-ARDS (dexamethasone 20 mg ×5 days then 10 mg ×5 days in moderate–severe ARDS — reduced mortality and ventilator days); benefit in COVID-19 ARDS (RECOVERY), severe CAP (CAPE COD). Not for influenza ARDS; avoid late (>14 days) initiation (LaSRS harm). **ESICM 2023/SCCM 2024:** conditional recommendation for corticosteroids in ARDS.

Advances in ventilator management

- Driving pressure-guided, mechanical power concept, EIT-guided PEEP, transpulmonary pressure monitoring, P0.1 and occlusion pressure (Pocc) to detect excessive effort (P-SILI), lung ultrasound scores, personalised phenotypes (hyper/hypoinflammatory), spontaneous breathing in mild ARDS.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Prone early (within 36 h) and long (≥16 h) — it is the only rescue therapy with a clear mortality benefit.

LANDMARK TRIALS & GUIDELINES

- ARDSNet ARMA NEJM 2000; Guérin C et al, PROSEVA NEJM 2013; Combes A et al, EOLIA NEJM 2018; Matthay MA et al, global definition AJRCCM 2024; Villar J et al, DEXA-ARDS Lancet Respir Med 2020.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. How to classify severity of ARDS? Conventional and advanced ventilatory strategies for ARDS. *[DNB May 2024 P2]*
2. Recruitment manoeuvre strategies for adults with ARDS. *[DNB Dec 2022 P3]*
3. Management strategies for refractory hypoxaemia in ARDS. *[DNB Dec 2025 P3]*
4. Differential diagnosis of acute respiratory distress syndrome. *[DNB Jun 2025 P3]*
5. Lung protective ventilation. *[DNB Oct 2024 P3]*
6. Lung protective mechanical ventilation. *[DNB Jun 2021 P3]*
7. Lung protective ventilatory strategy. *[NEET-SS/DNB 2024]*
8. Protective ventilatory strategy. *[DNB Jun 2020 P3]*

12.6 Ventilating Obstructive Airway Disease — COPD & Dynamic Hyperinflation

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B10 Respiratory failure in obstructive airway disease
- ✓ Set ventilator for COPD with acute respiratory failure
- ✓ Detect and manage dynamic hyperinflation in intubated COPD patients

IN BRIEF In intubated COPD patients, the main danger is dynamic hyperinflation from expiratory flow limitation. Settings aim to give a long expiratory time with modest minute ventilation, targeting the patient's baseline (compensated) PaCO2 rather than normocapnia.

Ventilator settings in COPD with acute respiratory failure

- **Mode:** volume or pressure A/C initially; **VT 6–8 mL/kg PBW**, **RR 10–14**, **high inspiratory flow 60–100 L/min** (Ti 0.6–0.8 s), **I:E ≥1:3**, PEEP: applied ≤ ~80% of measured iPEEP once triggering (initially 0–5), FiO2 to SpO2 88–92%.
- **Target pH** 7.30–7.35 and patient's baseline PaCO2 — over-ventilation causes post-hypercapnic alkalosis (seizures, arrhythmia, bicarbonate loss → weaning failure).

INVESTIGATIONS & TREATMENT AT A GLANCE

12.5 ARDS — Definition, Lung-Protective Ventilation, Prone Positioning & Refractory Hypoxaemia

‹ Back to topic (p 219)
Next topic 12.6 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
P/F ratio (Berlin / global 2023)	> 300	200–300 mild; 100–200 moderate; ≤ 100 severe	ARDS severity (global definition allows SpO_2/FiO_2 ≤ 315 and HFNC ≥ 30 L/min)
Plateau pressure	< 30 cmH_2O	> 30	Reduce Vt
Driving pressure	< 15 cmH_2O	> 15	—
Lung ultrasound / CXR	—	Bilateral opacities	Not fully explained by effusion/ collapse/oedema
Echo	Normal	LV dysfunction (alternative) / acute cor pulmonale	Haemodynamics

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Lung-protective ventilation	Vt 4–8 mL/kg PBW; Pplat < 30; PEEP per ARDSnet table	All ARDS	Permissive hypercapnia (pH > 7.20)
Prone positioning	≥ 12–16 h/day	P/F < 150 (PROSEVA)	Pressure injuries, tube displacement
Cisatracurium	0.15 mg/kg then 37.5 mg/h × 48 h (selected)	Severe ARDS with asynchrony	Not routine (ROSE)
Dexamethasone	20 mg daily × 5 d then 10 mg × 5 d (DEXA-ARDS)	Moderate–severe ARDS (conditional)	Hyperglycaemia
VV-ECMO	—	Refractory hypoxaemia (P/F < 80)	Bleeding, centre expertise

★ PRESCRIBING & INTERPRETATION PEARLS

- Conservative fluid strategy once shock resolved (FACTT).

- Bronchodilators via in-line MDI/vibrating mesh, steroids, antibiotics; early transition to PSV; extubate to NIV in hypercapnic patients (reduces reintubation).

Dynamic hyperinflation in intubated COPD

- **Mechanism:** expiratory flow limitation + short Te → EELV above relaxation volume → iPEEP.
- **Recognition:** expiratory flow not reaching zero, rising Pplat, hypotension after intubation (↓ venous return), high iPEEP on end-expiratory hold, ineffective triggering, increasing PaCO2 despite high MV.
- **Consequences:** barotrauma (pneumothorax), haemodynamic compromise/PEA arrest, increased WOB, asynchrony, weaning failure.
- **Management:** disconnect circuit to allow exhalation if crashing; ↓ VT and RR (minute ventilation), ↑ inspiratory flow, bronchodilators, adequate sedation, treat bronchospasm/secretions; external PEEP to match iPEEP in spontaneously triggering patients; consider larger ETT.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Hypotension right after intubating a COPD/asthma patient — disconnect the ventilator for 30 s before giving pressors.

LANDMARK TRIALS & GUIDELINES

- Laghi F, Tobin MJ. Ventilating obstructive lung disease (Crit Care Clin 2007).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Ventilator settings in patients with acute respiratory failure complicating COPD. *[DNB Dec 2022 P3]*
2. Dynamic hyperinflation in patients with COPD who are intubated for respiratory failure. *[DNB Dec 2022 P3]*

12.7 Weaning, Tracheostomy, VILI & Complications of Mechanical Ventilation

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B10 Mechanical ventilation; complications
- ✓ Assess readiness to wean and perform a spontaneous breathing trial
- ✓ Prevent ventilator-induced lung injury and other complications
- ✓ Critically appraise early vs late tracheostomy

IN BRIEF Weaning is the process of liberation from mechanical ventilation; 40% of ventilator time is spent weaning. Daily screening, sedation interruption and spontaneous breathing trials shorten ventilation. VILI results from volutrauma, barotrauma, atelectrauma, biotrauma and excessive mechanical power.

Readiness criteria (daily screen)

- Underlying cause improving; PaO2/FiO2 ≥150–200 on PEEP ≤5–8 and FiO2 ≤0.4–0.5; pH ≥7.25; haemodynamic stability (minimal/no vasopressors); able to initiate breaths; awake/arousable (RASS 0 to −1); adequate cough.
- **RSBI (f/VT) <105** breaths/min/L on T-piece predicts success (Yang–Tobin).

Methods of weaning

- **Spontaneous breathing trial (SBT):** 30–120 min on T-piece, CPAP or **low PS (5–8 cmH2O)** — PS 30 min had higher successful extubation than T-piece 2 h (Subirà 2019).
- **SBT failure signs:** RR >35, SpO2 <90%, HR >140 or ↑20%, SBP >180 or <90, agitation, diaphoresis, accessory muscles, paradox.
- **Weaning categories (WIND/ICC):** simple (first SBT success), difficult (up to 3 SBTs or 7 days), prolonged (>7 days).
- **Protocolised weaning** and paired SAT + SBT ("Wake up and Breathe" — Girard 2008) reduce ventilator days and mortality.

INVESTIGATIONS & TREATMENT AT A GLANCE

12.6 Ventilating Obstructive Airway Disease — COPD & Dynamic Hyperinflation

‹ Back to topic (p 221)
Next topic 12.7 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Auto-PEEP	0	> 5–10 cmH_2O	Dynamic hyperinflation
Plateau pressure	< 30	Rising	Hyperinflation
Expiratory flow	Reaches zero	Does not reach zero before next breath	Air trapping
pH / $PaCO_2$	—	Accept pH ≥ 7.20	Permissive hypercapnia
BP after intubation	Stable	Hypotension	Hyperinflation — disconnect briefly

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Low RR, low Vt, short Ti	RR 8–12, Vt 6–8 mL/kg, I:E 1:4–1:5, flow 60–80 L/min	Asthma/COPD on ventilator	—
Extrinsic PEEP	≤ 80% of measured auto-PEEP (COPD)	Triggering difficulty	Not in asthma unless measured
Bronchodilators in-line	Salbutamol MDI 4–8 puffs via spacer	Ventilated asthma/COPD	—
Ketamine	0.5–2 mg/kg/h	Refractory bronchospasm	Secretions

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Sudden hypotension after intubation in asthma → disconnect the ventilator for 30–60 s and compress the chest.

- **Prevent extubation failure:** post-extubation HFNC (low risk) or NIV + HFNC (high risk: >65 years, heart failure, COPD, hypercapnia, BMI >30); cuff-leak test and steroids for stridor risk.
- **Causes of weaning failure:** respiratory load (bronchospasm, auto-PEEP, oedema), cardiac (weaning-induced pulmonary oedema — BNP rise, echo), neuromuscular (ICU-acquired weakness, diaphragm dysfunction), neuropsychological (delirium), metabolic (alkalosis, hypophosphataemia, hypothyroidism), nutrition.

Prevention of ventilator-induced lung injury

- Low VT (6 mL/kg PBW), Pplat ≤30, driving pressure ≤15, adequate PEEP to avoid atelectrauma, avoid hyperoxia, limit mechanical power (RR, flow), avoid patient self-inflicted lung injury (high drive — sedation/NMB when needed), prone, protective ventilation even in patients without ARDS (surgery — lower VT reduces pulmonary complications).

Complications of mechanical ventilation

Category	Complications
Airway	Tube malposition/obstruction, laryngeal oedema, tracheal stenosis/malacia, TEF, sinusitis
Lung	VAP, VILI, barotrauma (pneumothorax, pneumomediastinum), oxygen toxicity, atelectasis
Cardiovascular	Hypotension (↓ venous return), RV failure, arrhythmia
Neuromuscular	Diaphragm atrophy (VIDD), ICU-acquired weakness, critical illness polyneuropathy/myopathy
Others	Sedation complications, delirium, GI bleed (stress ulcer), ileus, VTE, pressure ulcers, fluid retention, PTSD, post-intensive care syndrome

Early versus late tracheostomy — evidence-based critique

- **Potential advantages of tracheostomy:** comfort, less sedation, easier oral care and communication, lower dead space and resistance, faster weaning, earlier ICU discharge, possibly less VAP.
- **TracMan (UK, 2013):** early (≤4 days) vs late (≥10 days) — no difference in 30-day mortality; 45% of late group never needed tracheostomy; early group had fewer sedation days.
- **Meta-analyses:** early tracheostomy reduces sedation duration and possibly VAP and ICU stay, but not mortality.
- **Consensus:** decide after 7–10 days if prolonged ventilation is predicted; earlier in severe brain injury (SETPOINT2 neutral), high cervical cord injury, NM disease; percutaneous dilatational tracheostomy (bronchoscopy/USG-guided) is standard.

★ CLINICAL PEARLS & EXAM FOCUS

▸ A daily paired SAT + SBT is the single most effective weaning intervention ("Wake up and Breathe").

LANDMARK TRIALS & GUIDELINES

- Girard TD et al, ABC trial Lancet 2008; Young D et al, TracMan JAMA 2013; Thille AW et al, HIGH-WEAN JAMA 2019.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Methods of weaning for extubation. *[DNB Dec 2021 P3]*
2. Weaning strategies from ventilator support. *[DNB Jun 2020 P3]*
3. Prevention of ventilator-induced lung injury. *[DNB Dec 2021 P3]*
4. Complications of mechanical ventilation. *[DNB Jun 2020 P3]*
5. Early versus late tracheostomy – evidence based critique. *[BFUHS 2019 P4]*

INVESTIGATIONS & TREATMENT AT A GLANCE

12.7 Weaning, Tracheostomy, VILI & Complications of Mechanical Ventilation

‹ Back to topic (p 222)
Next topic 12.8 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
RSBI (f/Vt)	< 105 breaths/min/L	> 105	Weaning failure likely
SBT (30–120 min, PS 5–8 / T-piece)	Tolerated	RR > 35, SpO_2 < 90%, HR > 140, agitation	Failed SBT
Cuff-leak test	> 110 mL or > 12–24%	Absent leak	Post-extubation stridor risk — steroids
Diaphragm thickening fraction	> 30%	< 30%	Weaning failure
P0.1	1.5–3.5 cmH_2O	> 4	High drive — failure risk

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Methylprednisolone	20 mg IV 4-hourly × 4 doses (12 h pre-extubation)	Absent cuff leak	—
Post-extubation HFNC / NIV	HFNC in low risk; NIV prophylactic in high-risk/ hypercapnic	Prevent re-intubation	—
Tracheostomy	Percutaneous / surgical	Prolonged ventilation (> 10–14 days)	Bleeding, stenosis
VAP bundle	Head-up 30–45°, SAT/SBT daily, subglottic suction, oral care	All ventilated	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Pair spontaneous awakening (SAT) with breathing trials (SBT) daily — ABC trial.

12.8 Sepsis & Septic Shock; SOFA Score; Common Medical Emergencies in the Respiratory ICU

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B10 Management of sepsis; severity scoring
- ✓ Define sepsis and septic shock (Sepsis-3) and calculate SOFA
- ✓ Manage sepsis per Surviving Sepsis Campaign 2021
- ✓ Manage DKA and myxoedema coma encountered in the ICU

IN BRIEF Sepsis is life-threatening organ dysfunction (SOFA rise ≥2) caused by a dysregulated host response to infection; septic shock requires vasopressors to keep MAP ≥65 mmHg and lactate >2 mmol/L despite adequate fluids (mortality >40%). Pneumonia is the commonest source.

SOFA score

System	0	1	2	3	4
Respiratory — PaO2/FiO2	≥400	<400	<300	<200 with support	<100 with support
Coagulation — platelets (×10³/µL)	≥150	<150	<100	<50	<20
Liver — bilirubin (mg/dL)	<1.2	1.2–1.9	2.0–5.9	6.0–11.9	≥12
Cardiovascular	MAP ≥70	MAP <70	Dopamine ≤5 or any dobutamine	Dopamine >5 / NA ≤0.1	Dopamine >15 / NA >0.1
CNS — GCS	15	13–14	10–12	6–9	<6
Renal — creatinine (mg/dL) or urine output	<1.2	1.2–1.9	2.0–3.4	3.5–4.9 or <500 mL/day	≥5 or <200 mL/day

- Baseline SOFA assumed 0; acute increase ≥2 = organ dysfunction (~10% mortality). **qSOFA** (RR ≥22, altered mentation, SBP ≤100) — prognostic, not for screening (SSC 2021 advises against using qSOFA alone).

Surviving Sepsis Campaign 2021 — key recommendations

- **Within 1 hour (shock/probable sepsis):** measure lactate, blood cultures before antibiotics, broad-spectrum antibiotics (within 1 h for shock/high likelihood; within 3 h if possible sepsis without shock), 30 mL/kg balanced crystalloid for hypoperfusion within 3 h, vasopressors to MAP ≥65.
- **Fluids:** balanced crystalloids preferred; albumin if large volumes; dynamic measures of fluid responsiveness; capillary refill time to guide.
- **Vasopressors:** norepinephrine first (peripheral start acceptable); add vasopressin (0.03 U/min) when NA 0.25–0.5 µg/kg/min; dobutamine/adrenaline for cardiac dysfunction.
- **Corticosteroids:** hydrocortisone 200 mg/day if ongoing vasopressor requirement (NA ≥0.25 for ≥4 h) — ADRENAL, APROCCHSS.
- **Source control** within 6–12 h; de-escalation; procalcitonin to stop; prolonged β-lactam infusions.
- **Others:** lung-protective ventilation, conservative oxygen, VTE prophylaxis, stress ulcer prophylaxis if bleeding risk, glucose 144–180 mg/dL, early enteral nutrition, no IV vitamin C, discuss goals of care.

Management of diabetic ketoacidosis

- **Diagnosis:** glucose >250 mg/dL (or known diabetes), pH <7.3, HCO3 <18, ketonaemia (β-OHB >3 mmol/L); euglycaemic DKA with SGLT2i.
- **Treatment:** fluids — 0.9% saline/balanced 1 L in first hour, then 250–500 mL/h; **potassium** — if K <3.3 hold insulin and replace; 3.3–5.3 add 20–30 mmol/L; **insulin** — fixed-rate 0.1 U/kg/h IV (after K ≥3.3); add dextrose when glucose <250; bicarbonate only if pH <6.9; treat precipitant (infection, missed insulin, MI); monitor glucose hourly, electrolytes 2–4 hourly; switch to SC insulin with overlap when resolved (pH >7.3, HCO3 ≥18, AG closed).

Myxoedema coma

- Decompensated hypothyroidism: hypothermia, altered sensorium, **hypoventilation (hypercapnic respiratory failure)**, bradycardia, hypotension, hyponatraemia, hypoglycaemia; precipitants — infection, cold, sedatives, amiodarone.
- **Treatment:** ventilatory support (often needs MV), IV hydrocortisone 100 mg 8-hourly (before thyroxine — coexisting adrenal insufficiency), **IV levothyroxine 200–400 µg loading** then 50–100 µg daily (± liothyronine), passive rewarming, correct hyponatraemia/hypoglycaemia, treat precipitant; weaning difficulty from hypothyroid myopathy and macroglossia.

★ CLINICAL PEARLS & EXAM FOCUS

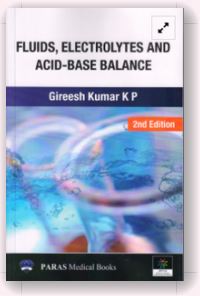
▸ Lactate >4 or vasopressor need = septic shock bundle now — antibiotics within the hour.

LANDMARK TRIALS & GUIDELINES

- Singer M et al, Sepsis-3 JAMA 2016; Evans L et al, SSC guidelines Crit Care Med 2021; Venkatesh B et al, ADRENAL NEJM 2018.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. The SOFA (Sequential Organ Failure Assessment) score. *[DNB Dec 2022 P3]*
2. The SOFA score to describe organ dysfunction/failure. *[DNB Dec 2021 P3]*
3. Sepsis and septic shock management. *[BFUHS 2020 P2]*
4. Management of diabetic ketoacidosis. *[DNB Oct 2024 P2]*
5. Myxoedema coma. *[VNSGU 2025 P2]*



ALSO FROM PARAS MEDICAL BOOKS

Fluids, Electrolytes & Acid-Base Balance

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12.9 Monitoring in Respiratory Critical Care — Pulse Oximetry, Capnography, Haemodynamics & Central Lines

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B10 Respiratory and haemodynamic monitoring
- ✓ Describe principles and limitations of pulse oximetry and capnography
- ✓ Describe haemodynamic monitoring and fluid responsiveness
- ✓ Prevent and manage complications of central venous catheters; use severity-of-illness scores

IN BRIEF Non-invasive monitoring (SpO2, EtCO2) and bedside haemodynamic assessment (echo, dynamic indices) guide therapy in respiratory failure. Each tool has well-defined pitfalls that must be recognised.

Pulse oximetry

- **Principle:** spectrophotometry — oxyHb absorbs more 940 nm (infrared), deoxyHb more 660 nm (red); ratio of pulsatile (AC) to non-pulsatile (DC) absorbance (plethysmography) calibrated against SaO2 (accuracy ±2–3% when SaO2 >80%).
- **Limitations:** poor perfusion/vasoconstriction, motion, nail polish, ambient light; **dyshaemoglobins** (COHb — falsely normal; MetHb — reads ~85%); severe anaemia; **skin pigmentation (overestimates SaO2 in darker skin — occult hypoxaemia)**; venous pulsation (TR); lag in detecting hypoventilation on supplemental O2 (plateau of ODC).
- Use: triage, titration (targets), sleep studies (ODI), 6MWT, NEWS2.

INVESTIGATIONS & TREATMENT AT A GLANCE

12.8 Sepsis & Septic Shock; SOFA Score; Common Medical Emergencies in the Respiratory ICU

‹ Back to topic (p 224)
Next topic 12.9 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Lactate	< 2 mmol/L	≥ 2 (and MAP < 65 on vasopressor = septic shock)	Sepsis-3
SOFA score	Baseline	Increase ≥ 2	Organ dysfunction — sepsis
qSOFA	0–1	≥ 2 (RR ≥ 22, altered mentation, SBP ≤ 100)	Screening only
Blood cultures (2 sets)	No growth	Positive	Before antibiotics
Procalcitonin	< 0.5	> 2	Bacterial sepsis; de-escalation guide

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Broad-spectrum antibiotics	Within 1 h (e.g. pip-taz 4.5 g IV)	Septic shock	De-escalate at 48–72 h
Crystalloid	30 mL/kg balanced crystalloid within 3 h	Hypotension / lactate ≥ 4	Fluid overload
Noradrenaline	0.05–0.5 µg/kg/min (MAP ≥ 65)	Septic shock	Peripheral line short-term OK
Vasopressin	0.03 U/min	Noradrenaline > 0.25 µg/kg/min	Digital ischaemia
Hydrocortisone	200 mg/day (50 mg 6-hourly)	Refractory shock	Hyperglycaemia

★ PRESCRIBING & INTERPRETATION PEARLS

- Surviving Sepsis 2021: start vasopressors early rather than waiting for full fluid resuscitation.

Capnography and end-tidal CO2

- **Principle:** infrared absorption by CO2; mainstream vs sidestream; waveform phases — I (dead space, zero), II (rapid rise — mixed), III (alveolar plateau, EtCO2 at end), 0 (inspiration).
- **Normal** EtCO2 35–45 mmHg; PaCO2–EtCO2 gradient 2–5 mmHg (widens with dead space — PE, low cardiac output, COPD, ARDS).
- **Clinical uses:** confirm ETT placement (gold standard — waveform for ≥6 breaths), CPR quality (EtCO2 <10 mmHg poor; sudden rise = ROSC), monitor ventilation in sedation/procedures, detect disconnection/obstruction, bronchospasm (**shark-fin** waveform), rebreathing (baseline rise), malignant hyperthermia (rising), PE (sudden fall), titrate ventilation in TBI, assess fluid responsiveness (EtCO2 rise with PLR).

Haemodynamic monitoring and fluid responsiveness

Method	Use / limitation
Clinical (CRT, mottling, urine output, lactate)	Perfusion targets (ANDROMEDA-SHOCK — CRT-guided)
Arterial line, pulse pressure variation (PPV >12%), SVV	Fluid responsiveness only in controlled ventilation, VT ≥8 mL/kg, sinus rhythm
Passive leg raise with CO/VTI rise ≥10%	Reliable in spontaneous breathing and arrhythmia
IVC collapsibility/distensibility (USG)	Limited reliability
Echocardiography (POCUS)	LV/RV function, tamponade, VTI, filling
CVP / ScvO2	CVP poor for responsiveness; ScvO2 <70% suggests low delivery
PA catheter (PAWP, CO, SvO2, PVR)	PH, complex shock, RV failure, transplant
Transpulmonary thermodilution (PiCCO)	CO, EVLW, GEDV

Complications of central venous catheters and prevention

CVC complications

Category	Complications	Prevention
Mechanical (insertion)	Arterial puncture, haematoma, pneumothorax (subclavian), haemothorax, arrhythmia, air embolism, malposition, nerve injury	USG guidance, Trendelenburg, experienced operator, guidewire control, CXR/USG confirmation
Infectious	CLABSI, exit-site infection	Insertion bundle: hand hygiene, maximal barrier precautions, 2% chlorhexidine–alcohol, avoid femoral (subclavian preferred for infection), daily review and prompt removal; maintenance bundle — scrub the hub, CHG dressings
Thrombotic	Catheter-related DVT, occlusion, PE, SVC syndrome	Smallest catheter, appropriate tip position, flushing
Late	Catheter fracture/embolism, erosion, cardiac tamponade	Proper tip at cavo-atrial junction

Severity-of-illness scoring systems

- **APACHE II** (12 physiological variables + age + chronic health, first 24 h; 0–71), **APACHE IV**, **SAPS II/III**, **SOFA** (organ dysfunction, serial), **MODS**, **qSOFA**, **NEWS2** (ward deterioration), **CURB-65/PSI** (CAP), **PESI** (PE), **Murray lung injury score**, **GCS**.
- Uses: benchmarking, research stratification, prognostication (not for individual decisions alone).

★ CLINICAL PEARLS & EXAM FOCUS

▸ A pulse oximeter can read 97% in CO poisoning — always check co-oximetry in smoke inhalation.

LANDMARK TRIALS & GUIDELINES

- Sjoding MW et al, racial bias in pulse oximetry NEJM 2020; O'Grady NP et al, CDC guidelines for prevention of intravascular catheter infections 2011.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Capnography and end tidal CO2. *[DNB Jun 2020 P3]*
2. Pulse oximetry. *[DNB Jun 2021 P1]*
3. Write a short note on pulse oximetry. *[BFUHS 2019 P1]*
4. Complications of central venous catheters and their prevention. *[DNB Dec 2021 P3]*

12.10 Airway Management & the Difficult Airway

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B10; Psychomotor — endotracheal intubation
- ✓ Predict and plan for a difficult airway
- ✓ Perform rapid sequence intubation and manage failed intubation (DAS/All India Difficult Airway Association algorithms)

IN BRIEF A difficult airway is one in which a trained clinician experiences difficulty with face-mask ventilation, laryngoscopy, intubation, supraglottic airway use or front-of-neck access. In respiratory failure, physiological difficulty (hypoxaemia, hypotension, acidosis) is as dangerous as anatomical difficulty.

Predicting difficulty

- **LEMON** (Look externally, Evaluate 3-3-2, Mallampati, Obstruction/obesity, Neck mobility); **MACOCHA** score in ICU (Mallampati III–IV, OSA, C-spine limitation, mouth opening <3 cm, coma, severe hypoxaemia, non-anaesthetist); difficult BMV — **MOANS**; difficult SGA — **RODS**; difficult cricothyrotomy — **SMART**.
- **Physiologically difficult:** hypoxaemia, hypotension, severe metabolic acidosis, RV failure.

Rapid sequence intubation in ICU

- Preparation (checklist, team, equipment incl. video laryngoscope, bougie, SGA, FONA kit), **pre-oxygenation** with NIV/HFNC (FiO2 1.0, 3–5 min), apnoeic oxygenation via HFNC, positioning (ramped, ear-to-sternal notch), fluid/pressor ready.
- Induction: ketamine 1–2 mg/kg or etomidate 0.2–0.3 mg/kg (haemodynamically unstable), propofol (stable); rocuronium 1.2 mg/kg or succinylcholine 1.5 mg/kg; video laryngoscope first-attempt success better (DEVICE trial 2023); bougie (BOUGIE trial).
- Confirm with **waveform capnography**; post-intubation sedation and ventilation.

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INVESTIGATIONS & TREATMENT AT A GLANCE

12.9 Monitoring in Respiratory Critical Care — Pulse Oximetry, Capnography, Haemodynamics & Central Lines

‹ Back to topic (p 225)
Next topic 12.10 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
SpO_2	94–98%	< 90%	Hypoxaemia (inaccurate < 80%, poor perfusion, CO)
$EtCO_2$	35–45 mmHg ($PaCO_2$ – $EtCO_2$ 2–5)	Gap > 10; sudden fall	Dead space ↑ (PE, low CO); tube dislodgement
Capnography waveform	Square plateau	Shark-fin; flat line	Bronchospasm; oesophageal intubation/arrest
Lung ultrasound	A-lines + sliding	B-lines, consolidation, absent sliding	Oedema, pneumonia, pneumothorax
$ScvO_2$	> 70%	< 70%	Low oxygen delivery
Pulse pressure variation	< 13%	> 13% (ventilated, sinus rhythm, Vt ≥ 8)	Fluid responsiveness

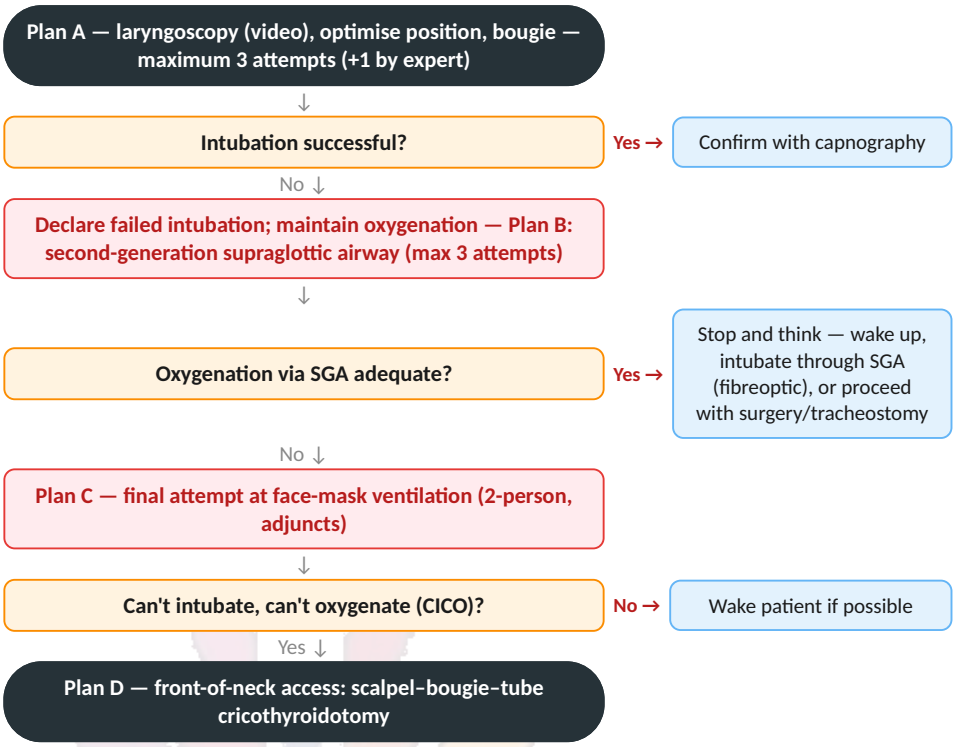
Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Capnography confirmation	Mandatory	Every intubation	—
Fluid challenge	250–500 mL over 10–15 min	Fluid-responsive hypotension	—
Arterial line	Radial	Shock, frequent ABG	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Sustained $EtCO_2$ < 10 mmHg after 20 min of CPR predicts poor outcome.

Algorithm 12.4 · Unanticipated difficult intubation (DAS/AIDAA)



Anticipated difficult airway

- Awake fibreoptic or video-laryngoscopic intubation with topical anaesthesia and minimal sedation (dexmedetomidine/remifentanil), double set-up for surgical airway, consider awake tracheostomy (upper airway tumour).

★ CLINICAL PEARLS & EXAM FOCUS

▸ Pre-oxygenate with NIV/HFNC and use a video laryngoscope — hypoxaemic patients desaturate within seconds of apnoea.

LANDMARK TRIALS & GUIDELINES

- Frerk C et al, DAS guidelines 2015; Myatra SN et al, All India Difficult Airway Association guidelines 2016; Prekker ME et al, DEVICE NEJM 2023.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Difficult airway. *[DNB Dec 2025 P3]*

Academy for Emergency, Trauma & Critical care Medicine

12.11 Ethics, End-of-Life Care & Brain Death in Critical Care

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B10 Ethical and end-of-life issues; Misc. end-of-life care
- ✓ Apply ethical principles to critical-care decision making and communication
- ✓ Describe withholding and withdrawal of life-sustaining treatment under Indian law (advance directives)
- ✓ Describe palliative care for respiratory diseases and brain-death certification

INVESTIGATIONS & TREATMENT AT A GLANCE

12.10 Airway Management & the Difficult Airway

‹ Back to topic (p 227)
Next topic 12.11 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
LEMON / MACOCHA score	Low	MACOCHA ≥ 3	Predicted difficult airway
Mallampati	I–II	III–IV	Difficult laryngoscopy
Thyromental distance	> 6.5 cm	< 6 cm	Difficult airway
Upper lip bite test	Class I	Class III	Difficult
Capnography after intubation	Sustained waveform	Absent	Oesophageal intubation

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Pre-oxygenation	NRB 15 L + nasal cannula 15 L (apnoeic oxygenation) × 3 min	All RSI	—
Ketamine	1–2 mg/kg IV	Induction in shock/asthma	Hypertension
Etomidate	0.3 mg/kg IV	Haemodynamically unstable	Adrenal suppression
Rocuronium	1.2 mg/kg IV	RSI paralysis	Sugammadex 16 mg/kg for reversal
Succinylcholine	1.5 mg/kg IV	RSI	Hyperkalaemia, MH; avoid in burns > 24 h, NMD

★ PRESCRIBING & INTERPRETATION PEARLS

- Difficult Airway Society: maximum three attempts each for laryngoscopy, then SGA, then front-of-neck access (CICO).

IN BRIEF Critical care must balance beneficence, non-maleficence, autonomy and justice. In India, the Supreme Court (Common Cause 2018, simplified 2023) recognised passive euthanasia and advance medical directives; ISCCM/IAPC guidelines provide a framework for limiting non-beneficial treatment.

Ethical principles and communication

- Autonomy (informed consent, capacity, surrogate decision-makers), beneficence, non-maleficence, justice (resource allocation, triage), dignity, truth-telling, confidentiality.
- Family meetings: prognosis, goals of care, shared decision making, cultural sensitivity, documentation; team consensus; conflict resolution (ethics committee, second opinion).

Withholding and withdrawal of life-sustaining treatment (India)

- **Common Cause vs Union of India (2018):** right to die with dignity; advance medical directives (living will) and passive euthanasia legal; **2023 modification** simplified process — primary and secondary medical boards at hospital level (decision within 48 h), no magistrate approval needed; consent of family/guardian.
- ICU practices: DNR/DNAR orders, no escalation, withdrawal of ventilation/vasopressors/dialysis with symptom control (opioids, benzodiazepines for dyspnoea and distress — principle of double effect), compassionate extubation.
- **MoHFW draft guidelines (2024)** on withdrawal of life support in terminally ill patients.

End-of-life care in respiratory diseases

- **Diseases:** COPD, ILD, lung cancer, MND, CF, PH — unpredictable trajectories; early integration of palliative care.
- **Symptoms:** dyspnoea (low-dose oral morphine 10–30 mg/day, fan therapy, breathing techniques, O2 if hypoxaemic, anxiolytics), cough (opioids), secretions (glycopyrrolate), anxiety, depression, fatigue, pain; NIV as palliation.
- Advance care planning, hospice/home care, bereavement support.

Brain death (brainstem death) — India

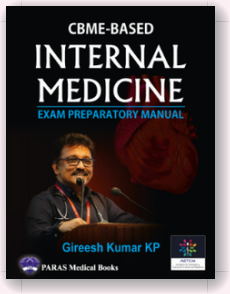
- **Transplantation of Human Organs and Tissues Act (THOTA) 1994, rules 2014:** board of 4 doctors (RMP in charge, independent specialist, neurologist/neurosurgeon, treating doctor), two sets of tests 6 h apart.
- **Prerequisites:** known irreversible cause, exclusion of reversible factors (drugs, hypothermia <35 °C, metabolic/endocrine, shock).
- **Tests:** coma (GCS 3), absent brainstem reflexes (pupillary, corneal, oculocephalic, oculovestibular, gag, cough), **apnoea test** (pre-oxygenate, disconnect with O2 insufflation; no respiratory effort with PaCO2 ≥60 mmHg or rise ≥20 above baseline).
- Ancillary tests when clinical tests incomplete; counsel family regarding organ donation (mandatory request).

★ CLINICAL PEARLS & EXAM FOCUS

▸ Withdrawal of non-beneficial treatment is legal and ethical in India — document the medical board decision and family consent.

LANDMARK TRIALS & GUIDELINES

- Common Cause v. Union of India, Supreme Court of India (2018; modified 2023); Myatra SN et al, ISCCM/IAPC end-of-life care guidelines, Indian J Crit Care Med 2014/2020.



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INVESTIGATIONS & TREATMENT AT A GLANCE

12.11 Ethics, End-of-Life Care & Brain Death in Critical Care

‹ Back to topic (p 228)
Next topic 13.1 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Brain-stem death tests (India, THOTA)	—	Absent brain-stem reflexes ×2 by 2 doctors, 6 h apart	Brain-stem death
Apnoea test	$PaCO_2$ rise with breathing	No breathing at $PaCO_2$ ≥ 60 mmHg (or ≥ 20 above baseline)	Confirms apnoea
Prerequisites	—	Exclude sedatives, hypothermia (< 32–34 °C), metabolic/endocrine causes	Valid testing
Prognostic scores (APACHE II, SOFA)	Low	High	Discussions

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Palliative sedation / symptom control	Morphine 2.5–5 mg SC 4-hourly; midazolam 2.5–5 mg SC	End-of-life dyspnoea/agitation	—
Glycopyrronium	200 µg SC 4–6-hourly	Death rattle	—
Advance directive / withdrawal	Per Supreme Court of India guidelines (2018, 2023)	Futile care	Documentation, medical board

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Communicate with families early and document shared decisions; consider organ donation (THOTA).

SECTION 13

Sleep-Related Breathing Disorders

Paper III

Physiology of sleep and its disorders; polysomnography (full-night and split-night) including CPAP titration and evaluation of daytime sleepiness; sleep apnoeas and other sleep-disordered breathing syndromes (NMC syllabus I-B8, II-B12).

13.1 Sleep Physiology, Polysomnography & Daytime Sleepiness Testing 230
13.2 Obstructive Sleep Apnoea — Epidemiology, Pathogenesis, Clinical Features & Diagnosis 232
13.3 Treatment of Obstructive Sleep Apnoea — PAP, Oral Appliances, Surgery & Pharmacotherapy 234
13.4 Central Sleep Apnoea, Obesity Hypoventilation, Overlap Syndrome & Narcolepsy 236

13.1 Sleep Physiology, Polysomnography & Daytime Sleepiness Testing

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-B8 Physiology of sleep; II-B12 Polysomnography
- ✓ Describe the stages and functions of sleep and respiratory changes during sleep
- ✓ Describe levels of polysomnography and scoring of respiratory events (AASM)
- ✓ Describe MSLT and MWT; effect of sleep on pulmonary disorders; sleep in pregnancy

IN BRIEF Sleep alternates between NREM (N1–N3) and REM in ~90-minute cycles. Sleep reduces ventilatory drive, upper-airway muscle tone and (in REM) accessory muscle activity — unmasking hypoventilation and hypoxaemia in lung, neuromuscular and chest-wall disease.

Stages of normal sleep (AASM)

Stage	% of sleep	EEG features	Other
Wake (W)	<5%	Alpha (8–13 Hz) with eyes closed	High EMG
N1	2–5%	Theta (4–7 Hz), vertex sharp waves	Slow rolling eye movements
N2	45–55%	Sleep spindles (12–14 Hz), K-complexes	—
N3 (slow-wave)	15–25% (↓ with age)	Delta (0.5–2 Hz, >75 µV) ≥20% of epoch	Growth hormone, restoration; first third of night
REM	20–25%	Low-voltage mixed frequency, sawtooth waves	Rapid eye movements, muscle atonia (except diaphragm), dreaming; later in night

- **Functions of sleep:** memory consolidation (REM, spindles), synaptic homeostasis, glymphatic clearance of metabolites, energy conservation, immune function, hormonal regulation (GH, cortisol rhythm, leptin/ghrelin), emotional processing.

Respiratory physiology during sleep

- Loss of wakefulness drive; ↓ hypoxic and hypercapnic responses (most in REM); ↑ upper airway resistance; ↓ minute ventilation (~10–15%) and ↑ PaCO2 (3–7 mmHg); REM atonia of intercostals/accessory muscles — diaphragm-dependent ventilation; ↓ FRC supine; ↓ cough reflex.

Effect of sleep in patients with pulmonary disorders (mechanism of respiratory failure in sleep)

- **COPD:** REM-related desaturation (hypoventilation + V/Q mismatch, low baseline SpO2 on steep ODC), overlap with OSA.
- **NM disease/diaphragm paralysis, kyphoscoliosis:** REM hypoventilation first → NREM → daytime hypercapnia.
- **Obesity:** OHS; **asthma:** nocturnal worsening (circadian β-tone, cortisol nadir, GERD, allergens); **ILD:** nocturnal desaturation; **CF; heart failure:** CSA/CSR.
- Consequences: pulmonary hypertension, arrhythmia, morning headache, poor sleep quality.

Levels of sleep studies (AASM)

Level	Description	Channels
I	Attended in-lab polysomnography — gold standard	EEG, EOG, chin EMG, ECG, airflow (thermistor + nasal pressure), effort belts (RIP), SpO2, body position, leg EMG, snoring, video
II	Unattended full PSG (home)	Same as level I
III	Home sleep apnoea test (HSAT) / cardiorespiratory polygraphy	≥4 channels: airflow, effort, SpO2, HR (no EEG) — REI
IV	Continuous single/dual bio-parameter	Oximetry ± airflow; peripheral arterial tonometry (WatchPAT — uses PAT, often grouped separately)

- HSAT suitable for high pretest probability of moderate–severe OSA without major comorbidity; not for suspected CSA, hypoventilation, NM disease, severe cardiopulmonary disease, insomnia/other sleep disorders.
- **Split-night study:** diagnostic first part (AHI ≥40/h in ≥2 h, or ≥20 with clinical judgement) followed by CPAP titration in the same night.

Scoring of respiratory events (AASM 2012/v3)

Event	Definition
Obstructive apnoea	≥90% drop in airflow ≥10 s with continued respiratory effort
Central apnoea	≥90% drop ≥10 s with absent effort
Mixed apnoea	Absent effort initially, effort resumes during event
Hypopnoea	≥30% airflow drop ≥10 s with ≥3% desaturation or arousal (recommended); CMS requires ≥4%
RERA	Increasing effort/flattening leading to arousal, not meeting apnoea/hypopnoea
Hypoventilation (sleep)	PaCO2 (or surrogate) >55 mmHg for ≥10 min, or ≥10 mmHg rise to >50 for ≥10 min
Cheyne–Stokes breathing	≥3 consecutive central apnoeas/hypopnoeas with crescendo–decrescendo cycle ≥40 s, ≥5/h over ≥2 h

Evaluation of daytime sleepiness

- **Epworth Sleepiness Scale** (0–24; >10 excessive sleepiness).
- **MSLT (multiple sleep latency test):** after overnight PSG (≥6 h sleep), 5 nap opportunities at 2-h intervals; mean sleep latency ≤8 min = pathological sleepiness; **≥2 sleep-onset REM periods (SOREMPs)** (including PSG SOREMP) + MSL ≤8 min = **narcolepsy**; stop REM-suppressing drugs 2 weeks before, sleep diary/actigraphy 1–2 weeks.
- **MWT (maintenance of wakefulness test):** 4 × 40-min trials staying awake in dim room — assesses treatment response and fitness to drive/occupational safety (mean latency <8 min abnormal).

Sleep deficiency in pregnancy

- Prevalence of insomnia, restless legs (iron/folate deficiency), nocturia, GERD, back pain; **sleep-disordered breathing** increases with weight gain, rhinitis (oestrogen), upper-airway oedema and reduced FRC — snoring in up to 1/3 in third trimester.

- **Associations:** gestational hypertension/pre-eclampsia, gestational diabetes, preterm birth, fetal growth restriction, caesarean delivery, postpartum depression.
- **Management:** sleep hygiene, left lateral position, treat RLS (iron), screen symptomatic women (STOP-Bang less validated), CPAP for OSA (safe; may improve BP), avoid sedative-hypnotics where possible.

★ CLINICAL PEARLS & EXAM FOCUS

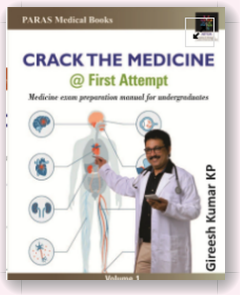
▸ SOREMPs on MSLT + MSL ≤8 min = narcolepsy (provided OSA is treated and drugs stopped).

LANDMARK TRIALS & GUIDELINES

- Berry RB et al, AASM Manual for the Scoring of Sleep (v3, 2023); Kapur VK et al, AASM guideline on diagnostic testing for OSA JCSM 2017.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Stages and functions of sleep. *[DNB Dec 2025 P3]*
2. Stages of normal sleep. *[DNB Oct 2023 P3]*
3. Levels of polysomnography (PSG) for suspected OSA. *[DNB Oct 2024 P3]*
4. MSLT. *[DNB Oct 2023 P4]*
5. Effect of sleep in patients with pulmonary disorder. *[DNB Jun 2020 P3]*
6. Outline the mechanism of respiratory failure in sleep. *[BFUHS 2022 P1]*
7. Sleep deficiency in pregnancy. *[DNB Oct 2023 P4]*



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13.2 Obstructive Sleep Apnoea — Epidemiology, Pathogenesis, Clinical Features & Diagnosis

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B12 Sleep apnoeas
- ✓ Describe the epidemiology and risk factors of OSA
- ✓ Explain pathogenesis including the four endotypes and the role of cephalometry
- ✓ Describe clinical manifestations, systemic complications, differential diagnosis and diagnostic criteria

IN BRIEF OSA is recurrent upper-airway collapse during sleep causing intermittent hypoxaemia, arousals and sleep fragmentation. It affects ~1 billion people worldwide; in India ~11% of adults have OSA (higher in obese, men). Untreated OSA causes hypertension, cardiovascular disease, accidents and poor quality of life.

Epidemiology and risk factors

- Prevalence (AHI ≥5) 9–38% of adults; moderate–severe in 6–17%; men 2–3× (equal after menopause); increases with age.
- **Risk factors:** obesity (BMI, neck circumference >43 cm men, >38 cm women — strongest), male sex, age, craniofacial anatomy (retrognathia, micrognathia, high-arched palate — **Asians have higher OSA at lower BMI due to skeletal restriction**), tonsillar hypertrophy/macroglossia, nasal obstruction, family history, alcohol/sedatives, smoking, supine sleep, menopause, pregnancy, hypothyroidism, acromegaly, Down syndrome, PCOS, stroke, heart failure (fluid shift), end-stage renal disease.

INVESTIGATIONS & TREATMENT AT A GLANCE

13.1 Sleep Physiology, Polysomnography & Daytime Sleepiness Testing

‹ Back to topic (p 230)
Next topic 13.2 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
AHI (PSG)	< 5/h	5–15 mild; 15–30 moderate; ≥ 30 severe	OSA severity
ODI	< 5/h	Raised	Oxygen desaturation index
Epworth Sleepiness Scale	≤ 10	> 10 (> 16 severe)	Daytime sleepiness
MSLT mean sleep latency	> 10 min	≤ 8 min + ≥ 2 SOREMPs	Narcolepsy
MWT	> 30–40 min	< 8 min	Inability to stay awake (driving)
Sleep efficiency	> 85%	< 85%	Insomnia/fragmentation

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Sleep hygiene	Regular schedule; avoid alcohol, screens	All	—
CBT-I	6–8 sessions	Chronic insomnia	First-line over hypnotics
Avoid sedatives in OSA	—	OSA	Worsen apnoea

★ PRESCRIBING & INTERPRETATION PEARLS

- Score apnoeas as ≥ 90% flow drop for ≥ 10 s; hypopnoea ≥ 30% drop with ≥ 3% desaturation or arousal (AASM).

Pathogenesis

- **Anatomical:** small, collapsible pharynx (high critical closing pressure — Pcrit), fat deposition (tongue, parapharyngeal), reduced lung volume (traction).
- **Non-anatomical endotypes (PALM):** P — high **Pcrit/anatomy**; A — low **arousal threshold**; L — high **loop gain** (unstable control); M — poor **muscle responsiveness** (genioglossus).
- **Cycle:** sleep onset → ↓ dilator tone → collapse → apnoea → hypoxaemia/hypercapnia → increased effort → arousal → airway opens → hyperventilation → sleep → repeat; consequences — intermittent hypoxia (oxidative stress, sympathetic activation), intrathoracic pressure swings, sleep fragmentation.

Role of MRI and cephalometry in OSA

- **Lateral cephalometry:** skeletal and soft-tissue landmarks — mandibular plane–hyoid distance (MP-H) increased (low hyoid), reduced posterior airway space (PAS <11 mm), long soft palate, retrognathia (SNA/SNB angles); guides maxillomandibular advancement and oral appliance therapy.
- **MRI:** 3D volumetric assessment of airway and surrounding soft tissues (tongue fat, lateral pharyngeal walls, parapharyngeal fat pads), dynamic cine-MRI during sleep identifies level and pattern of collapse; research and surgical planning (with drug-induced sleep endoscopy — DISE).

Clinical manifestations

- **Nocturnal:** loud habitual snoring, witnessed apnoeas, choking/gasping, nocturia, restless sleep, sweating, GERD, insomnia (COMISA).
- **Daytime:** excessive sleepiness (drowsy driving), unrefreshing sleep, morning headache, poor concentration, memory loss, irritability, depression, decreased libido.
- **Signs:** obesity, large neck, crowded oropharynx (Mallampati III–IV, Friedman tongue position), retrognathia, nasal obstruction, hypertension (resistant).

Differential diagnosis of OSA

Condition	Distinguishing feature
Primary snoring	No apnoeas, no sleepiness
Central sleep apnoea / CSR	Absent effort; HF, opioids
Obesity hypoventilation	Daytime hypercapnia
Narcolepsy, idiopathic hypersomnia	MSLT findings, cataplexy
Insufficient sleep, shift work	History, actigraphy
Periodic limb movement disorder, RLS	Leg movements on PSG
Nocturnal asthma/GERD/panic, nocturnal seizures	Clinical, PSG/EEG
Depression, hypothyroidism, drugs	Clinical/lab

Diagnostic criteria (ICSD-3)

Criterion	Requirement
A	AHI/RDI ≥5/h on PSG (or REI on HSAT) **plus** symptoms (sleepiness, non-restorative sleep, fatigue, insomnia, waking gasping, witnessed apnoeas/snoring) **or** comorbidity (hypertension, mood disorder, cognitive dysfunction, CAD, stroke, CHF, AF, type 2 diabetes)
B	AHI ≥15/h regardless of symptoms

- **Severity:** mild 5–14, moderate 15–29, severe ≥30 events/h. Screening questionnaires: STOP-Bang (≥3 high risk), Berlin, NoSAS; ESS for sleepiness.

Systemic complications of OSA

System	Complications
Cardiovascular	Hypertension (resistant), AF, bradyarrhythmias, CAD/MI, HF, stroke, sudden death at night, pulmonary hypertension
Metabolic	Insulin resistance, type 2 diabetes, dyslipidaemia, NAFLD/MASLD, metabolic syndrome (**Syndrome Z** = OSA + metabolic syndrome)
Neuropsychiatric	Cognitive impairment, depression, motor vehicle and occupational accidents
Others	GERD, nocturia, erectile dysfunction, glaucoma, CKD progression, perioperative complications (difficult airway, post-op respiratory events), pregnancy complications

★ CLINICAL PEARLS & EXAM FOCUS

- ▸ An Indian patient may have severe OSA at a BMI of 25 — craniofacial crowding matters as much as weight.

LANDMARK TRIALS & GUIDELINES

- Benjafield AV et al, global prevalence, Lancet Respir Med 2019; Eckert DJ et al, PALM endotypes AJRCCM 2013; Sharma SK et al, Indian Initiative on OSA (INOSA) guidelines 2015.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Clinical manifestations of OSA and its differential diagnosis. *[DNB Jun 2025 P3]*
2. Diagnostic criteria for OSA. *[DNB Jun 2025 P3]*
3. Risk factors for obstructive sleep apnoea. *[DNB Jun 2025 P3]*
4. Pathogenesis of obstructive sleep apnoea. *[DNB Dec 2025 P3]*
5. Epidemiology and risk factors for OSA. *[DNB Dec 2021 P3]*
6. Diagnosis of obstructive sleep apnoea. *[DNB Jun 2020 P3]*
7. Systemic complications of OSA. *[DNB Oct 2023 P3]*
8. Role of MRI cephalometry in OSA. *[DNB Oct 2023 P3]*

13.3 Treatment of Obstructive Sleep Apnoea — PAP, Oral Appliances, Surgery & Pharmacotherapy

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B12 Sleep apnoeas; Therapeutic — CPAP
- ✓ Prescribe PAP therapy and manage adherence and side effects
- ✓ Choose alternatives — oral appliances, positional therapy, surgery, hypoglossal nerve stimulation
- ✓ Describe medical (drug) management of OSA and prevention

IN BRIEF CPAP is first-line for moderate–severe OSA and symptomatic mild OSA: it splints the upper airway, abolishes events, improves sleepiness, quality of life and blood pressure. Adherence (≥4 h/night on ≥70% of nights) is the main challenge. Tirzepatide (2024) is the first drug approved for OSA with obesity.

Positive airway pressure

- **CPAP:** fixed pressure from in-lab titration (eliminate apnoeas, hypopnoeas, RERAs, snoring in all positions including supine REM) or **auto-PAP** (APAP 4–20 cmH2O) at home for uncomplicated OSA.
- **Bilevel PAP** for high pressure intolerance (>15 cmH2O), coexisting hypoventilation (OHS, COPD overlap, NM).
- **Indications:** AHI ≥15, or AHI ≥5 with symptoms or comorbidities; occupational drivers.
- **Benefits:** reduced sleepiness (ESS by ~2–3 points), improved QoL, mood, BP (2–3 mmHg; more in resistant HTN), fewer accidents; cardiovascular event reduction not proven in SAVE (low adherence) but suggested with good adherence.

INVESTIGATIONS & TREATMENT AT A GLANCE

13.2 Obstructive Sleep Apnoea — Epidemiology, Pathogenesis, Clinical Features & Diagnosis

‹ Back to topic (p 232)
Next topic 13.3 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
STOP-BANG	0–2 low risk	5–8 high risk	OSA screening
AHI (PSG/HSAT)	< 5/h	≥ 5 with symptoms or ≥ 15	OSA diagnosis
Nadir SpO_2 / T90	> 90%; T90 < 1%	Nadir < 80%; T90 > 10%	Hypoxic burden
Neck circumference	< 40 cm	> 43 cm (men), > 40 (women)	Risk
TSH	0.4–4.0	Raised	Hypothyroidism (cause)
HbA1c / BP	Normal	Diabetes, resistant HTN	Comorbidities

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Weight reduction	≥ 10% body weight	Obese OSA	—
Positional therapy	Side-sleeping devices	Positional OSA	—
Tirzepatide	10–15 mg SC weekly	Moderate–severe OSA with obesity (SURMOUNT-OSA)	GI effects
Treat nasal obstruction	Intranasal steroid (fluticasone 100 µg each nostril)	Rhinitis	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Untreated OSA increases road-traffic accident risk 2–3-fold — advise on driving.

- **Side effects:** mask leak, nasal congestion/dryness (heated humidification), aerophagia, claustrophobia, skin pressure; improve with mask fitting, education, telemonitoring, CBT.

Alternatives to PAP

Therapy	Indication / comment
Weight loss (diet, exercise, GLP-1/GIP agonists, bariatric surgery)	All overweight patients; bariatric surgery reduces AHI substantially
Positional therapy (vibration devices, tennis-ball)	Supine-predominant OSA (supine AHI ≥2× non-supine)
Mandibular advancement device	Mild–moderate OSA, PAP intolerance; dental review, custom titratable
Upper airway surgery	Tonsillectomy (children, adults with large tonsils), septoplasty/turbinate reduction (improves PAP tolerance), UPPP (variable success), multilevel surgery, maxillomandibular advancement (most effective, ~85% success)
Hypoglossal nerve stimulation	Moderate–severe OSA (AHI 15–65/100), BMI <32–40, PAP intolerant, no complete concentric collapse on DISE
Avoid alcohol/sedatives, treat nasal obstruction, smoking cessation	All

Medical management of OSA

- **Tirzepatide** (dual GIP/GLP-1 agonist) — SURMOUNT-OSA (2024): AHI reduction ~25–29 events/h with ~18–20% weight loss; FDA-approved for moderate–severe OSA with obesity.
- **Pharmacological endotype targeting: atomoxetine + oxybutynin** (AD109/aroxybutynin — improves genioglossus activity; phase 3 SynAIRgy 2025), carbonic anhydrase inhibitors (acetazolamide, sulthiame — lower loop gain), hypnotics (low arousal threshold — eszopiclone, trazodone: limited), oxygen (high loop gain).
- **Excessive sleepiness despite CPAP:** solriamfetol, modafinil/armodafinil, pitolisant (after optimising CPAP adherence).
- **Treat contributing conditions:** hypothyroidism (levothyroxine), acromegaly, nasal steroids for rhinitis, avoid opioids.

Prevention of sleep-related disorders

- Primary: healthy weight, physical activity, avoid alcohol/sedatives at night, smoking cessation, sleep hygiene (regular schedule, 7–9 h), management of nasal allergies, avoid sleep deprivation, screen high-risk groups (commercial drivers, obese, resistant HTN, AF, stroke, pre-bariatric).
- Secondary: early diagnosis and treatment; tertiary: adherence programmes, managing complications.
- Public health: drowsy driving awareness, occupational screening, school start times for adolescents.

★ CLINICAL PEARLS & EXAM FOCUS

▸ The best PAP is the one the patient uses ≥4 h every night — check download data at 1 month.

LANDMARK TRIALS & GUIDELINES

- Malhotra A et al, SURMOUNT-OSA NEJM 2024; McEvoy RD et al, SAVE NEJM 2016; Patil SP et al, AASM PAP guideline JCSM 2019.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Treatment of OSA. *[DNB Jun 2025 P3]*
2. Role of positive airway pressure (PAP) in managing OSA. *[DNB Oct 2024 P3]*
3. Medical management of obstructive sleep apnoea. *[BFUHS 2023 P2]*
4. Prevention of sleep related disorders. *[BFUHS 2019 P3]*

INVESTIGATIONS & TREATMENT AT A GLANCE

13.3 Treatment of Obstructive Sleep Apnoea — PAP, Oral Appliances, Surgery & Pharmacotherapy

‹ Back to topic (p 234)
Next topic 13.4 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
CPAP titration AHI	< 5/h on therapy	Residual AHI > 10	Pressure adjustment
CPAP adherence (download)	≥ 4 h/night on ≥ 70% nights	Lower	Adherence intervention
Mask leak	< 24 L/min (device-specific)	High	Mask refit
DISE (drug-induced sleep endoscopy)	—	Site of collapse	Surgery/HGNS planning
BMI for HGNS	< 32–35 kg/m²	Higher	Eligibility

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
CPAP / APAP	4–20 cmH_2O (APAP); fixed 8–12 typical	Moderate–severe OSA	Dryness — heated humidification
Mandibular advancement device	Custom, titrated	Mild–moderate OSA, CPAP intolerance	TMJ pain, dental shift
Hypoglossal nerve stimulation	Implant	Moderate–severe OSA, CPAP failure, BMI < 32–35	Surgery
Uvulopalatopharyngoplasty / MMA	Surgical	Selected anatomical cases	Variable success
Solriamfetol / modafinil	Solriamfetol 37.5–150 mg; modafinil 100–200 mg	Residual sleepiness on CPAP	Hypertension

★ PRESCRIBING & INTERPRETATION PEARLS

- Most CPAP problems are mask fit and nasal symptoms — fix those before abandoning therapy.

13.4 Central Sleep Apnoea, Obesity Hypoventilation, Overlap Syndrome & Narcolepsy

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B12 Other sleep-disordered breathing syndromes
- ✓ Classify and treat central sleep apnoea syndromes
- ✓ Diagnose and manage obesity hypoventilation syndrome
- ✓ Recognise overlap syndrome and narcolepsy

IN BRIEF Central apnoeas result from transient loss of ventilatory drive (high loop gain — hypocapnic, or low drive — hypercapnic). OHS is obesity (BMI ≥30) with daytime hypercapnia (PaCO2 ≥45 mmHg) after excluding other causes — 90% also have OSA. Untreated OHS carries high mortality.

Central sleep apnoea syndromes

Type	Mechanism	Treatment
CSA with Cheyne–Stokes breathing	Heart failure, stroke — high loop gain, hypocapnia, circulatory delay	Optimise HF (GDMT, CRT), CPAP; ASV only if LVEF >45%; O2; phrenic nerve stimulation (remede)
High-altitude periodic breathing	Hypoxic drive	Descent, acetazolamide
Drug/opioid-induced CSA	Depressed drive, ataxic breathing	Reduce opioids, ASV/bilevel with backup
Primary (idiopathic) CSA	High chemosensitivity	CPAP, acetazolamide, hypnotics
Treatment-emergent CSA (complex apnoea)	Emerging on CPAP for OSA	Often resolves with time; ASV if persistent
Hypercapnic (hypoventilation) CSA	NM disease, CCHS (PHOX2B), brainstem lesions	NIV with backup rate

Obesity hypoventilation syndrome

- **Diagnostic criteria:** BMI ≥30 kg/m², awake **PaCO2 ≥45 mmHg**, exclusion of other causes of hypoventilation (COPD, NM, chest wall, hypothyroidism). Screening — serum bicarbonate ≥27 mmol/L (ATS 2019) in obese with sleep-disordered breathing → ABG.
- **Pathogenesis:** excess load (reduced compliance, low FRC, increased WOB), blunted chemosensitivity (leptin resistance), sleep-disordered breathing (OSA and REM hypoventilation) → bicarbonate retention resets drive.
- **Clinical:** severe obesity, sleepiness, morning headache, plethora, cor pulmonale, oedema, hypoxaemia; often first presents as acute-on-chronic hypercapnic failure ("Pickwickian" — misdiagnosed as COPD).
- **Management:** PAP — **CPAP first if severe OSA coexists** (Pickwick, ATS 2019) with switch to **NIV** if persistent hypercapnia/no OSA/acute presentation; weight loss (bariatric surgery — target 25–30% loss), avoid sedatives; O2 only with PAP; discharge hospitalised patients on NIV until titration study.

Overlap syndrome

- Coexistence of COPD and OSA (10–15% of COPD patients). More profound nocturnal desaturation, earlier hypercapnia, PH, cor pulmonale, and higher mortality and exacerbations than either alone.
- **Treatment:** CPAP reduces mortality and hospitalisation (Marin 2010); bilevel if hypercapnic; optimise COPD therapy; LTOT if indicated.

Narcolepsy

- Chronic hypersomnolence disorder due to **loss of hypocretin (orexin)-producing hypothalamic neurons** (type 1 — autoimmune, HLA-DQB1*06:02).

- **Tetrad:** excessive daytime sleepiness, **cataplexy** (sudden loss of tone with emotions — type 1), sleep paralysis, hypnagogic/hypnopompic hallucinations; disrupted night sleep.
- **Diagnosis:** PSG + MSLT (MSL ≤8 min, ≥2 SOREMPs); CSF hypocretin ≤110 pg/mL (type 1).
- **Treatment:** scheduled naps, sleep hygiene; modafinil/armodafinil, solriamfetol, pitolisant, methylphenidate; **sodium oxybate** (sleepiness + cataplexy); venlafaxine/SSRIs for cataplexy; orexin receptor 2 agonists in trials.

★ CLINICAL PEARLS & EXAM FOCUS

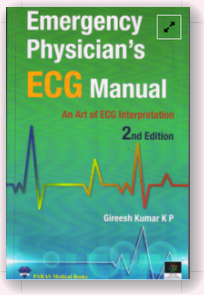
▸ Obese patient with serum HCO3 ≥27 and daytime sleepiness — get an ABG; untreated OHS is a hidden killer.

LANDMARK TRIALS & GUIDELINES

- Mokhlesi B et al, ATS OHS guideline AJRCCM 2019; Masa JF et al, Pickwick Lancet 2019; Marin JM et al, overlap syndrome AJRCCM 2010.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Central sleep apnoea syndrome. *[DNB Jun 2025 P3]*
2. Obesity hypoventilation syndrome. *[DNB Jun 2025 P3; DNB Dec 2021 P3]*
3. Diagnostic criteria for obesity hypoventilation syndrome. *[DNB Oct 2024 P3]*
4. Obesity hypoventilation syndrome and its management. *[BFUHS 2020 P2]*
5. Overlap syndrome. *[BFUHS 2020 P2]*
6. Syndrome Z. *[DNB Oct 2023 P3]*
7. Narcolepsy. *[DNB Oct 2023 P3]*



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INVESTIGATIONS & TREATMENT AT A GLANCE

13.4 Central Sleep Apnoea, Obesity Hypoventilation, Overlap Syndrome & Narcolepsy

‹ Back to topic (p 236)
Next topic 14.1 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Central apnoea index	< 5/h	≥ 5/h, > 50% central	CSA
Cheyne–Stokes pattern	—	Crescendo-decrescendo cycles ≥ 40 s	Heart failure / stroke
Awake $PaCO_2$ / bicarbonate	$PaCO_2$ < 45; HCO_3 < 27	$PaCO_2$ > 45 with BMI ≥ 30	Obesity hypoventilation syndrome
LVEF (echo)	≥ 50%	≤ 45%	ASV contraindicated (SERVE-HF)
CSF hypocretin	> 200 pg/mL	≤ 110 pg/mL	Narcolepsy type 1
Overlap (COPD + OSA)	—	Both present	Higher mortality — treat both

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
CPAP	8–12 cmH_2O	OHS with severe OSA	—
BiPAP ST	IPAP 14–20, EPAP 6–10, backup RR	OHS without OSA, CPAP failure	—
Adaptive servo-ventilation	Auto	CSA with LVEF > 45%	Avoid if LVEF ≤ 45%
Acetazolamide	250 mg at bedtime	High-altitude periodic breathing / CSA	—
Narcolepsy treatment	Modafinil 200–400 mg; pitolisant 9–36 mg; sodium oxybate	Narcolepsy	Abuse potential (oxybate)

★ PRESCRIBING & INTERPRETATION PEARLS

- OHS: BMI ≥ 30 + awake $PaCO_2$ > 45 mmHg after excluding other causes; screen with serum bicarbonate.

SECTION 14

Thoracic Imaging & Pulmonary Procedures

Paper I / IV · Clinical / Viva

Interpretation of plain radiography, ultrasound, CT, PET and MRI; fibreoptic and rigid bronchoscopy (diagnostic and therapeutic), BAL, cryobiopsy, EBUS and navigation; medical thoracoscopy, pleural biopsy, tube thoracostomy, pleurodesis and image-guided FNAC (NMC diagnostic tests and therapeutic procedures). Also the core of the clinical/viva examination (instruments and X-rays).

14.1 Chest Radiograph Interpretation — Collapse, Silhouette & Classic Signs 238
14.2 CT & HRCT Signs in Pulmonary Medicine 240
14.3 Thoracic Ultrasound — Effusion, Pneumothorax & Trauma 241
14.4 PET-CT, MRI & Interventional Radiology in Chest Medicine 242
14.5 Flexible & Rigid Bronchoscopy, BAL, Transbronchial Lung Biopsy & Cryobiopsy 244
14.6 EBUS, Navigational & Virtual Bronchoscopy 245
14.7 Medical Thoracoscopy & Pleural Biopsy (Abrams, Cope, Image-Guided) 246
14.8 Tube Thoracostomy, Pleurodesis (incl. Autologous Blood Patch) & Image-Guided FNAC 248

14.1 Chest Radiograph Interpretation — Collapse, Silhouette & Classic Signs

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** Diagnostic tests — imaging; Psychomotor — X-ray interpretation
- ✓ Interpret a chest radiograph systematically
- ✓ Recognise direct and indirect signs of lobar collapse and other classic signs
- ✓ Describe the role of fluoroscopy

IN BRIEF A systematic approach — technical adequacy, lines and tubes, airway, bones and soft tissues, cardiac and mediastinal contours, hila, lungs (zones), pleura, diaphragm, hidden areas — avoids missed lesions. Lobar collapse produces a reproducible pattern of direct and indirect signs.

Systematic reading

- **Technical:** projection (PA/AP), rotation (medial clavicle ends equidistant from spinous process), inspiration (6 anterior/10 posterior ribs), penetration (vertebrae visible behind heart).
- **Review areas:** apices, behind heart, below diaphragm, hila, costophrenic angles, bones (rib metastases), soft tissue (mastectomy).

Signs of lobar collapse

Direct signs	Indirect signs
Displacement of fissures	Elevation of hemidiaphragm
Crowding of vessels and bronchi	Mediastinal shift (trachea, heart) towards collapse
Increased density of collapsed lobe	Hilar displacement (up in upper lobe, down in lower lobe collapse)
Loss of aeration	Compensatory hyperinflation of remaining lobes (hyperlucency)
—	Rib crowding; shift of anterior junction line; juxtaphrenic peak (upper lobe)

Collapse of individual lobes

Lobe	Appearance
RUL	Elevated horizontal fissure, triangular opacity at right apex; **Golden S sign** (reverse S — central mass)
LUL	Veil-like hazy opacity over left hemithorax (anterior collapse), **Luftsichel sign** (aerated superior segment of LLL outlining aortic knob)
RML	Silhouetting of right heart border; lateral view — wedge between horizontal and oblique fissures
Lingula	Silhouetting of left heart border
RLL / LLL	Triangular opacity behind heart (sail sign), loss of hemidiaphragm outline; LLL — **"sail" behind heart**, loss of descending aorta outline
Total lung	Opaque hemithorax with shift towards (vs effusion — shift away)

Classic CXR signs

Sign	Meaning
Silhouette sign	Loss of border = lesion abutting that structure (RML–right heart, lingula–left heart, lower lobes–diaphragm)
Air bronchogram	Alveolar filling with patent airways (pneumonia, oedema, adenocarcinoma, lymphoma)
Cervicothoracic sign	Mass above clavicles with clear lateral margins = posterior (in lung) vs anterior (blurred)
Hilum overlay / convergence	Mass anterior/posterior to hilum vs hilar vascular enlargement
Deep sulcus sign	Pneumothorax on supine film
Westermark / Hampton's hump	PE oligaemia / infarct
Continuous diaphragm sign	Pneumomediastinum
Bat-wing, Kerley lines	Pulmonary oedema
Finger-in-glove	Mucoid impaction (ABPA, bronchial atresia)
Monod / air crescent	Aspergilloma / recovering IPA

Fluoroscopy

- Real-time X-ray imaging — **sniff test** for diaphragm paralysis, guidance for transbronchial biopsy and peripheral nodule sampling, pleural procedures, bronchial stent placement, foreign-body localisation (air trapping — mediastinal shift on expiration), swallow studies (aspiration, TEF), pulmonary angiography and BAE. Radiation safety — ALARA, lead protection, pulsed fluoroscopy.

★ CLINICAL PEARLS & EXAM FOCUS

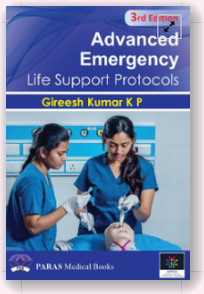
▸ Opaque hemithorax: trachea pulled towards = collapse; pushed away = effusion; central = consolidation or effusion + collapse (tumour).

LANDMARK TRIALS & GUIDELINES

- Felson B. Chest Roentgenology (1973); Hansell DM et al, Fleischner glossary of terms Radiology 2008.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Indirect radiological signs of collapse. *[DNB Oct 2023 P4]*
2. Fluoroscopy. *[Manipal 2014 P1]*



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14.2 CT & HRCT Signs in Pulmonary Medicine

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** Diagnostic tests — imaging
- ✓ Recognise key CT/HRCT signs and their differential diagnoses

IN BRIEF HRCT (thin sections ≤1.5 mm, high spatial frequency reconstruction, prone and expiratory images) characterises diffuse lung disease; CECT and CTPA evaluate masses, mediastinum, pleura and vasculature. Signs are best interpreted in combination with distribution and clinical context.

Important CT signs

Sign	Description	Causes
Tree-in-bud	Centrilobular branching nodular opacities — impacted bronchioles	Endobronchial TB, infectious bronchiolitis, aspiration, ABPA, CF, DPB, NTM
Split pleura sign	Enhancing separated visceral and parietal pleura around fluid	Empyema (also exudative effusion, haemothorax)
Halo sign	GGO surrounding a nodule — haemorrhage	Invasive aspergillosis (neutropenia), mucor, GPA, haemorrhagic metastases
Reversed halo (atoll)	Central GGO with peripheral consolidation ring	COP, mucormycosis, TB, sarcoid, infarct
Crazy paving	GGO + septal thickening	PAP, PJP, DAH, ARDS, lipoid pneumonia, mucinous adenocarcinoma
Mosaic attenuation	Patchy lung density differences	Small airway disease (air trapping on expiration), vascular (CTEPH — vessel size differs), infiltrative GGO
Signet ring	Bronchus larger than accompanying artery	Bronchiectasis
Air crescent	Air surrounding fungal ball/necrotic lung	Aspergilloma, recovering IPA
CT angiogram sign	Vessels visible within low-density consolidation	Mucinous adenocarcinoma, lymphoma, lipoid pneumonia
Comet tail	Curved vessels into subpleural mass	Round atelectasis

INVESTIGATIONS & TREATMENT AT A GLANCE

14.1 Chest Radiograph Interpretation — Collapse, Silhouette & Classic Signs

‹ Back to topic (p 238)
Next topic 14.2 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
CXR quality (RIPE)	Rotation: spinous process midway; 9–10 posterior ribs; full lung fields	Rotated, under-inspired	Interpret with caution
Cardiothoracic ratio (PA)	< 0.5	> 0.5	Cardiomegaly
Silhouette sign	Borders visible	Lost right heart border (RML), left heart border (lingula), hemidiaphragm (lower lobes)	Localises opacity
Lobar collapse signs	Normal fissures	Shifted fissure, sail sign (LLL), Golden S (RUL + mass)	Collapse
Deep sulcus sign (supine)	Absent	Present	Pneumothorax in supine film
Kerley B lines	Absent	Present	Interstitial oedema / lymphangitis

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Chest drain for pneumothorax	8–14 F Seldinger	Confirmed pneumothorax	—
Bronchoscopy	—	Lobar collapse with suspected obstruction	—
Radiology reference	Radiopaedia	Image review	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Always compare with previous films — change over time is the most useful sign.

Sign	Description	Causes
Galaxy / cluster sign	Coalescent nodules	Sarcoidosis, TB
Feeding vessel sign	Vessel leading into nodule	Septic emboli, metastases
Headcheese (three-density)	GGO + normal + air trapping	Hypersensitivity pneumonitis
Honeycombing	Clustered subpleural cysts in rows	UIP (IPF, RA, asbestosis)
Traction bronchiectasis	Irregular dilated bronchi within fibrosis	Fibrotic ILD

- **CT protocols:** HRCT (ILD), CECT (masses, nodes, pleura), CTPA (PE), CT angiography (haemoptysis — bronchial arteries, sequestration), LDCT (screening), dual-energy CT (perfusion), virtual bronchoscopy, quantitative CT (emphysema %LAA-950, airway wall).

★ CLINICAL PEARLS & EXAM FOCUS

▸ Tree-in-bud in an Indian patient = active endobronchial TB until proven otherwise.

LANDMARK TRIALS & GUIDELINES

- Hansell DM et al, Fleischner Society glossary of terms for thoracic imaging, Radiology 2008 (update 2024).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Tree-in-bud sign and split pleura sign. *[DNB Oct 2023 P4]*

14.3 Thoracic Ultrasound — Effusion, Pneumothorax & Trauma

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** Diagnostic tests — ultrasound examination
- ✓ Describe normal lung ultrasound artefacts and pathological patterns
- ✓ Use ultrasound to diagnose and guide management of pleural effusion and pneumothorax
- ✓ Apply chest ultrasound in trauma (eFAST) and in the BLUE protocol

IN BRIEF Lung ultrasound is a rapid, radiation-free bedside tool more sensitive than CXR for effusion and pneumothorax. BTS recommends ultrasound guidance for all pleural procedures involving fluid, reducing complications.

Lung ultrasound signs

Sign	Meaning
Bat sign	Ribs with pleural line between (orientation)
Lung sliding	Visceral pleura moving on parietal — excludes pneumothorax at that point
A-lines	Horizontal reverberation artefacts — normal aerated lung (or pneumothorax if no sliding)
B-lines	Vertical comet-tail artefacts from pleura to screen bottom, move with sliding; ≥3 per field = interstitial syndrome (oedema, ILD, ARDS)
Seashore sign (M-mode)	Normal sliding
Barcode/stratosphere sign (M-mode)	Absent sliding — pneumothorax (or adhesions, apnoea, mainstem intubation)
Lung point	Alternating sliding and no sliding — **pathognomonic of pneumothorax** (100% specific)
Lung pulse	Cardiac pulsation transmitted without sliding — excludes pneumothorax (atelectasis)
Quad sign, sinusoid sign	Pleural effusion
Tissue-like sign / hepatisation, dynamic air bronchogram	Consolidation (pneumonia vs static in atelectasis)

INVESTIGATIONS & TREATMENT AT A GLANCE

14.2 CT & HRCT Signs in Pulmonary Medicine

‹ Back to topic (p 240)
Next topic 14.3 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Interlobular septal thickening	Barely seen	Smooth: oedema/alveolar proteinosis; nodular: lymphangitis/sarcoid	Pattern
Tree-in-bud	Absent	Centrilobular branching	Infectious bronchiolitis, TB, aspiration
Ground-glass opacity	Absent	Present	Active alveolitis, infection, haemorrhage, oedema
Honeycombing	Absent	Subpleural clustered cysts	UIP
Mosaic attenuation + expiratory air-trapping	Absent	Present	Small-airway disease (HP, BO)
Halo / reversed halo	Absent	Halo — angioinvasive fungus; reversed halo — OP / mucor	Pattern

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Contrast (iodinated)	1–1.5 mL/kg (300–350 mgI/mL)	CTPA/CECT	AKI risk; metformin hold if eGFR < 30
Premedication (prior contrast reaction)	Prednisolone 50 mg 13, 7, 1 h + diphenhydramine 50 mg 1 h before	Previous moderate reaction	—
Low-dose CT protocol	~ 1–1.5 mSv	Screening / follow-up	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Prone HRCT distinguishes dependent atelectasis from early fibrosis.

Ultrasonography in pleural effusion

- **Detection:** as little as 5–20 mL (CXR needs 200 mL PA); differentiates effusion from consolidation/thickening.
- **Characterisation:** anechoic (transudate or exudate), complex non-septated, complex septated (exudate — parapneumonic/TB), homogeneously echogenic (empyema, haemothorax); pleural nodularity/thickening >1 cm, diaphragmatic nodules → malignancy.
- **Volume estimation:** maximal interpleural distance (mm) × 20 ≈ mL (Balik formula).
- **Guidance:** site marking/real-time guidance for thoracentesis and drains (reduces pneumothorax and organ puncture), identify intercostal vessels (Doppler), diaphragm and organ position; detect trapped lung, assess lung re-expansion.

Ultrasonography in pneumothorax

- Algorithm: absent sliding → look for B-lines/lung pulse (exclude) → A-lines + barcode sign → search for **lung point** (confirms and estimates size: anterior = small, lateral = large).
- Sensitivity 86–98% vs supine CXR ~50%; useful after procedures, in ICU and trauma; limitations — subcutaneous emphysema, bullae, adhesions, obesity.

Chest ultrasonography in trauma patients

- **eFAST:** pericardial tamponade, haemothorax (anechoic above diaphragm, spine sign), pneumothorax, haemoperitoneum; also rib/sternal fractures, pulmonary contusion (B-lines/consolidation), diaphragmatic rupture, ETT placement; repeatable, bedside, during resuscitation.

BLUE protocol (acute dyspnoea)

- A-profile + DVT → PE; A-profile + no DVT + PLAPS → pneumonia; B-profile (bilateral B-lines with sliding) → cardiogenic oedema; A-profile with abolished sliding + lung point → pneumothorax; A'/B' profiles, C-profile (anterior consolidation) → pneumonia; normal A-profile with no PLAPS → COPD/asthma. Accuracy ~90%.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Lung point = pneumothorax (100% specific); sliding or B-lines at a point excludes pneumothorax under the probe.

LANDMARK TRIALS & GUIDELINES

- Volpicelli G et al, International evidence-based recommendations for point-of-care lung ultrasound, Intensive Care Med 2012; Lichtenstein DA, BLUE protocol Chest 2008.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Ultrasonography in pleural effusion. *[DNB Jun 2025 P4]*
2. Ultrasonography in pneumothorax. *[DNB Jun 2025 P4]*
3. USG in pneumothorax. *[VNSGU 2025 P3]*
4. Chest ultrasonography in trauma patients. *[DNB Oct 2023 P4]*

14.4 PET-CT, MRI & Interventional Radiology in Chest Medicine

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** Diagnostic tests — PET, MRI; Therapeutic — guided procedures
- ✓ Describe indications and pitfalls of PET-CT in pulmonary medicine
- ✓ Describe the role of thoracic MRI
- ✓ Describe interventional radiological procedures used in respiratory medicine

INVESTIGATIONS & TREATMENT AT A GLANCE

14.3 Thoracic Ultrasound — Effusion, Pneumothorax & Trauma

‹ Back to topic (p 241)
Next topic 14.4 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Lung sliding	Present	Absent (+ barcode sign on M-mode)	Pneumothorax
Lung point	—	Present	Pneumothorax — specific
B-lines	≤ 2 per zone	≥ 3 per zone, bilateral	Interstitial syndrome / oedema
Effusion volume estimate	—	Interpleural distance × 20 = mL (supine)	Volume
Septations / echogenicity	Anechoic	Septated/complex	Exudate, empyema
eFAST	Negative	Free fluid / pneumothorax	Trauma

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
USG-guided thoracentesis	—	All effusions	Reduces pneumothorax risk
USG-guided ICD insertion	Triangle of safety	Effusion / pneumothorax	—
Diuresis for B-line pattern	Furosemide 40 mg IV	Cardiogenic oedema	—

★ **PRESCRIBING & INTERPRETATION PEARLS**

- BLUE protocol: A-profile + DVT → PE; A-profile without DVT → COPD/asthma; B-profile → pulmonary oedema.

IN BRIEF FDG-PET-CT combines metabolic and anatomical imaging for staging lung cancer and characterising nodules; MRI is valuable for chest wall, superior sulcus, mediastinal cystic lesions and cardiac sarcoid; interventional radiology delivers biopsies, drainage, embolisation and ablation.

PET-CT

- FDG uptake reflects glucose metabolism (SUV); **indications:** staging NSCLC (mediastinal and distant), SPN characterisation (≥8 mm solid), radiotherapy planning, response assessment, recurrence, mesothelioma, lymphoma, sarcoidosis activity (cardiac sarcoid with dietary preparation), fever of unknown origin, large-vessel vasculitis, CPA activity.
- **False positives:** TB, fungal, sarcoidosis, pneumonia, silicosis nodes, rheumatoid nodules, post-radiation, talc pleurodesis; **false negatives:** <8 mm, AIS/lepidic adenocarcinoma, carcinoid (use Ga-68 DOTATATE), mucinous, hyperglycaemia. In India, granulomatous disease lowers specificity of PET-positive nodes — tissue confirmation by EBUS is mandatory.

Thoracic MRI

- Superior sulcus (Pancoast) tumour — brachial plexus, vertebral, subclavian invasion; chest wall/diaphragm invasion; mediastinal cysts (fluid signal), thymic hyperplasia vs thymoma (chemical shift), cardiac sarcoid and myocarditis, pulmonary vascular imaging (MRA, perfusion) in PH/CTEPH without radiation, CF in children (radiation-free monitoring), brain MRI for staging; hyperpolarised gas (Xe-129) ventilation imaging (research).

Interventional radiological procedures in respiratory medicine

Procedure	Indication
CT/USG-guided transthoracic needle biopsy (FNAC/core)	Peripheral lung lesions, pleural masses, mediastinal masses
Image-guided pleural drainage, IPC placement	Effusion, empyema, pneumothorax
Bronchial artery embolisation	Haemoptysis
Pulmonary artery embolisation / coil	PAVM, Rasmussen aneurysm, pseudoaneurysm
Catheter-directed thrombolysis/ thrombectomy	Massive/submassive PE
IVC filter	VTE with contraindication to anticoagulation
SVC stenting	SVC obstruction
Thoracic duct embolisation	Chylothorax
Percutaneous ablation (RFA, MWA, cryoablation)	Inoperable early lung cancer, metastases
Lung abscess/cyst drainage	Non-resolving abscess
Pulmonary angiography / RHC	CTEPH, PH

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★ CLINICAL PEARLS & EXAM FOCUS

▸ PET-positive mediastinal nodes in India are often granulomatous — always confirm by EBUS before denying surgery.

LANDMARK TRIALS & GUIDELINES

- Silvestri GA et al, ACCP lung cancer staging guideline Chest 2013.

INVESTIGATIONS & TREATMENT AT A GLANCE

14.4 PET-CT, MRI & Interventional Radiology in Chest Medicine

‹ Back to topic (p 242)
Next topic 14.5 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
PET SUVmax	< 2.5	> 2.5 (malignancy likely)	False positives: TB, sarcoid, infection
MRI (superior sulcus, chest wall)	—	Brachial plexus/vertebral invasion	Pancoast staging
Blood glucose before PET	< 150–200 mg/dL	Higher	Reduces accuracy
eGFR before contrast	> 30	< 30	Contrast nephropathy risk
INR / platelets before biopsy	INR < 1.5; platelets > 50 000	Abnormal	Correct before

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
CT-guided biopsy	18–20 G core	Peripheral lesion	Pneumothorax 15–25%
Bronchial artery embolisation	—	Haemoptysis	Spinal ischaemia
Radiofrequency/microwave ablation	—	Small tumours, unfit for surgery	Pneumothorax
Gadolinium caution	—	eGFR < 30	Nephrogenic systemic fibrosis (group II agents safer)

★ PRESCRIBING & INTERPRETATION PEARLS

- Hold metformin 48 h after iodinated contrast if eGFR < 30 or AKI.

14.5 Flexible & Rigid Bronchoscopy, BAL, Transbronchial Lung Biopsy & Cryobiopsy

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** Diagnostic tests — bronchoscopy; Psychomotor
- ✓ State indications, contraindications and complications of diagnostic flexible bronchoscopy
- ✓ Describe bronchoscopic approach in diffuse lung disease including transbronchial cryobiopsy
- ✓ Implement airborne infection control in the bronchoscopy suite

IN BRIEF Flexible bronchoscopy is the core pulmonology procedure — for inspection, sampling (washings, BAL, brushings, TBLB, TBNA, cryobiopsy) and therapy. Rigid bronchoscopy is the platform for interventional airway procedures and massive haemoptysis.

Indications for diagnostic flexible bronchoscopy

Category	Indications
Suspected malignancy	Mass/collapse, haemoptysis, unexplained cough, abnormal sputum cytology, staging (EBUS), vocal cord palsy, SVC syndrome
Infection	Non-resolving pneumonia, immunocompromised host, smear-negative TB (BAL — Xpert/culture), VAP sampling
Diffuse lung disease	BAL, TBLB, cryobiopsy (sarcoid, HP, lymphangitis, PAP, eosinophilic pneumonia)
Airway	Stridor, localised wheeze, foreign body, tracheal stenosis, TEF, BPF, airway trauma, inhalation injury
Others	Haemoptysis localisation, chylothorax/pulmonary haemorrhage (DAH), ETT placement, post-transplant surveillance

- **Contraindications:** uncooperative patient, refractory hypoxaemia, unstable cardiac status (recent MI <6 weeks relative), uncorrected coagulopathy (for biopsy: platelets <50,000, INR >1.5, clopidogrel — stop 5–7 days; aspirin acceptable), severe PH (for biopsy), raised ICP.
- **Complications:** hypoxaemia, bleeding (TBLB 1–4%), pneumothorax (TBLB 1–5%), bronchospasm, laryngospasm, fever, arrhythmias, lidocaine toxicity (max 8 mg/kg), infection transmission, death (<0.01%).

Diagnostic bronchoscopy in diffuse lung disease

- **BAL** (see 5.1) — diagnostic in infection, DAH, PAP, AEP/CEP, lymphangitis; supportive for sarcoid (CD4/CD8), HP (lymphocytes).
- **Forceps TBLB:** 4–6 biopsies from one lobe under fluoroscopy; yield high for bronchocentric/perilymphatic disease (sarcoid 60–90%, lymphangitis, HP, OP, infection); poor for UIP (small, crushed samples).
- **Transbronchial lung cryobiopsy (TBLC):** cryoprobe (1.9/2.4 mm) freezes tissue (3–6 s) and samples larger (~10–40 mm^2) well-preserved specimens; diagnostic yield ~80% in fibrotic ILD (COLDICE — 77% agreement with surgical biopsy at MDD); complications — pneumothorax (~10%), moderate–severe bleeding (prophylactic endobronchial balloon), exacerbation of ILD, mortality ~0.3%; preferred over SLB in experienced centres (ATS 2022 conditional recommendation).
- Avoid in severe PH, FVC <50%, DLCO <35%, uncontrolled coagulopathy.

Rigid bronchoscopy

- Under general anaesthesia (jet ventilation); large working channel — CAO debulking, stents, massive haemoptysis, foreign-body removal (children), dilatation of stenosis, laser.

Airborne infection control in a bronchoscopy suite

- **Facility:** negative pressure room (≥12 ACH, exhaust outdoors/HEPA), separate recovery area, UVGI.
- **Patient:** screen for TB/COVID-19 symptoms, schedule suspected TB at end of list, mask before/after.

- **Staff:** N95/FFP2 (fit-tested), eye protection, gowns, gloves; minimise personnel; avoid aerosol generation (nebulised lidocaine — use spray-as-you-go).
- **Equipment:** high-level disinfection (glutaraldehyde/OPA/peracetic acid, ≥20 min for mycobacteria) or sterilisation; leak test, manual cleaning, drying, storage hanging; single-use bronchoscopes for high-risk; surveillance cultures; track pseudo-outbreaks.
- Biomedical waste segregation (BMW rules 2016).

★ CLINICAL PEARLS & EXAM FOCUS

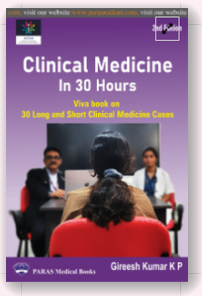
▸ Cryobiopsy gives surgical-quality tissue for ILD — but always have a balloon blocker ready for bleeding.

LANDMARK TRIALS & GUIDELINES

- Du Rand IA et al, BTS bronchoscopy guideline Thorax 2013; Troy LK et al, COLDICE Lancet Respir Med 2020; Mehta AC et al, AABIP/ACCP infection control guidance.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. What are the indications for diagnostic flexible bronchoscopy? *[BFUHS 2019 P1]*
2. Diagnostic bronchoscopy in diffuse lung disease. *[DNB Oct 2023 P4]*
3. Transbronchial cryobiopsy (cryo-TBB). *[DNB Dec 2022 P4]*
4. Measures to control airborne infection in a bronchoscopy suite. *[BFUHS 2022 P2]*



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14.6 EBUS, Navigational & Virtual Bronchoscopy

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** Diagnostic tests — bronchoscopy; Recent advances
- ✓ Describe the current status of EBUS in respiratory medicine
- ✓ Describe guided bronchoscopy for peripheral lesions — radial EBUS, electromagnetic navigation, robotic bronchoscopy and virtual bronchoscopy

IN BRIEF Endobronchial ultrasound combined with real-time needle aspiration has transformed mediastinal diagnosis and staging. Guided bronchoscopy platforms aim to reach peripheral nodules safely, with yields approaching transthoracic biopsy and lower pneumothorax rates.

Endobronchial ultrasound — current status

- **CP-EBUS-TBNA:** first-line for mediastinal/hilar staging of lung cancer (ACCP, ESTS), diagnosis of sarcoidosis, TB lymphadenitis, lymphoma (with EBUS-guided cryobiopsy/core), metastatic disease; sampling of paratracheal/central masses; molecular profiling (adequate for NGS in ~90%).
- **EBUS-IFB / cryo-node biopsy** (2023–24) improves diagnosis of lymphoma and sarcoid.
- **EUS-B-FNA** (via the same scope from oesophagus) adds stations 8, 9, left adrenal and left lower paratracheal.
- **Radial EBUS:** 360° image confirms concentric/eccentric probe position in peripheral nodules (concentric — yield ~80%).

INVESTIGATIONS & TREATMENT AT A GLANCE

14.5 Flexible & Rigid Bronchoscopy, BAL, Transbronchial Lung Biopsy & Cryobiopsy

‹ Back to topic (p 244)
Next topic 14.6 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Pre-bronchoscopy SpO_2	> 90% on O_2	< 90%	Hypoxaemia risk
Platelets / INR (TBLB)	Platelets > 50 000; INR < 1.5	Abnormal	Bleeding
BAL return	> 30% instilled	Low return	Non-diagnostic
BAL cell count	Macrophages > 85%	Lymphocytes > 15%, neutrophils > 3%, eosinophils > 1%	Pattern
Cryobiopsy yield	—	Diagnostic in ~ 80% of ILD	Alternative to SLB

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Lignocaine (topical)	1–2% spray; max 8 mg/kg	Airway anaesthesia	Toxicity
Midazolam	1–2 mg IV, titrate (max ~ 5 mg)	Conscious sedation	Respiratory depression
Fentanyl	25–50 µg IV	Cough suppression/analgesia	—
Cold saline / adrenaline 1:20 000	5–10 mL aliquots	Biopsy bleeding	—
Stop clopidogrel	5–7 days before TBLB	Antiplatelet therapy	Cardiology input

★ **PRESCRIBING & INTERPRETATION PEARLS**

- BAL: instil 3–5 aliquots of 20–50 mL warm saline from a wedged middle-lobe or lingula segment (diffuse disease).

- Training: simulators, competency (≥25–50 procedures), EBUS-STAT assessment tool.

Guided bronchoscopy for peripheral lesions

Technology	Principle	Yield
Radial EBUS ± guide sheath	Ultrasound confirmation	60–75%
Virtual bronchoscopic navigation	CT-reconstructed airway map guides route	70–75% (with rEBUS)
Electromagnetic navigation (ENB)	Patient in EM field, sensor-tipped catheter tracked on CT map	~70% (NAVIGATE)
Robotic-assisted bronchoscopy (Monarch, Ion)	Stable thin articulating catheter, shape-sensing	80–90% in early studies
Cone-beam CT / augmented fluoroscopy	Real-time 3D confirmation (tool-in-lesion)	Improves all platforms
Bronchoscopic transparenchymal nodule access	Tunnel through parenchyma	Selected

Virtual bronchoscopy

- Non-invasive 3D reconstruction of the tracheobronchial tree from thin-section CT (surface/volume rendering, fly-through).
- **Uses:** planning navigation routes and biopsy, evaluating central airway stenosis length and beyond obstruction (where scope cannot pass), stent sizing, follow-up of stents, post-transplant anastomosis, children with foreign bodies (screening), patients unfit for bronchoscopy.
- **Limitations:** no tissue sampling, no mucosal colour/texture, artefacts from secretions/motion, radiation, cannot assess dynamic collapse unless expiratory CT.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Combine navigation with radial EBUS and cone-beam CT — "tool-in-lesion" confirmation is the strongest predictor of diagnostic yield.

LANDMARK TRIALS & GUIDELINES

- Wahidi MM et al, ACCP guideline on technical aspects of EBUS-TBNA Chest 2016; Folch EE et al, NAVIGATE J Thorac Oncol 2019.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Endobronchial ultrasound: current status in diagnosis in respiratory medicine. *[DNB Dec 2021 P4]*
2. Endobronchial ultrasound. *[Manipal 2014 P1]*
3. Virtual bronchoscopy. *[BFUHS 2023 P2]*

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14.7 Medical Thoracoscopy & Pleural Biopsy (Abrams, Cope, Image-Guided)

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** Diagnostic tests — medical thoracoscopy; Therapeutic — pleural biopsy
- ✓ State indications, technique and complications of medical thoracoscopy, including pre-operative evaluation and anaesthesia
- ✓ Describe closed pleural biopsy needles (Abrams, Cope) and image-guided biopsy

IN BRIEF Medical (local anaesthetic) thoracoscopy — pleuroscopy — allows visual inspection of the pleural space, targeted parietal pleural biopsies and talc poudrage in one sitting, with diagnostic sensitivity >90% for malignancy and TB.

INVESTIGATIONS & TREATMENT AT A GLANCE

14.6 EBUS, Navigational & Virtual Bronchoscopy

‹ Back to topic (p 245)
Next topic 14.7 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
EBUS-TBNA nodal stations	—	2R/L, 4R/L, 7, 10, 11 accessible	Staging
Short-axis node size	< 10 mm	≥ 10 mm (or PET-avid)	Sample
ROSE adequacy	—	Lymphocytes / tumour cells present	Adequacy
Navigational yield	—	60–70% for peripheral nodules	Diagnostic yield
Molecular testing from EBUS	—	Adequate tissue for EGFR/ALK/PD-L1	Ensure ≥ 4 passes

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Moderate sedation / GA	Midazolam + fentanyl or propofol	EBUS	—
22/21 G needle passes	≥ 3 per station	Staging	—
Sample N3 → N2 → N1 order	—	Staging	Avoid contamination

★ PRESCRIBING & INTERPRETATION PEARLS

- EBUS-TBNA has largely replaced mediastinoscopy for initial staging.

Indications for medical thoracoscopy

- **Diagnostic:** undiagnosed exudative effusion after fluid analysis (malignancy, TB, mesothelioma), pleural thickening, staging of lung cancer with effusion, empyema (early loculated — adhesiolysis).
- **Therapeutic:** talc poudrage pleurodesis (MPE, recurrent pneumothorax), breaking loculations in early empyema, IPC placement under vision.
- **Contraindications:** obliterated pleural space (adhesions, fibrothorax — no lateral window on USG), uncorrected coagulopathy, refractory hypoxaemia, unstable cardiac status, inability to lie lateral, severe PH, persistent cough.

Pre-operative evaluation and anaesthetic requirements

- **Evaluation:** clinical stability, CXR/CT, thoracic USG (entry site, adhesions — sliding, free fluid), ECG, CBC, coagulation (platelets >60,000, INR <1.5), stop anticoagulants/clopidogrel, SpO2 >90% on minimal O2, consent; consider pre-procedure drainage if massive effusion (induces pneumothorax — "artificial pneumothorax" technique).
- **Anaesthesia:** local anaesthesia (lidocaine 1–2% to skin, intercostal space, parietal pleura — max 3–4.5 mg/kg plain), conscious sedation (midazolam + fentanyl/pethidine; or propofol with anaesthetist), lateral decubitus, monitoring (SpO2, ECG, BP), supplemental O2; spontaneously breathing patient (no single-lung ventilation needed — unlike VATS).

Procedure

- Entry in 4th–7th ICS mid-axillary line (USG-marked) → trocar (5–7 mm rigid or semi-rigid pleuroscope) → aspirate fluid → inspect (nodules, sago grains, plaques) → biopsy parietal pleura over ribs (≥6–10 samples) → talc poudrage (2–4 g) if indicated → chest drain → CXR.
- **Post-procedural complications:** pain, fever (talc), subcutaneous emphysema, persistent air leak, re-expansion oedema, bleeding, infection/empyema, port-site tumour seeding (mesothelioma), arrhythmia, hypoxaemia; mortality <0.1%.

Closed pleural biopsy needles

Needle	Design	Use
Abrams	Outer cannula with distal side notch, inner cutting trocar rotated to close notch — reverse-bevel hooking of pleura	Classic; 4–6 biopsies from 3, 6, 9 o'clock positions (avoid 12 — intercostal bundle)
Cope	Outer cannula + hooked inner biopsy trocar	Similar; older
Raja	Self-retaining flap	Modification
Tru-cut / core needle (image-guided)	Spring-loaded cutting core under USG/CT	Pleural thickening/nodules — malignancy yield 85–90%

- **Yield:** closed pleural biopsy for TB 60–80% (histology + culture ~90%), for malignancy ~50% (patchy distribution) — replaced by image-guided biopsy and thoracoscopy for malignancy; still valuable in high TB-prevalence settings with free-flowing fluid.
- **Complications:** pneumothorax (5–10%), vasovagal, haemothorax, site pain, infection, liver/spleen puncture.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Closed Abrams biopsy is reasonable for suspected TB pleuritis in India; for suspected malignancy go straight to thoracoscopy or image-guided core biopsy.

LANDMARK TRIALS & GUIDELINES

- Rahman NM et al, BTS pleural procedures and thoracic ultrasound guideline 2010 (Asciak 2023 update); Metintas M et al, CT-guided Abrams vs thoracoscopy, Chest 2010.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Enumerate indications, procedure and post-procedural complications of thoracoscopy. *[VNSGU 2025 P4]*
2. Pre-operative evaluation and anaesthetic requirements of medical thoracoscopy. *[BFUHS 2020 P1]*
3. Abrams and Cope's pleural biopsy needles. *[DNB Oct 2023 P4]*
4. Pleural biopsy. *[DNB Dec 2021 P4; Manipal 2014 P1]*

INVESTIGATIONS & TREATMENT AT A GLANCE

14.7 Medical Thoracoscopy & Pleural Biopsy (Abrams, Cope, Image-Guided)

‹ Back to topic (p 246)
Next topic 14.8 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Pleural biopsy yield (thoracoscopy)	—	~ 95% for malignancy and TB	Best pleural diagnostic test
Abrams biopsy yield (TB)	—	~ 70–80% (granulomas)	Blind biopsy in TB-endemic areas
Image-guided biopsy	—	~ 85% for malignancy with thickening	Alternative
Pre-procedure INR / platelets	INR < 1.5; platelets > 60 000	Abnormal	Correct

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Lignocaine (local)	1–2%, max 3 mg/kg (7 with adrenaline)	Local anaesthesia	Toxicity
Talc poudrage	4 g	MPE at thoracoscopy	Fever
Conscious sedation	Midazolam 1–2 mg + fentanyl 25–50 µg	Medical thoracoscopy	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Send biopsy for histology, mycobacterial culture and NAAT in India.

14.8 Tube Thoracostomy, Pleurodesis (incl. Autologous Blood Patch) & Image-Guided FNAC

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** Therapeutic procedures — tube thoracostomy, FNAC
- ✓ Perform and troubleshoot intercostal chest drain insertion
- ✓ Describe chemical pleurodesis agents and autologous blood patch pleurodesis
- ✓ Describe image-guided and blind FNAC of lung masses

IN BRIEF Chest drains should be inserted in the "safe triangle" under ultrasound guidance, using Seldinger small-bore tubes for most indications. Pleurodesis obliterates the pleural space using sclerosants (talc most effective) or autologous blood (for persistent air leak).

Intercostal chest drain

- **Indications:** pneumothorax (tension after decompression, SSP, ventilated, large symptomatic), complicated parapneumonic effusion/empyema, haemothorax, MPE (for pleurodesis), chylothorax, post-operative.
- **Safe triangle:** anterior border of latissimus dorsi, lateral border of pectoralis major, line horizontal to nipple (5th ICS), apex below axilla; insert just above the upper border of the rib.
- **Size:** small-bore (10–14 F, Seldinger) for most effusions, pneumothorax, empyema (with flushing); large-bore (24–32 F) for haemothorax, trauma, massive air leak.
- **Underwater seal:** tube 2–3 cm under water; observe swinging (patent) and bubbling (air leak); suction −10 to −20 cmH2O for persistent leak/incomplete re-expansion; never clamp a bubbling drain.
- **Complications:** malposition (intraparenchymal, fissural, subcutaneous), organ injury (liver, spleen, heart), bleeding (intercostal artery), infection, re-expansion oedema, subcutaneous emphysema, blockage, dislodgement, vasovagal.

Pleurodesis agents

Agent	Comment
Talc (graded large-particle)	Most effective (poudrage or slurry 4–5 g); ARDS risk with small particles
Tetracycline / doxycycline / minocycline	Less effective; pain
Bleomycin	Expensive; less effective than talc
Iodopovidone	Cheap, effective in low-resource settings; pain, hypotension
Silver nitrate, OK-432	Selected/regional
Autologous blood patch	Persistent air leak, PSP/SSP recurrence in non-surgical patients

Autologous blood patch pleurodesis

- **Mechanism:** blood clot seals the air leak ("patch") and induces pleural inflammation/fibrin adhesion.
- **Technique:** aseptically draw 50–120 mL (1–2 mL/kg) of patient's venous blood without anticoagulant, inject immediately through the chest drain, flush with saline; elevate drain tubing on a hook above patient level (loop) for 2–4 h to prevent blood reflux while allowing air to escape (avoid clamping — tension risk); may repeat after 24–48 h.
- **Success:** 60–90% cessation of air leak within 12–48 h; works even with unexpanded lung (unlike chemical agents).
- **Complications:** fever, empyema (infection ~5–9%), tension pneumothorax (if drain occluded by clot), pleural effusion.

FNAC of lung masses (blind and image-guided)

- **Image-guided (CT/USG) transthoracic needle aspiration (TTNA) / core biopsy:** peripheral nodules/masses, pleural-based lesions, chest wall; diagnostic accuracy 85–95% for malignancy; core biopsy gives histology, IHC and molecular testing.
- **Blind FNAC:** large peripheral masses abutting chest wall, palpable nodes (supraclavicular, cervical) — quick, cheap; now replaced by USG guidance for safety.

- **Complications:** pneumothorax (15–25%; 2–7% need drain — risk with emphysema, deep lesions, multiple pleural passes), haemoptysis/pulmonary haemorrhage (~5%), air embolism (rare, catastrophic), tumour seeding (rare); contraindications — uncooperative, coagulopathy, severe emphysema/bullae in path, PAH, contralateral pneumonectomy, suspected hydatid/AVM.
- Rapid on-site evaluation (ROSE) improves adequacy.

★ CLINICAL PEARLS & EXAM FOCUS

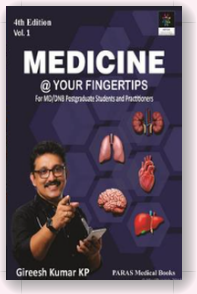
▸ Do not clamp a bubbling chest drain; elevate the tubing after a blood patch rather than clamping.

LANDMARK TRIALS & GUIDELINES

- Havelock T et al, BTS pleural procedures guideline 2010; Manley K et al, autologous blood patch review, Respirology 2012.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Autologous blood patch pleurodesis. *[DNB Dec 2021 P4]*



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INVESTIGATIONS & TREATMENT AT A GLANCE

14.8 Tube Thoracostomy, Pleurodesis (incl. Autologous Blood Patch) & Image-Guided FNAC

‹ Back to topic (p 248)
Next topic 15.1 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Triangle of safety	—	Anterior border latissimus, lateral border pectoralis major, 5th ICS	Insertion site
Air leak (bubbling)	Absent	Persistent > 5 days	Surgery/blood patch
Drain output	—	> 200 mL/h × 2–4 h after haemothorax	Surgical exploration
Lung expansion on CXR	Full	Trapped lung	IPC rather than pleurodesis

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Small-bore ICD	10–14 F Seldinger	Pneumothorax, effusion, empyema	—
Talc slurry	4–5 g in 50 mL saline	Pleurodesis	Clamp 1 h; fever
Autologous blood patch	50–100 mL	Persistent air leak	Infection
Lignocaine	Max 3 mg/kg	Local anaesthesia	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Never clamp a bubbling chest drain.

SECTION 15

Special Environments, Systemic Disease, Pregnancy & Preventive Pulmonology

Paper II / IV

High altitude, diving and aerospace medicine; extrapulmonary manifestations of pulmonary diseases and pulmonary manifestations of systemic diseases; respiratory disease in pregnancy; vaccination and preventive pulmonology; quality-of-life assessment and chronic cough syndromes; obesity-related pulmonary disorders (NMC syllabus II-B11, II-B13, II-B14).

15.1 High-Altitude Illness — AMS, HACE, HAPE & Chronic Mountain Sickness 250
15.2 Diving, Aviation & Aerospace Medicine; Miners 252
15.3 Extrapulmonary Manifestations & Pulmonary Complications of Systemic Disease 253
15.4 Respiratory Diseases in Pregnancy 254
15.5 Vaccination in Pulmonary Diseases 255
15.6 Chronic Cough Syndromes (UACS, GERD) & Quality-of-Life Questionnaires 257
15.7 Obesity-Related Pulmonary Disorders 259

15.1 High-Altitude Illness — AMS, HACE, HAPE & Chronic Mountain Sickness

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B13 Special environments (high altitude)
- ✓ Describe the pathophysiology of HAPE and HACE
- ✓ Recognise, treat and prevent HAPE and AMS
- ✓ Describe chronic mountain sickness

IN BRIEF High-altitude illness occurs above ~2500 m in unacclimatised individuals ascending rapidly — highly relevant to Indian soldiers, pilgrims (Amarnath, Kailash, Kedarnath) and trekkers (Ladakh, Sikkim). HAPE is the commonest cause of death from altitude; descent and oxygen are the definitive treatment.

Pathophysiology

- **AMS/HACE:** hypoxaemia → cerebral vasodilatation, increased blood–brain barrier permeability (VEGF), impaired venous outflow → vasogenic (HACE: cytotoxic + vasogenic) oedema; HACE = end-stage AMS.
- **HAPE:** exaggerated, **uneven hypoxic pulmonary vasoconstriction** → high PA pressure (>35–40 mmHg) → overperfusion of non-constricted areas → **capillary stress failure** → protein-rich **non-cardiogenic (hydrostatic → permeability) oedema**; impaired alveolar Na+ transport (ENaC); low hypoxic ventilatory response; susceptibility — previous HAPE, rapid ascent, exertion, cold, respiratory infection, PFO, genetic factors, re-entry HAPE (residents returning).

High-altitude illnesses

Feature	AMS	HACE	HAPE
Onset	6–12 h after ascent	24–72 h, progression of AMS	2–4 days after arrival
Symptoms	Headache + GI upset, fatigue, dizziness (Lake Louise score ≥3)	Ataxia (tandem gait), altered consciousness, confusion	Exertional dyspnoea → dyspnoea at rest, dry cough → frothy pink sputum, reduced exercise tolerance
Signs	Few	Ataxia, papilloedema, retinal haemorrhages	Tachypnoea, tachycardia, crackles (right middle zone first), cyanosis, low SpO2 for altitude, low-grade fever
Imaging	—	MRI: T2 hyperintensity of splenium of corpus callosum	Patchy asymmetric infiltrates (right middle zone), normal heart size

Treatment of HAPE

- **Descent** at least 300–1000 m (most effective) — do not let patient exert (carry); **supplemental oxygen** to SpO2 >90% (may allow treatment in place with good facilities); **portable hyperbaric chamber (Gamow bag)** if descent impossible.
- **Nifedipine SR 30 mg 12-hourly** (pulmonary vasodilator) when descent/O2 unavailable; PDE5 inhibitors (tadalafil/sildenafil) alternative; dexamethasone if coexisting HACE; avoid diuretics; CPAP/EPAP masks as adjunct.
- **Indian Armed Forces protocol:** O2 + nifedipine + evacuation to lower altitude.

Treatment of AMS/HACE

- Stop ascent; mild AMS — rest, analgesics, acetazolamide 250 mg BD; moderate–severe — descent, O2, dexamethasone 8 mg then 4 mg 6-hourly; HACE — immediate descent, O2, dexamethasone, hyperbaric bag.

Prevention

- **Gradual ascent:** above 3000 m, increase sleeping altitude by ≤500 m/day with a rest day every 3–4 days; "climb high, sleep low"; staged acclimatisation (Indian Army — staging at 2700–3600 m for 6 days, then above 3600 m for 4 days).
- **Acetazolamide** 125 mg BD (AMS prevention) starting 1 day before; dexamethasone 2 mg 6-hourly alternative.
- **HAPE prophylaxis** (prior HAPE): nifedipine SR 30 mg BD or tadalafil 10 mg BD / salmeterol (adjunct); avoid exertion, alcohol, sleeping pills; recognise early symptoms.

Chronic mountain sickness (Monge's disease)

- In long-term high-altitude residents (Andes, Tibet less, Ladakh): **excessive erythrocytosis** (Hb ≥21 g/dL men, ≥19 women), hypoxaemia, headache, dizziness, tinnitus, sleep disturbance, paraesthesia, cyanosis, dilated veins; pulmonary hypertension and cor pulmonale; Qinghai score.
- **Treatment:** relocation to low altitude (curative), venesection, acetazolamide (stimulates ventilation), treat sleep-disordered breathing, ACE inhibitors; **high-altitude pulmonary hypertension (HAPH)** — PH without excessive erythrocytosis.

★ **CLINICAL PEARLS & EXAM FOCUS**

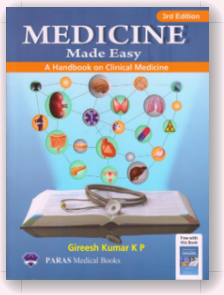
▸ HAPE patient = "descend, oxygen, nifedipine" — and never let them walk down themselves.

LANDMARK TRIALS & GUIDELINES

- Luks AM et al, Wilderness Medical Society clinical practice guidelines for acute altitude illness (2019, 2024 update); Bärtsch P, Swenson ER. Acute high-altitude illnesses, NEJM 2013.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Treatment of high altitude pulmonary oedema. *[DNB Jun 2025 P2]*
2. Recognition, management and prevention of high-altitude pulmonary oedema. *[BFUHS 2022 P2]*
3. Mechanisms for the development of HAPE and HACE. *[BFUHS 2022 P1]*
4. High altitude pulmonary oedema. *[VNSGU 2025 P3]*
5. Chronic mountain sickness. *[Manipal 2014 P2]*



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15.2 Diving, Aviation & Aerospace Medicine; Miners

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B13 Aerospace medicine; special environments (diving, miners)
- ✓ Describe pulmonary barotrauma, decompression sickness and arterial gas embolism
- ✓ Assess fitness to fly in respiratory patients (hypoxic challenge)
- ✓ Describe respiratory hazards in miners

IN BRIEF Pressure changes follow Boyle's law (volume inversely proportional to pressure) and Henry's law (gas solubility proportional to partial pressure). Divers face barotrauma and decompression illness; air travellers face cabin hypoxia (cabin altitude up to 8000 ft — PIO2 equivalent to 15% O2 at sea level).

Diving medicine

- **Pulmonary barotrauma of ascent:** breath-holding during ascent → alveolar rupture → pneumothorax, pneumomediastinum, **arterial gas embolism (AGE)** (stroke-like within minutes of surfacing).
- **Decompression sickness:** nitrogen bubbles on rapid ascent — type I (joint pain "bends", skin, lymphatic), type II (neurological — spinal cord, "chokes" — cardiopulmonary).
- **Treatment:** 100% O2, supine, fluids, urgent **recompression in hyperbaric chamber** (US Navy Table 6).
- **Fitness to dive:** contraindications — bullae/cysts, previous spontaneous pneumothorax (unless bilateral surgical pleurectomy), uncontrolled/exercise-induced asthma (controlled asthma may dive with normal spirometry and no EIB), COPD, ILD, PAVM/ significant shunt, epilepsy.
- Immersion pulmonary oedema (SIPE) — swimmers/divers.

Aviation — fitness to fly

- Cabin pressurised to 6000–8000 ft: PaO2 falls to ~55–65 mmHg in healthy; patients with lung disease may desaturate severely.
- **Assessment (BTS 2022):** resting SpO2 at sea level, history of in-flight symptoms; **hypoxic challenge test** (breathe 15% O2 for 20 min) — PaO2 <6.6 kPa (50 mmHg) or SpO2 <85% → in-flight O2 (2 L/min).
- **Contraindications to flying:** current pneumothorax (fly ≥7 days after radiological resolution; 2 weeks after surgery), infectious TB (until non-infectious — 2 weeks of effective treatment and smear conversion), recent thoracic surgery, unstable disease, severe PH (O2), large bullae (relative); VTE prophylaxis on long flights for high-risk.
- **Aerospace medicine:** high-G effects (atelectasis with 100% O2 — "acceleration atelectasis"), hypoxia training, space flight (fluid shifts, microgravity effects on V/Q, decompression risk during EVA), pilot medical standards (Institute of Aerospace Medicine, Bengaluru).

Miners

- Coal (CWP, COPD), metal mining (silicosis, TB), uranium (radon — lung cancer), asbestos mining (banned in India), mica, iron ore; also heat stress, CO and methane explosions, diesel exhaust, noise, hypobaric conditions in high-altitude mines.

INVESTIGATIONS & TREATMENT AT A GLANCE

15.1 High-Altitude Illness — AMS, HACE, HAPE & Chronic Mountain Sickness

‹ Back to topic (p 250)
Next topic 15.2 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Lake Louise AMS score	< 3	≥ 3 with headache	AMS
SpO_2 at altitude	Expected for altitude	Markedly lower than peers	HAPE
CXR / lung USG	Clear	Patchy infiltrates, B-lines	HAPE
Neurological exam	Normal	Ataxia, altered consciousness	HACE
Haemoglobin	Normal for altitude	> 21 g/dL men, > 19 women	Chronic mountain sickness

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Descent	≥ 300–1000 m	All severe altitude illness	Definitive
Acetazolamide	Prevention 125 mg bd; treatment 250 mg bd	AMS	Sulfa caution
Dexamethasone	8 mg then 4 mg 6-hourly	HACE, moderate–severe AMS	—
Nifedipine SR	30 mg bd	HAPE	Hypotension
Portable hyperbaric bag (Gamow) / O_2	2 psi	When descent impossible	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Ascend no more than 300–500 m/day above 3000 m, with a rest day every 3–4 days.

- Mines Act 1952 — periodic medical examination (CXR per ILO, spirometry every 5 years), notification of pneumoconiosis; DGMS (Directorate General of Mines Safety).

★ CLINICAL PEARLS & EXAM FOCUS

▸ Previous spontaneous pneumothorax = no diving (unless bilateral surgical pleurectomy); flying is permitted 1 week after full resolution.

LANDMARK TRIALS & GUIDELINES

- Coker RK et al, BTS clinical statement on air travel 2022; British Thoracic Society guidelines on respiratory aspects of fitness for diving (Thorax 2003).

15.3 Extrapulmonary Manifestations & Pulmonary Complications of Systemic Disease

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B11 Extrapulmonary manifestations of pulmonary diseases
- ✓ Enumerate extrapulmonary manifestations of pulmonary diseases
- ✓ Describe pulmonary complications of renal disease, acute pancreatitis, liver disease and other systemic illnesses

IN BRIEF Lung disease has systemic effects (clubbing, cor pulmonale, paraneoplastic syndromes, cachexia, polycythaemia), and systemic diseases commonly involve the lungs — the interface examined in DNB Paper II.

Extrapulmonary manifestations of pulmonary diseases

Pulmonary disease	Extrapulmonary manifestations
COPD	Muscle wasting, osteoporosis, CVD, anaemia/polycythaemia, depression, cor pulmonale
Lung cancer	Paraneoplastic syndromes, HPOA, metastases, cachexia, VTE
TB	Erythema nodosum, phlyctenular conjunctivitis, Poncet's, amyloidosis, SIADH, adrenal insufficiency
Sarcoidosis	Uveitis, skin, cardiac, neuro, hypercalcaemia, arthritis, parotitis
Bronchiectasis/CF	Amyloidosis, clubbing, brain abscess, sinusitis, pancreatic insufficiency (CF), infertility
OSA	Hypertension, arrhythmia, diabetes, stroke, cognitive impairment
Chronic hypoxaemia	Polycythaemia, PH, fluid retention, neuropsychological deficits

Pulmonary complications of renal diseases

Category	Complications
Pulmonary–renal syndromes	Anti-GBM, AAV, SLE, cryoglobulinaemia
Fluid / uraemia	Pulmonary oedema ("uraemic lung" — bat-wing), uraemic pleuritis, fibrothorax
Dialysis-related	Hypoxaemia (complement activation — cuprophane membranes), hydrothorax (peritoneal dialysis), sleep apnoea, metastatic pulmonary calcification
Nephrotic syndrome	Transudative effusions, PE (renal vein thrombosis, AT-III loss), infections
Transplant	Opportunistic infections, drug toxicity (sirolimus pneumonitis), PTLD
Others	Urinothorax (obstructive uropathy), metabolic acidosis (Kussmaul), PH (AV fistula, volume), TB risk, calciphylaxis

INVESTIGATIONS & TREATMENT AT A GLANCE

15.2 Diving, Aviation & Aerospace Medicine; Miners

‹ Back to topic (p 252)
Next topic 15.3 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Pre-dive spirometry / CT	Normal	Bullae, obstruction	Contraindication to diving
Fitness to fly — SpO_2 at sea level	> 95%	< 92%	Needs in-flight O_2 / HCT
Hypoxic challenge test (FiO_2 15%)	PaO_2 > 55 mmHg	< 50 mmHg	In-flight O_2 2 L/min
Pneumothorax status	Resolved	Present	Do not fly (until 1 week after resolution)
Coal miners — CXR/ spirometry	Normal	CWP, obstruction	Surveillance

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Hyperbaric oxygen	Treatment tables (USN Table 6)	Decompression sickness, arterial gas embolism	—
In-flight oxygen	2 L/min	Hypoxic challenge positive	Arrange with airline
100% O_2	15 L/min	DCS before recompression	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Do not fly for 1 week after full resolution of pneumothorax (or 24 h after surgical cure — BTS).

Pulmonary complications of acute pancreatitis

Complication	Features
Hypoxaemia (early)	V/Q mismatch, atelectasis, diaphragmatic splinting
Pleural effusion (left > bilateral)	Exudate with high amylase
ARDS	Inflammatory mediators (phospholipase A2 — surfactant destruction), in severe pancreatitis — major cause of death
Pancreaticopleural fistula	Large recurrent amylase-rich effusion; MRCP; ERCP stent/octreotide/surgery
Atelectasis, pneumonia, mediastinal pseudocyst	—
Abdominal compartment syndrome	Raised airway pressures

Pulmonary manifestations of other systemic diseases

- **Liver:** hepatic hydrothorax, HPS, PoPH, α1-ATD, PBC (lymphocytic interstitial pneumonitis), IBD (bronchiectasis, OP, tracheal stenosis), **Budd–Chiari syndrome** (hepatic venous outflow obstruction — hepatomegaly, ascites, pain; myeloproliferative neoplasms, hypercoagulability; pleural effusion, PE risk; anticoagulation, angioplasty/TIPS, transplant).
- **Haematological:** sickle cell acute chest syndrome, PH in haemolytic anaemias, leukaemic infiltration, DAH in thrombocytopenia, extramedullary haematopoiesis (paravertebral masses).
- **Endocrine:** hypothyroidism (OSA, effusions, hypoventilation), acromegaly (OSA, large lungs), Cushing's (infections), diabetes (TB, mucor, melioidosis).
- **Neuromuscular, CTD, vasculitis** (see 7.6, 7.7, 11.9).
- **Erythema nodosum** — see 7.5.

★ **CLINICAL PEARLS & EXAM FOCUS**

▸ Recurrent left-sided effusion with very high amylase in an alcoholic = pancreaticopleural fistula.

LANDMARK TRIALS & GUIDELINES

- Zinner MJ et al, review; Lee YCG, pleural manifestations of systemic disease (Clin Chest Med).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Pulmonary complications in renal diseases. *[DNB Oct 2023 P2]*
2. Pulmonary complications of acute pancreatitis. *[DNB Jun 2021 P2]*
3. Budd–Chiari syndrome. *[VNSGU 2025 P2]*

15.4 Respiratory Diseases in Pregnancy

‹ NMC curriculum

Previous-year Qs ›

◉ **CBME COMPETENCIES — the postgraduate should be able to:**

✓ **NMC syllabus area:** I-B12 Physiological changes in pregnancy; Preventive aspects
✓ Manage asthma, TB, pneumonia, PE and OSA in pregnancy
✓ Describe preventive measures for respiratory disease in pregnancy

IN BRIEF Respiratory illness in pregnancy threatens two lives. Most respiratory drugs are safe; under-treatment (especially of asthma and TB) is more dangerous to the fetus than the medications. Imaging needed for diagnosis (CXR, CTPA) should not be withheld.

INVESTIGATIONS & TREATMENT AT A GLANCE

15.3 Extrapulmonary Manifestations & Pulmonary Complications of Systemic Disease

‹ Back to topic (p 253)
Next topic 15.4 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Pulmonary manifestations of RA / SLE / SSc	—	ILD, pleuritis, shrinking lung, PH	Systemic disease
Sickle cell — acute chest syndrome	—	New infiltrate + fever/chest pain/hypoxia	ACS
CKD — uraemic pleuritis	—	Exudative haemorrhagic effusion	—
IBD — airway disease	—	Bronchiectasis, bronchiolitis	—
Liver disease — HPS / PoPH	—	Orthodeoxia; mPAP > 20	See 9.3

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Acute chest syndrome	O_2, analgesia, ceftriaxone + azithromycin, exchange transfusion	Sickle cell disease	Incentive spirometry
Hydroxyurea	15–35 mg/kg/day	Prevention of ACS	Myelosuppression
Treat systemic disease	Immunosuppression per disease	CTD lung	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Shrinking-lung syndrome in SLE → diaphragmatic dysfunction with small lung volumes.

Common respiratory conditions in pregnancy

Condition	Key management points
Asthma (4–8%; 1/3 worsen)	Continue ICS/ICS-LABA (budesonide best data), SABA/ICS-formoterol reliever; OCS for exacerbations; avoid uncontrolled asthma (pre-eclampsia, preterm, low birth weight)
Tuberculosis	HRZE safe; avoid streptomycin; pyridoxine; DR-TB — see 4.8
Pneumonia / influenza / COVID-19	Higher severity in third trimester; oseltamivir early; β-lactams/macrolides safe; avoid FQ/tetracyclines; dexamethasone (or methylprednisolone) for COVID; vaccines
PE/VTE (leading cause of maternal death in high-income countries)	Clinical prediction (pregnancy-adapted YEARS), CUS legs first, CXR → V/Q or CTPA; LMWH (no DOAC/warfarin); thrombolysis only for life-threatening
OSA	CPAP safe; screening in obese/hypertensive
Amniotic fluid embolism	Sudden hypoxia, hypotension, DIC at delivery — supportive, A-OK protocol
Peripartum cardiomyopathy, tocolytic-induced and pre-eclampsia pulmonary oedema	Diuretics, O2, echo
Cystic fibrosis, ILD, PH	PH — pregnancy contraindicated (mortality up to 30%); counsel/contraception

Preventive measures

- Preconception counselling (optimise asthma, TB treatment, PH avoidance of pregnancy, CF/ILD assessment), smoking and second-hand smoke cessation, avoid biomass exposure.
- **Vaccination in pregnancy:** influenza (any trimester), Tdap (27–36 weeks), COVID-19, RSV (RSVpreF 32–36 weeks — infant protection); avoid live vaccines.
- TB screening (symptom) at antenatal visits, especially PLHIV, contacts; TPT where indicated.
- VTE risk assessment and thromboprophylaxis (RCOG), early mobilisation after caesarean.
- Air pollution avoidance, nutrition (iron, folate), weight management, OSA screening.

★ CLINICAL PEARLS & EXAM FOCUS

▸ The fetus suffers more from maternal hypoxaemia than from almost any respiratory drug or imaging — treat pregnant women with lung disease fully.

LANDMARK TRIALS & GUIDELINES

- GINA 2025 (asthma in pregnancy); RCOG Green-top guideline 37a/b on VTE in pregnancy (2015).

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Respiratory diseases in pregnancy and their preventive measures. *[DNB Jun 2021 P1]*

15.5 Vaccination in Pulmonary Diseases

‹ NMC curriculum

Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B14 Vaccination in pulmonary diseases
- ✓ Describe vaccines recommended for chronic respiratory disease — pneumococcal, influenza, RSV, COVID-19, pertussis, zoster
- ✓ Explain mechanisms and evidence for each, and critically review pneumococcal and influenza immunisation

IN BRIEF Vaccination prevents exacerbations, hospitalisation and death in COPD, asthma, bronchiectasis, ILD and the immunocompromised. Adult vaccination coverage in India is low; pulmonologists must actively recommend vaccines.

INVESTIGATIONS & TREATMENT AT A GLANCE

15.4 Respiratory Diseases in Pregnancy

‹ Back to topic (p 254)
Next topic 15.5 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
ABG in pregnancy	$PaCO_2$ 28–32; pH 7.40–7.47	$PaCO_2$ ≥ 35–40	Respiratory failure
CTPA / V/Q in pregnancy	—	PE	Fetal radiation low (< 1 mGy)
D-dimer (pregnancy)	Raised normally	—	Use YEARS / pregnancy-adapted algorithm
Spirometry / PEF	Normal	Reduced	Asthma control
Xpert (TB)	Not detected	Detected	TB in pregnancy

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
ICS / LABA / SABA	Usual doses	Asthma in pregnancy	Continue — uncontrolled asthma riskier
LMWH (enoxaparin)	1 mg/kg bd (treatment)	VTE in pregnancy	Warfarin and DOACs contraindicated
HRZE + pyridoxine	Standard	TB in pregnancy	Avoid aminoglycosides
Oseltamivir	75 mg bd	Influenza in pregnancy	Treat early
Prednisolone	40–50 mg × 5 days	Acute asthma	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Pregnant women with influenza or COVID are high-risk — treat early and vaccinate.

Vaccines in chronic respiratory disorders

Vaccine	Type / mechanism	Recommendation (adults with chronic lung disease)
Influenza (inactivated quadrivalent/trivalent; recombinant; high-dose)	Inactivated split/subunit — strain-specific antibodies (HA)	Annually, before season (India: April–May for southern, Sept–Oct north); reduces COPD exacerbations and mortality
Pneumococcal conjugate (PCV13, PCV15, PCV20, PCV21)	Polysaccharide conjugated to carrier protein (CRM197) — T-cell dependent, memory, mucosal immunity	PCV20 or PCV21 single dose; or PCV15 followed by PPSV23 ≥1 year later
Pneumococcal polysaccharide (PPSV23)	T-cell independent, no memory, poor in <2 years	After PCV15/13; broad serotype cover
RSV (RSVPreF3-AS01, RSVpreF, mRNA-1345)	Prefusion F protein	≥60–75 years and 50–59 with chronic lung disease (single dose)
COVID-19	mRNA, protein subunit, vector	Updated doses for high-risk
Tdap	Toxoid + acellular pertussis	Once in adulthood (COPD/asthma — pertussis causes exacerbations), each pregnancy
Recombinant zoster (RZV)	Glycoprotein E + AS01B	≥50 years, immunosuppressed ≥18 (2 doses)
BCG	Live attenuated	Neonates (see 4.15)

Trivalent (and quadrivalent) influenza vaccine

- Contains two influenza A (H1N1, H3N2) and one B (Victoria) strains; WHO recommends composition twice yearly (southern/northern hemisphere); B/Yamagata removed from 2024 (trivalent again).
- **Efficacy:** 40–60% in healthy adults (lower in elderly — use high-dose/adjuvanted); in COPD reduces exacerbations, hospitalisation and death (Cochrane); in asthma reduces severe outcomes; safe in pregnancy and with ICS.
- Adverse effects: local pain, low-grade fever; GBS ~1 per million; contraindication — previous severe allergy (egg allergy not a contraindication).

Critical review — pneumococcal and influenza immunisation

- **Pneumococcal:** PPSV23 prevents invasive pneumococcal disease (IPD) in adults (~50–70%) but evidence for non-bacteraemic pneumonia is inconsistent; conjugate vaccines (CAPiTA — PCV13 prevented 46% of vaccine-type CAP and 75% IPD in ≥65 years); serotype replacement and coverage gaps (Indian serotype data limited); higher-valency PCVs (PCV20/21) simplify schedules; cost–effectiveness and low uptake in India; limited RCT evidence specifically in COPD for hard outcomes.
- **Influenza:** good observational and RCT data for reduced exacerbations and hospitalisation in COPD; effectiveness varies with strain match and age (immunosenescence); tropical seasonality complicates timing in India; healthcare worker vaccination protects patients; uptake <2% in Indian adults.
- **Way forward:** adult immunisation schedules (Indian Chest Society/NCCP, API), opportunistic vaccination at clinic visits, addressing hesitancy and cost.

★ **CLINICAL PEARLS & EXAM FOCUS**

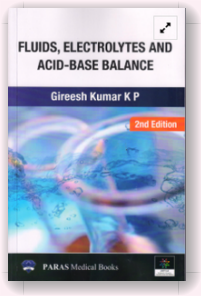
▸ "Influenza every year, one conjugate pneumococcal dose, RSV once after 60" — the three vaccines every COPD patient should get.

LANDMARK TRIALS & GUIDELINES

- Bonten MJM et al, CAPiTA NEJM 2015; Kopsaftis Z et al, Cochrane influenza vaccine in COPD 2018; ACIP adult immunization schedule 2025.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Pneumococcal vaccination. *[DNB Jun 2025 P2]*
2. Write a note on currently available vaccines in chronic respiratory disorders; their mechanisms and role. *[DNB May 2024 P2]*
3. Trivalent influenza vaccine. *[BFUHS 2020 P3]*
4. Prophylactic vaccination in respiratory disorders. *[BFUHS 2020 P4]*
5. Influenza vaccination. *[BFUHS 2019 P2]*
6. Critical review of pneumococcal and influenza immunization. *[BFUHS 2019 P4]*
7. Vaccination against pulmonary infections. *[Manipal 2014 P2]*



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15.6 Chronic Cough Syndromes (UACS, GERD) & Quality-of-Life Questionnaires

‹ NMC curriculum

Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** Misc. — Assessment of quality of life; I-B13 Cough
- ✓ Approach chronic cough including upper airway cough syndrome and GERD-related cough
- ✓ Describe disease-specific and generic quality-of-life questionnaires used in respiratory medicine

IN BRIEF Chronic cough (>8 weeks) in non-smokers with normal CXR is mostly due to asthma/eosinophilic bronchitis, upper airway cough syndrome and GERD — often multiple. Refractory cough reflects cough hypersensitivity and responds to neuromodulators.

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INVESTIGATIONS & TREATMENT AT A GLANCE

15.5 Vaccination in Pulmonary Diseases

‹ Back to topic (p 255)
Next topic 15.6 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Anti-HBs titre	> 10 mIU/mL	< 10	Revaccinate (HCW, dialysis)
Pneumococcal antibody levels	≥ 1.3 µg/mL	Low	Immunodeficiency assessment
CD4 (live vaccines)	> 200	< 200	Avoid live vaccines
Pregnancy status	—	Pregnant	Tdap, influenza, RSV (32–36 weeks) recommended; live vaccines avoided

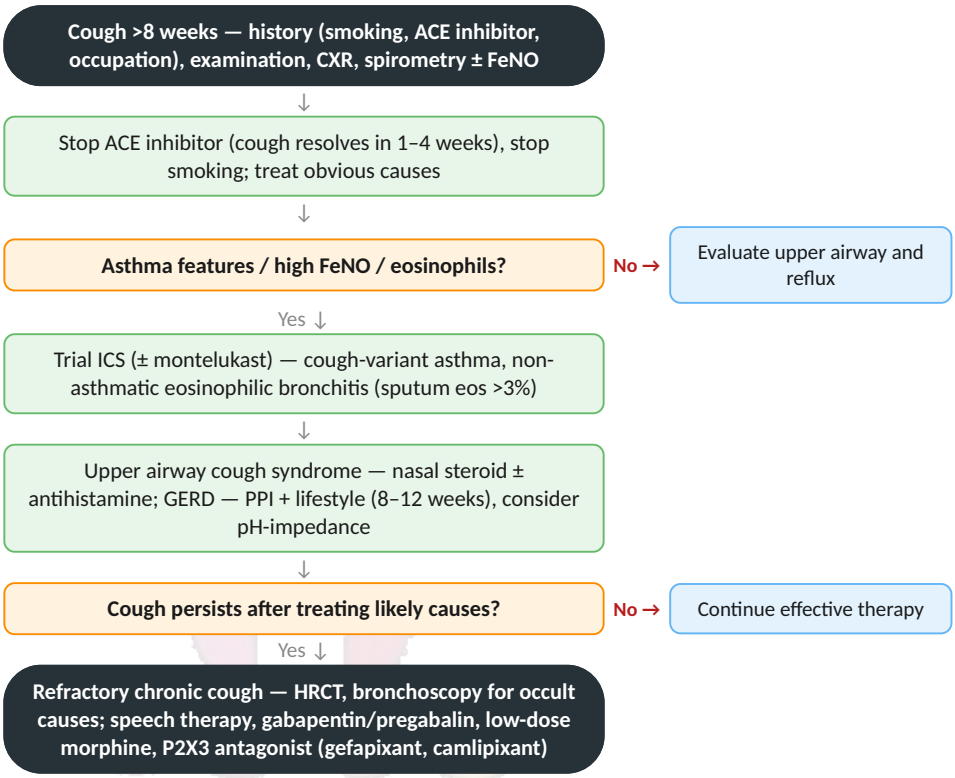
Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Influenza (quadrivalent)	0.5 mL IM annually	All chronic lung disease, ≥ 65, HCW, pregnancy	—
Pneumococcal PCV20	0.5 mL IM once	≥ 65 or chronic lung disease	—
RSV vaccine	Single 0.5 mL IM	≥ 60 y with chronic disease; pregnancy 32–36 wk (Abrysvo)	GBS (rare)
Tdap	0.5 mL IM	COPD/asthma adults not vaccinated in adolescence; each pregnancy	—
Recombinant zoster	2 doses 2–6 months apart	≥ 50 y, immunosuppressed ≥ 18 y	—
BCG	0.1 mL ID at birth	National schedule	Not in immunodeficiency

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Vaccinate before starting immunosuppression or biologics whenever possible.

Algorithm 15.1 · Approach to chronic cough (normal CXR)



Upper airway cough syndrome (UACS; postnasal drip)

- Cough from upper airway conditions — allergic/non-allergic rhinitis, chronic rhinosinusitis, post-viral; sensation of drainage, throat clearing, cobblestoning of posterior pharynx.
- **Treatment:** first-generation antihistamine–decongestant (non-allergic), intranasal steroids, antihistamines (allergic), saline irrigation, ipratropium nasal spray, treat sinusitis (antibiotics/surgery).

GERD and the lung

- **Mechanisms:** micro-aspiration of acid/pepsin/bile and oesophageal–tracheobronchial reflex (vagal).
- **Associations:** chronic cough, asthma (poorly controlled), IPF (micro-aspiration hypothesis), aspiration pneumonia, laryngopharyngeal reflux (hoarseness), bronchiectasis, lung transplant BOS.
- **Diagnosis:** clinical; 24-h pH-impedance (non-acid reflux), endoscopy for alarm features.
- **Treatment:** lifestyle (weight loss, head elevation, avoid late meals), PPI (benefit in cough mostly when heartburn/acid reflux present), prokinetics; fundoplication for selected (lung transplant candidates); routine anti-acid therapy not recommended in IPF.

Quality-of-life questionnaires

Instrument	Use
St George's Respiratory Questionnaire (SGRQ)	COPD/bronchiectasis; 0–100; MCID 4
Chronic Respiratory Questionnaire (CRQ)	Dyspnoea, fatigue, emotion, mastery; MCID 0.5
COPD Assessment Test (CAT)	8 items, 0–40; ≥10 symptomatic (GOLD); MCID 2
mMRC dyspnoea scale	0–4 grading of breathlessness
Asthma Control Test (ACT) / ACQ / AQLQ	Asthma control and QoL
Leicester Cough Questionnaire (LCQ), CQLQ	Chronic cough

Instrument	Use
K-BILD, ATAQ-IPF	ILD
QOL-B	Bronchiectasis
SF-36, EQ-5D, WHOQOL-BREF	Generic (health economics — QALYs)
Epworth (ESS), FOSQ	Sleep
emPHasis-10	Pulmonary hypertension

- Properties: validity, reliability, responsiveness, MCID; translated/validated Indian versions (Hindi, Malayalam SGRQ/CAT).

★ CLINICAL PEARLS & EXAM FOCUS

- The commonest causes of chronic cough with normal CXR in non-smokers: asthma/eosinophilic bronchitis, UACS and GERD — "the pathogenic triad".

LANDMARK TRIALS & GUIDELINES

- Morice AH et al, ERS chronic cough guideline Eur Respir J 2020; Irwin RS et al, CHEST cough guidelines 2006–2018.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. GERD. *[DNB Oct 2024 P2]*
2. Upper airway cough syndrome. *[DNB Oct 2024 P2]*

15.7 Obesity-Related Pulmonary Disorders

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** II-B4, II-B13 Obesity-related pulmonary disorders
- ✓ Describe the effects of obesity on respiratory mechanics and gas exchange
- ✓ List obesity-related pulmonary disorders and their management

IN BRIEF Obesity reduces FRC and ERV, increases work of breathing, and predisposes to OSA, OHS, asthma, PE, aspiration and perioperative complications. Weight loss improves nearly all obesity-related lung disorders.

Respiratory physiology in obesity

Parameter	Effect
FRC, ERV	Markedly reduced (exponential with BMI) — airway closure, basal atelectasis, V/Q mismatch, hypoxaemia (worse supine)
TLC, VC	Mildly reduced (severe obesity)
FEV1/FVC	Normal or high
Compliance	Reduced chest wall and lung compliance
Work of breathing / O2 cost	Increased; respiratory muscle inefficiency
Ventilatory drive	Increased (eucapnic) or blunted (OHS)
DLCO	Normal or increased (blood volume)

Obesity-related pulmonary disorders

Disorder	Notes
OSA, OHS	See 13.2–13.4; syndrome Z (OSA + metabolic syndrome)
Asthma (obese phenotype)	T2-low, poor ICS response — weight loss

INVESTIGATIONS & TREATMENT AT A GLANCE

15.6 Chronic Cough Syndromes (UACS, GERD) & Quality-of-Life Questionnaires

‹ Back to topic (p 257)
Next topic 15.7 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
CXR	Normal	Abnormal	Must be normal before diagnosing UACS/GERD/asthma cough
Spirometry / FeNO	Normal	Variability / FeNO > 25 ppb	Cough-variant asthma / NAEB
Sputum eosinophils	< 3%	≥ 3%	Non-asthmatic eosinophilic bronchitis
24-h pH-impedance	Acid exposure < 4%	> 6% with symptom association	GERD cough
Leicester Cough Questionnaire	21	Low	QoL
ACE-inhibitor use	No	Yes	Stop — cough resolves in 1–4 weeks

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Intranasal steroid + antihistamine	Fluticasone 100 µg/nostril daily + cetirizine 10 mg	UACS	—
ICS trial	Budesonide 400 µg bd × 4–8 weeks	Asthma/NAEB cough	—
PPI	Omeprazole 20–40 mg bd × 8 weeks (with lifestyle)	GERD cough with heartburn	Not for isolated cough without reflux
Gabapentin / pregabalin	Gabapentin up to 1800 mg/day	Refractory chronic cough	Sedation
Speech-language therapy	4 sessions	Refractory cough	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Chronic cough = > 8 weeks in adults; stop ACE inhibitors first.

Disorder	Notes
Dyspnoea / exercise limitation	Deconditioning, mechanics
PE/VTE	Prophylaxis dose adjustment
Aspiration, GERD	—
Perioperative complications	Difficult airway, rapid desaturation, atelectasis — pre-oxygenation with CPAP, reverse Trendelenburg, PEEP
Pulmonary hypertension	Group 2/3
Pneumonia severity (influenza, COVID-19)	Obesity paradox in some cohorts

Management principles

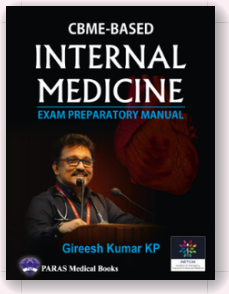
- Weight loss: diet, exercise, pharmacotherapy (GLP-1 RAs, tirzepatide), bariatric surgery (improves OSA, OHS, asthma, lung volumes); PAP for OSA/OHS; asthma optimisation; perioperative planning; VTE prophylaxis; rehabilitation.

★ CLINICAL PEARLS & EXAM FOCUS

▸ Supine position worsens hypoxaemia in obesity — intubate and ventilate in reverse Trendelenburg with PEEP.

LANDMARK TRIALS & GUIDELINES

- Dixon AE, Peters U. The effect of obesity on lung function, Expert Rev Respir Med 2018.



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INVESTIGATIONS & TREATMENT AT A GLANCE

15.7 Obesity-Related Pulmonary Disorders

‹ Back to topic (p 259)
Next topic 16.1 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
BMI	18.5–24.9 (Asian 18.5–22.9)	≥ 30 (Asian ≥ 25 obese)	Obesity
ERV / FRC	Normal	Reduced	Obesity effect
Awake $PaCO_2$ / HCO_3	< 45 / < 27	> 45 / ≥ 27	OHS
STOP-BANG / PSG	Low / AHI < 5	High / AHI ≥ 5	OSA
Waist circumference	< 90 cm men, < 80 cm women (Asian)	Higher	Metabolic risk

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Weight loss (diet + exercise)	5–10% target	All	—
GLP-1 RA (semaglutide)	0.25 → 2.4 mg SC weekly	Obesity	GI effects, pancreatitis
Tirzepatide	2.5 → 15 mg SC weekly	Obesity, OSA	—
Bariatric surgery	—	BMI ≥ 35 (≥ 32.5 Asian) with comorbidity	—
CPAP / NIV	Per titration	OSA / OHS	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Obesity reduces ERV first — FEV1/FVC is usually preserved.

SECTION 16

Surgical Aspects of Pulmonary Medicine

Paper II / III

Pre- and post-operative evaluation and management of thoracic and extrathoracic surgical patients; chest trauma and trauma-related lung dysfunction; lung transplantation; upper airway obstruction syndromes (NMC syllabus III, II-B4).

16.1 Pre-Operative Evaluation for Lung Resection & Extrathoracic Surgery 261
16.2 Chest Trauma & Trauma-Related Lung Dysfunction 263
16.3 Lung Transplantation 264
16.4 Upper Airway Obstruction & Approach to Stridor 265

16.1 Pre-Operative Evaluation for Lung Resection & Extrathoracic Surgery

‹ NMC curriculum
Previous-year Qs ›

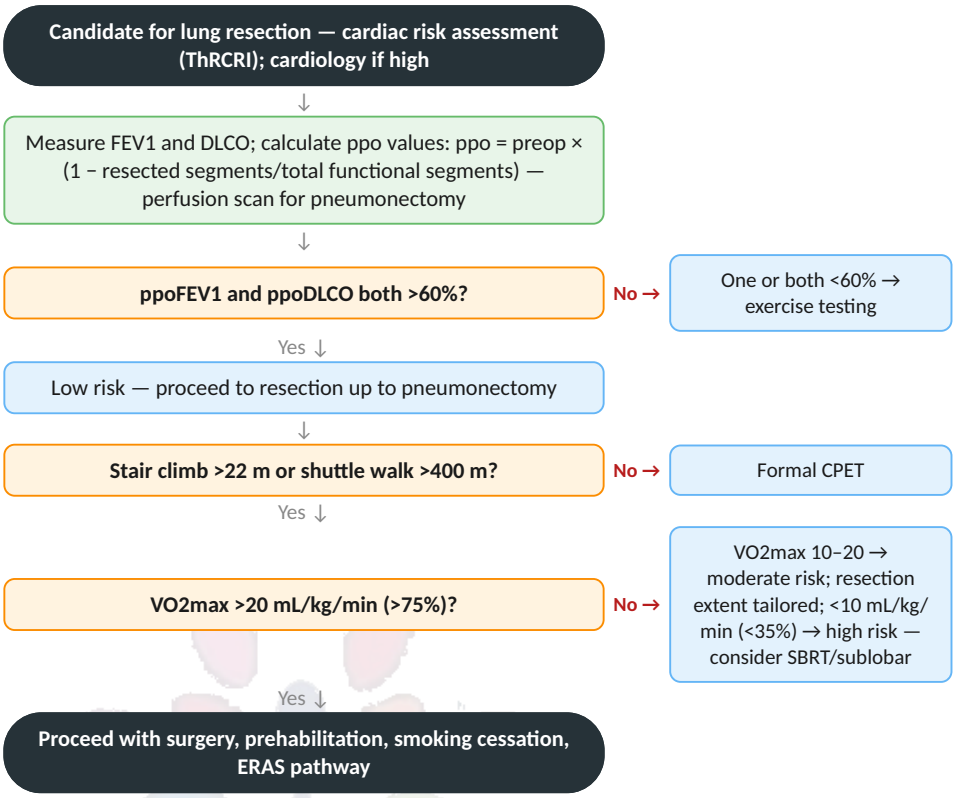
◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** III Pre- and post-operative evaluation
- ✓ Assess physiological fitness for lung resection (ERS/ESTS, ACCP algorithms)
- ✓ Perform pre-anaesthetic respiratory evaluation for extrathoracic surgery and reduce post-operative pulmonary complications

IN BRIEF Lung resection candidacy depends on cardiac risk, predicted post-operative (ppo) FEV1 and DLCO and, when borderline, exercise capacity (VO2max). For non-thoracic surgery, post-operative pulmonary complications (PPCs) are predicted by patient and procedure factors (ARISCAT) and reduced by optimisation and lung-protective anaesthesia.

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Algorithm 16.1 · Physiological evaluation before lung resection (ERS/ESTS/ACCP)



Pre-anaesthetic evaluation for extrathoracic surgeries

- **History:** dyspnoea, exercise tolerance (METs <4), cough/sputum, recent respiratory infection (defer elective surgery 2–4 weeks; 7 weeks after COVID-19), smoking, OSA symptoms (STOP-Bang), COPD/asthma control, steroid use.
- **Risk factors for PPCs:** age >60, ASA ≥II, COPD, HF, OSA, low SpO2, anaemia, albumin <3.5 g/dL, functional dependence; surgery — upper abdominal/thoracic/aortic/neuro/head-neck, emergency, duration >2–3 h, general anaesthesia. **ARISCAT score** (age, SpO2, respiratory infection in last month, anaemia, incision site, duration, emergency).
- **Investigations:** spirometry only if diagnosis uncertain or to optimise (not routine), ABG if hypercapnia suspected, CXR selectively, echo if PH suspected, PSG if high OSA risk.
- **Optimisation:** smoking cessation (ideally >8 weeks; any duration helpful), bronchodilators/ICS, short OCS course for uncontrolled asthma/COPD (hydrocortisone stress dose if chronic steroids), treat infection, inspiratory muscle training/prehabilitation, OSA — bring CPAP, avoid opioids, regional anaesthesia preferred.
- **Intra-operative:** lung-protective ventilation (VT 6–8 mL/kg, PEEP), avoid residual neuromuscular blockade (quantitative monitoring, sugammadex), short-acting agents, minimally invasive surgery.
- **Post-operative:** early mobilisation, incentive spirometry/deep breathing, adequate analgesia (epidural/regional), head-up, CPAP/HFNC in high risk, VTE prophylaxis.

★ CLINICAL PEARLS & EXAM FOCUS

▸ ppoFEV1 and ppoDLCO >60% — operate; <30% — high risk; in between, VO2max decides.

LANDMARK TRIALS & GUIDELINES

- Brunelli A et al, ERS/ESTS guidelines on fitness for radical therapy Eur Respir J 2009; ACCP guideline Chest 2013; Canet J et al, ARISCAT Anesthesiology 2010.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Pre-anaesthetic evaluation for extra-thoracic surgeries. *[DNB Oct 2023 P4]*

INVESTIGATIONS & TREATMENT AT A GLANCE

16.1 Pre-Operative Evaluation for Lung Resection & Extrathoracic Surgery

‹ Back to topic (p 261)
Next topic 16.2 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
FEV1 / DLCO (ppo)	ppo > 60%	< 60% → stair/shuttle; < 30% high risk	ERS/ESTS algorithm
Stair climbing	> 22 m	< 12 m	High risk
Shuttle walk	> 400 m	< 25 shuttles (250 m)	CPET needed
VO_2max	> 20 mL/kg/min	< 10 mL/kg/min	Prohibitive risk
ARISCAT score	< 26	≥ 45	Post-op pulmonary complication risk (extrathoracic surgery)
STOP-BANG	Low	High	Undiagnosed OSA

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Smoking cessation	≥ 4–8 weeks before surgery	All smokers	—
Prehabilitation	2–4 weeks exercise, inspiratory muscle training	High-risk resection	—
Optimise bronchodilators	Per COPD/asthma plan	Obstructive disease	—
Post-op lung expansion	Incentive spirometry, early mobilisation	All	—

★ PRESCRIBING & INTERPRETATION PEARLS

- ppoFEV1 = pre-op FEV1 × (1 − segments resected/19).

16.2 Chest Trauma & Trauma-Related Lung Dysfunction

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** III Chest trauma
- ✓ Identify and manage life-threatening thoracic injuries (ATLS primary survey)
- ✓ Manage pulmonary contusion, rib fractures, haemopneumothorax and tracheobronchial injury

IN BRIEF Chest trauma causes ~25% of trauma deaths; most injuries are managed with simple measures — airway, oxygen, analgesia, chest drain. Fewer than 10% of blunt and 15–30% of penetrating injuries need thoracotomy.

Life-threatening injuries (primary survey)

Injury	Recognition	Immediate action
Airway obstruction / tracheobronchial disruption	Stridor, haemoptysis, massive surgical emphysema, persistent air leak	Airway control, bronchoscopy, surgical repair
Tension pneumothorax	Distress, hypotension, absent breath sounds, tracheal deviation, distended neck veins	Needle/finger decompression (4th/5th ICS anterior axillary in adults), chest drain
Open pneumothorax (sucking wound)	Wound with air movement	Three-sided occlusive dressing, chest drain
Massive haemothorax	>1500 mL or ongoing >200 mL/h	Large-bore drain, volume, thoracotomy
Flail chest with contusion	Paradoxical segment, hypoxaemia	O2, analgesia, ventilation/fixation (see 11.9)
Cardiac tamponade	Beck's triad, FAST	Pericardiocentesis/thoracotomy

Potentially life-threatening (secondary survey)

Injury	Notes
Pulmonary contusion	Hypoxaemia evolves over 24–72 h; avoid fluid overload; NIV/MV; pneumonia, ARDS
Rib/sternal fractures	Elderly — high mortality; multimodal analgesia, regional blocks, SSRF
Blunt aortic injury	Widened mediastinum, CTA; endovascular repair
Diaphragmatic rupture	Left > right; bowel in chest; surgical repair
Oesophageal rupture	Pneumomediastinum, left effusion with food/low pH
Blunt cardiac injury	ECG, troponin, echo

Trauma-related lung dysfunction

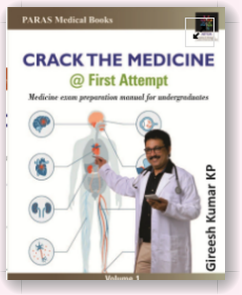
- ARDS (contusion, transfusion, fat embolism, aspiration, sepsis), VAP, atelectasis, retained haemothorax/empyema, post-traumatic pneumatocele, BPF, chylothorax, VTE/PE, chronic pain, restrictive impairment.
- **Chest ultrasound** in trauma (eFAST — see 14.3).

★ CLINICAL PEARLS & EXAM FOCUS

▸ Elderly with ≥3 rib fractures — admit, give regional analgesia early; mortality doubles with each extra fracture.

LANDMARK TRIALS & GUIDELINES

- ATLS 10th edition (American College of Surgeons 2018); Pieracci FM et al, SSRF consensus J Trauma 2020.



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16.3 Lung Transplantation

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** III Lung transplantation
- ✓ Select and refer candidates for lung transplantation (ISHLT 2021)
- ✓ Describe complications and outcomes after lung transplantation

IN BRIEF Lung transplantation improves survival and quality of life in selected patients with end-stage lung disease; median survival is ~6–7 years. Indian programmes (Chennai, Hyderabad, Kochi) have expanded since 2017. Early referral is key.

Indications and timing of referral (ISHLT 2021)

Disease	Refer when
COPD	BODE 5–6, FEV1 <25%, PaCO2 >50, PH, progressive despite optimal therapy
IPF / fibrotic ILD	At diagnosis of IPF/PPF; FVC <80% or DLCO <40%; any decline; O2 need
Cystic fibrosis	FEV1 <50% with rapid decline, FEV1 <30%, PH, frequent exacerbations
PAH	Intermediate/high risk despite therapy, WHO FC III–IV, progressive disease
Others	Bronchiectasis, LAM, PLCH, sarcoidosis, α1-ATD, post-COVID fibrosis, retransplant (BOS)

Selection of candidates

- **Absolute contraindications (selected):** recent malignancy (within 2–5 years, except low-risk skin cancer), untreatable major organ dysfunction (heart, liver, kidney — unless combined transplant), uncorrected CAD, active infection (uncontrolled), active TB, substance abuse/smoking within 6 months, non-adherence, BMI ≥35, severe frailty, lack of social support, psychiatric instability.
- **Relative:** age >65–70, BMI 30–34.9, osteoporosis, colonisation with resistant organisms (B. cenocepacia, M. abscessus), prior thoracic surgery, HIV (controlled acceptable), hepatitis.
- **Evaluation:** PFT, 6MWT, CPET, echo/RHC, coronary angiography, CT, V/Q, bone density, renal/liver function, infectious serologies, psychosocial assessment, nutrition, rehabilitation; Lung Allocation Score/composite allocation score.

Complications

- **Early:** primary graft dysfunction (grade 3 — ECMO), surgical (anastomotic dehiscence, stenosis), acute cellular rejection, antibody-mediated rejection, infections (bacterial, CMV, fungal — Aspergillus), haemorrhage, phrenic palsy.
- **Late: chronic lung allograft dysfunction (CLAD)** — BOS (obstructive; azithromycin, ECP, retransplant) and RAS (restrictive; worse), infections, malignancy (skin, PTLD), CKD (calcineurin inhibitors), diabetes, osteoporosis, GERD.
- **Immunosuppression:** tacrolimus + mycophenolate + prednisolone; prophylaxis — co-trimoxazole, valganciclovir, antifungal.

INVESTIGATIONS & TREATMENT AT A GLANCE

16.2 Chest Trauma & Trauma-Related Lung Dysfunction

‹ Back to topic (p 263)
Next topic 16.3 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
eFAST	Negative	Haemothorax / pneumothorax / pericardial fluid	Trauma
CXR / CT chest	—	Rib fractures, contusion, widened mediastinum (> 8 cm)	Injury pattern
ABG / P/F	> 300	< 300	Contusion / ARDS
Haemothorax drainage	—	> 1500 mL initial or > 200 mL/h × 2–4 h	Thoracotomy
Troponin / ECG	Normal	Raised / arrhythmia	Blunt cardiac injury

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Needle decompression	14 G, 4th–5th ICS mid-axillary (adults)	Tension pneumothorax	—
ICD	28–32 F	Haemothorax	—
Analgesia	Multimodal; epidural/serratus block	Rib fractures	Avoid hypoventilation
Tranexamic acid	1 g IV over 10 min, then 1 g over 8 h (within 3 h)	Haemorrhagic trauma (CRASH-2)	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Elderly patients with ≥ 3 rib fractures need HDU care — high pneumonia and mortality risk.

★ **CLINICAL PEARLS & EXAM FOCUS**

▸ Refer IPF patients for transplant at diagnosis — many deteriorate too fast to be listed later.

LANDMARK TRIALS & GUIDELINES

• Leard LE et al, ISHLT consensus on selection of lung transplant candidates, J Heart Lung Transplant 2021.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Selection of lung transplant candidates. *[DNB Dec 2022 P4]*

16.4 Upper Airway Obstruction & Approach to Stridor

‹ NMC curriculum
Previous-year Qs ›

◉ **CBME COMPETENCIES — the postgraduate should be able to:**

✓ **NMC syllabus area:** II-B4 Upper airways obstruction syndromes
✓ Localise upper airway obstruction clinically and physiologically
✓ Approach a patient with stridor and manage emergently

IN BRIEF Stridor is a high-pitched noise from turbulent flow in a narrowed large airway — inspiratory (extrathoracic: larynx, upper trachea), expiratory (intrathoracic trachea/main bronchi) or biphasic (fixed/glottic–subglottic). It signals at least 50–70% luminal narrowing and can precede complete obstruction.

Causes of upper airway obstruction in adults

Category	Causes
Infective	Epiglottitis/supraglottitis, Ludwig's angina, retropharyngeal abscess, diphtheria, laryngeal TB
Neoplastic	Laryngeal/tracheal cancer, thyroid malignancy, oesophageal cancer, mediastinal masses, adenoid cystic carcinoma
Post-intubation / tracheostomy	Subglottic/tracheal stenosis, granulation, tracheomalacia
Inflammatory	GPA (subglottic stenosis), relapsing polychondritis, amyloidosis, sarcoid, IgG4
Functional / neurological	Bilateral vocal cord palsy (thyroidectomy), VCD/ILO, laryngospasm, myasthenia
Other	Foreign body, angioedema (ACE inhibitor, C1-INH deficiency), anaphylaxis, trauma/burns, goitre, tracheobronchomalacia

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INVESTIGATIONS & TREATMENT AT A GLANCE

16.3 Lung Transplantation

‹ Back to topic (p 264)
Next topic 16.4 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Lung allocation / referral criteria	—	COPD BODE 7–10; IPF DLCO < 40%; CF FEV1 < 30%	Referral
Spirometry post-transplant	Stable FEV1	Decline ≥ 20% from baseline	CLAD/BOS
Tacrolimus trough	8–12 ng/mL early; 5–10 later	Toxic/sub-therapeutic	TDM
CMV PCR	Undetectable	Rising	CMV infection
Donor-specific antibodies	Negative	Positive	Antibody-mediated rejection

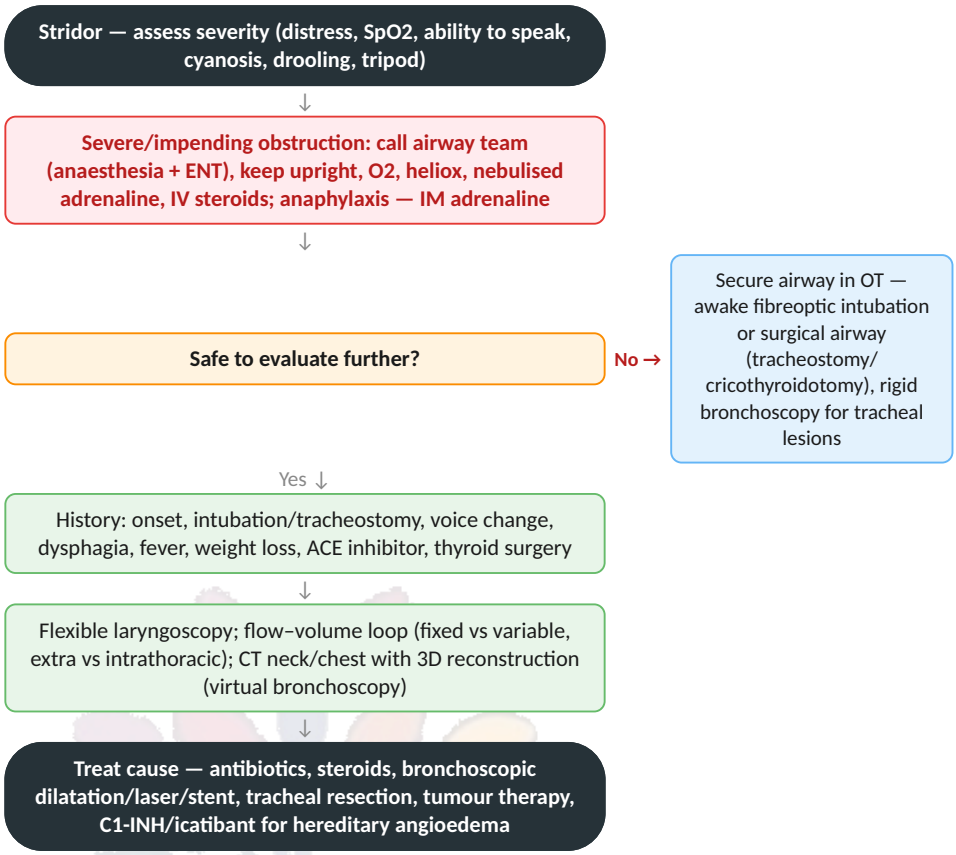
Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Tacrolimus	0.05–0.1 mg/kg/day in 2 doses	Maintenance immunosuppression	Nephrotoxicity, interactions (rifampicin ↓, azoles ↑)
Mycophenolate	1–1.5 g bd	Maintenance	Cytopenia
Prednisolone	0.5 mg/kg, taper to 5–10 mg	Maintenance	—
Valganciclovir	900 mg daily (prophylaxis 6–12 months)	CMV prophylaxis	Myelosuppression
Methylprednisolone	10–15 mg/kg IV × 3 days	Acute cellular rejection	—

★ PRESCRIBING & INTERPRETATION PEARLS

- Azithromycin may slow BOS progression (neutrophilic reversible allograft dysfunction).

Algorithm 16.2 · Approach to a patient with stridor



★ CLINICAL PEARLS & EXAM FOCUS

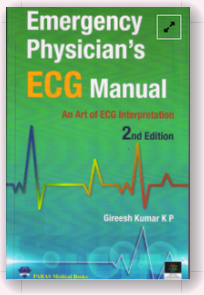
- Never send a stridorous patient for CT alone — airway first, imaging second.

LANDMARK TRIALS & GUIDELINES

- Aboussouan LS, Stoller JK. Diagnosis and management of upper airway obstruction, Clin Chest Med 1994; Frerk C et al, DAS 2015.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Approach to a patient with stridor. *[BFUHS 2022 P2]*



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INVESTIGATIONS & TREATMENT AT A GLANCE

16.4 Upper Airway Obstruction & Approach to Stridor

‹ Back to topic (p 265)
Next topic 17.1 ›

Most important investigations — reference and abnormal values

Investigation	Reference (normal) value	Abnormal value / diagnostic finding	Interpretation
Flow–volume loop	Normal	Flattened inspiratory (variable extrathoracic) / both limbs (fixed)	Upper airway obstruction
Laryngoscopy	Normal	Laryngeal oedema, mass, vocal-cord palsy	Cause
CT neck/chest	—	Tracheal stenosis, goitre, mass	Cause
Peak flow / Empey index	—	Empey > 10	UAO
SpO_2	Normal	Falls late	Do not rely on SpO_2 in stridor

Treatment — drug, dose and caution

Drug / intervention	Adult dose	Indication	Caution
Nebulised adrenaline	1 mg/mL × 5 mL (1:1000)	Stridor / croup / angio-oedema	Tachycardia
Dexamethasone	0.15–0.6 mg/kg (max 10–16 mg)	Croup, airway oedema	—
Heliox	70:30	Temporising	Limited FiO_2
Adrenaline IM	0.5 mg (1:1000)	Anaphylactic angio-oedema	—
Icatibant / C1-esterase inhibitor	Icatibant 30 mg SC	Hereditary angio-oedema (bradykinin)	Not responsive to adrenaline

★ PRESCRIBING & INTERPRETATION PEARLS

- Stridor at rest means airway narrowed by > 50% — secure the airway early in experienced hands.

SECTION 17

Research Methodology, Epidemiology & Biostatistics

Paper I / IV

Epidemiological terms, definitions and methods; study designs; research methodology, statistics, clinical trials, bias and ethics (NMC syllabus I-E1, I-E5, cognitive domain 9; thesis requirements).

17.1 Epidemiological Terms & Study Designs 267
17.2 Biostatistics — Central Tendency, Dispersion, Hypothesis Testing & Sampling 268
17.3 Clinical Trials, Bias, Blinding, Randomisation, Outcomes & Research Ethics 270

17.1 Epidemiological Terms & Study Designs

‹ NMC curriculum
Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-E1, I-E5 Epidemiological methods and study designs
- ✓ Define incidence, prevalence, rates and measures of association
- ✓ Classify study designs and state their advantages and disadvantages
- ✓ Distinguish systematic review from meta-analysis

IN BRIEF Study design is chosen by the research question. Descriptive studies generate hypotheses; analytical observational studies (case-control, cohort) test associations; randomised controlled trials test interventions; systematic reviews and meta-analyses synthesise evidence.

Key epidemiological measures

Term	Definition
Incidence	New cases / population at risk over time (rate or cumulative)
Prevalence	All existing cases at a point (point) or period / population — P ≈ I × D
Attack rate, case fatality rate	Outbreaks; deaths/cases
Relative risk (RR)	Incidence in exposed / unexposed (cohort, RCT)
Odds ratio (OR)	Odds of exposure in cases / controls (case-control); approximates RR for rare disease
Attributable risk	Incidence exposed − unexposed
NNT	1 / absolute risk reduction
Sensitivity, specificity, PPV, NPV, likelihood ratios	Diagnostic accuracy

Study designs — hierarchy and features

Design	Description	Advantages	Disadvantages
Case report / series	Description of patients	New diseases, rare events	No comparison

Design	Description	Advantages	Disadvantages
Cross-sectional	Exposure and outcome measured at one time	Quick, prevalence, multiple variables, cheap	No temporality; prevalence–incidence bias
Ecological	Group-level data	Hypothesis generation	Ecological fallacy
Case-control	Cases with disease vs controls without; look back at exposure	Rare diseases, long latency, quick, cheap, multiple exposures, small sample	Recall and selection bias, OR only (no incidence), temporality unclear, poor for rare exposures
Cohort (prospective/ retrospective)	Exposed vs unexposed followed for outcome	Temporality, incidence, RR, multiple outcomes, rare exposures	Costly, long, loss to follow-up, not for rare diseases
RCT	Random allocation to intervention vs control	Gold standard for causality; controls confounding	Cost, ethics, generalisability, time
Systematic review / meta-analysis	Synthesis of studies	Highest level of evidence	Heterogeneity, publication bias

Systematic review vs meta-analysis

- **Systematic review:** structured, reproducible search and appraisal of all studies on a focused question (PRISMA, PROSPERO registration), with risk-of-bias assessment; may be qualitative.
- **Meta-analysis:** statistical pooling of results from studies in a systematic review — fixed or random effects, forest plot, heterogeneity (I^2 >50% substantial), funnel plot (publication bias), sensitivity analysis.
- **Role:** resolve conflicting results, increase power and precision, inform guidelines (GRADE), identify research gaps; limitations — garbage in, garbage out; heterogeneity; publication bias.

★ **CLINICAL PEARLS & EXAM FOCUS**

▸ Case-control = start from outcome, look back at exposure, report OR; cohort = start from exposure, follow forward, report RR.

LANDMARK TRIALS & GUIDELINES

- Page MJ et al, PRISMA 2020 statement BMJ 2021.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Cross-sectional study. *[DNB Jun 2025 P1]*
2. Case-control study. *[DNB Jun 2025 P1]*
3. Types of studies in clinical research. *[DNB Oct 2024 P1]*
4. Advantages and disadvantages of case-control studies. *[DNB Dec 2022 P1]*
5. Outline the difference between a systematic review and meta-analysis; their role in clinical medicine. *[BFUHS 2022 P1]*

17.2 Biostatistics — Central Tendency, Dispersion, Hypothesis Testing & Sampling

‹ NMC curriculum
Previous-year Qs ›

◉ **CBME COMPETENCIES — the postgraduate should be able to:**

- ✓ **NMC syllabus area:** I-E5 Statistics
- ✓ Describe measures of central tendency and dispersion including standard deviation
- ✓ Explain null hypothesis, P value, statistical significance, odds ratio and confidence intervals
- ✓ Describe methods of sampling

IN BRIEF Statistics summarise data (descriptive) and draw inferences about populations from samples (inferential). A P value is the probability of obtaining results at least as extreme as those observed if the null hypothesis were true — it is not the probability that the hypothesis is true.

INVESTIGATIONS & TREATMENT AT A GLANCE

17.1 Epidemiological Terms & Study Designs

‹ Back to topic (p 267)
Next topic 17.2 ›

Key epidemiological measures — definitions and interpretation

Measure	Formula / definition	Example / typical value	Interpretation
Incidence rate	New cases ÷ person-time at risk	TB India 187 per lakh per year	Risk of new disease
Prevalence	Existing cases ÷ population	NTPS 2019–21: 312 per lakh	Burden
Relative risk (cohort)	Incidence exposed ÷ unexposed	RR 2 = double risk	Strength of association
Odds ratio (case-control)	(a×d) ÷ (b×c)	Approximates RR when disease rare	Association
Attributable risk	Incidence exposed − unexposed	—	Excess risk due to exposure
Number needed to treat	1 ÷ absolute risk reduction	NNT 20	Clinical usefulness

Study designs — choosing the right design

Study design	When to use	Strength	Limitation
Randomised controlled trial	Interventions	Highest for causation	Cost, generalisability
Cohort	Exposure → outcome; incidence	Temporal sequence	Loss to follow-up, time
Case-control	Rare diseases	Quick, cheap	Recall bias
Cross-sectional	Prevalence	Snapshot	No temporality
Systematic review / meta-analysis	Pooling evidence	Top of evidence pyramid	Heterogeneity, publication bias

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Bradford Hill criteria: strength, consistency, specificity, temporality, gradient, plausibility, coherence, experiment, analogy.

Measures of central tendency and dispersion

Measure	Description	Use
Mean	Sum/n	Normal distribution; affected by outliers
Median	Middle value	Skewed data
Mode	Most frequent	Categorical
Range	Max − min	Crude
Interquartile range	Q3 − Q1	With median, skewed data
Variance / standard deviation	SD = $\sqrt{[\Sigma(x - \text{mean})^2/(n-1)]}$	Spread around mean; normal curve — ±1 SD 68%, ±2 SD 95%, ±3 SD 99.7%
Standard error of mean	SD/√n	Precision of the sample mean; for 95% CI (mean ± 1.96 SE)
Coefficient of variation	SD/mean × 100	Compare variability across scales

Hypothesis testing

- **Null hypothesis (H0):** no difference/association; **alternative (H1):** difference exists.
- **Type I error (α):** rejecting a true H0 (false positive) — usually 0.05; **type II error (β):** failing to reject a false H0 (false negative) — usually 0.1–0.2; **power** = 1 − β (80–90%).
- **P value:** P <0.05 = statistically significant (conventional); does not measure effect size or clinical importance; multiple testing inflates type I error (Bonferroni correction).
- **Statistical vs clinical significance:** large samples make trivial differences significant; use confidence intervals and MCID.
- **Confidence interval (95%):** range expected to contain the true value in 95% of repeated samples; if CI of a ratio excludes 1 (or difference excludes 0) — significant.
- **Odds ratio:** (a/c)/(b/d) = ad/bc; OR 1 = no association, >1 risk factor, <1 protective; report with 95% CI; logistic regression gives adjusted OR.

Choosing a statistical test

Data	Two groups	>2 groups	Paired
Continuous, normal	Unpaired t-test	ANOVA	Paired t-test
Continuous, non-normal	Mann–Whitney U	Kruskal–Wallis	Wilcoxon signed-rank
Categorical	Chi-square / Fisher's exact	Chi-square	McNemar
Association	Pearson (normal) / Spearman correlation; regression	—	—
Survival	Kaplan–Meier, log-rank, Cox regression (HR)	—	—

Methods of sampling

Type	Method	Comment
Simple random	Every unit equal chance (random number tables/ software)	Needs sampling frame
Systematic	Every kth unit	Easy; periodicity bias
Stratified random	Random within strata (age, sex)	Representative subgroups
Cluster	Random selection of groups (villages)	Large surveys (WHO 30×7 immunisation); design effect
Multistage	Combination	National surveys (NFHS)
Non-probability	Convenience, purposive, quota, snowball	Qualitative/hard-to-reach; not generalisable

- **Sample size** depends on α, power, effect size, variability/prevalence, design (cluster effect), dropout.

★ CLINICAL PEARLS & EXAM FOCUS

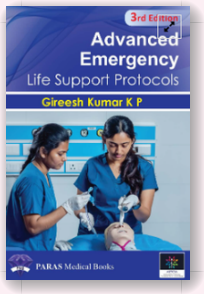
▸ "P <0.05" tells you the result is unlikely to be chance — the confidence interval tells you whether it matters.

LANDMARK TRIALS & GUIDELINES

- Wasserstein RL, Lazar NA. ASA statement on P values, Am Stat 2016.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Standard deviation. *[DNB Jun 2025 P1]*
2. Null hypothesis. *[DNB Oct 2024 P1]*
3. Measures of central tendency and dispersion in data analysis. *[DNB Oct 2024 P1]*
4. Odds ratio. *[DNB Dec 2021 P1]*
5. P value. *[DNB Dec 2021 P1]*
6. Methods of sampling. *[DNB Dec 2021 P1]*
7. The concept of statistical significance. *[DNB Jun 2021 P1]*



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17.3 Clinical Trials, Bias, Blinding, Randomisation, Outcomes & Research Ethics

‹ NMC curriculum | Previous-year Qs ›

◉ CBME COMPETENCIES — the postgraduate should be able to:

- ✓ **NMC syllabus area:** I-E5 Research methodology; Cognitive 9; Affective 3
- ✓ Describe phases of clinical trials and methods of randomisation and blinding
- ✓ Classify bias and describe its control; define outcome parameters in interventional studies
- ✓ Apply research ethics (ICMR 2017, CTRI) and teaching methodology

IN BRIEF Randomised controlled trials minimise selection bias and confounding; blinding minimises performance and detection bias. Ethical research in India follows ICMR National Ethical Guidelines (2017), New Drugs and Clinical Trials Rules (2019) and mandatory CTRI registration.

Phases of clinical trials

Phase	Participants	Purpose
Pre-clinical	Laboratory/animals	Pharmacology, toxicity
Phase 0	Few humans, micro-doses	Pharmacokinetics (exploratory)
Phase I	20–80 healthy volunteers (patients in oncology)	Safety, tolerability, PK, MTD
Phase II	100–300 patients	Efficacy signals, dose-finding, side effects
Phase III	1000s of patients, RCTs	Confirm efficacy vs standard, ADRs — basis for approval
Phase IV	Post-marketing, general population	Long-term safety, rare ADRs, pharmacovigilance, new indications

Randomisation methods

- **Simple** (coin toss, computer), **block** (balanced numbers in blocks of 4/6), **stratified** (by prognostic factors), **cluster** (groups), **minimisation**, **adaptive**; **allocation concealment** (sealed opaque envelopes, central web/telephone) prevents selection bias.

INVESTIGATIONS & TREATMENT AT A GLANCE

17.2 Biostatistics — Central Tendency, Dispersion, Hypothesis Testing & Sampling

‹ Back to topic (p 268)
Next topic 17.3 ›

Most important investigations — reference and abnormal values

Statistic	Definition / formula	Usual threshold	Interpretation
Sensitivity	TP ÷ (TP + FN)	High for rule-out (SnNout)	Proportion of diseased detected
Specificity	TN ÷ (TN + FP)	High for rule-in (SpPin)	Proportion of non-diseased negative
PPV / NPV	TP ÷ (TP+FP); TN ÷ (TN+FN)	Depend on prevalence	Post-test probability
Likelihood ratio	LR+ = Sn ÷ (1 − Sp); LR− = (1 − Sn) ÷ Sp	LR+ > 10, LR− < 0.1 strong	Changes probability
p-value	Probability of data if H_0 true	< 0.05	Statistical significance (not clinical)
95% confidence interval	Range of plausible values	Excludes 1 (ratio) or 0 (difference)	Precision
Power	1 − β	≥ 80%	Ability to detect true effect

Choosing the statistical test

Data type / question	Test	Assumption	Example
Two means (normal)	Student t-test (paired/unpaired)	Normality	FEV1 before/after drug
> 2 means	ANOVA	Normality, equal variance	FEV1 across 3 groups
Non-normal / ordinal	Mann–Whitney U, Wilcoxon, Kruskal–Wallis	—	CAT scores
Proportions	Chi-square / Fisher exact (expected < 5)	—	Cure rates
Correlation	Pearson (normal) / Spearman	—	FEV1 vs 6MWD
Survival	Kaplan–Meier, log-rank, Cox regression	Proportional hazards	Time to exacerbation

★ **PRESCRIBING & INTERPRETATION PEARLS**

- Sample size rises with smaller effect size, higher variability, higher power and lower alpha.

Blinding

- Single (participant), double (participant + investigator), triple (+ outcome assessor/analyst); placebo, double-dummy technique; open-label with blinded outcome assessment (PROBE).

Types of bias

Bias	Example	Control
Selection	Non-random allocation; volunteer bias	Randomisation, allocation concealment
Information / measurement	Recall bias (case-control), observer bias	Blinding, standardised tools
Confounding	Smoking confounds alcohol–lung cancer association	Randomisation, restriction, matching, stratification, multivariable analysis
Attrition	Loss to follow-up	ITT analysis, follow-up strategies
Publication / reporting	Positive results published	Trial registration, protocol publication
Lead-time and length-time	Screening studies	Mortality endpoints, RCT

Defining outcome parameters in interventional studies

- **Primary outcome:** single, pre-specified, clinically relevant, measurable — drives sample size (e.g., all-cause mortality, exacerbation rate, FEV1 change, time to culture conversion).
- **Secondary outcomes:** supportive (QoL, hospitalisation, adverse events); **safety outcomes; surrogate outcomes** (FEV1, CD4, biomarkers — must be validated); **composite outcomes** (combine events — interpret components); **patient-reported outcomes** (SGRQ, CAT); **MCID** defines clinical relevance.
- Good outcomes are SMART (specific, measurable, achievable, relevant, time-bound) and pre-registered; analysis — ITT vs per-protocol; superiority vs non-inferiority margin.

Research ethics and governance

- **Principles (Belmont/Helsinki):** respect for persons (informed consent — written, audio-video for vulnerable in regulatory trials), beneficence, justice; ICMR 2017 — 12 general principles; Institutional Ethics Committee approval (registered with CDSCO/DHR); CTRI registration before enrolment; compensation for trial-related injury (NDCT Rules 2019); data privacy (DPDP Act 2023); conflict of interest declaration; plagiarism checks; authorship (ICMJE).

Thesis and teaching

- NMC requires a thesis protocol in the first 6 months, data collection ≥1 year, submission 6 months before exams; one poster, one paper presentation and one publication/submission during PG training; maintain a log book.
- **Teaching methodology:** learning objectives (Bloom's taxonomy), small-group teaching, bedside teaching, flipped classroom, simulation, feedback (Pendleton), assessment (formative — mini-CEX, DOPS, CbD; summative), AETCOM.

★ CLINICAL PEARLS & EXAM FOCUS

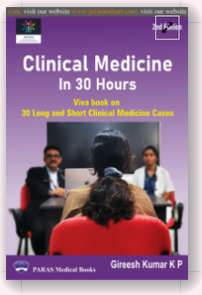
▸ Intention-to-treat preserves randomisation — analyse patients in the groups to which they were randomised.

LANDMARK TRIALS & GUIDELINES

- ICMR National Ethical Guidelines for Biomedical and Health Research Involving Human Participants (2017); CONSORT 2025 statement; New Drugs and Clinical Trials Rules, 2019.

PREVIOUS-YEAR QUESTIONS ON THIS TOPIC · full list ›

1. Phases in clinical trials. *[DNB Oct 2024 P1]*
2. Bias and blinding. *[DNB Dec 2022 P1]*
3. Randomisation methods. *[DNB Dec 2021 P1]*
4. Defining outcome parameters in interventional studies. *[DNB Jun 2021 P1]*



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INVESTIGATIONS & TREATMENT AT A GLANCE

17.3 Clinical Trials, Bias, Blinding, Randomisation, Outcomes & Research Ethics

‹ Back to topic (p 270)

Most important investigations — reference and abnormal values

Concept	Definition	Example	Control measure
Selection bias	Non-comparable groups	Healthier volunteers	Randomisation
Allocation concealment	Hiding next assignment	Sealed opaque envelopes, central randomisation	Prevents selection bias
Blinding	Participants/assessors unaware	Double-blind placebo	Reduces performance/detection bias
Intention-to-treat	Analyse as randomised	All randomised	Preserves randomisation
Confounding	Third variable related to exposure and outcome	Smoking in TB–diabetes studies	Randomisation, stratification, regression
Non-inferiority margin	Largest acceptable loss of efficacy	6–12% in TB trials	CI must lie within margin

Ethics and regulation of clinical research in India

Requirement (India)	Regulation / body	Key point	Reference
Ethics committee approval	ICMR National Ethical Guidelines 2017	Before enrolment	Registered EC (CDSCO)
Trial registration	CTRI (ctri.nic.in)	Prospective registration mandatory	Before first participant
Informed consent	New Drugs and Clinical Trials Rules 2019	Audio-visual consent for vulnerable groups (as applicable)	Language understood by participant
Serious adverse event reporting	NDCT Rules 2019	Report within 24 h; compensation provisions	Sponsor / investigator
Good Clinical Practice	ICH-GCP E6(R3)	Data integrity, monitoring	—

★ **PRESCRIBING & INTERPRETATION PEARLS**

- CONSORT (RCTs), STROBE (observational), STARD (diagnostic) and PRISMA (systematic reviews) guide reporting.

Previous-Year Question Bank

Questions from the DNB Respiratory Medicine (National Board of Examinations) theory papers of 2020–2025 and from MD Pulmonary / Respiratory Medicine university examinations (BFUHS, WBUHS, VNSGU, Manipal) and NEET-SS (DNB) 2024, arranged topic-wise. Each topic heading links to the page where it is answered; the source of every question is given in brackets (P1–P4 = theory paper).

PREVIOUS-YEAR QUESTION BANK · SECTION 1

Applied Anatomy, Embryology & Developmental Anomalies

1.1 Development of the Lung & Postnatal Growth

Theory › p.10

1. Development of the airways and blood vessels. *[DNB Jun 2025 P1]*
2. Post natal lung development. *[DNB Dec 2022 P1]*
3. Briefly describe the stages of development of lung and the airways. *[BFUHS 2022 P1]*

1.2 Airway Structure, Epithelium, Cilia & Alveolar Cells

Theory › p.12

1. Airway wall structure at the three principal levels. *[DNB Jun 2025 P1]*
2. Pneumocytes. *[DNB Jun 2025 P1]*
3. Ultrastructure of a cilium with diagram. *[DNB Jun 2025 P1]*
4. Ultrastructure of a cilium. *[DNB Dec 2022 P1]*
5. Epithelium lining of lower respiratory tract. *[DNB Dec 2022 P1]*
6. Type 2 alveolar epithelial cell structure and functions. *[DNB Dec 2021 P1]*
7. Epithelium of conducting airways. *[DNB Dec 2021 P1]*
8. Alveolar epithelium. *[DNB Dec 2021 P1]*
9. Bronchial mucus. *[DNB Jun 2025 P1]*

1.3 Bronchopulmonary Segments, Secondary Lobule & Fissures

Theory › p.13

1. Bronchopulmonary segments of human lung. *[DNB Jun 2025 P1]*
2. Discuss anatomy of bronchopulmonary segments with diagram and its clinical significance. *[WBUHS 2022 P1]*
3. Anatomy of secondary pulmonary lobule. *[DNB Oct 2024 P1]*
4. Secondary pulmonary lobule. *[DNB Oct 2023 P1]*
5. Secondary lobule. *[DNB Dec 2021 P1]*
6. Secondary lobule of lung. *[BFUHS 2020 P1]*
7. Azygos fissure. *[DNB Oct 2024 P1]*

1.4 Pulmonary & Bronchial Circulation and Lymphatics

Theory › p.15

1. Blood supply of lung. *[DNB Oct 2023 P1]*
2. The bronchial circulation. *[DNB Jun 2021 P1]*
3. Discuss normal pulmonary circulation. Describe various interventional radiological procedures used in respiratory medicine. *[VNSGU 2025 P4]*

1.5 Mediastinum & Pleura: Anatomy and Innervation

Theory › p.16

1. Anatomy of mediastinum. *[DNB Jun 2021 P1]*
2. Anatomy of the anterior mediastinum and investigations for its mass lesion. *[DNB Oct 2023 P1]*
3. Nerve supply of pleura. *[BFUHS 2020 P1]*

1.6 Diaphragm, Respiratory Muscles & Phrenic Nerve

Theory › p.17

1. Discuss structure, innervation and action of diaphragm. *[DNB Oct 2024 P1]*
2. Role of intercostal muscles. *[DNB Jun 2025 P1]*
3. Phrenic nerve. *[DNB Oct 2023 P1]*
4. Respiratory muscles. *[DNB Oct 2023 P1]*

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1.7 Developmental Anomalies I — Agenesis, Sequestration, CPAM, Bronchogenic Cyst

Theory › p.18

1. Definition and types of pulmonary sequestration. *[DNB Oct 2024 P1]*
2. Congenital agenesis of lung. *[DNB Oct 2023 P1]*
3. Stocker's classification of congenital pulmonary airway malformation (CPAM). *[DNB Oct 2023 P1]*
4. Bronchogenic cyst. *[DNB Oct 2023 P1]*
5. Tracheal bronchus. *[DNB Oct 2023 P1]*
6. Clinical manifestation of pulmonary sequestration. *[DNB Dec 2022 P2]*
7. Management of intrapulmonary sequestration. *[DNB Dec 2022 P2]*
8. Lung sequestration. *[DNB Jun 2021 P1]*
9. Enumerate developmental disorders of lung. Discuss diagnosis and treatment of lung sequestration. *[WBUHS 2022 P1]*
10. Describe in brief the various developmental anomalies of lung. *[VNSGU 2025 P1]*
11. Presentation and diagnosis of bronchogenic cyst. *[DNB Dec 2021 P2]*
12. Differential diagnosis and management of bronchogenic cysts. *[DNB Jun 2021 P2]*

1.8 Developmental Anomalies II — TEF, Scimitar, PAVM, Unilateral Hyperlucent Lung

Theory › p.20

1. Tracheo-esophageal fistula (TEF): types and clinical features. *[DNB Oct 2024 P1; DNB Oct 2023 P1]*
2. Scimitar syndrome. *[DNB Oct 2023 P1]*
3. Macleod syndrome. *[DNB Jun 2021 P1]*
4. Management of a radiologically unilateral hyperlucent lung. *[DNB Jun 2021 P2]*
5. Pulmonary arterio-venous malformation. *[DNB Dec 2025 P2]*

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PREVIOUS-YEAR QUESTION BANK · SECTION 2

Respiratory Physiology & Pulmonary Function

2.1 Lung Volumes & Capacities and Their Measurement

Theory › p.22

1. Measurement of residual volume (RV). *[DNB Jun 2025 P1]*
2. Static and dynamic lung volumes. *[DNB Oct 2023 P1]*
3. Functional residual capacity (FRC) and residual volume (RV). *[DNB Dec 2021 P1]*
4. Measurement of lung volume. *[DNB Dec 2021 P1]*
5. Closing volume. *[DNB Dec 2021 P1; VNSGU 2025 P1; Manipal 2014 P1]*
6. Vital capacity. *[Manipal 2014 P1]*
7. Minute ventilation. *[Manipal 2014 P1]*

2.2 Spirometry & Flow–Volume Loops

Theory › p.24

1. Slow vital capacity. *[DNB Oct 2024 P1]*
2. Interpretation of spirometry in obstructive and restrictive lung disease. *[DNB Oct 2024 P1]*
3. Office spirometry. *[DNB Oct 2023 P1]*
4. Flow volume loops in diagnosis of upper airway diseases. *[DNB Oct 2023 P1]*
5. Describe indications, contraindications, pre-procedure instructions and different manoeuvres for spirometry. *[DNB May 2024 P2]*
6. Draw and describe important features of flow volume loops. How are they useful in differentiating airway obstruction at different levels? *[WBUHS 2022 P1]*
7. Write a short note on indications and contraindications of spirometry. *[BFUHS 2020 P1]*
8. Characteristic patterns of disordered respiratory function. *[DNB Jun 2025 P4]*
9. Preserved Ratio Impaired Spirometry (PRISm). *[VNSGU 2025 P4]*
10. Mid expiratory flow rate. *[Manipal 2014 P1]*

2.3 Diffusion & DLCO

Theory › p.26

1. Factors affecting diffusion of gases from alveolus to pulmonary circulation. *[DNB Jun 2025 P1]*
2. Diffusing capacity for carbon monoxide (DLCO). *[DNB Jun 2025 P1]*
3. Diffusion capacity of carbon monoxide (DLCO). *[DNB Oct 2024 P1]*
4. Significance of changes in DLCO. *[DNB Dec 2021 P1]*
5. Methods to measure and factors influencing DLCO. *[DNB Oct 2023 P4]*
6. Define DLCO. How is it measured? What factors influence it? What is its clinical utility? *[WBUHS 2022 P1]*
7. Mechanism of gas exchange at the alveolar level. *[DNB Jun 2021 P1]*

2.4 Respiratory Mechanics — Compliance, Resistance, Surfactant & Atelectasis

Theory › p.28

1. What is lung compliance and methods of measuring lung compliance? *[DNB Oct 2023 P1]*
2. Measurement of compliance. *[DNB Dec 2021 P1]*
3. Assessment of lung compliance. *[DNB Jun 2021 P1]*
4. Airways resistance. *[DNB Dec 2021 P1]*
5. Measurement of airway resistance. *[BFUHS 2019 P2]*
6. Dynamic compression of the airways. *[DNB Dec 2021 P1]*
7. What is surfactant? Discuss its clinical utility. *[WBUHS 2022 P1]*
8. Write a short note on surfactant. *[BFUHS 2022 P1]*
9. Discuss the composition and function of pulmonary surfactant. *[BFUHS 2019 P1]*
10. Hysteresis. *[BFUHS 2022 P1]*
11. Types of acquired atelectasis. *[DNB Dec 2022 P1]*

2.5 Ventilation, Dead Space, V/Q Matching & Mechanisms of Hypoxaemia

Theory › p.29

1. Describe the causes of ventilation-perfusion (VA/Q) mismatch and its effects. *[DNB Jun 2025 P1]*
2. Alveolar air equation. *[DNB Jun 2025 P1]*
3. Measurement of physiological shunt. *[DNB Jun 2025 P1]*
4. Causes of hypoxaemia. *[DNB Jun 2025 P1]*

5. Alveolar–arterial gradient. *[DNB Dec 2022 P1]*
6. Dead space. *[DNB Dec 2022 P1]*
7. Pathophysiology of arterial hypoxemia. *[DNB Oct 2024 P3]*
8. Describe the pathophysiological mechanisms of hypoxia. *[BFUHS 2022 P1]*
9. Discuss the normal gas exchange mechanism. Discuss the aetiopathogenesis and management of CO2 narcosis in COPD patients. *[VNSGU 2025 P3]*

2.6 Gas Transport — Oxygen and Carbon Dioxide Dissociation Curves

Theory › p.32

1. Oxygen dissociation curve. *[DNB Oct 2024 P1]*
2. Oxygen haemoglobin dissociation curve. *[DNB Dec 2022 P1; BFUHS 2020 P1]*
3. Draw and describe oxyhemoglobin dissociation curve. Explain its clinical implications. *[WBUHS 2022 P1]*
4. Factors affecting carbon dioxide dissociation curve. *[DNB Dec 2022 P1]*
5. Carbon dioxide transport. *[DNB Dec 2021 P1]*
6. Blood gas transport in the lung parenchyma. *[DNB Oct 2023 P1]*
7. Define Bohr and Haldane effect. *[BFUHS 2019 P1]*

2.7 Control of Ventilation, Chemoreceptors & Reflexes

Theory › p.33

1. Discuss anatomy and physiology of the ventilatory control system. *[DNB Oct 2024 P1]*
2. Chemoreceptors. *[DNB Oct 2023 P1]*
3. Chemoreceptors for control of breathing. *[NEET-SS/DNB 2024]*
4. Chemical factors affecting respiration. *[DNB Dec 2022 P1]*
5. Cough reflex. *[NEET-SS/DNB 2024]*
6. Cough reflex pathways. *[Manipal 2014 P1]*
7. J receptors. *[Manipal 2014 P1]*
8. Discuss in short about Cheyne–Stokes respiration. *[BFUHS 2019 P1]*
9. Loop gain. *[DNB Dec 2025 P3]*
10. Discuss the causes, clinical features and management of hyperventilation syndromes. *[Manipal 2014 P2]*

2.8 Acid–Base Balance & ABG Interpretation; Electrolytes

Theory › p.35

1. Respiratory acidosis. *[DNB Oct 2024 P1; NEET-SS/DNB 2024; Manipal 2014 P1]*
2. Henderson–Hasselbalch equation. *[BFUHS 2020 P1]*
3. Metabolic alkalosis: causes and management. *[WBUHS 2022 P1]*
4. ABGs associated with COPD. *[DNB Oct 2023 P3]*
5. Hyponatraemia. *[Manipal 2014 P2]*
6. Management of hyponatraemia. *[BFUHS 2019 P1]*

2.9 Non-Respiratory Functions & Pulmonary Defence Mechanisms

Theory › p.37

1. Alveolar macrophages. *[DNB Jun 2025 P1]*
2. Role of macrophages in the lung. *[DNB Oct 2024 P1]*
3. Write a short note on macrophages in the lung. *[BFUHS 2019 P1]*
4. Mucociliary escalator. *[DNB Jun 2025 P1; WBUHS 2022 P1]*
5. Mucociliary clearance mechanism. *[DNB Oct 2024 P1]*
6. Non respiratory functions of the lungs. *[DNB Oct 2023 P1]*
7. Non respiratory functions of lungs. *[DNB Jun 2021 P1]*
8. Antiproteinases and lung. *[DNB Dec 2022 P1]*

2.10 Exercise Physiology, CPET & Six-Minute Walk Test

Theory › p.39

1. Describe in brief cardiopulmonary exercise testing. *[BFUHS 2022 P1]*
2. Six-minute walk test. *[DNB Jun 2021 P1; BFUHS 2022 P2]*

2.11 Bronchoprovocation, Oscillometry, Small Airway Tests, FeNO & Respiratory Muscle Tests

Theory › p.40

1. Broncho-provocation testing. *[DNB Dec 2021 P1]*
2. Impulse oscillometry. *[DNB Dec 2021 P1; DNB Dec 2025 P2; BFUHS 2022 P4]*
3. Evaluation of small airway function. *[DNB Oct 2023 P4]*

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4. Current status of exhaled nitric oxide measurement. *[DNB Dec 2022 P4]*
5. Applications of exhaled breath nitric oxide in respiratory conditions. *[DNB Dec 2025 P4]*
6. FENO. *[BFUHS 2022 P4]*
7. Clinical applications of exhaled breath analysis in respiratory medicine. *[BFUHS 2020 P4]*
8. Inflammatory markers in bronchial asthma. *[BFUHS 2019 P4]*

2.12 Physiological Changes in Pregnancy, Ageing & High Altitude

Theory › p.41

1. Respiratory physiologic changes of pregnancy. *[DNB Dec 2022 P1]*
2. Pulmonary physiological changes during pregnancy. *[BFUHS 2023 P2]*
3. Aging lung. *[DNB Dec 2022 P1]*
4. Effect of high altitude on rapid ascent. *[DNB Dec 2022 P1]*
5. Discuss in brief about high altitude physiology and related clinical disorders. *[BFUHS 2019 P1]*

2.13 Physiological Basis of Pulmonary Symptoms & Signs — Dyspnoea, Cough, Clubbing

Theory › p.43

1. Mechanism of dyspnoea. *[DNB Dec 2021 P1]*
2. Respiratory causes of breathlessness. *[DNB Jun 2025 P2]*
3. Clubbing and hypertrophic osteoarthropathy. *[DNB Jun 2025 P2]*

2.14 Aerosol Kinetics & Inhaled Drug Delivery Devices

Theory › p.44

1. Factors governing airway deposition of aerosols. *[WBUHS 2022 P1]*
2. Advantages and disadvantages of different modes of inhalation therapy. *[WBUHS 2022 P1]*
3. Enumerate various inhaled drug delivery devices and their principal mechanisms. *[DNB May 2024 P2]*
4. Write a note on the role of inhaled antibiotics in various respiratory disorders. *[DNB May 2024 P2]*

2.15 Pulmonary Circulation Physiology — West Zones, Starling Resistor, HPV & Fluids

Theory › p.45

1. The Starling resistor model of the pulmonary circulation. *[DNB Oct 2024 P1]*
2. Intravenous fluid therapy in critically ill patients. *[BFUHS 2020 P1]*

2.16 Principles of Electrocardiography & Cardiac Changes in Lung Disease

Theory › p.47

No question from this topic in the papers reviewed — study it from the chapter; it is a likely future question.

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PREVIOUS-YEAR QUESTION BANK · SECTION 3

Microbiology, Pathology, Immunology & Pharmacology

3.1 Mycobacteria — Biology, Staining, Culture & Phenotypic DST

Theory › p.49

1. Staining techniques for acid fast bacilli (AFB). *[DNB Oct 2023 P3]*
2. Liquid culture and DST for acid fast bacilli. *[WBUHS 2022 P3]*
3. Utility of immunohistochemistry in respiratory diseases. *[DNB Jun 2021 P1]*

3.2 Molecular Diagnosis of TB — CBNAAT, Truenat, LPA & Whole-Genome Sequencing

Theory › p.51

1. Rapid molecular drug resistance testing (genotypic tests) for tuberculosis. *[DNB Dec 2021 P3]*
2. Describe rapid molecular tests for diagnosis of drug resistant TB. *[WBUHS 2020 P3]*
3. Write a short note on genotypic tests for drug-resistant tuberculosis. *[BFUHS 2022 P1]*
4. Whole genome sequencing (WGS). *[WBUHS 2022 P3]*
5. Discuss molecular methods in diagnosis of tuberculosis and their clinical utility. *[WBUHS 2022 P3]*
6. Newer molecular methods for assessing drug resistance in tuberculosis. *[BFUHS 2019 P2]*
7. GeneXpert Ultra. *[BFUHS 2023 P2]*
8. Polymerase chain reaction. *[Manipal 2014 P3]*

3.3 Respiratory Viruses & Their Laboratory Diagnosis

Theory › p.52

No question from this topic in the papers reviewed — study it from the chapter; it is a likely future question.

3.4 Respiratory Fungi — Classification & Laboratory Diagnosis

Theory › p.53

No question from this topic in the papers reviewed — study it from the chapter; it is a likely future question.

3.5 Bacteria, Atypical Organisms, Anaerobes & Biomarkers of Infection

Theory › p.54

1. Biomarkers of infection. *[NEET-SS/DNB 2024]*
2. Procalcitonin. *[VNSGU 2025 P2]*

3.6 Pathology — Inflammation, Granuloma & Emphysema

Theory › p.56

1. Types of lung emphysema. *[DNB Dec 2022 P1]*
2. Granuloma formation in tuberculosis. *[DNB Dec 2022 P1]*

3.7 Hypersensitivity, Allergy Testing & Immunology of Lung Disease

Theory › p.57

1. What is the role of eosinophil in lung disease? *[BFUHS 2022 P1]*
2. Skin prick allergy testing. *[BFUHS 2020 P4]*
3. Hygiene hypothesis. *[Manipal 2014 P4]*

3.8 Pharmacology of First-Line Anti-TB Drugs & Drug-Induced Liver Injury

Theory › p.58

1. Isoniazid. *[Manipal 2014 P3]*
2. How will you manage a case of antitubercular drug induced jaundice? *[WBUHS 2022 P3]*

3.9 Second-Line & Newer Anti-TB Drugs — Bedaquiline, Delamanid, Pretomanid, Linezolid, Fluoroquinolones

Theory › p.60

1. Bedaquiline and delamanid. *[DNB Oct 2024 P3]*
2. Delamanid. *[WBUHS 2020 P3]*
3. Pretomanid. *[WBUHS 2022 P3; VNSGU 2025 P1]*
4. Fluoroquinolones in tuberculosis. *[Manipal 2014 P3]*
5. New anti-tuberculosis drugs. *[Manipal 2014 P3]*

3.10 Bronchodilators & Inhaled Corticosteroids

Theory › p.61

1. Role of ultra-LABA in obstructive airway diseases. *[DNB Oct 2024 P2]*
2. Pleiotropic actions of theophylline. *[DNB Oct 2024 P2]*

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3. Methylxanthines use in respiratory disease. *[BFUHS 2020 P1]*
4. Umeclidinium. *[VNSGU 2025 P4]*

3.11 Antimicrobials, Antifungals & Macrolides in Respiratory Medicine

Theory › p.63

1. Antifungal drugs in respiratory disorders. *[DNB Dec 2025 P1]*
2. Role of macrolides in respiratory medicine. *[BFUHS 2022 P2]*
3. Drugs for Pneumocystis pneumonia. *[DNB Oct 2023 P2]*

3.12 Antineoplastic, Immunosuppressant & Biologic Drugs; Pharmacovigilance

Theory › p.64

No question from this topic in the papers reviewed — study it from the chapter; it is a likely future question.

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PREVIOUS-YEAR QUESTION BANK · SECTION 4

Tuberculosis

4.1 Epidemiology of TB, End TB Strategy & the National TB Elimination Programme

Theory › p.66

1. Magnitude of TB burden in India. *[DNB Jun 2020 P3]*
2. The WHO End TB strategy, with targets for 2030 (SDG) and 2035. *[WBUHS 2022 P3]*
3. End TB strategy. *[BFUHS 2020 P4]*
4. Active case finding. *[WBUHS 2020 P3]*
5. Economic impact of tuberculosis. *[BFUHS 2020 P3]*
6. TB-free district. *[WBUHS 2022 P3]*

4.2 Pathogenesis — Primary & Post-Primary TB, Wallgren Timetable

Theory › p.68

1. Primary pulmonary tuberculosis. *[DNB Jun 2021 P3]*
2. Wallgren timetable of tuberculosis. *[DNB Jun 2021 P3]*
3. Describe pathophysiology of post primary tuberculosis. Discuss complications, sequelae and management of post primary tuberculosis. *[VNSGU 2025 P1]*
4. Radiological changes in primary and post primary tuberculosis. *[WBUHS 2022 P3]*

4.3 Diagnosis of Pulmonary TB — NTEP Algorithm, Radiology & Differential Diagnosis

Theory › p.70

No question from this topic in the papers reviewed — study it from the chapter; it is a likely future question.

4.4 Latent TB Infection — TST, IGRA & TB Preventive Treatment

Theory › p.71

1. Eligible population and strategy for tuberculosis preventive therapy (TPT). *[DNB Jun 2025 P3]*
2. Expanded population and strategy for tuberculosis preventive therapy (TPT). *[DNB Jun 2025 P3]*
3. Contraindications to tuberculosis preventive therapy. *[DNB Dec 2025 P3]*
4. What are the newer diagnostic methods of latent tuberculosis? *[DNB May 2024 P2]*
5. Compare tuberculin skin test (TST) and IGRA. *[WBUHS 2022 P3]*
6. What is Mantoux test and its role in primary pulmonary tuberculosis? *[DNB Oct 2023 P3]*
7. What are the causes of false positive tuberculin skin test? *[BFUHS 2022 P3]*
8. Criteria to start TPT (tuberculosis preventive treatment). *[BFUHS 2022 P3]*
9. Regimens used in TB preventive therapy. *[VNSGU 2025 P4]*
10. Isoniazid preventive therapy (IPT). *[DNB Jun 2020 P3]*
11. Latent TB infection: diagnosis and management. *[BFUHS 2019 P4]*
12. Mantoux test. *[Manipal 2014 P3]*

4.5 Treatment of Drug-Susceptible TB under NTEP

Theory › p.74

1. Factors promoting emergence of drug resistance during anti-tubercular therapy. *[BFUHS 2020 P3]*

4.6 Drug-Resistant TB — Definitions, Risk Factors, Surveillance & Diagnostic Algorithm

Theory › p.76

1. Integrated diagnostic and treatment algorithm for drug resistant tuberculosis. *[DNB Jun 2025 P3]*
2. Various diagnostic methods (phenotypic and genotypic) used in the diagnosis of drug resistant tuberculosis and their role in programmatic management of DR-TB. *[DNB Oct 2024 P3]*
3. Risk factors for drug resistant tuberculosis. *[DNB Jun 2020 P3]*
4. Salient findings of National Drug Resistance Survey (NDRS) 2014–16. *[DNB Jun 2020 P3]*
5. Drug resistant TB centres under RNTCP – roles and responsibilities. *[DNB Dec 2022 P3]*
6. Discuss in detail the drug resistance in tuberculosis. *[Manipal 2014 P3]*
7. XDR tuberculosis. *[Manipal 2014 P3]*

4.7 Treatment of MDR/RR-TB — BPaLM, Shorter & Longer Oral Regimens

Theory › p.78

1. Classification of drugs for drug resistant tuberculosis under NTEP. *[DNB Dec 2025 P1]*
2. Grouping of anti-TB drugs for designing longer all oral MDR-TB regimen. *[DNB Dec 2021 P3]*
3. Shorter oral regimen and its exclusion criteria for MDR/RR-TB patients. *[DNB Dec 2021 P3]*

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4. Shorter MDR-TB regimen and its exclusion criteria as per NTEP of India. *[DNB Dec 2022 P4]*
5. Describe classification of second line anti-TB drugs for management of MDR/RR-TB as per WHO guidelines. *[WBUHS 2020 P3]*
6. BPaL regimen. *[BFUHS 2022 P4]*
7. Newer regimens for MDR and XDR tuberculosis including injection-free regimens. *[BFUHS 2019 P4]*

4.8 TB in Special Situations — Pregnancy, Renal Failure, Liver Disease, Diabetes & the Elderly

Theory › p.79

1. Management of tuberculosis in pregnant and lactating women. *[DNB Dec 2021 P3]*
2. Management of tuberculosis in pregnancy. *[WBUHS 2020 P3; WBUHS 2022 P3]*
3. Management of MDR-TB in pregnancy. *[DNB Jun 2020 P3]*
4. Management of TB in patients with liver disorders. *[DNB Jun 2020 P3]*
5. Management of TB in patients with renal failure and severe renal insufficiency. *[DNB Jun 2020 P3]*
6. Management of TB in patients with severe renal insufficiency. *[DNB Dec 2022 P3]*
7. Describe management of TB in renal failure. *[WBUHS 2020 P3]*
8. A 16-week pregnant woman with persistent cough: Xpert MTB trace positive, rifampicin resistance indeterminate. Discuss further management. *[BFUHS 2022 P2]*

4.9 HIV–TB Co-infection, ART Interactions & IRIS

Theory › p.81

1. HIV co-infection among TB patients – magnitude and management. *[DNB Dec 2022 P3]*
2. HIV co-infection among TB patients. *[DNB Jun 2020 P3]*
3. Anti-retroviral therapy in HIV infected TB patients. *[DNB Jun 2020 P3]*
4. Initiating ART in patients with DR-TB. *[DNB Dec 2021 P3]*
5. Immune reconstitution inflammatory syndrome. *[DNB Oct 2024 P3]*
6. Paradoxical reaction. *[DNB Jun 2025 P3]*
7. Discuss the radiological features of HIV–TB co-infection. *[WBUHS 2020 P3]*
8. Discuss TB in HIV patient with respect to epidemiology, diagnosis and treatment. *[WBUHS 2022 P3]*
9. Managing HIV/TB co-infection. *[BFUHS 2019 P3]*

4.10 Paediatric TB & Childhood Contact Management

Theory › p.82

1. Consideration of newer drugs for treatment of tuberculosis in children. *[DNB Dec 2021 P3]*
2. Evaluation and management of children in contact with tuberculosis patient. *[DNB Jun 2020 P3]*
3. Discuss diagnosis and management of paediatric TB under RNTCP. *[WBUHS 2020 P3]*
4. Congenital tuberculosis. *[Manipal 2014 P3]*

4.11 Miliary TB & TB Meningitis

Theory › p.84

1. Management of miliary tuberculosis patient. *[DNB Dec 2021 P3]*
2. Management of a patient with altered sensorium with miliary shadow on chest X-ray. *[DNB Oct 2024 P3]*
3. Discuss miliary TB, its etiopathogenesis, diagnosis, complication and management. *[VNSGU 2025 P1]*
4. Sequelae of tuberculous meningitis. *[DNB Oct 2023 P3]*

4.12 Pleural & Pericardial Tuberculosis

Theory › p.86

1. Diagnosis of adult pleural tuberculosis. *[DNB Jun 2025 P3]*
2. Adenosine deaminase – diagnostic utility. *[DNB Jun 2021 P3]*
3. Discuss pathogenesis, clinical features, diagnosis and management of tuberculosis of pericardium. *[WBUHS 2022 P3]*

4.13 Extrapulmonary TB — Lymph Node, Abdominal, Spinal, Genitourinary, Endobronchial & Cutaneous

Theory › p.87

1. Differential diagnosis of lymphadenopathy. *[DNB Jun 2025 P3]*
2. Management of recurrent cervical lymphadenitis. *[DNB Oct 2024 P3]*
3. Tuberculous lymphadenitis. *[DNB Oct 2023 P3]*
4. Clinical manifestation and diagnosis of abdominal tuberculosis. *[DNB Oct 2024 P3]*
5. Investigations of ileocaecal tuberculosis. *[DNB Oct 2023 P3]*
6. Gastrointestinal tuberculosis. *[Manipal 2014 P3]*
7. Endocrine and metabolic manifestations of tuberculosis. *[DNB Oct 2023 P3]*
8. Endobronchial TB. *[DNB Oct 2023 P3]*

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9. Tuberculous endobronchitis. *[Manipal 2014 P3]*
10. Tuberculids. *[DNB Dec 2025 P3]*
11. Diagnosis and management of Pott's spine. *[DNB Dec 2025 P3]*
12. Management of a case of genito-urinary tuberculosis. *[DNB Jun 2020 P3]*
13. What are the rheumatic syndromes resulting from tuberculosis? *[BFUHS 2022 P3]*

4.14 Complications & Post-TB Lung Disease; Corticosteroids and Surgery in TB

Theory › p.89

1. Complications of pulmonary TB. *[DNB Oct 2023 P3]*
2. Post-TB sequelae. *[WBUHS 2020 P3]*
3. Discuss role of surgery in pleuro-pulmonary tuberculosis. *[WBUHS 2020 P3]*
4. Role of corticosteroids in management of tuberculosis. *[DNB Dec 2021 P3]*
5. Role of steroids in tuberculosis. *[DNB Jun 2021 P3]*
6. Role of steroids in the management of TB. *[DNB Jun 2020 P3]*
7. Role of corticosteroids in management of various extrapulmonary tuberculosis. *[DNB Oct 2024 P3]*
8. Role of corticosteroid in management of tuberculosis. *[WBUHS 2022 P3]*

4.15 Patient Support, Infection Control, Stigma, BCG & TB Vaccines

Theory › p.91

1. Ni-kshay Poshan Yojana (NPY). *[DNB Jun 2025 P3]*
2. Ni-kshay Mitra. *[DNB Jun 2025 P3]*
3. Discuss role of nutrition in tuberculosis and Nikshay Poshan Yojana. *[VNSGU 2025 P1]*
4. What is airborne infection control and describe AIIR? *[BFUHS 2022 P3]*
5. Airborne infection control measures in TB. *[BFUHS 2019 P3]*
6. Stigma related to pulmonary tuberculosis. *[BFUHS 2020 P3]*
7. Cough etiquette for reducing transmission of respiratory pathogens. *[BFUHS 2020 P3]*
8. BCG vaccination. *[DNB Jun 2021 P1]*
9. BCG vaccine. *[BFUHS 2020 P1]*
10. Discuss newer vaccines against TB. *[WBUHS 2020 P3]*
11. Prevention of tuberculosis. *[Manipal 2014 P3]*

4.16 Non-Tuberculous Mycobacteria

Theory › p.92

1. Criteria of diagnosis of NTM lung disease. *[DNB Jun 2025 P3]*
2. Treatment of pulmonary NTM disease. *[DNB Jun 2025 P3]*
3. Diagnosis criteria of non-tubercular mycobacterial pulmonary disease (NTM-PD). *[DNB Oct 2024 P3]*
4. Describe the treatment guidelines of Mycobacterium avium complex in pulmonary diseases. *[DNB May 2024 P2]*
5. Describe non-tuberculous mycobacteria, its diagnosis and management. *[WBUHS 2020 P3]*
6. Classification of non-tubercular mycobacteria. *[BFUHS 2020 P1]*
7. Swimming pool granuloma. *[VNSGU 2025 P1]*

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PREVIOUS-YEAR QUESTION BANK · SECTION 5

Non-Tuberculous Infections of the Lung

5.1 Approach to Pulmonary Infection, Antibiotic Principles & Bronchoalveolar Lavage

Theory › p.95

1. Discuss the diagnostic utility of bronchoalveolar lavage. *[BFUHS 2022 P2]*

5.2 Community-Acquired Pneumonia

Theory › p.97

1. Aetiology and severity assessment of community acquired pneumonia. *[DNB Dec 2022 P2]*
2. Management of CAP in out-patient setting. *[DNB Dec 2022 P2]*
3. Empiric antibiotic selection in adult outpatients with community-acquired pneumonia. *[DNB Jun 2025 P2]*
4. Corticosteroids in severe pneumonia. *[DNB Oct 2023 P3]*
5. Macrolides in severe community-acquired pneumonia and sepsis. *[DNB Oct 2023 P3]*

5.3 Hospital-Acquired & Ventilator-Associated Pneumonia

Theory › p.99

1. Predisposing factors of nosocomial pneumonia. *[DNB Jun 2020 P3]*
2. Ventilator associated pneumonia and its management. *[BFUHS 2020 P2; BFUHS 2022 P4]*

5.4 Atypical & Tropical Pneumonias — Mycoplasma, Legionella, Melioidosis, Scrub Typhus, Leptospirosis

Theory › p.100

1. Mycoplasma pneumonia. *[DNB Jun 2021 P2]*
2. Melioidosis. *[Manipal 2014 P2]*

5.5 Viral Pneumonias — Influenza, COVID-19, RSV — and Vaccines

Theory › p.101

1. Vaccination against COVID-19: types of vaccines and side effects. *[DNB Jun 2021 P1]*
2. Vaccine hesitancy. *[DNB Jun 2021 P1]*
3. Caring for the critically ill patient with COVID-19. *[DNB Oct 2023 P4]*
4. Respiratory syncytial virus. *[Manipal 2014 P2]*

5.6 Aspiration Pneumonia, Lung Abscess & Lipoid Pneumonia

Theory › p.103

1. Diagnosis and management of aspiration pneumonia. *[DNB Dec 2021 P2]*
2. Differential diagnosis of lung abscess. *[DNB Jun 2025 P2]*
3. Discuss the microbiology, diagnosis and treatment of lung abscess. *[Manipal 2014 P2]*
4. Lipoid pneumonia. *[Manipal 2014 P2]*

5.7 Bronchiectasis, Cystic Fibrosis & Primary Ciliary Dyskinesia

Theory › p.104

1. Management of bronchiectasis. *[DNB Dec 2021 P2]*
2. Reversible bronchiectasis. *[DNB Oct 2023 P2]*
3. Management of a case of cystic fibrosis of lung. *[DNB Oct 2023 P2]*
4. Describe the approach of evaluating and managing a young male with bilateral bronchiectasis. *[DNB May 2024 P2]*
5. Complications of bronchiectasis. *[DNB Dec 2025 P3]*
6. Ciliary dyskinesia. *[VNSGU 2025 P2]*

5.8 Pulmonary Aspergillosis Spectrum & Mucormycosis

Theory › p.106

1. Non-surgical treatments of mycetoma lung. *[DNB Jun 2025 P2]*
2. COVID-19 associated fungal infections. *[BFUHS 2022 P4]*
3. Fungal sinusitis. *[Manipal 2014 P2]*

5.9 Pneumocystis, Cryptococcosis, Histoplasmosis & Other Fungal Pneumonias

Theory › p.108

1. Diagnosis and management of fungal pneumonias. *[BFUHS 2019 P4]*

5.10 Parasitic Lung Disease — Hydatid, Amoebiasis, Paragonimiasis & Zoonoses

Theory › p.109

1. Hydatid cyst. *[WBUHS 2020 P2]*

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2. Radiological findings in hydatid disease of lung. *[BFUHS 2020 P2]*
3. Hydatid disease of lung. *[BFUHS 2019 P3]*
4. Thoracic amoebiasis. *[Manipal 2014 P2]*

5.11 Pulmonary Infections in the Immunocompromised Host (HIV, Transplant, Immunosuppressants)

Theory › p.110

1. HIV and pulmonary infections. *[DNB Jun 2021 P2]*
2. A 40-year-old lady who underwent allogeneic stem cell transplantation five months ago presents with cough and dyspnoea of three weeks. How will you manage her? *[BFUHS 2023 P2]*
3. Prevention of respiratory infections in patients receiving immunosuppressants. *[BFUHS 2020 P3]*

5.12 Recurrent & Non-Resolving Pneumonia; MRSA Pneumonia

Theory › p.113

1. Recurrent pneumonia. *[DNB Oct 2023 P2]*
2. Methicillin resistant staphylococcal pneumonia. *[DNB Oct 2023 P2]*

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PREVIOUS-YEAR QUESTION BANK · SECTION 6

Obstructive Airway Diseases

6.1 Asthma — Pathogenesis, Phenotypes & Diagnosis

Theory › p.115

1. Obesity as a risk factor related to asthma. *[DNB Dec 2022 P1]*

6.2 Management of Chronic Asthma — GINA Tracks, MART & Control Assessment

Theory › p.117

1. Classification of control of asthma. *[DNB Dec 2022 P2]*
2. Why inhaled corticosteroids are recommended to be commenced from the time of diagnosis of bronchial asthma? *[DNB Jun 2025 P2]*

6.3 Acute Severe Asthma — Assessment, ICU Management & Mechanical Ventilation

Theory › p.118

1. Management of acute severe asthma in ICU. *[DNB Dec 2022 P2]*
2. Severity assessment of an acute exacerbation of asthma. *[DNB Dec 2021 P3]*
3. Role of mechanical ventilation during an acute asthma exacerbation. *[DNB Dec 2021 P3]*

6.4 Difficult-to-Treat & Severe Asthma — Phenotyping and Biologics

Theory › p.120

1. The importance of phenotyping in the management of chronic severe asthma. *[DNB Oct 2023 P2]*
2. Difficult-to-treat asthma. *[BFUHS 2020 P2]*
3. Use of anti-interleukin-5 agents for treatment of severe asthma. *[BFUHS 2023 P2]*
4. Monoclonal antibody in bronchial asthma. *[Manipal 2014 P4]*

6.5 Allergic Bronchopulmonary Aspergillosis

Theory › p.122

1. Criteria for diagnosis and clinical staging of ABPA. *[DNB Oct 2024 P2]*
2. Allergic bronchopulmonary aspergillosis. *[DNB Oct 2023 P2]*
3. Diagnosis and management of allergic bronchopulmonary aspergillosis. *[DNB Jun 2021 P2]*
4. Describe diagnosis and management of allergic bronchopulmonary aspergillosis. *[WBUHS 2020 P2]*
5. Enumerate eosinophilic lung diseases. Discuss pathogenesis, clinical features and management of ABPA. *[VNSGU 2025 P3]*

6.6 Occupational Asthma, Allergen Immunotherapy & Inducible Laryngeal Obstruction

Theory › p.124

1. Write a note on various causes, newer diagnostic methods and treatment modalities of occupational asthma. *[DNB May 2024 P3]*
2. Describe the definition and classification of occupational asthma. *[BFUHS 2022 P3]*
3. Vocal cord dysfunction. *[NEET-SS/DNB 2024]*

6.7 COPD — Definition, Pathogenesis, Risk Factors & Assessment

Theory › p.125

1. Assessment of COPD severity. *[DNB Dec 2022 P2]*
2. Risk factors for COPD in non-smokers. *[BFUHS 2019 P3]*
3. Differences between men and women with COPD. *[DNB Oct 2023 P4]*

6.8 Pharmacotherapy of Stable COPD

Theory › p.127

1. Algorithm for the initiation of pharmacological management of COPD. *[DNB Jun 2025 P2]*
2. Adverse effects of inhaled corticosteroids in COPD. *[DNB Jun 2025 P2]*
3. Significance of blood eosinophil in COPD management. *[DNB Jun 2025 P4]*
4. Role of inhaled corticosteroids for treating patients having COPD. *[BFUHS 2022 P2]*
5. Inhaled corticosteroids for patients with stable COPD. *[BFUHS 2019 P2]*
6. Current status of fixed triple drug inhaler combinations in obstructive pulmonary disease. *[BFUHS 2020 P4]*
7. Pharmacological agents useful in preventing COPD exacerbations. *[BFUHS 2020 P3]*
8. Emerging pharmacotherapies for COPD. *[Manipal 2014 P4]*

6.9 Acute Exacerbation of COPD & Role of NIV

Theory › p.129

1. Management of acute exacerbation of COPD. *[BFUHS 2020 P2]*
2. Role of NIV in acute exacerbation of COPD. *[DNB Oct 2024 P3]*
3. Non-invasive mechanical ventilation indications for patient of acute exacerbation of COPD. *[DNB Dec 2021 P3]*

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6.10 Systemic Manifestations, Comorbidities, Hyperinflation & Cor Pulmonale

Theory › p.131

1. Systemic manifestations of COPD. *[DNB Jun 2025 P2]*
2. Clinical significance of hyperinflation in COPD. *[DNB Jun 2025 P2]*
3. Osteoporosis and skeletal muscle dysfunction in COPD. *[DNB Oct 2023 P2]*
4. Complications of COPD. *[DNB Oct 2023 P4]*
5. Aetiopathogenesis, clinical features and complications of cor pulmonale. *[DNB Oct 2023 P2]*

6.11 Asthma–COPD Overlap & Small Airway Diseases (Bronchiolitis)

Theory › p.132

1. Asthma–COPD overlap syndrome. *[DNB Oct 2024 P3]*

6.12 Oxygen Therapy, LTOT, Oxygen Toxicity & Hyperbaric Oxygen

Theory › p.133

1. Nocturnal and long-term oxygen therapy. *[DNB Oct 2023 P2]*
2. Describe oxygen therapy and mention oxygen toxicity. *[WBUHS 2020 P2]*
3. Oxygen toxicity. *[DNB Dec 2025 P4]*
4. Pulmonary oxygen toxicity. *[DNB Jun 2021 P3]*
5. Hyperbaric oxygen therapy. *[BFUHS 2019 P3]*

6.13 Pulmonary Rehabilitation & Airway Clearance Techniques

Theory › p.135

1. Role of rehabilitation in chronic obstructive pulmonary disease. *[DNB Oct 2024 P2]*
2. Evidence for pulmonary rehabilitation in COPD. *[DNB Jun 2021 P2]*
3. Role of pulmonary rehabilitation in COPD. *[BFUHS 2022 P4]*
4. Pulmonary rehabilitation in COPD. *[BFUHS 2019 P4]*
5. Discuss in detail about pulmonary rehabilitation in respiratory diseases. *[VNSGU 2025 P4]*
6. Airway clearance techniques. *[NEET-SS/DNB 2024]*

6.14 Interventions for Emphysema — LVRS, Bronchoscopic Lung Volume Reduction & Bullous Disease

Theory › p.136

1. Bronchoscopic lung volume reduction (bLVR). *[DNB Dec 2022 P4]*
2. Lung volume reduction surgery. *[WBUHS 2020 P2]*

6.15 Alpha-1 Antitrypsin Deficiency

Theory › p.137

1. Alpha 1 antitrypsin deficiency. *[DNB Dec 2021 P1]*

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PREVIOUS-YEAR QUESTION BANK · SECTION 7

Interstitial Lung Diseases & Immunological Disorders

7.1 Classification & Approach to Interstitial Lung Disease; HRCT Patterns

Theory › p.139

1. Classification of interstitial lung diseases. *[NEET-SS/DNB 2024]*
2. Diagnostic classification of interstitial lung disease in clinical practice. *[DNB Oct 2023 P4]*
3. Role of thoracic HRCT scanning in evaluating interstitial lung disease. *[BFUHS 2019 P2]*
4. Describe the newer classification of interstitial lung diseases. *[WBUHS 2020 P2]*
5. Molecular markers and the promise of precision medicine for interstitial lung disease. *[DNB Oct 2023 P4]*

7.2 Idiopathic Pulmonary Fibrosis — Diagnosis, Severity, Antifibrotics & Acute Exacerbation

Theory › p.141

1. Clinical features and HRCT criteria of idiopathic pulmonary fibrosis (IPF). *[DNB Oct 2024 P2]*
2. Assessment of disease severity in idiopathic pulmonary fibrosis. *[DNB Dec 2022 P2]*
3. Antifibrotics in treatment of IPF. *[DNB Jun 2025 P2]*
4. Acute exacerbation of idiopathic pulmonary fibrosis (AE-IPF). *[DNB Jun 2025 P3]*
5. Management of acute exacerbation in a patient with idiopathic pulmonary fibrosis. *[DNB Dec 2022 P2]*
6. Nintedanib and IPF. *[BFUHS 2020 P2]*
7. Newer anti-fibrotics for interstitial lung diseases. *[BFUHS 2020 P4]*
8. Current status of anti-fibrotic drugs in treating interstitial lung disease. *[BFUHS 2023 P2]*
9. Management of idiopathic pulmonary fibrosis. *[BFUHS 2019 P4]*
10. Classify ILDs. Discuss clinical features, management and complications of IPF. *[VNSGU 2025 P3]*
11. Discuss the etiopathogenesis, pathology and management of IPF. *[Manipal 2014 P4]*

7.3 Other Idiopathic Interstitial Pneumonias — NSIP, COP, RB-ILD, DIP, AIP, PPFE; Progressive Pulmonary Fibrosis

Theory › p.144

1. Classification of idiopathic interstitial pneumonias. *[DNB Dec 2022 P2]*
2. Cryptogenic organizing pneumonia. *[DNB Jun 2021 P2]*
3. Respiratory bronchiolitis-ILD. *[DNB Dec 2025 P2]*
4. Smoking related interstitial pulmonary fibrosis. *[DNB Dec 2022 P1]*
5. Non-specific interstitial pneumonia. *[Manipal 2014 P4]*

7.4 Hypersensitivity Pneumonitis

Theory › p.145

1. Hypersensitivity pneumonitis. *[DNB Jun 2025 P2]*
2. Prognosis and treatment of hypersensitivity pneumonitis. *[DNB Dec 2021 P2]*
3. Approach to hypersensitivity pneumonitis. *[BFUHS 2022 P4]*
4. Discuss management of hypersensitivity pneumonitis. *[WBUHS 2020 P2]*
5. Farmer's lung. *[VNSGU 2025 P3]*

7.5 Sarcoidosis

Theory › p.147

1. Approach to the initial treatment of pulmonary sarcoidosis in adults. *[DNB Jun 2025 P2]*
2. Chest radiographic stages of pulmonary sarcoidosis and their implications for treatment. *[DNB Dec 2022 P2]*
3. Indications and treatment of pulmonary sarcoidosis with alternatives to glucocorticoids. *[DNB Dec 2022 P2]*
4. Treatment of sarcoidosis. *[DNB Dec 2021 P2]*
5. Diagnosis of sarcoidosis. *[DNB Jun 2021 P2]*
6. Treatment of symptomatic pulmonary sarcoidosis. *[DNB Jun 2021 P2]*
7. Erythema nodosum. *[DNB Jun 2021 P1]*

7.6 Connective Tissue Disease–Associated ILD & IPAF

Theory › p.149

1. Clinical manifestations of systemic sclerosis. *[DNB Oct 2024 P2]*
2. Determinants of disease progression in systemic sclerosis-associated interstitial lung disease. *[DNB Dec 2022 P2]*
3. Treatment of interstitial lung disease in systemic sclerosis. *[DNB Dec 2022 P2]*
4. Pleuropulmonary complications of rheumatoid arthritis. *[DNB Dec 2021 P2]*

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5. Briefly describe management of rheumatoid lung. *[DNB May 2024 P3]*
6. Respiratory system involvement in SLE. *[DNB Jun 2021 P2]*
7. Management of interstitial pneumonia with autoimmune features. *[BFUHS 2022 P2]*

7.7 Pulmonary Vasculitis & Diffuse Alveolar Haemorrhage

Theory › p.150

1. Granulomatosis with polyangiitis. *[DNB Dec 2021 P2]*
2. Describe Wegener's granulomatosis. *[WBUHS 2020 P2]*
3. Management of EGPA. *[DNB Dec 2025 P3]*
4. ANCA-associated pulmonary vasculitis. *[Manipal 2014 P4]*
5. Enumerate various causes of diffuse alveolar hemorrhage; how to suspect the diagnosis and its management. *[DNB May 2024 P3]*
6. Diffuse alveolar haemorrhage. *[DNB Oct 2023 P2]*
7. Therapy of immune mediated alveolar haemorrhage (DAH). *[DNB Dec 2021 P2]*

7.8 Eosinophilic Lung Diseases — AEP, CEP, Tropical Pulmonary Eosinophilia & HES

Theory › p.152

1. Clinical features, diagnosis and treatment of acute eosinophilic pneumonia. *[DNB Oct 2024 P2]*
2. Chronic eosinophilic pneumonia. *[DNB Dec 2021 P2]*
3. Tropical pulmonary eosinophilia. *[DNB Dec 2021 P2]*
4. Classification of pulmonary eosinophilias. *[DNB Jun 2021 P2]*
5. Differential diagnosis of hypereosinophilic syndromes (HES). *[DNB Dec 2022 P2]*
6. Eosinophilic lung disease. *[BFUHS 2022 P4]*

7.9 Drug-Induced & Transfusion-Related Lung Disease; Radiation Injury

Theory › p.153

1. Methotrexate-induced pneumonitis. *[DNB Dec 2021 P2]*
2. Bevacizumab induced lung complications. *[DNB Dec 2021 P2]*
3. Blood transfusion induced lung injuries. *[DNB Oct 2023 P2]*

7.10 Cystic Lung Diseases — LAM, PLCH, BHD & LIP

Theory › p.155

1. Pulmonary and extrapulmonary manifestations of cystic interstitial lung diseases. *[DNB Jun 2025 P2]*
2. HRCT chest findings in cystic interstitial lung diseases. *[DNB Jun 2025 P2]*

7.11 Pulmonary Alveolar Proteinosis, Alveolar Microlithiasis & Amyloidosis

Theory › p.156

1. Pulmonary alveolar proteinosis (PAP). *[DNB Oct 2023 P2]*
2. Describe whole lung lavage in pulmonary alveolar proteinosis. *[WBUHS 2020 P2]*
3. Whole lung lavage. *[BFUHS 2020 P4]*

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PREVIOUS-YEAR QUESTION BANK · SECTION 8

Occupational & Environmental Lung Disease, Smoking & Inhalational Injury

8.1 Approach to Occupational Lung Disease; Silicosis

Theory › p.159

1. Radiological features of silicosis. *[DNB Jun 2021 P1]*
2. Describe pathogenesis of silicosis and diseases associated with exposure to silica dust. *[BFUHS 2022 P3]*
3. Silicosis. *[Manipal 2014 P4]*
4. Describe pulmonary diseases caused by inhaled inorganic dust. *[WBUHS 2020 P2]*

8.2 Coal Workers' Pneumoconiosis, Asbestos-Related Diseases, Byssinosis & Other Dusts

Theory › p.161

1. Radiographic features of asbestosis in respiratory system. *[DNB Oct 2023 P2]*
2. Asbestos related pleural disorders. *[BFUHS 2023 P2]*
3. Primary and secondary prevention of coal workers' pneumoconiosis. *[BFUHS 2020 P3]*

8.3 Air Pollution, Ozone & Climate Change — Respiratory and Extrapulmonary Effects

Theory › p.162

1. Describe the lung problems of poor ambient air quality; national standards of ambient air quality. *[DNB May 2024 P3]*
2. Respiratory effects of ambient air pollution. *[BFUHS 2020 P3]*
3. Extrapulmonary effects of air pollution. *[BFUHS 2020 P4]*
4. Ozone effect. *[VNSGU 2025 P3]*

8.4 Health Hazards of Smoking & Smoking Cessation

Theory › p.164

1. Describe the pharmacological and non-pharmacological approach for smoking cessation. *[DNB May 2024 P2]*
2. Smoking cessation therapy. *[DNB Jun 2021 P1]*
3. Cigarettes and other tobacco products act (COTPA). *[BFUHS 2020 P3]*
4. Quit smoking clinic activities. *[BFUHS 2019 P3]*

8.5 Toxic Inhalational Injury, Chemical & Biological Agents, Radiation Sickness & Strangulation

Theory › p.165

1. What are the chemical warfare agents (CWAs) and describe their types. *[BFUHS 2022 P3]*
2. What are the key features of biological agents which can be used as a bioweapon? *[BFUHS 2022 P3]*
3. Clinical manifestation and management of acute radiation sickness. *[BFUHS 2022 P3]*
4. Strangulation and its effect on respiration. *[DNB Oct 2023 P2]*

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PREVIOUS-YEAR QUESTION BANK · SECTION 9

Pulmonary Circulation — Hypertension, Embolism, Oedema & Haemoptysis

9.1 Pulmonary Hypertension — Definition, Classification & Diagnosis

Theory › p.168

1. Classification, symptoms and management of pulmonary hypertension. *[DNB Oct 2023 P2]*
2. Discuss the causes, classification system and management of pulmonary hypertension. *[VNSGU 2025 P2]*

9.2 Pulmonary Arterial Hypertension — Risk Stratification & Treatment; CTD-PAH and Drug-Induced PAH

Theory › p.170

1. Describe the outline of management of Group 1 pulmonary arterial hypertension. *[DNB May 2024 P4]*
2. Pharmacotherapy in pulmonary arterial hypertension. *[DNB Oct 2024 P2]*
3. Pulmonary arterial hypertension secondary to drugs and toxins. *[DNB Oct 2023 P4]*
4. Approach to PAH in a patient with connective tissue disease. *[BFUHS 2019 P4]*
5. Nitric oxide in pulmonary diseases. *[Manipal 2014 P4]*

9.3 CTEPH, Portopulmonary Hypertension & Hepatopulmonary Syndrome

Theory › p.172

1. Hepatopulmonary syndrome diagnosis and definitive management. *[DNB Oct 2024 P2]*
2. Portopulmonary hypertension. *[DNB Dec 2021 P2]*

9.4 Pulmonary Embolism — Diagnosis & Risk Stratification

Theory › p.173

1. Diagnosis of pulmonary embolism. *[DNB Jun 2021 P2; BFUHS 2019 P3]*
2. Diagnosis of pulmonary thromboembolism. *[NEET-SS/DNB 2024]*
3. Wells clinical prediction score model. *[DNB Dec 2021 P2]*

9.5 Management of PE & VTE; Prophylaxis, HIT, DOACs & Fat Embolism

Theory › p.175

1. Management of pulmonary embolism. *[DNB Oct 2024 P2; DNB Jun 2021 P2]*
2. Prevention of venous thromboembolism. *[DNB Dec 2021 P3]*
3. Heparin-induced thrombocytopenia. *[DNB Dec 2021 P3]*
4. Newer anticoagulants in pulmonary embolism. *[BFUHS 2019 P4]*
5. Write in brief about venous thromboembolism. *[BFUHS 2019 P1]*
6. Fat embolism. *[BFUHS 2019 P2]*

9.6 Pulmonary Oedema; Cardiac–Pulmonary Interactions

Theory › p.177

1. Management of pulmonary oedema. *[DNB Oct 2024 P2]*
2. Differentiate between cardiogenic and non-cardiogenic pulmonary oedema. *[BFUHS 2019 P1]*
3. Re-expansion pulmonary oedema. *[DNB Oct 2023 P2]*
4. 2D ECHO in respiratory medicine. *[DNB Oct 2023 P4]*
5. Lymphatic (Kerley) lines. *[NEET-SS/DNB 2024]*
6. Jugular pulse and the superior vena cava syndrome. *[NEET-SS/DNB 2024]*

9.7 Haemoptysis — Causes, Massive Haemoptysis & Bronchial Artery Embolisation

Theory › p.178

1. Enumerate the different causes of haemoptysis and management of massive haemoptysis. *[WBUHS 2020 P2]*
2. Discuss causes and management of massive hemoptysis. *[WBUHS 2022 P1]*
3. Describe the causes of haemoptysis; discuss management of massive haemoptysis. *[VNSGU 2025 P2]*
4. Management of recurrent haemoptysis. *[DNB Jun 2021 P2]*
5. Enumerate the causes, diagnostic approach and management of haemoptysis. *[Manipal 2014 P1]*

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PREVIOUS-YEAR QUESTION BANK · SECTION 10

Lung Cancer & Other Thoracic Tumours

10.1 Epidemiology, Risk Factors, Genetic Susceptibility & Lung Cancer Screening

Theory › p.181

1. Genetic susceptibility to lung cancer. *[DNB Jun 2021 P1]*
2. Patient selection and implementation in lung cancer screening. *[DNB Oct 2023 P4]*
3. Role of vaccines in lung cancer. *[BFUHS 2019 P3]*

10.2 WHO Classification, Pathology, Tumour Markers & Liquid Biopsy

Theory › p.183

1. Tumour markers in lung cancer. *[DNB Oct 2023 P2]*
2. Liquid biopsy tests for lung cancer patients. *[DNB Dec 2022 P4]*

10.3 Clinical Features, Paraneoplastic Syndromes & Electrolyte Disorders

Theory › p.184

1. Electrolyte imbalance in small cell lung carcinoma. *[DNB Oct 2024 P2]*
2. Paraneoplastic syndrome in squamous cell lung carcinoma. *[DNB Oct 2024 P2]*
3. Paraneoplastic syndromes. *[DNB Jun 2021 P2]*

10.4 Diagnosis & Staging — TNM 9th Edition, EBUS & Mediastinal Staging

Theory › p.186

1. Staging of lung cancer. *[BFUHS 2020 P4]*
2. Discuss classification systems for staging of lung cancer. *[VNSGU 2025 P4]*
3. Endobronchial ultrasound for evaluating mediastinal lymphadenopathy. *[BFUHS 2019 P2]*

10.5 Treatment of NSCLC — Surgery, SBRT, Chemoradiation, Targeted Therapy & Immunotherapy

Theory › p.187

1. Factors affecting prognosis of non-small cell lung carcinoma (NSCLC). *[DNB Dec 2022 P2]*
2. Oral tyrosine kinase inhibitors for lung adenocarcinoma. *[BFUHS 2019 P2]*
3. Immunotherapy in lung cancer. *[BFUHS 2020 P2]*
4. Stereotactic body radiotherapy (SBRT). *[DNB Dec 2025 P2]*

10.6 Small Cell Lung Cancer

Theory › p.189

1. Treatment of small cell lung carcinoma (SCLC). *[DNB Dec 2022 P2]*
2. Recent TNM staging of carcinoma lung; diagnosis and management of small cell carcinoma; paraneoplastic syndromes with SCLC. *[VNSGU 2025 P3]*

10.7 Solitary Pulmonary Nodule & Incidental Nodules

Theory › p.190

1. Solitary lung lesion. *[DNB Jun 2025 P2]*
2. Differential diagnosis of incidental pulmonary nodule. *[DNB Dec 2022 P2]*
3. Solitary pulmonary nodule: definition, differential diagnosis and management. *[BFUHS 2020 P2]*

10.8 Carcinoid Tumours, Metastatic Lung Disease & Other Tumours

Theory › p.192

1. Management of lung carcinoid tumours with localised disease. *[DNB Dec 2022 P2]*
2. Symptoms of carcinoid syndrome and their management in patients with unresectable disease. *[DNB Dec 2022 P2]*

10.9 Interventional Bronchoscopy — Central Airway Obstruction, Heat Ablation & Palliation

Theory › p.193

1. Bronchoscopic management of central airway obstruction. *[DNB Jun 2021 P2]*
2. Write a detailed note on various bronchoscopic heat-ablative modalities. *[DNB May 2024 P2]*
3. Bronchoscopic palliation for lung cancer. *[BFUHS 2023 P2]*

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PREVIOUS-YEAR QUESTION BANK · SECTION 11

Pleura, Mediastinum, Diaphragm & Chest Wall

11.1 Pleural Physiology & Approach to Pleural Effusion

Theory › p.196

1. Differentiating transudative from exudative pleural effusions. *[NEET-SS/DNB 2024]*
2. Pleural effusions in the critically ill and 'at-bleeding-risk' population. *[DNB Oct 2023 P4]*
3. Uraemic pleuritis. *[Manipal 2014 P2]*
4. Dressler's syndrome. *[Manipal 2014 P2]*

11.2 Parapneumonic Effusion, Empyema & Bronchopleural Fistula

Theory › p.198

1. Bacterial empyema. *[DNB Dec 2021 P2]*

11.3 Malignant & Recurrent Pleural Effusions, Hepatic Hydrothorax & Indwelling Pleural Catheters

Theory › p.200

1. Enumerate various causes of recurrent non-malignant pleural effusion. *[DNB May 2024 P2]*
2. Describe portable devices useful for management of pleural effusion. *[DNB May 2024 P2]*
3. Management options in malignant pleural effusion. *[BFUHS 2022 P2]*
4. Hepatic hydrothorax. *[BFUHS 2019 P2]*
5. Enumerate causes of ascites; aetiology, clinical features and management of hepatic hydrothorax. *[VNSGU 2025 P2]*

11.4 Malignant Pleural Mesothelioma

Theory › p.202

1. Treatment of pleural mesothelioma. *[DNB Oct 2024 P2]*
2. Mesothelioma. *[Manipal 2014 P4]*

11.5 Chylothorax, Pseudochylothorax, Haemothorax, Fibrothorax & Trapped Lung

Theory › p.203

1. Difference between chylothorax and pseudochylothorax and causes of chylothorax. *[DNB Oct 2024 P2]*
2. Complications of hemothorax. *[DNB Jun 2025 P2]*
3. Hemothorax. *[Manipal 2014 P4]*

11.6 Pneumothorax — Primary, Secondary, Recurrent, Iatrogenic; Persistent Air Leak

Theory › p.204

1. Re-absorption of pleural gases in a pneumothorax. *[DNB Dec 2022 P1]*
2. Recurrent pneumothorax. *[DNB Oct 2023 P2]*
3. Management of recurrent pneumothorax. *[DNB Jun 2021 P2]*
4. Treatment of pneumothorax. *[DNB Dec 2021 P2]*
5. Etiology and management of iatrogenic pneumothorax. *[DNB Oct 2024 P2]*
6. Management of persistent air leak. *[NEET-SS/DNB 2024]*
7. Bronchopleural fistula. *[Manipal 2014 P4]*

11.7 Mediastinal Masses, Mediastinitis & Pneumomediastinum

Theory › p.206

1. Write a note on etiology, clinical features, evaluation and management of fibrosing mediastinitis. *[DNB May 2024 P2]*
2. Spontaneous pneumomediastinum. *[DNB Dec 2021 P2]*
3. Etiology of pneumomediastinum. *[NEET-SS/DNB 2024]*
4. Clinical assessment and management of thymoma. *[DNB Dec 2021 P2]*
5. Germ cell tumours of mediastinum. *[DNB Jun 2021 P2]*

11.8 Diseases of the Diaphragm — Paralysis, Eventration & Elevated Hemidiaphragm

Theory › p.207

1. Diaphragmatic eventration. *[DNB Oct 2023 P1]*
2. Causes of unilateral elevation of the hemidiaphragm. *[DNB Jun 2025 P2]*

11.9 Chest Wall Disorders — Kyphoscoliosis, Thoracoplasty, Flail Chest, Marfan Syndrome & Neuromuscular Restriction

Theory › p.209

1. Kyphoscoliosis: definition, causes and treatment. *[DNB Dec 2021 P2]*

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2. Thoracoplasty. *[DNB Dec 2021 P2]*
3. Flail chest. *[DNB Oct 2024 P1]*
4. Clinical signs of Marfan's syndrome. *[DNB Jun 2021 P2]*

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PREVIOUS-YEAR QUESTION BANK · SECTION 12

Critical Care Pulmonary Medicine

12.1 Respiratory Failure — Types, Mechanisms & Approach to Acute Hypoxaemia

Theory › p.211

1. Hypercapnic respiratory failure. *[DNB Oct 2023 P1]*
2. Acute vs chronic respiratory failure. *[DNB Oct 2023 P3]*
3. Diagnosis and management of type I respiratory failure. *[DNB Oct 2024 P3]*
4. Differential diagnosis in a patient presenting with type II respiratory failure on ABG. *[DNB Oct 2024 P3]*
5. Evaluation and management of the non-ventilated, hospitalised adult patient with acute hypoxaemia. *[DNB Jun 2025 P3]*
6. Define respiratory failure. Discuss different types of respiratory failure. *[WBUHS 2022 P1]*

12.2 Oxygen Delivery Devices, High-Flow Nasal Cannula & ROX Index

Theory › p.213

1. Oxygen delivery devices. *[DNB Oct 2024 P3]*
2. What is ROX index? *[BFUHS 2022 P3]*

12.3 Non-Invasive Ventilation

Theory › p.215

1. Non-invasive ventilation in adults with acute respiratory failure: benefits and contraindications. *[DNB Jun 2025 P3]*
2. Benefits and contraindications of non-invasive ventilation in adults with acute respiratory failure. *[DNB Dec 2022 P3]*
3. Describe the indications of NIV and role of NIV in chronic respiratory failure. *[WBUHS 2020 P2]*
4. Role of NIV in acute respiratory failure. *[BFUHS 2022 P4]*

12.4 Mechanical Ventilation — Modes, Waveforms, Alarms & Auto-PEEP

Theory › p.216

1. Modes and alarms in pressure ventilation. *[DNB Oct 2023 P3]*
2. Interrelationship between flow, time, volume and respiratory frequency in mechanical ventilation. *[DNB Oct 2023 P3]*
3. Approach to a ventilated patient with sudden desaturation and raised plateau pressure. *[DNB Oct 2024 P3]*
4. Pressure support ventilation. *[BFUHS 2023 P2]*
5. Newer modes of mechanical ventilation. *[BFUHS 2020 P4]*
6. Auto-PEEP. *[BFUHS 2022 P1]*

12.5 ARDS — Definition, Lung-Protective Ventilation, Prone Positioning & Refractory Hypoxaemia

Theory › p.219

1. How to classify severity of ARDS? Conventional and advanced ventilatory strategies for ARDS. *[DNB May 2024 P2]*
2. Recruitment manoeuvre strategies for adults with ARDS. *[DNB Dec 2022 P3]*
3. Management strategies for refractory hypoxaemia in ARDS. *[DNB Dec 2025 P3]*
4. Differential diagnosis of acute respiratory distress syndrome. *[DNB Jun 2025 P3]*
5. Lung protective ventilation. *[DNB Oct 2024 P3]*
6. Lung protective mechanical ventilation. *[DNB Jun 2021 P3]*
7. Lung protective ventilatory strategy. *[NEET-SS/DNB 2024]*
8. Protective ventilatory strategy. *[DNB Jun 2020 P3]*
9. Low tidal volume ventilation strategy in ARDS. *[BFUHS 2019 P3]*
10. Permissive hypercapnia. *[DNB Oct 2024 P3]*
11. Physiological effect of prone ventilation on oxygenation. *[DNB Dec 2021 P3]*
12. Prone positioning for refractory ARDS. *[BFUHS 2019 P2]*
13. Role of corticosteroids in ARDS. *[DNB Jun 2020 P3]*
14. Advances in ventilator management for patients with ARDS. *[DNB Oct 2023 P4]*
15. ECCO2R (extracorporeal carbon dioxide removal). *[VNSGU 2025 P4]*

12.6 Ventilating Obstructive Airway Disease — COPD & Dynamic Hyperinflation

Theory › p.221

1. Ventilator settings in patients with acute respiratory failure complicating COPD. *[DNB Dec 2022 P3]*
2. Dynamic hyperinflation in patients with COPD who are intubated for respiratory failure. *[DNB Dec 2022 P3]*

12.7 Weaning, Tracheostomy, VILI & Complications of Mechanical Ventilation

Theory › p.222

1. Methods of weaning for extubation. *[DNB Dec 2021 P3]*
2. Weaning strategies from ventilator support. *[DNB Jun 2020 P3]*
3. Prevention of ventilator-induced lung injury. *[DNB Dec 2021 P3]*
4. Complications of mechanical ventilation. *[DNB Jun 2020 P3]*
5. Early versus late tracheostomy – evidence based critique. *[BFUHS 2019 P4]*

12.8 Sepsis & Septic Shock; SOFA Score; Common Medical Emergencies in the Respiratory ICU

Theory › p.224

1. The SOFA (Sequential Organ Failure Assessment) score. *[DNB Dec 2022 P3]*
2. The SOFA score to describe organ dysfunction/failure. *[DNB Dec 2021 P3]*
3. Sepsis and septic shock management. *[BFUHS 2020 P2]*
4. Management of diabetic ketoacidosis. *[DNB Oct 2024 P2]*
5. Myxoedema coma. *[VNSGU 2025 P2]*

12.9 Monitoring in Respiratory Critical Care — Pulse Oximetry, Capnography, Haemodynamics & Central Lines

Theory › p.225

1. Capnography and end tidal CO2. *[DNB Jun 2020 P3]*
2. Pulse oximetry. *[DNB Jun 2021 P1]*
3. Write a short note on pulse oximetry. *[BFUHS 2019 P1]*
4. Complications of central venous catheters and their prevention. *[DNB Dec 2021 P3]*

12.10 Airway Management & the Difficult Airway

Theory › p.227

1. Difficult airway. *[DNB Dec 2025 P3]*

12.11 Ethics, End-of-Life Care & Brain Death in Critical Care

Theory › p.228

No question from this topic in the papers reviewed — study it from the chapter; it is a likely future question.

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PREVIOUS-YEAR QUESTION BANK · SECTION 13

Sleep-Related Breathing Disorders

13.1 Sleep Physiology, Polysomnography & Daytime Sleepiness Testing

Theory › p.230

1. Stages and functions of sleep. *[DNB Dec 2025 P3]*
2. Stages of normal sleep. *[DNB Oct 2023 P3]*
3. Levels of polysomnography (PSG) for suspected OSA. *[DNB Oct 2024 P3]*
4. MSLT. *[DNB Oct 2023 P4]*
5. Effect of sleep in patients with pulmonary disorder. *[DNB Jun 2020 P3]*
6. Outline the mechanism of respiratory failure in sleep. *[BFUHS 2022 P1]*
7. Sleep deficiency in pregnancy. *[DNB Oct 2023 P4]*

13.2 Obstructive Sleep Apnoea — Epidemiology, Pathogenesis, Clinical Features & Diagnosis

Theory › p.232

1. Clinical manifestations of OSA and its differential diagnosis. *[DNB Jun 2025 P3]*
2. Diagnostic criteria for OSA. *[DNB Jun 2025 P3]*
3. Risk factors for obstructive sleep apnoea. *[DNB Jun 2025 P3]*
4. Pathogenesis of obstructive sleep apnoea. *[DNB Dec 2025 P3]*
5. Epidemiology and risk factors for OSA. *[DNB Dec 2021 P3]*
6. Diagnosis of obstructive sleep apnoea. *[DNB Jun 2020 P3]*
7. Systemic complications of OSA. *[DNB Oct 2023 P3]*
8. Role of MRI cephalometry in OSA. *[DNB Oct 2023 P3]*
9. Causes and management of sleep apnoea hypopnoea syndrome. *[DNB Oct 2023 P3]*
10. Discuss obstructive sleep apnoea syndrome. *[WBUHS 2020 P2]*
11. Sleep disordered breathing: risk factors, pathogenesis, clinical features and management of OSA. *[VNSGU 2025 P2]*
12. Sleep related breathing disorders. *[BFUHS 2022 P4]*

13.3 Treatment of Obstructive Sleep Apnoea — PAP, Oral Appliances, Surgery & Pharmacotherapy

Theory › p.234

1. Treatment of OSA. *[DNB Jun 2025 P3]*
2. Role of positive airway pressure (PAP) in managing OSA. *[DNB Oct 2024 P3]*
3. Medical management of obstructive sleep apnoea. *[BFUHS 2023 P2]*
4. Prevention of sleep related disorders. *[BFUHS 2019 P3]*

13.4 Central Sleep Apnoea, Obesity Hypoventilation, Overlap Syndrome & Narcolepsy

Theory › p.236

1. Central sleep apnoea syndrome. *[DNB Jun 2025 P3]*
2. Obesity hypoventilation syndrome. *[DNB Jun 2025 P3; DNB Dec 2021 P3]*
3. Diagnostic criteria for obesity hypoventilation syndrome. *[DNB Oct 2024 P3]*
4. Obesity hypoventilation syndrome and its management. *[BFUHS 2020 P2]*
5. Overlap syndrome. *[BFUHS 2020 P2]*
6. Syndrome Z. *[DNB Oct 2023 P3]*
7. Narcolepsy. *[DNB Oct 2023 P3]*

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PREVIOUS-YEAR QUESTION BANK · SECTION 14

Thoracic Imaging & Pulmonary Procedures

14.1 Chest Radiograph Interpretation — Collapse, Silhouette & Classic Signs

Theory › p.238

1. Indirect radiological signs of collapse. *[DNB Oct 2023 P4]*
2. Fluoroscopy. *[Manipal 2014 P1]*

14.2 CT & HRCT Signs in Pulmonary Medicine

Theory › p.240

1. Tree-in-bud sign and split pleura sign. *[DNB Oct 2023 P4]*

14.3 Thoracic Ultrasound — Effusion, Pneumothorax & Trauma

Theory › p.241

1. Ultrasonography in pleural effusion. *[DNB Jun 2025 P4]*
2. Ultrasonography in pneumothorax. *[DNB Jun 2025 P4]*
3. USG in pneumothorax. *[VNSGU 2025 P3]*
4. Chest ultrasonography in trauma patients. *[DNB Oct 2023 P4]*

14.4 PET-CT, MRI & Interventional Radiology in Chest Medicine

Theory › p.242

No question from this topic in the papers reviewed — study it from the chapter; it is a likely future question.

14.5 Flexible & Rigid Bronchoscopy, BAL, Transbronchial Lung Biopsy & Cryobiopsy

Theory › p.244

1. What are the indications for diagnostic flexible bronchoscopy? *[BFUHS 2019 P1]*
2. Diagnostic bronchoscopy in diffuse lung disease. *[DNB Oct 2023 P4]*
3. Transbronchial cryobiopsy (cryo-TBB). *[DNB Dec 2022 P4]*
4. Measures to control airborne infection in a bronchoscopy suite. *[BFUHS 2022 P2]*

14.6 EBUS, Navigational & Virtual Bronchoscopy

Theory › p.245

1. Endobronchial ultrasound: current status in diagnosis in respiratory medicine. *[DNB Dec 2021 P4]*
2. Endobronchial ultrasound. *[Manipal 2014 P1]*
3. Virtual bronchoscopy. *[BFUHS 2023 P2]*

14.7 Medical Thoracoscopy & Pleural Biopsy (Abrams, Cope, Image-Guided)

Theory › p.246

1. Enumerate indications, procedure and post-procedural complications of thoracoscopy. *[VNSGU 2025 P4]*
2. Pre-operative evaluation and anaesthetic requirements of medical thoracoscopy. *[BFUHS 2020 P1]*
3. Abrams and Cope's pleural biopsy needles. *[DNB Oct 2023 P4]*
4. Pleural biopsy. *[DNB Dec 2021 P4; Manipal 2014 P1]*

14.8 Tube Thoracostomy, Pleurodesis (incl. Autologous Blood Patch) & Image-Guided FNAC

Theory › p.248

1. Autologous blood patch pleurodesis. *[DNB Dec 2021 P4]*

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PREVIOUS-YEAR QUESTION BANK · SECTION 15

Special Environments, Systemic Disease, Pregnancy & Preventive Pulmonology

15.1 High-Altitude Illness — AMS, HACE, HAPE & Chronic Mountain Sickness

Theory › p.250

1. Treatment of high altitude pulmonary oedema. *[DNB Jun 2025 P2]*
2. Recognition, management and prevention of high-altitude pulmonary oedema. *[BFUHS 2022 P2]*
3. Mechanisms for the development of HAPE and HACE. *[BFUHS 2022 P1]*
4. High altitude pulmonary oedema. *[VNSGU 2025 P3]*
5. Chronic mountain sickness. *[Manipal 2014 P2]*

15.2 Diving, Aviation & Aerospace Medicine; Miners

Theory › p.252

No question from this topic in the papers reviewed — study it from the chapter; it is a likely future question.

15.3 Extrapulmonary Manifestations & Pulmonary Complications of Systemic Disease

Theory › p.253

1. Pulmonary complications in renal diseases. *[DNB Oct 2023 P2]*
2. Pulmonary complications of acute pancreatitis. *[DNB Jun 2021 P2]*
3. Budd–Chiari syndrome. *[VNSGU 2025 P2]*

15.4 Respiratory Diseases in Pregnancy

Theory › p.254

1. Respiratory diseases in pregnancy and their preventive measures. *[DNB Jun 2021 P1]*

15.5 Vaccination in Pulmonary Diseases

Theory › p.255

1. Pneumococcal vaccination. *[DNB Jun 2025 P2]*
2. Write a note on currently available vaccines in chronic respiratory disorders; their mechanisms and role. *[DNB May 2024 P2]*
3. Trivalent influenza vaccine. *[BFUHS 2020 P3]*
4. Prophylactic vaccination in respiratory disorders. *[BFUHS 2020 P4]*
5. Influenza vaccination. *[BFUHS 2019 P2]*
6. Critical review of pneumococcal and influenza immunization. *[BFUHS 2019 P4]*
7. Vaccination against pulmonary infections. *[Manipal 2014 P2]*

15.6 Chronic Cough Syndromes (UACS, GERD) & Quality-of-Life Questionnaires

Theory › p.257

1. GERD. *[DNB Oct 2024 P2]*
2. Upper airway cough syndrome. *[DNB Oct 2024 P2]*

15.7 Obesity-Related Pulmonary Disorders

Theory › p.259

No question from this topic in the papers reviewed — study it from the chapter; it is a likely future question.

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PREVIOUS-YEAR QUESTION BANK · SECTION 16

Surgical Aspects of Pulmonary Medicine

16.1 Pre-Operative Evaluation for Lung Resection & Extrathoracic Surgery

Theory › p.261

1. Pre-anaesthetic evaluation for extra-thoracic surgeries. *[DNB Oct 2023 P4]*

16.2 Chest Trauma & Trauma-Related Lung Dysfunction

Theory › p.263

No question from this topic in the papers reviewed — study it from the chapter; it is a likely future question.

16.3 Lung Transplantation

Theory › p.264

1. Selection of lung transplant candidates. *[DNB Dec 2022 P4]*

16.4 Upper Airway Obstruction & Approach to Stridor

Theory › p.265

1. Approach to a patient with stridor. *[BFUHS 2022 P2]*

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PREVIOUS-YEAR QUESTION BANK · SECTION 17

Research Methodology, Epidemiology & Biostatistics

17.1 Epidemiological Terms & Study Designs

Theory › p.267

1. Cross-sectional study. *[DNB Jun 2025 P1]*
2. Case-control study. *[DNB Jun 2025 P1]*
3. Types of studies in clinical research. *[DNB Oct 2024 P1]*
4. Advantages and disadvantages of case-control studies. *[DNB Dec 2022 P1]*
5. Outline the difference between a systematic review and meta-analysis; their role in clinical medicine. *[BFUHS 2022 P1]*

17.2 Biostatistics — Central Tendency, Dispersion, Hypothesis Testing & Sampling

Theory › p.268

1. Standard deviation. *[DNB Jun 2025 P1]*
2. Null hypothesis. *[DNB Oct 2024 P1]*
3. Measures of central tendency and dispersion in data analysis. *[DNB Oct 2024 P1]*
4. Odds ratio. *[DNB Dec 2021 P1]*
5. P value. *[DNB Dec 2021 P1]*
6. Methods of sampling. *[DNB Dec 2021 P1]*
7. The concept of statistical significance. *[DNB Jun 2021 P1]*

17.3 Clinical Trials, Bias, Blinding, Randomisation, Outcomes & Research Ethics

Theory › p.270

1. Phases in clinical trials. *[DNB Oct 2024 P1]*
2. Bias and blinding. *[DNB Dec 2022 P1]*
3. Randomisation methods. *[DNB Dec 2021 P1]*
4. Defining outcome parameters in interventional studies. *[DNB Jun 2021 P1]*

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NMC Curriculum — MD Pulmonary Medicine

The syllabus of the NMC Guidelines for Competency Based Postgraduate Training Programme for MD in Pulmonary Medicine, reproduced in the layout of this book. Every syllabus item is hyperlinked to the page(s) where it is covered.

A. Cognitive domain — subject-specific competencies

C1	Demonstrate sound knowledge of common pulmonary diseases, their clinical manifestations including emergent situations, investigative procedures to confirm diagnosis, and their epidemiology	2.13 · p.43 · 14.1 · p.238 · 4.1 · p.66
C2	Demonstrate comprehensive knowledge of the various modes of therapy used in treatment of pulmonary diseases	6.2 · p.117 · 6.8 · p.127 · 2.14 · p.44
C3	Describe the mode of action of commonly used drugs, their doses, side-effects/ toxicity, indications, contraindications and interactions	3.8 · p.58 · 3.9 · p.60 · 3.10 · p.61 · 3.11 · p.63 · 3.12 · p.64
C4	Describe medical and surgical modes of management and offer a comprehensive plan inclusive of the National Tuberculosis (Elimination) Programme	4.5 · p.74 · 4.7 · p.78 · 16.1 · p.261
C5	Manage common pulmonary emergencies and understand the basics of intensive care	12.1 · p.211 · 9.7 · p.178 · 6.3 · p.118 · 11.6 · p.204
C6	Practise ethically, show empathy and adopt a humane approach towards patients and families	12.11 · p.228
C7	Recognise national priorities and implement National Health Programmes including tuberculosis	4.1 · p.66 · 4.15 · p.91
C8	Demonstrate competence in medical management	5.1 · p.95
C9	Inculcate good reading habits, search medical literature and develop basic concepts of research	17.1 · p.267 · 17.3 · p.270

B. Affective domain

B1	Function as part of a team; cooperate with colleagues to provide the best possible diagnosis or opinion	12.11 · p.228
B2	Adopt ethical principles; respect patients' rights including right to information and second opinion	12.11 · p.228
B3	Communication skills — reports, opinions, interaction with patients, relatives, peers and teaching	17.3 · p.270

C. Psychomotor domain — skills

S1	Interview the patient and record history chronologically	2.13 · p.43
S2	Conduct clinical examination; elicit and interpret findings; diagnose common pulmonary disorders and emergencies	2.13 · p.43 · 15.3 · p.253
S3	Perform office procedures: sputum collection and AFB examination, chest X-ray interpretation, lung function tests	3.1 · p.49 · 14.1 · p.238 · 2.2 · p.24
S4	Interpret and manage blood gas abnormalities	2.8 · p.35 · 12.1 · p.211
S5	Develop management plans for pulmonary diseases	6.2 · p.117 · 6.8 · p.127 · 4.5 · p.74
S6	Assist/perform bronchoscopy, pleural aspiration and biopsy, physiotherapy, intubation, pneumothorax drainage	14.5 · p.244 · 14.7 · p.246 · 14.8 · p.248 · 12.10 · p.227 · 6.13 · p.135
S7	Recognise ICU emergencies; perform basic critical-care monitoring and therapeutic procedures	12.9 · p.225 · 12.4 · p.216

S8	Collect, compile, analyse, interpret and present research data	17.2 · p.268
S9	Teach pulmonary medicine to undergraduates and postgraduates	17.3 · p.270

Diagnostic tests — performance and interpretation

D1	Sputum and body-fluid examination: ZN stain for AFB; culture for bacteria, fungi and viruses	3.1 · p.49 · 3.3 · p.52 · 3.4 · p.53 · 3.5 · p.54
D2	Newer diagnostic techniques for tuberculosis including molecular techniques	3.2 · p.51 · 4.3 · p.70
D3	FNAC of lung masses (blind and image-guided)	14.8 · p.248 · 10.4 · p.186
D4	Arterial blood gas analysis and pulse oximetry	2.8 · p.35 · 12.9 · p.225
D5	Imaging: plain radiography, ultrasound, CT, PET, MRI	14.1 · p.238 · 14.2 · p.240 · 14.3 · p.241 · 14.4 · p.242
D6	Sputum cytology; simple haematological, immunological and serological tests	10.2 · p.183 · 3.7 · p.57
D7	Polysomnography (full-night and split-night) incl. CPAP titration; evaluation of daytime sleepiness	13.1 · p.230 · 13.3 · p.234
D8	Cardiopulmonary exercise testing	2.10 · p.39
D9	Pulmonary function tests: spirometry, lung volumes, diffusion, body plethysmography	2.1 · p.22 · 2.2 · p.24 · 2.3 · p.26
D10	Bronchoprovocation tests	2.11 · p.40
D11	BCG vaccination; Mantoux testing; interferon-gamma release assays	4.15 · p.91 · 4.4 · p.71
D12	Bronchoscopy: fibreoptic/rigid, diagnostic and therapeutic	14.5 · p.244 · 14.6 · p.245 · 10.9 · p.193
D13	ECG, 2D and Doppler echocardiography; venous Doppler ultrasound	2.16 · p.47 · 9.6 · p.177 · 9.4 · p.173
D14	Skin tests for hypersensitivity; sputum induction and non-invasive monitoring of airway inflammation	3.7 · p.57 · 2.11 · p.40
D15	Medical thoracoscopy	14.7 · p.246

Therapeutic procedures

T1	Fine-needle aspiration and other guided procedures	14.8 · p.248 · 14.4 · p.242
T2	Tube thoracostomy; pleural biopsy; lymph node biopsy	14.8 · p.248 · 14.7 · p.246
T3	Cardiopulmonary rehabilitation exercises; postural drainage	6.13 · p.135
T4	Administration of inhalation therapy and oxygen therapy	2.14 · p.44 · 6.12 · p.133 · 12.2 · p.213
T5	Administration of CPAP / BiPAP	12.3 · p.215 · 13.3 · p.234
T6	Monitoring and emergency procedures in intensive care	12.9 · p.225 · 12.10 · p.227

Syllabus I-A. Anatomy and histology of the respiratory system

IA1	Development and anatomy of the respiratory system	1.1 · p.10 · 1.2 · p.12 · 1.3 · p.13 · 1.4 · p.15 · 1.5 · p.16
IA2	Applied embryology of lungs, mediastinum and diaphragm	1.1 · p.10 · 1.5 · p.16 · 1.6 · p.17
IA3	Developmental anomalies	1.7 · p.18 · 1.8 · p.20

Syllabus I-B. Physiology and biochemistry

IB1	Assessment of pulmonary functions	2.1 · p.22 · 2.2 · p.24 · 2.3 · p.26 · 2.11 · p.40
IB2	Control of ventilation; pulmonary mechanics	2.7 · p.33 · 2.4 · p.28
IB3	Ventilation, pulmonary blood flow, gas exchange and transport	2.5 · p.29 · 2.6 · p.32 · 2.15 · p.45
IB4	Non-respiratory metabolic functions of lung	2.9 · p.37
IB5	Principles of electrocardiography	2.16 · p.47
IB6	Inhalation kinetics and its implication in aerosol therapy, sputum induction	2.14 · p.44
IB7	Acid–base and electrolyte balance	2.8 · p.35 · 2.16 · p.47
IB8	Physiology of sleep and its disorders	13.1 · p.230
IB9	Pulmonary innervation and reflexes	2.7 · p.33 · 1.5 · p.16
IB10	Pulmonary defence mechanisms	2.9 · p.37
IB11	Principles of exercise physiology and testing	2.10 · p.39
IB12	Physiological changes in pregnancy, high altitude, ageing	2.12 · p.41 · 15.1 · p.250
IB13	Physiological basis of pulmonary symptoms	2.13 · p.43

Syllabus I-C. Microbiology

IC1	Mycobacterium tuberculosis and other mycobacteria	3.1 · p.49 · 4.16 · p.92
IC2	Bacteria causing pulmonary diseases; atypical organisms; anaerobes	3.5 · p.54 · 5.4 · p.100 · 5.6 · p.103
IC3	Laboratory diagnosis of non-tubercular infections of respiratory tract	3.5 · p.54
IC4	Laboratory diagnosis of TB including staining, culture and drug sensitivity testing	3.1 · p.49 · 3.2 · p.51
IC5	Virulence and pathogenicity of mycobacteria	3.1 · p.49 · 4.2 · p.68
IC6	Respiratory viruses: viral diseases and diagnostic methods	3.3 · p.52 · 5.5 · p.101
IC7	Respiratory fungi: classification, laboratory diagnosis of pulmonary mycoses	3.4 · p.53 · 5.8 · p.106 · 5.9 · p.108
IC8	Opportunistic infections in the immunocompromised	5.11 · p.110
IC9	HIV and AIDS: virology, immunopathogenesis, diagnosis	4.9 · p.81 · 5.11 · p.110
IC10	Parasitic lung diseases	5.10 · p.109

Syllabus I-D. Pathology

ID1	Acute and chronic inflammation; pathogenetic mechanisms	3.6 · p.56
ID2	Pathology of tuberculosis	3.6 · p.56 · 4.2 · p.68
ID3	Pathology of pneumonias and bronchopulmonary suppuration	5.2 · p.97 · 5.6 · p.103
ID4	Chronic bronchitis, emphysema, asthma, other airway diseases	3.6 · p.56 · 6.1 · p.115 · 6.7 · p.125
ID5	Occupational lung diseases including pneumoconiosis	8.1 · p.159 · 8.2 · p.161
ID6	Interstitial lung diseases incl. sarcoidosis, CTD, vasculitis, eosinophilias	7.1 · p.139 · 7.5 · p.147 · 7.6 · p.149 · 7.7 · p.150 · 7.8 · p.152
ID7	Tumours of the lung, mediastinum and pleura	10.2 · p.183 · 11.4 · p.202 · 11.7 · p.206

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Syllabus I-E. Epidemiology

IE1	Epidemiological terms, definitions and methods	17.1 · p.267
IE2	Epidemiology of TB, pneumoconiosis, asthma, lung cancer, COPD	4.1 · p.66 · 8.1 · p.159 · 6.1 · p.115 · 10.1 · p.181 · 6.7 · p.125
IE3	National TB Programme (RNTCP → NTEP); epidemiological aspects of BCG	4.1 · p.66 · 4.15 · p.91
IE4	Pollution-related pulmonary diseases	8.3 · p.162
IE5	Research methodology, statistics and study designs	17.1 · p.267 · 17.2 · p.268 · 17.3 · p.270

Syllabus I-F. Allergy and immunology

IF1	Hypersensitivity mechanisms in pulmonary diseases	3.7 · p.57
IF2	Diagnostic tests in allergic lung disease: in-vitro, in-vivo, bronchial provocation	3.7 · p.57 · 2.11 · p.40
IF3	Immunology of tuberculosis, sarcoidosis and other immunological diseases	3.7 · p.57 · 4.2 · p.68 · 7.5 · p.147

Syllabus I-G. Pharmacology

IG1	Antimicrobial drugs	3.11 · p.63 · 5.1 · p.95
IG2	Antitubercular drugs	3.8 · p.58 · 3.9 · p.60
IG3	Antineoplastic and immunosuppressant drugs	3.12 · p.64 · 10.5 · p.187
IG4	Bronchodilator and anti-inflammatory drugs	3.10 · p.61
IG5	Drugs used in viral, fungal and parasitic infections	3.11 · p.63
IG6	Pharmacokinetics and drug interactions; pharmacovigilance	3.12 · p.64 · 4.9 · p.81

Syllabus II-A1. Tuberculosis

TB1	Aetiopathogenesis	4.2 · p.68
TB2	Diagnostic methods	4.3 · p.70 · 3.2 · p.51
TB3	Differential diagnosis	4.3 · p.70
TB4	Management of pulmonary TB; NTEP (RNTCP, DOTS, DOTS-Plus); International Standards of TB Care	4.5 · p.74 · 4.1 · p.66
TB5	Complications of tuberculosis	4.14 · p.89
TB6	Tuberculosis in children	4.10 · p.82
TB7	Geriatric tuberculosis	4.8 · p.79
TB8	Pleural and pericardial effusion and empyema	4.12 · p.86
TB9	Mycobacteria other than tuberculosis	4.16 · p.92
TB10	Extrapulmonary tuberculosis	4.13 · p.87 · 4.11 · p.84
TB11	HIV and TB; interactions of antitubercular drugs with antiretrovirals	4.9 · p.81
TB12	Diabetes mellitus and tuberculosis	4.8 · p.79
TB13	Management of MDR and XDR tuberculosis	4.6 · p.76 · 4.7 · p.78

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Syllabus II-A2. Non-tuberculous infections

NT1	Approach to a patient with pulmonary infection	5.1 · p.95
NT2	Community-acquired pneumonia	5.2 · p.97
NT3	Hospital-acquired and ventilator-associated pneumonia	5.3 · p.99
NT4	Unusual and atypical pneumonias — bacterial, viral, fungal, parasitic, rickettsial, anaerobic	5.4 · p.100 5.5 · p.101 5.9 · p.108 5.6 · p.103
NT5	Bronchiectasis, lung abscess and other pulmonary suppurations	5.7 · p.104 5.6 · p.103
NT6	AIDS and opportunistic infections in the immunocompromised host	5.11 · p.110
NT7	Principles governing use of antibiotics	5.1 · p.95
NT8	Other pneumonias, parasitic infections, zoonoses	5.10 · p.109 5.12 · p.113

Syllabus II-B3. Immunological disorders

IM1	Immune defence mechanisms of the lung	2.9 · p.37
IM2	Sarcoidosis	7.5 · p.147
IM3	Hypersensitivity pneumonitis	7.4 · p.145
IM4	Eosinophilic pneumonias and tropical eosinophilia	7.8 · p.152
IM5	Pulmonary vasculitides	7.7 · p.150
IM6	Connective tissue diseases involving the respiratory system	7.6 · p.149
IM7	Interstitial lung disease of other aetiologies; reactions of the interstitium to injury and drugs	7.1 · p.139 7.2 · p.141 7.3 · p.144 7.9 · p.153
IM8	Occupational and environmental pulmonary diseases	8.1 · p.159 8.2 · p.161

Syllabus II-B4. Other non-infectious disorders of lungs and airways

NI1	Aspiration and inhalational (non-occupational) diseases	5.6 · p.103 8.5 · p.165
NI2	Drug-induced pulmonary diseases	7.9 · p.153
NI3	Bullous lung disease	6.14 · p.136
NI4	Uncommon diseases; pulmonary haemorrhagic syndromes	7.7 · p.150 7.11 · p.156
NI5	PLCH, LAM, PAP, alveolar microlithiasis	7.10 · p.155 7.11 · p.156
NI6	Cystic fibrosis and disorders of ciliary motility	5.7 · p.104
NI7	Obesity-related pulmonary disorders	15.7 · p.259 13.4 · p.236
NI8	Upper airway obstruction syndromes	16.4 · p.265
NI9	Air-pollution induced disease, toxic lung and inhalational injuries	8.3 · p.162 8.5 · p.165
NI10	Health hazards of smoking	8.4 · p.164

Syllabus II-B5. Pulmonary circulatory disorders

PC1	Pulmonary hypertension and cor pulmonale	9.1 · p.168 9.2 · p.170 9.3 · p.172 6.10 · p.131
PC2	Pulmonary oedema	9.6 · p.177

PC3	Pulmonary thromboembolic disease and infarction	9.4 · p.173 · 9.5 · p.175
PC4	Cardiac problems in pulmonary patients; pulmonary complications of cardiac disease	9.6 · p.177

Syllabus II-B6. Obstructive diseases

OB1	Asthma incl. ABPA, specific allergen immunotherapy and immunomodulation	6.1 · p.115 · 6.2 · p.117 · 6.3 · p.118 · 6.4 · p.120 · 6.5 · p.122 · 6.6 · p.124
OB2	COPD and diseases of small airways	6.7 · p.125 · 6.8 · p.127 · 6.9 · p.129 · 6.10 · p.131 · 6.11 · p.132 · 6.15 · p.137
OB3	Long-term oxygen therapy, inhalation therapy, pulmonary rehabilitation	6.12 · p.133 · 2.14 · p.44 · 6.13 · p.135

Syllabus II-B7. Tumours of the lungs

LT1	Neoplastic and non-neoplastic lesions: epidemiology, natural history, staging, treatment	10.1 · p.181 · 10.2 · p.183 · 10.3 · p.184 · 10.4 · p.186 · 10.5 · p.187 · 10.6 · p.189 · 10.8 · p.192 · 10.9 · p.193
LT2	Solitary pulmonary nodule	10.7 · p.190

Syllabus II-B8. Diseases of the mediastinum

MD1	Non-neoplastic disorders	11.7 · p.206
MD2	Benign and malignant neoplasms and cysts	11.7 · p.206 · 1.7 · p.18

Syllabus II-B9. Disorders of the pleura

PL1	Pleural dynamics and effusions	11.1 · p.196
PL2	Non-neoplastic and neoplastic pleural diseases	11.2 · p.198 · 11.3 · p.200 · 11.4 · p.202
PL3	Pneumothorax	11.6 · p.204
PL4	Pyothorax and bronchopleural fistula	11.2 · p.198 · 11.6 · p.204
PL5	Fibrothorax	11.5 · p.203

Syllabus II-B10. Critical care pulmonary medicine

CC1	Management of emergencies in pulmonary diseases	9.7 · p.178 · 6.3 · p.118 · 6.9 · p.129
CC2	Acute respiratory distress syndrome	12.5 · p.219
CC3	Respiratory failure in obstructive airway disease and other diseases	12.1 · p.211 · 12.6 · p.221
CC4	Management of sepsis	12.8 · p.224
CC5	Respiratory and haemodynamic monitoring	12.9 · p.225
CC6	Non-invasive and mechanical ventilation	12.3 · p.215 · 12.4 · p.216 · 12.7 · p.222
CC7	Principles of critical care; complications; severity-of-illness scores	12.8 · p.224 · 12.9 · p.225
CC8	Ethical and end-of-life issues in critical care	12.11 · p.228

Syllabus II-B11 to B14

EX1	Extrapulmonary manifestations of pulmonary diseases	15.3 · p.253 · 2.13 · p.43

SL1	Polysomnography	13.1 · p.230
SL2	Sleep apnoeas and other sleep-disordered breathing	13.2 · p.232 · 13.3 · p.234 · 13.4 · p.236
MI1	Diseases of the diaphragm and chest wall	11.8 · p.207 · 11.9 · p.209
MI2	Oxygen therapy; end-of-life care	6.12 · p.133 · 12.11 · p.228
MI3	Aerospace medicine; special environments (high altitude, diving, miners)	15.1 · p.250 · 15.2 · p.252
MI4	Quality-of-life questionnaires; health impacts of global warming	15.6 · p.257 · 8.3 · p.162
PV1	Smoking cessation principles and strategies	8.4 · p.164
PV2	Cardiopulmonary rehabilitation; preventive aspects	6.13 · p.135 · 15.5 · p.255
PV3	Vaccination in pulmonary diseases	15.5 · p.255

Syllabus III. Surgical aspects

SU1	Pre- and post-operative evaluation and management of thoracic surgical patients	16.1 · p.261
SU2	Chest trauma / trauma-related lung dysfunction	16.2 · p.263
SU3	Lung transplantation	16.3 · p.264

Assessment (summative)

Theory — four papers: I General pulmonary medicine & basic sciences; II Clinical pulmonary medicine incl. medical emergencies; III Clinical pulmonary medicine incl. critical care; IV Recent advances & research methodology. Clinical — one long and two short cases; oral/viva voce. Thesis to be accepted before the examination; 50% in theory and practical separately to pass.

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Abbreviations

AASM American Academy of Sleep Medicine
ABG Arterial blood gas
ABPA Allergic bronchopulmonary aspergillosis
ACE Angiotensin-converting enzyme
ACF Active case finding
ACO Asthma–COPD overlap
ACTH Adrenocorticotropic hormone
ADA Adenosine deaminase
ADR Adverse drug reaction
AE-IPF Acute exacerbation of idiopathic pulmonary fibrosis
AECOPD Acute exacerbation of COPD
AEP Acute eosinophilic pneumonia
AFB Acid-fast bacilli
AHI Apnoea–hypopnoea index
AIIR Airborne infection isolation room
AIP Acute interstitial pneumonia
AKI Acute kidney injury
ALK Anaplastic lymphoma kinase
AMS Acute mountain sickness
ANCA Anti-neutrophil cytoplasmic antibody
APC Argon plasma coagulation
APS Antiphospholipid syndrome
AQI Air Quality Index
ARDS Acute respiratory distress syndrome
ART Antiretroviral therapy
ASV Adaptive servo-ventilation
ATS American Thoracic Society
ATT Anti-tuberculosis treatment
BAE Bronchial artery embolisation
BAL Bronchoalveolar lavage
BCG Bacille Calmette–Guérin
BDQ Bedaquiline
BHD Birt–Hogg–Dubé syndrome
BiPAP Bilevel positive airway pressure
BLVR Bronchoscopic lung volume reduction
BMI Body mass index
BNP B-type natriuretic peptide
BOS Bronchiolitis obliterans syndrome
BPF Bronchopleural fistula
BTS British Thoracic Society
CAO Central airway obstruction
CAP Community-acquired pneumonia
CAPA COVID-19-associated pulmonary aspergillosis
CAT COPD Assessment Test
CBNAAT Cartridge-based nucleic acid amplification test
CECT Contrast-enhanced CT
CF Cystic fibrosis
CFTR Cystic fibrosis transmembrane conductance regulator
CKD Chronic kidney disease
CMV Cytomegalovirus
COP Cryptogenic organising pneumonia
COPD Chronic obstructive pulmonary disease
COTPA Cigarettes and Other Tobacco Products Act
CPA Chronic pulmonary aspergillosis
CPAM Congenital pulmonary airway malformation
CPAP Continuous positive airway pressure
CPET Cardiopulmonary exercise testing
CPFE Combined pulmonary fibrosis and emphysema
CRP C-reactive protein
CSA Central sleep apnoea
CT Computed tomography
CTD Connective tissue disease
CTEPH Chronic thromboembolic pulmonary hypertension
CTPA CT pulmonary angiography
CWP Coal workers' pneumoconiosis
CXR Chest X-ray
DAD Diffuse alveolar damage
DAH Diffuse alveolar haemorrhage
DBT Direct benefit transfer
DILI Drug-induced liver injury
DIP Desquamative interstitial pneumonia
DKA Diabetic ketoacidosis
DLCO Diffusing capacity of the lung for carbon monoxide
DOAC Direct oral anticoagulant
DOTS Directly observed treatment, short-course
DR-TB Drug-resistant tuberculosis
DST Drug susceptibility testing
DVT Deep vein thrombosis
EBUS Endobronchial ultrasound
ECCO2R Extracorporeal CO2 removal
ECMO Extracorporeal membrane oxygenation
EGFR Epidermal growth factor receptor
EGPA Eosinophilic granulomatosis with polyangiitis
EPAP Expiratory positive airway pressure
EPTB Extrapulmonary tuberculosis
ERS European Respiratory Society
ERV Expiratory reserve volume
ESC European Society of Cardiology
ETT Endotracheal tube
FDC Fixed-dose combination
FeNO Fractional exhaled nitric oxide
FEV1 Forced expiratory volume in 1 second
FNAC Fine-needle aspiration cytology
FQ Fluoroquinolone
FRC Functional residual capacity
FVC Forced vital capacity
GERD Gastro-oesophageal reflux disease
GGO Ground-glass opacity
GINA Global Initiative for Asthma
GM Galactomannan
GOLD Global Initiative for Chronic Obstructive Lung Disease
GPA Granulomatosis with polyangiitis
HACE High-altitude cerebral oedema
HAP Hospital-acquired pneumonia
HAPE High-altitude pulmonary oedema
HES Hypereosinophilic syndrome
HFNC High-flow nasal cannula
HHT Hereditary haemorrhagic telangiectasia
HIT Heparin-induced thrombocytopenia
HP Hypersensitivity pneumonitis
HPS Hepatopulmonary syndrome
HRCT High-resolution computed tomography
HSCT Haematopoietic stem cell transplantation
IASLC International Association for the Study of Lung Cancer
ICD Intercostal drain
ICMR Indian Council of Medical Research
ICS Inhaled corticosteroid
IGRA Interferon-gamma release assay

ILD Interstitial lung disease
ILO International Labour Organization; inducible laryngeal obstruction
IOS Impulse oscillometry
IPAF Interstitial pneumonia with autoimmune features
IPAP Inspiratory positive airway pressure
IPC Indwelling pleural catheter
IPF Idiopathic pulmonary fibrosis
IRIS Immune reconstitution inflammatory syndrome
LABA Long-acting β2-agonist
LAM Lymphangioleiomyomatosis; lipoarabinomannan
LAMA Long-acting muscarinic antagonist
LDCT Low-dose CT
LFT Liver function tests
LIP Lymphocytic interstitial pneumonia
LLN Lower limit of normal
LMWH Low-molecular-weight heparin
LPA Line probe assay
LTOT Long-term oxygen therapy
LVRS Lung volume reduction surgery
MAC Mycobacterium avium complex
MART Maintenance and reliever therapy
MCID Minimal clinically important difference
MDD Multidisciplinary discussion
MDR-TB Multidrug-resistant tuberculosis
MGIT Mycobacteria growth indicator tube
MIP Maximal inspiratory pressure
MPE Malignant pleural effusion
MRSA Methicillin-resistant Staphylococcus aureus
MSLT Multiple sleep latency test
MV Mechanical ventilation
NAAT Nucleic acid amplification test
NIV Non-invasive ventilation
NMC National Medical Commission
NPY Ni-kshay Poshan Yojana
NSCLC Non-small cell lung cancer
NSIP Non-specific interstitial pneumonia
NTEP National Tuberculosis Elimination Programme
NTM Non-tuberculous mycobacteria
OCS Oral corticosteroid
ODC Oxyhaemoglobin dissociation curve
OHS Obesity hypoventilation syndrome
OP Organising pneumonia
OSA Obstructive sleep apnoea
PAH Pulmonary arterial hypertension
PAP Pulmonary alveolar proteinosis; positive airway pressure
PAVM Pulmonary arteriovenous malformation
PAWP Pulmonary artery wedge pressure
PCD Primary ciliary dyskinesia
PCV Pneumococcal conjugate vaccine
PE Pulmonary embolism
PEEP Positive end-expiratory pressure
PEF Peak expiratory flow
PET Positron emission tomography
PFT Pulmonary function test
PH Pulmonary hypertension
PJP Pneumocystis jirovecii pneumonia
PLCH Pulmonary Langerhans cell histiocytosis
PLHIV People living with HIV
PPF Progressive pulmonary fibrosis
PRISm Preserved ratio impaired spirometry
PSG Polysomnography
PSV Pressure support ventilation
PTLD Post-TB lung disease; post-transplant lymphoproliferative disorder
PVR Pulmonary vascular resistance
RCT Randomised controlled trial
REM Rapid eye movement (sleep)
RNTCP Revised National TB Control Programme
RR Respiratory rate; relative risk
RR-TB Rifampicin-resistant tuberculosis
RSV Respiratory syncytial virus
RV Residual volume; right ventricle
SABA Short-acting β2-agonist
SBRT Stereotactic body radiotherapy
SBT Spontaneous breathing trial
SCLC Small cell lung cancer
SIADH Syndrome of inappropriate antidiuretic hormone
SLE Systemic lupus erythematosus
SOFA Sequential Organ Failure Assessment
SPN Solitary pulmonary nodule
SSc Systemic sclerosis
SVC Superior vena cava
TBLB Transbronchial lung biopsy
TBM Tuberculous meningitis
TBNA Transbronchial needle aspiration
TEF Tracheo-oesophageal fistula
TKI Tyrosine kinase inhibitor
TLC Total lung capacity
TNM Tumour–node–metastasis staging
TPE Tropical pulmonary eosinophilia
TPT TB preventive treatment
TRALI Transfusion-related acute lung injury
TST Tuberculin skin test
UIP Usual interstitial pneumonia
USG Ultrasonography
V/Q Ventilation–perfusion
VAP Ventilator-associated pneumonia
VATS Video-assisted thoracoscopic surgery
VC Vital capacity
VILI Ventilator-induced lung injury
VT Tidal volume
VTE Venous thromboembolism
WHO World Health Organization
XDR-TB Extensively drug-resistant tuberculosis

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PART II

Advanced Theory, Clinical Algorithms & Image Atlas

Extended answers · step-by-step decision algorithms · model-answer frameworks · Radiopaedia image links · Merck Manual Professional links

Part I gives you the core of every CBME competency. Part II takes the topics that examiners return to year after year and develops them to the depth expected in a 10-mark MD/DNB answer and in the clinical examination. Each entry is cross-linked to its core topic in Part I (tap ‹ **Core topic**) and to the previous-year questions of its section.

- **Algorithms** — every major emergency and diagnostic pathway drawn as a decision chart.
- **Model answer frameworks** — the order in which to write a long answer, so that no marks are left on the table.
- **Radiopaedia links** — tap any pink chip to open articles and real cases (CXR, CT, HRCT, ultrasound) on *radiopaedia.org*; tap “cases” to go straight to image collections.
- **Merck Manual links** — tap any blue chip to open the corresponding disease chapter search in the *Merck Manual Professional Version*.

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Contents — Part II

Section 1 · Applied Anatomy, Embryology & Developmental Anomalies 315

E1.1 Bronchoscopic Anatomy & Segmental Nomenclature for the Bronchoscopist 315
E1.2 Mediastinal Compartments (ITMIG) & IASLC Lymph-Node Map 317
E1.3 Applied Anatomy for Pleural Procedures, Intercostal Drains & Tracheostomy 318
E1.4 Congenital Thoracic Malformations — Imaging-Based Approach 321
E1.5 Drug Chart — Drugs for Bronchoscopy, Pleural & Airway Procedures 322

Section 2 · Respiratory Physiology & Pulmonary Function 324

E2.1 Stepwise Interpretation of Pulmonary Function Tests (ATS/ERS 2022) 324
E2.2 Diffusing Capacity (DLCO) — Interpretation & Clinical Patterns 327
E2.3 Approach to Hypoxaemia — A–a Gradient Algorithm & Worked Examples 328
E2.4 Arterial Blood Gas — Systematic Six-Step Analysis 331
E2.5 Cardiopulmonary Exercise Testing (CPET) — Interpretation Algorithm 332
E2.6 Respiratory Muscle Weakness — Physiology & Clinical Evaluation 334
E2.7 Approach to Chronic Breathlessness — Physiology to Bedside 336
E2.8 Drug Chart — Drugs Used in Pulmonary Function & Physiological Testing 337
E2.9 Oxygen Transport, the Dissociation Curve & Tissue Delivery 338

Section 3 · Microbiology, Pathology, Immunology & Pharmacology 341

E3.1 Reading TB Laboratory Reports — Smear, CBNAAT, Truenat, LPA, Culture & WGS 341
E3.2 Anti-TB Drug Adverse Events — Recognition & Management Algorithms 342
E3.3 Inhaler Devices, Aerosol Science & Device Selection 345
E3.4 Biologics in Airway & Lung Disease — Pharmacology Table 347
E3.5 Antifungal Pharmacology & Therapeutic Drug Monitoring 348
E3.6 Drug Chart — First-Line & Second-Line Anti-TB Drugs (NTEP Weight Bands) 350
E3.7 Corticosteroids & Immunosuppression in Respiratory Practice — Safe Prescribing 352

Section 4 · Tuberculosis 354

E4.1 Diagnosis of TB in Special Groups — Children, EPTB & Smear-Negative TB 354
E4.2 DS-TB Treatment Monitoring, Outcomes & Programmatic Indicators 355
E4.3 Drug-Resistant TB — Regimen Selection Algorithm (NTEP / WHO 2024–25) 357
E4.4 TB Preventive Treatment & Contact Investigation — Algorithm 360
E4.5 Tuberculous Pleural Effusion — Diagnostic & Management Algorithm 361
E4.6 Post-TB Lung Disease, Haemoptysis & Surgery in TB 362
E4.7 Drug Chart — Drugs in TB Prevention, Adjunctive & Supportive Care 364

Section 5 · Non-Tuberculous Infections of the Lung 365

E5.1 Community-Acquired Pneumonia — Severity, Site of Care & Empirical Therapy 365
E5.2 HAP & VAP — Diagnosis, Risk-Based Empirical Therapy & De-escalation 367
E5.3 Non-Resolving Pneumonia — Systematic Approach 368
E5.4 Bronchiectasis — Evaluation, Exacerbation & Long-term Management 370
E5.5 Chronic Pulmonary Aspergillosis & Mucormycosis — Diagnostic Algorithms 371
E5.6 Pulmonary Infiltrates in the Immunocompromised Host — Algorithm 372
E5.7 Drug Chart — Antimicrobials for Respiratory Infections 375
E5.8 Viral Pneumonias, Influenza & COVID-19 — Management Algorithms 376
E5.9 Tropical & Zoonotic Pneumonias in India 377

Section 6 · Obstructive Airway Diseases 380

E6.1 Diagnosis of Asthma — GINA 2024–25 Algorithm 380
E6.2 Stepwise Management of Chronic Asthma — GINA Track 1 & Track 2 382
E6.3 Acute Severe Asthma — Emergency Department Algorithm 383
E6.4 COPD — GOLD 2025 Assessment & Pharmacological Algorithm 386
E6.5 Acute Exacerbation of COPD — Hospital Algorithm & NIV Decisions 387
E6.6 ABPA — ISHAM-AWG 2024 Criteria & Management 389
E6.7 Long-Term Oxygen Therapy & Pulmonary Rehabilitation — Prescribing Algorithm 391
E6.8 Drug Chart — Inhaled & Oral Drugs for Asthma and COPD 392
E6.9 Severe Asthma — Phenotyping, Comorbidities & the Difficult-Asthma Clinic 393
E6.10 Asthma–COPD Overlap, Small-Airway Disease & Bronchiolitis 395

Section 7 · Interstitial Lung Diseases & Immunological Disorders 397

AETCM Academy for Emergency, Trauma & Critical care Medicine

E7.1 ILD — Diagnostic Algorithm & the Multidisciplinary Discussion 397
E7.2 HRCT Patterns in Fibrosing ILD — UIP Categories 399
E7.3 IPF & Progressive Pulmonary Fibrosis — Treatment & Acute Exacerbation Algorithm 400
E7.4 Hypersensitivity Pneumonitis — ATS/JRS/ALAT 2020 Algorithm 402
E7.5 Sarcoidosis — Organ Assessment & Treatment Algorithm (ERS 2021) 403
E7.6 CTD-ILD, Diffuse Alveolar Haemorrhage & Cystic Lung Disease — Algorithms 404
E7.7 Drug Chart — Immunosuppressants & Antifibrotics in ILD 407
E7.8 Eosinophilic Lung Disease & Pulmonary Vasculitis — Approach 408

Section 8 · Occupational & Environmental Lung Disease, Smoking & Inhalational Injury 410

E8.1 Occupational History, ILO Classification & Silicosis in India 410
E8.2 Asbestos-Related Diseases & Other Pneumoconioses 411
E8.3 Smoking Cessation — 5A/5R Model & Pharmacotherapy 413
E8.4 Toxic Inhalational Injury, Smoke Inhalation & Air Pollution 415
E8.5 Drug Chart — Smoking Cessation & Inhalational Injury Antidotes 416

Section 9 · Pulmonary Circulation — Hypertension, Embolism, Oedema & Haemoptysis 417

E9.1 Pulmonary Hypertension — ESC/ERS 2022 Diagnostic Algorithm 417
E9.2 PAH — Risk Stratification & Treatment Algorithm 418
E9.3 Pulmonary Embolism — Diagnostic Algorithm 420
E9.4 PE — Risk Stratification, Reperfusion & Anticoagulation Duration 422
E9.5 Massive Haemoptysis — Emergency Algorithm 423
E9.6 Drug Chart — Anticoagulants, Thrombolytics & PAH-Specific Therapy 424
E9.7 CTEPH & Pulmonary Oedema — Diagnostic and Management Pathways 426

Section 10 · Lung Cancer & Other Thoracic Tumours 428

E10.1 Solitary Pulmonary Nodule — Fleischner 2017 & BTS Management Algorithm 428
E10.2 Lung Cancer — Diagnosis & Staging Algorithm (TNM 9th Edition) 429
E10.3 NSCLC & SCLC — Stage-Wise Treatment Algorithm 431
E10.4 Thoracic Oncological Emergencies — SVCO, Central Airway Obstruction & Paraneoplastic Crises 433
E10.5 Lung Cancer Screening & Prevention 434
E10.6 Drug Chart — Systemic Therapy in Thoracic Oncology 435
E10.7 Immunotherapy Toxicity & Thoracic Radiation Injury 437

Section 11 · Pleura, Mediastinum, Diaphragm & Chest Wall 439

E11.1 Pleural Effusion — Diagnostic Algorithm Beyond Light's Criteria 439
E11.2 Pleural Infection & Empyema — BTS 2023 Algorithm & MIST-2 440
E11.3 Malignant Pleural Effusion & Pneumothorax — Management Algorithms 442
E11.4 Chylothorax, Pseudochylothorax, Haemothorax & Mesothelioma 444
E11.5 Diaphragm, Mediastinitis & Chest Wall — Clinical Algorithms 445
E11.6 Drug Chart — Drugs Used in Pleural Disease 447
E11.7 Pneumothorax — Physiology, Special Situations & Recurrence Prevention 448

Section 12 · Critical Care Pulmonary Medicine 450

E12.1 Acute Hypoxaemic Respiratory Failure — Oxygen, HFNC & NIV Decision Algorithm 450
E12.2 Mechanical Ventilation — Initial Settings & Waveform Troubleshooting 451
E12.3 ARDS — Berlin to Global Definition & Management Algorithm 454
E12.4 Weaning, Spontaneous Breathing Trials & Extubation Failure 457
E12.5 Sepsis & Septic Shock — Surviving Sepsis Campaign Bundle 458
E12.6 Difficult Airway — Prediction & Unanticipated Difficult Intubation Algorithm 459
E12.7 Respiratory ICU Monitoring — Capnography, Lung Ultrasound & Haemodynamics 461
E12.8 Drug Chart — Respiratory ICU Drugs 462
E12.9 Ventilation in Obstructive Airway Disease — Auto-PEEP & Permissive Hypercapnia 463
E12.10 Extracorporeal Support & Rescue Therapies in Respiratory Failure 465
E12.11 Non-Invasive Ventilation — Modes, Interfaces & Setting Up at the Bedside 466

Section 13 · Sleep-Related Breathing Disorders 468

E13.1 Obstructive Sleep Apnoea — Diagnostic Algorithm (HSAT vs PSG) 468
E13.2 PAP Therapy, Alternatives & Troubleshooting 469
E13.3 Obesity Hypoventilation, Central Sleep Apnoea & Hypersomnolence 470
E13.4 Drug Chart — Drugs in Sleep-Disordered Breathing 473
E13.5 Sleep Physiology & the Polysomnogram — Reading the Report 473

Section 14 · Thoracic Imaging & Pulmonary Procedures 476

E14.1 Systematic Chest Radiograph Interpretation — with Radiopaedia Image Links 476
E14.2 HRCT Patterns — Nodules, Lines, Cysts & Ground Glass 478
E14.3 Thoracic Ultrasound — BLUE Protocol, Effusion, Pneumothorax & eFAST 479
E14.4 Bronchoscopy, EBUS-TBNA & Cryobiopsy — Indications, Technique & Complications 481
E14.5 Medical Thoracoscopy & Intercostal Drains — Procedure Guide 482
E14.6 Imaging Atlas — Key Radiological Signs for Viva (Radiopaedia Links) 483
E14.7 Drug Chart — Contrast Agents, Radiopharmaceuticals & Procedure Premedication 486
E14.8 Interventional Pulmonology — Stents, Valves, Thermoplasty & Whole-Lung Lavage 486

Section 15 · Special Environments, Systemic Disease, Pregnancy & Preventive Pulmonology 488

E15.1 High-Altitude Illness & Flight Fitness — Algorithms 488
E15.2 Respiratory Disease in Pregnancy — Management Algorithm 489
E15.3 Pulmonary Manifestations of Systemic Disease 491
E15.4 Chronic Cough — Diagnostic Algorithm & Treatment of Refractory Cough 493
E15.5 Vaccination in Respiratory Disease 494
E15.6 Drug Chart — Altitude, Cough, Vaccines & Systemic Disease 495
E15.7 Obesity, Metabolic Disease & the Lung 496

Section 16 · Surgical Aspects of Pulmonary Medicine 498

E16.1 Pre-operative Pulmonary Evaluation for Lung Resection — Three-Legged Stool 498
E16.2 Chest Trauma — Primary Survey, Thoracostomy & Operative Triggers 499
E16.3 Lung Transplantation — Referral, Listing & Complications 501
E16.4 Upper Airway Obstruction & Stridor — Emergency Algorithm 503
E16.5 Drug Chart — Peri-operative, Trauma & Transplant Drugs 504

Section 17 · Research Methodology, Epidemiology & Biostatistics 506

E17.1 Study Designs, Bias & Confounding — Applied to Respiratory Research 506
E17.2 Biostatistics for the Pulmonologist — Tests, Sample Size & Diagnostic Accuracy 507
E17.3 Critical Appraisal, Trial Ethics & Writing the Thesis Protocol 509
E17.4 Drug Chart — Pharmacovigilance, Drug Trials & Reporting in India 512

Section 18 · Clinical Examination in Respiratory Medicine — Long Case, Short Case & Viva 513

E18.1 Respiratory History Taking for the Long Case 513
E18.2 General Physical Examination — Respiratory Focus 514
E18.3 Examination of the Respiratory System — Inspection & Palpation 516
E18.4 Examination — Percussion, Auscultation & Putting It Together 518
E18.5 Presenting the Long Case & Common Viva Questions 520
E18.6 Short Cases, Spotters & Procedure Stations 522
E18.7 Rapid Revision — One-Page Answers to the Twenty Commonest Questions 525
E18.8 Bedside Interpretation Stations — ABG, CXR, Spirometry & Ventilator Graphics 526

Section 19 · Oxygen Delivery, Non-Invasive Support & Mechanical Ventilation — A Practical Handbook 528

E19.1 Oxygen Delivery Devices — Principles & How to Choose 528
E19.2 Device 1 · Nasal Cannula (Nasal Prongs) 531
E19.3 Device 2 · Simple Face Mask 532
E19.4 Device 3 · Partial Rebreathing Mask 534
E19.5 Device 4 · Non-Rebreather Mask (NRB) 535
E19.6 Device 5 · Reservoir Cannula (Oxymizer) & Pulse-Dose Devices 536
E19.7 Device 6 · Venturi Mask (Air-Entrainment Mask) 538
E19.8 Device 7 · High-Flow Nasal Cannula (HFNC) 539
E19.9 Device 8 · Tracheostomy Mask, T-Piece & Face Tent 541
E19.10 Device 9 · Bag-Valve-Mask & Manual Ventilation Devices 543
E19.11 Device 10 · Oxygen Sources — Cylinders, Concentrators & Liquid Oxygen 544
E19.12 Oxygen Prescription, Weaning & Hazards 545
E19.13 Non-Invasive Ventilation 1 · Principles, CPAP vs BiPAP 548
E19.14 Non-Invasive Ventilation 2 · Indications, Contraindications & Interfaces 549
E19.15 Non-Invasive Ventilation 3 · Setting Up CPAP & BiPAP at the Bedside 551
E19.16 Non-Invasive Ventilation 4 · Troubleshooting & Weaning 553
E19.17 Non-Invasive Ventilation 5 · Domiciliary NIV, CPAP for OSA & Cough Augmentation 554
E19.18 Invasive Mechanical Ventilation 1 · Indications & The Four Basic Questions 555
E19.19 Invasive Mechanical Ventilation 2 · Modes Explained 557
E19.20 Invasive Mechanical Ventilation 3 · Disease-Specific Ventilator Settings 559
E19.21 Invasive Mechanical Ventilation 4 · Alarms, Complications & Daily Care 560
E19.22 Rapid Sequence Intubation 1 · Preparation & The Seven Ps 562

E19.23 Rapid Sequence Intubation 2 · Drug Table with Doses & Cautions 563
E19.24 Rapid Sequence Intubation 3 · Delayed & Awake Techniques, Failed Airway 564
E19.25 Quick Reference — Oxygen, NIV & Ventilation on One Spread 567

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PART II · SECTION 1

Applied Anatomy, Embryology & Developmental Anomalies

Advanced theory & algorithms

E1.1 Bronchoscopic Anatomy & Segmental Nomenclature for the Bronchoscopist 315
E1.2 Mediastinal Compartments (ITMIG) & IASLC Lymph-Node Map 317
E1.3 Applied Anatomy for Pleural Procedures, Intercostal Drains & Tracheostomy 318
E1.4 Congenital Thoracic Malformations — Imaging-Based Approach 321
E1.5 Drug Chart — Drugs for Bronchoscopy, Pleural & Airway Procedures 322

E1.1 Bronchoscopic Anatomy & Segmental Nomenclature for the Bronchoscopist

‹ Core topic 1.3 · p.13
Previous-year Qs ›

IN BRIEF Every bronchoscopy report, every EBUS station and every lobar collapse on a radiograph is described in the language of segmental anatomy. Learn the airway tree as the bronchoscope sees it — from the carina outwards, clockwise, with the patient supine.

The airway as seen through the bronchoscope

- **Trachea:** 10–12 cm long, 16–20 C-shaped cartilage rings, posterior membranous wall (trachealis). Carina at T4–T5 (sternal angle); normal carina is sharp — widening suggests subcarinal nodes or left atrial enlargement.
- **Right main bronchus:** shorter (≈2.5 cm), wider and more vertical (≈25° from the midline) — the usual site of aspirated foreign bodies and of an endotracheal tube advanced too far.
- **Left main bronchus:** longer (≈5 cm), passes under the aortic arch, ≈45° from the midline; the left recurrent laryngeal nerve hooks around the arch just above it.
- **Right upper lobe (RUL) orifice** arises 1–2 cm from the carina at 3 o'clock (patient supine, operator at the head) — the classic "Mercedes-Benz" trifurcation into apical (B1), posterior (B2) and anterior (B3).
- **Bronchus intermedius** continues 2–3 cm to give the right middle lobe (RML: lateral B4, medial B5) anteriorly and the right lower lobe (RLL) posteriorly; the superior segment of RLL (B6) arises opposite the RML orifice.
- **Left upper lobe** divides into upper division (apicoposterior B1+2, anterior B3) and lingula (superior B4, inferior B5) — the lingula is the left-sided counterpart of the middle lobe.
- **Basal segments:** right — medial (B7), anterior (B8), lateral (B9), posterior (B10); left — anteromedial (B7+8), lateral (B9), posterior (B10).

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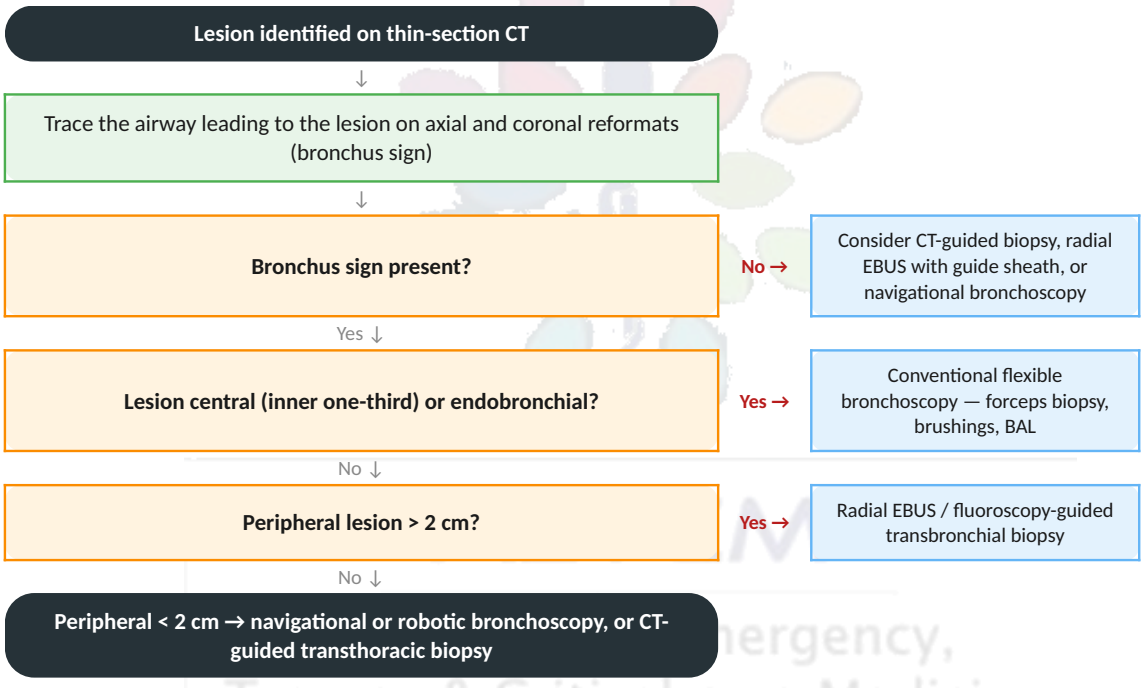
Segmental bronchi (Jackson–Huber nomenclature) and their applied relevance

Lobe	Segments	Applied relevance
RUL	Apical B1, posterior B2, anterior B3	Post-primary TB (apical and posterior), silicosis, upper-lobe fibrosis
RML	Lateral B4, medial B5	Middle-lobe syndrome — long, narrow bronchus surrounded by nodes; silhouettes the right heart border
RLL	Superior B6, medial basal B7, anterior B8, lateral B9, posterior B10	Aspiration supine → B6 and B10; aspiration upright → basal segments
LUL	Apicoposterior B1+2, anterior B3, superior lingular B4, inferior lingular B5	Lingular collapse silhouettes the left heart border
LLL	Superior B6, anteromedial B7+8, lateral B9, posterior B10	LLL collapse — "sail sign" behind the heart, loss of the medial hemidiaphragm

Where things go — aspiration and gravity

- **Supine patient:** posterior segment of the upper lobes (B2) and superior segment of the lower lobes (B6) — the commonest sites of aspiration pneumonia and lung abscess in alcoholics and after seizures.
- **Upright patient:** basal segments of the right lower lobe (B9, B10).
- **Lateral decubitus:** posterior and axillary sub-segments of the upper lobe on the dependent side.

Algorithm E1.1.1 · Locating a lesion for bronchoscopy from the CT



★ CLINICAL PEARLS & EXAM FOCUS

- Describe bronchoscopic findings systematically: vocal cords → trachea → carina → right side → left side; abnormal side last to avoid seeding.
- A "positive bronchus sign" on CT roughly doubles the diagnostic yield of bronchoscopy for a peripheral lesion.
- The RML bronchus is long and narrow, with a ring of lymph nodes — hence recurrent RML collapse (Brock's middle-lobe syndrome) with TB nodes.

RADIOPAEDIA · IMAGE LINKS Bronchopulmonary segments cases | Middle lobe syndrome cases | Bronchus sign cases

E1.2 Mediastinal Compartments (ITMIG) & IASLC Lymph-Node Map

‹ Core topic 1.5 · p.16
Previous-year Qs ›

IN BRIEF The modern three-compartment classification of the International Thymic Malignancy Interest Group (ITMIG) is CT-based and replaces the old four-compartment radiographic scheme. The IASLC lymph-node map is the language of lung-cancer staging and EBUS.

ITMIG mediastinal compartments and their usual masses

Compartment	Boundaries / contents	Common lesions
Prevascular (anterior)	Sternum to anterior pericardium; thymus, fat, lymph nodes, left brachiocephalic vein	"4 Ts" — thymoma/thymic carcinoma, teratoma and other germ-cell tumours, "terrible" lymphoma, thyroid (retrosternal goitre)
Visceral (middle)	Pericardium, heart, great vessels, trachea, oesophagus, nodes; posterior boundary is a line 1 cm behind the anterior vertebral margin	Lymphadenopathy (TB, sarcoid, lymphoma, metastases), bronchogenic and pericardial cysts, oesophageal lesions, aortic aneurysm
Paravertebral (posterior)	Paravertebral soft tissue, posterior to the visceral compartment line	Neurogenic tumours (schwannoma, neurofibroma, ganglioneuroma, neuroblastoma), extramedullary haematopoiesis, paraspinal abscess (Pott's)

IASLC lymph-node stations (2009 map, used in TNM 8th and 9th editions)

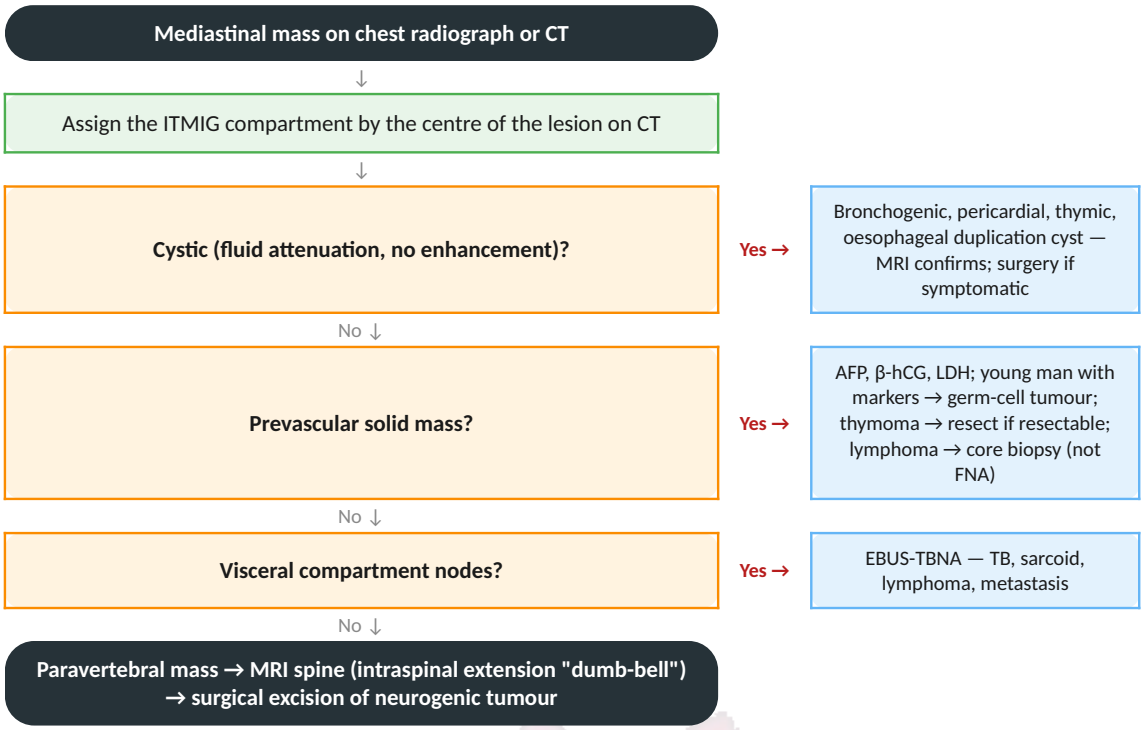
Lymph-node stations — how they are reached

Station	Name	Access
1	Low cervical, supraclavicular, sternal notch	Ultrasound-guided FNA
2R / 2L	Upper paratracheal	EBUS, cervical mediastinoscopy
3a / 3p	Prevascular / retrotracheal	CT-guided or EUS (3p)
4R / 4L	Lower paratracheal (4L lies left of the tracheal midline, medial to the ligamentum arteriosum)	EBUS, mediastinoscopy
5	Subaortic (aorto-pulmonary window)	Anterior mediastinotomy (Chamberlain), VATS; not reached by EBUS
6	Para-aortic (ascending aorta / phrenic)	Chamberlain procedure, VATS
7	Subcarinal	EBUS, EUS, mediastinoscopy
8	Para-oesophageal (below carina)	EUS-B / EUS
9	Pulmonary ligament	EUS, VATS
10	Hilar	EBUS
11–14	Interlobar, lobar, segmental, subsegmental (N1)	EBUS (10, 11), surgical

- **N1** = stations 10–14 ipsilateral; **N2** = ipsilateral mediastinal or subcarinal (2, 3, 4, 5, 6, 7, 8, 9); **N3** = contralateral mediastinal/hilar or any scalene/supraclavicular (1).
- The 9th edition subdivides N2 into **N2a** (single N2 station) and **N2b** (multiple N2 stations).

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Algorithm E1.2.1 · Approach to a mediastinal mass on CT



★ **CLINICAL PEARLS & EXAM FOCUS**

- **Anterior mediastinal mass + myasthenia** = thymoma until proved otherwise; check anti-AChR antibodies.
- Never give general anaesthesia lightly to a patient with a large anterior mass — risk of complete airway or SVC collapse on induction.
- Hilum overlay sign: hilar vessels visible through a mass → the mass is anterior or posterior, not hilar.

RADIOPAEDIA · IMAGE LINKS Mediastinal compartments · cases · Hilum overlay sign · cases · Thymoma · cases · IASLC lymph node map · cases

MERCK MANUAL PROFESSIONAL Mediastinal masses

E1.3 Applied Anatomy for Pleural Procedures, Intercostal Drains & Tracheostomy

‹ Core topic 1.6 · p.17
Previous-year Qs ›

IN BRIEF Most complications of thoracentesis, chest-drain insertion and tracheostomy are anatomical. Knowing the neurovascular bundle, the safe triangle and the tracheal landmarks prevents them.

The intercostal space

- The neurovascular bundle runs in the **costal groove on the inferior border of each rib**, in the order vein–artery–nerve (VAN) from above downwards, between the internal and innermost intercostal muscles.
- Needles and drains are therefore passed **just above the upper border of the lower rib**.
- Posteriorly (within 6 cm of the spine) the intercostal artery is tortuous and lies unprotected in the middle of the space — avoid medial posterior punctures.

- Collateral branches run along the upper border of the lower rib in the mid-axillary line — another reason for ultrasound guidance.

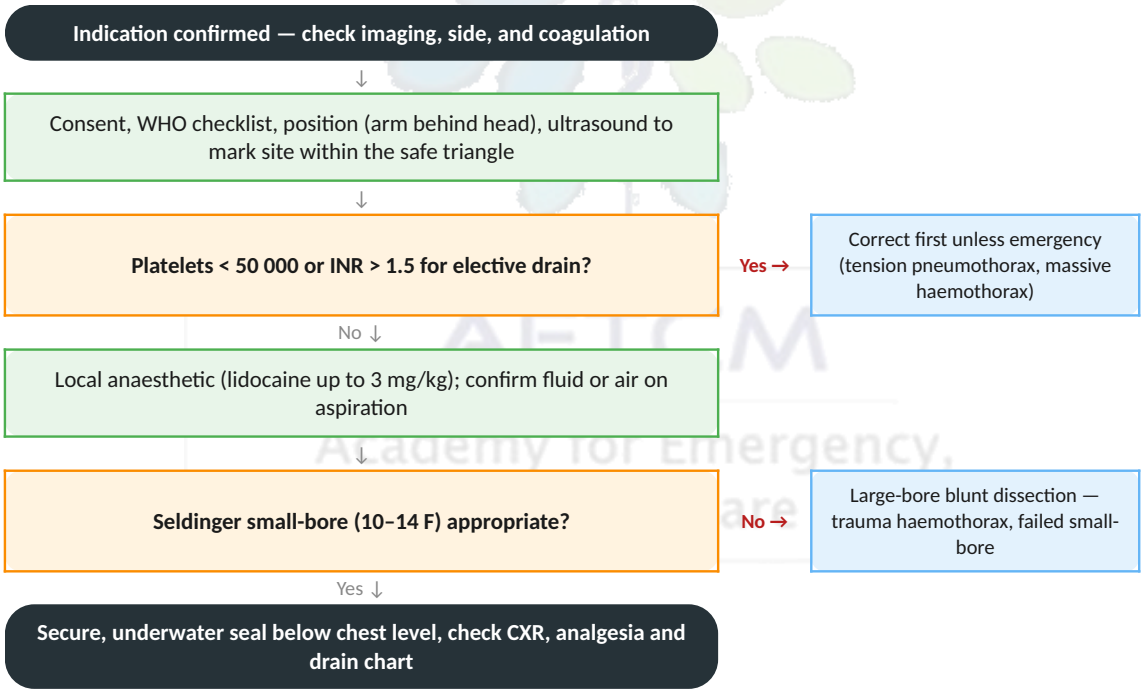
The safe triangle

- **Boundaries:** anterior border of latissimus dorsi, lateral border of pectoralis major, a horizontal line at the level of the nipple (5th intercostal space); apex below the axilla.
- Avoids the long thoracic nerve (behind the mid-axillary line), the internal mammary vessels and the diaphragm, liver and spleen.
- The 4th or 5th intercostal space in the mid-axillary line is standard for blunt dissection drains.

Applied anatomy of common thoracic procedures

Procedure	Landmark	Structure at risk
Thoracentesis	1–2 spaces below the top of the effusion, posterior axillary line, ultrasound-marked	Intercostal artery, lung, liver/spleen (low punctures)
Needle decompression	2nd intercostal space mid-clavicular line, or 4th–5th space anterior axillary line (ATLS 10th ed. adults)	Internal mammary artery if too medial
Intercostal drain	Safe triangle, above the rib	Long thoracic nerve, intercostal bundle, diaphragm
Pleural biopsy (Abrams)	Over the rib, cutting notch downwards	Intercostal bundle above
Percutaneous tracheostomy	Between 1st–2nd or 2nd–3rd tracheal rings	Thyroid isthmus, anterior jugular veins, brachiocephalic artery (too low)
Cricothyroidotomy	Cricothyroid membrane	Cricothyroid arteries (upper third)

Algorithm E1.3.1 · Safe chest-drain insertion (BTS 2023)



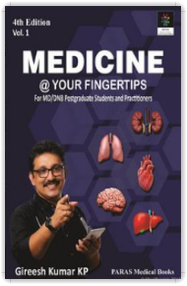
★ CLINICAL PEARLS & EXAM FOCUS

- Never insert a drain without aspirating air or fluid first — "no aspirate, no drain".
- Re-expansion pulmonary oedema: drain no more than 1.5 L at one go, stop if chest discomfort or cough.
- Tracheostomy below the 4th ring risks tracheo-innominate fistula — herald bleed after day 3 is an emergency.

RADIOPAEDIA · IMAGE LINKS Intercostal chest drain cases · Safe triangle cases · Tracheostomy tube position cases

MERCK MANUAL PROFESSIONAL How to do thoracentesis · Tube thoracostomy

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E1.4 Congenital Thoracic Malformations — Imaging-Based Approach

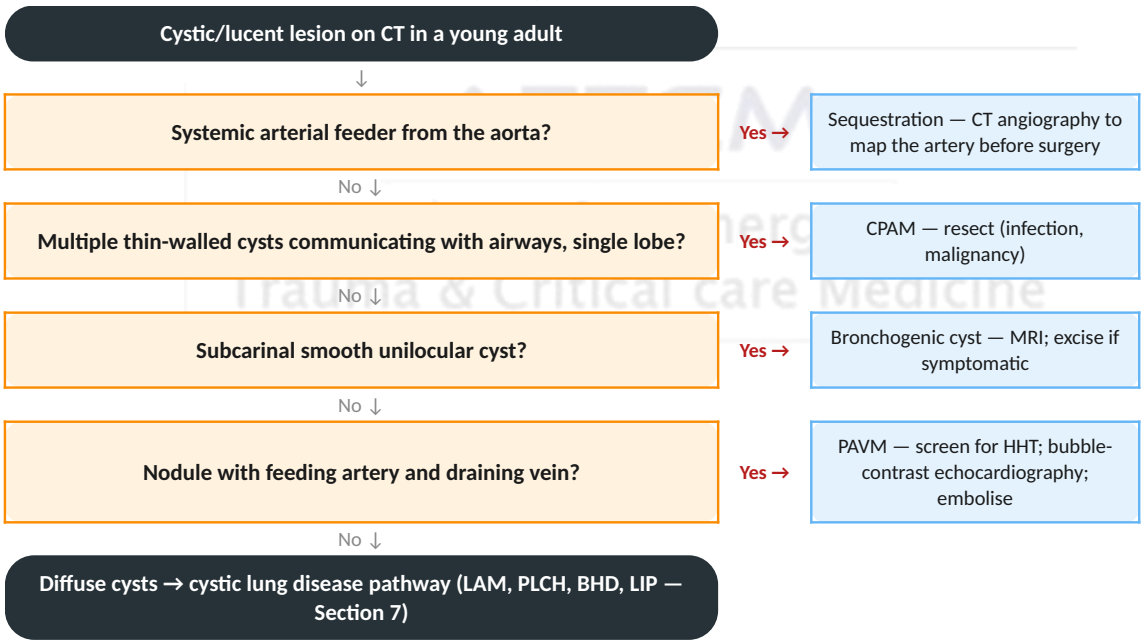
‹ Core topic 1.7 · p.18
Previous-year Qs ›

IN BRIEF Congenital lung lesions present across the age spectrum — antenatally on ultrasound, as respiratory distress in the newborn, as recurrent pneumonia in childhood and as incidental CT findings in adults. Imaging decides the diagnosis; the arterial supply decides the surgery.

Key congenital lesions — distinguishing features

Lesion	Pathology	Imaging clue	Management
CPAM (CCAM)	Hamartomatous cysts communicating with the airway; Stocker types 0–4	Multicystic air-filled lesion, pulmonary arterial supply	Resection (infection and malignancy risk — type 4 ↔ pleuropulmonary blastoma)
Intralobar sequestration	Within visceral pleura; pulmonary venous drainage	Left lower lobe, systemic artery from descending aorta	Lobectomy / segmentectomy; recurrent infection in adults
Extralobar sequestration	Own pleural covering; systemic venous drainage	Infants, associated anomalies (CDH), left base	Excision
Bronchogenic cyst	Foregut duplication	Subcarinal/paratracheal, water or higher attenuation (protein), no enhancement	Excision if symptomatic
Congenital lobar overinflation	Bronchial cartilage deficiency	LUL > RML > RUL hyperlucency with mediastinal shift in the newborn	Lobectomy if symptomatic
Scimitar syndrome	Partial anomalous venous return from right lung to IVC	Curved vein along the right heart border, small right lung, dextroposition	Repair if Qp:Qs > 1.5
Pulmonary AVM	Direct artery-to-vein connection; HHT in 80%	Nodule with feeding artery and draining vein	Embolisation if feeding artery ≥ 2–3 mm

Algorithm E1.4.1 · Adult with an incidental cystic or lucent lung lesion



★ CLINICAL PEARLS & EXAM FOCUS

- Recurrent left lower-lobe pneumonia in a young adult → think intralobar sequestration; ask for CT angiography, not just HRCT.
- PAVM causes paradoxical embolism — stroke and brain abscess in a young patient with cyanosis, clubbing and orthodeoxia.
- Curaçao criteria for HHT: epistaxis, telangiectasia, visceral AVMs, first-degree relative (≥ 3 = definite).

RADIOPAEDIA · IMAGE LINKS Pulmonary sequestration cases | Congenital pulmonary airway malformation cases | Scimitar syndrome cases | Pulmonary arteriovenous malformation cases

MERCK MANUAL PROFESSIONAL Hereditary hemorrhagic telangiectasia | Pulmonary arteriovenous malformation

E1.5 Drug Chart — Drugs for Bronchoscopy, Pleural & Airway Procedures

Previous-year Qs ›

IN BRIEF Drugs the pulmonologist uses every day at the procedure table — local anaesthesia, sedation, reversal, and topical haemostasis.

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Procedural drugs

Class	Drug	Dose	Indication	Caution
Topical local anaesthetic	Lidocaine 1–2% (spray/"spray-as-you-go"), 4% nebulised	Maximum 8.2 mg/kg total topical (BTS); usual < 400 mg	Airway anaesthesia for bronchoscopy	Toxicity — perioral numbness, seizures, arrhythmia; lower doses in elderly, liver/heart failure
Infiltrative local anaesthetic	Lidocaine 1% (± adrenaline)	3 mg/kg (7 mg/kg with adrenaline)	Chest drain, thoracoscopy, pleural biopsy	Aspirate before injecting; intercostal vessels
Benzodiazepine sedative	Midazolam	1–2 mg IV, titrate in 0.5–1 mg; usual ≤ 5 mg (≤ 2–3.5 mg in elderly)	Conscious sedation for bronchoscopy, EBUS	Respiratory depression; reverse with flumazenil
Opioid analgesic	Fentanyl	25–50 µg IV, repeat to ≤ 100–200 µg	Cough suppression and analgesia during EBUS/ thoracoscopy	Chest-wall rigidity, apnoea with midazolam; reverse with naloxone
Short-acting hypnotic	Propofol	0.5–1 mg/kg bolus, then infusion	Deep sedation — rigid bronchoscopy, EBUS	Hypotension; airway skills mandatory
Benzodiazepine antagonist	Flumazenil	0.2 mg IV, repeat to 1 mg	Reversal of midazolam	Seizures in chronic benzodiazepine users; re-sedation
Opioid antagonist	Naloxone	0.1–0.4 mg IV, repeat every 2–3 min	Opioid-induced respiratory depression	Short half-life — re-narcotisation
Anticholinergic	Glycopyrrolate	0.2 mg IV/IM	Reduce secretions (rigid bronchoscopy)	Tachycardia; not routinely needed (BTS)
Topical haemostatic	Cold saline; adrenaline 1:10 000–1:20 000	1–2 mL aliquots via scope	Bleeding after biopsy	Arrhythmia with adrenaline; balloon tamponade if brisk
Antifibrinolytic	Tranexamic acid	500 mg in 5 mL topical; 1 g IV	Post-biopsy bleeding, haemoptysis	Thrombosis risk systemic use
Anticoagulant hold	Warfarin / DOACs / clopidogrel	Stop warfarin 5 days (INR < 1.5); DOAC 48 h; clopidogrel 7 days before TBLB/EBUS-TBNA	Pre-procedure planning	Bridge high thrombotic-risk patients; aspirin may continue

★ **CLINICAL PEARLS & EXAM FOCUS**

- BTS bronchoscopy guideline: offer sedation to all unless contraindicated; oxygen to keep SpO2 > 90%.
- Lidocaine toxicity is cumulative — count the gel, spray and solution together.

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PART II · SECTION 2

Respiratory Physiology & Pulmonary Function

Advanced theory & algorithms

E2.1 Stepwise Interpretation of Pulmonary Function Tests (ATS/ERS 2022) 324
E2.2 Diffusing Capacity (DLCO) — Interpretation & Clinical Patterns 327
E2.3 Approach to Hypoxaemia — A–a Gradient Algorithm & Worked Examples 328
E2.4 Arterial Blood Gas — Systematic Six-Step Analysis 331
E2.5 Cardiopulmonary Exercise Testing (CPET) — Interpretation Algorithm 332
E2.6 Respiratory Muscle Weakness — Physiology & Clinical Evaluation 333
E2.7 Approach to Chronic Breathlessness — Physiology to Bedside 336
E2.8 Drug Chart — Drugs Used in Pulmonary Function & Physiological Testing 337
E2.9 Oxygen Transport, the Dissociation Curve & Tissue Delivery 338

E2.1 Stepwise Interpretation of Pulmonary Function Tests (ATS/ERS 2022)

‹ Core topic 2.2 · p.24
Previous-year Qs ›

IN BRIEF The 2022 ATS/ERS technical standard on interpretive strategies replaced fixed cut-offs and "percent predicted" with the lower limit of normal (LLN, 5th percentile) and z-scores, recommended the race-neutral GLI Global reference equations, and grades severity by z-score rather than by % predicted.

Before interpretation — is the test acceptable?

- **Acceptability:** good start (back-extrapolated volume < 5% of FVC or 100 mL), no cough in the first second, no early termination (plateau < 25 mL in 1 s or FET ≥ 15 s), no glottic closure or leak.
- **Repeatability:** the two largest FVC and FEV1 values within 150 mL (100 mL if FVC ≤ 1 L).
- At least three acceptable manoeuvres; grade the session A–F and report the grade.

Key definitions

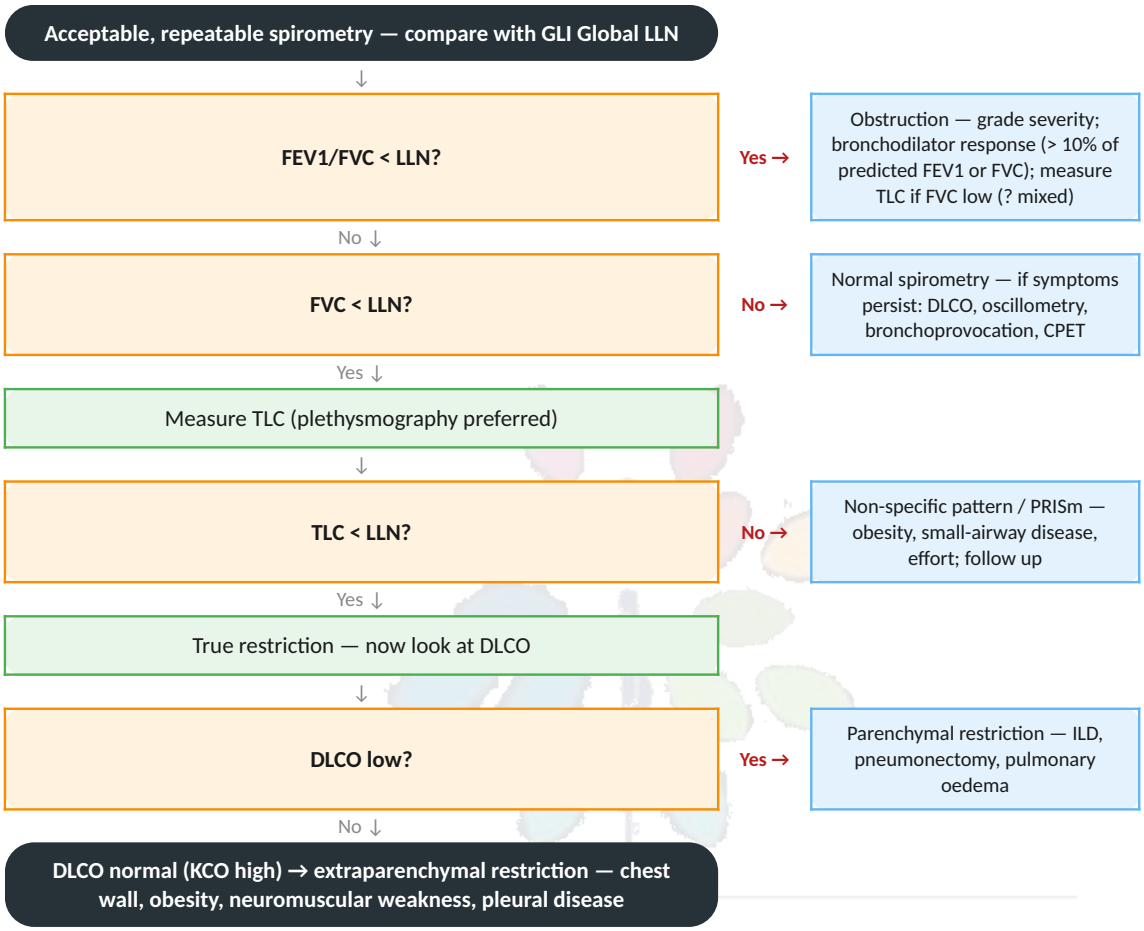
- **Obstruction:** FEV1/FVC (or FEV1/VC) < LLN (z-score < −1.645). GOLD continues to use a fixed post-bronchodilator ratio < 0.70 for COPD — a fixed ratio over-diagnoses obstruction in the elderly and under-diagnoses it in the young.
- **Restriction:** TLC < LLN by body plethysmography or gas dilution — spirometry alone can only suggest restriction (low FVC with normal ratio).
- **PRISm (preserved ratio impaired spirometry):** FEV1/FVC normal but FEV1 < LLN — a heterogeneous group with increased risk of later COPD and cardiovascular death.
- **Mixed defect:** FEV1/FVC < LLN and TLC < LLN.
- **Non-specific pattern:** low FVC and FEV1, normal ratio, normal TLC — seen in obesity, early small-airway disease, poor effort.

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Severity grading by z-score (ATS/ERS 2022)

z-score of FEV1 (or other index)	Severity
> −1.645	Within normal limits
−1.65 to −2.5	Mild
−2.51 to −4.0	Moderate
< −4.0	Severe

Algorithm E2.1.1 · Interpreting spirometry, lung volumes and DLCO



Bronchodilator responsiveness — the new definition

- ATS/ERS 2022: a change in FEV1 or FVC of **> 10% of the individual's predicted value** after 400 µg salbutamol (measured 10–15 min later).
- The older criterion (≥ 12% and ≥ 200 mL from baseline) is still used by GINA for the diagnosis of asthma — quote both in the examination.

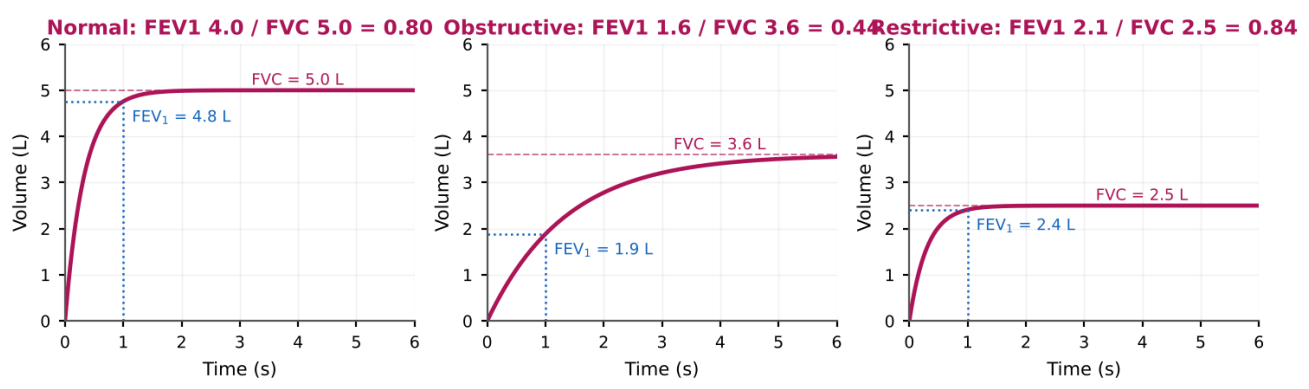


Figure 2.1 · Volume–time spirograms — normal, obstructive and restrictive patterns

Flow–volume loop shapes worth recognising

- **Obstructive:** "scooped-out" concave expiratory limb; reduced peak flow.
- **Restrictive:** tall, narrow "witch's hat" loop — normal shape, small volume.
- **Variable extrathoracic obstruction:** flattened inspiratory limb (vocal-cord palsy, OSA, laryngeal tumour).
- **Variable intrathoracic obstruction:** flattened expiratory limb (tracheomalacia, tumour of the lower trachea).
- **Fixed upper-airway obstruction:** both limbs flattened — "box" loop (post-intubation tracheal stenosis, goitre). Empey index FEV1 (mL)/PEF (L/min) > 10 suggests upper-airway obstruction.

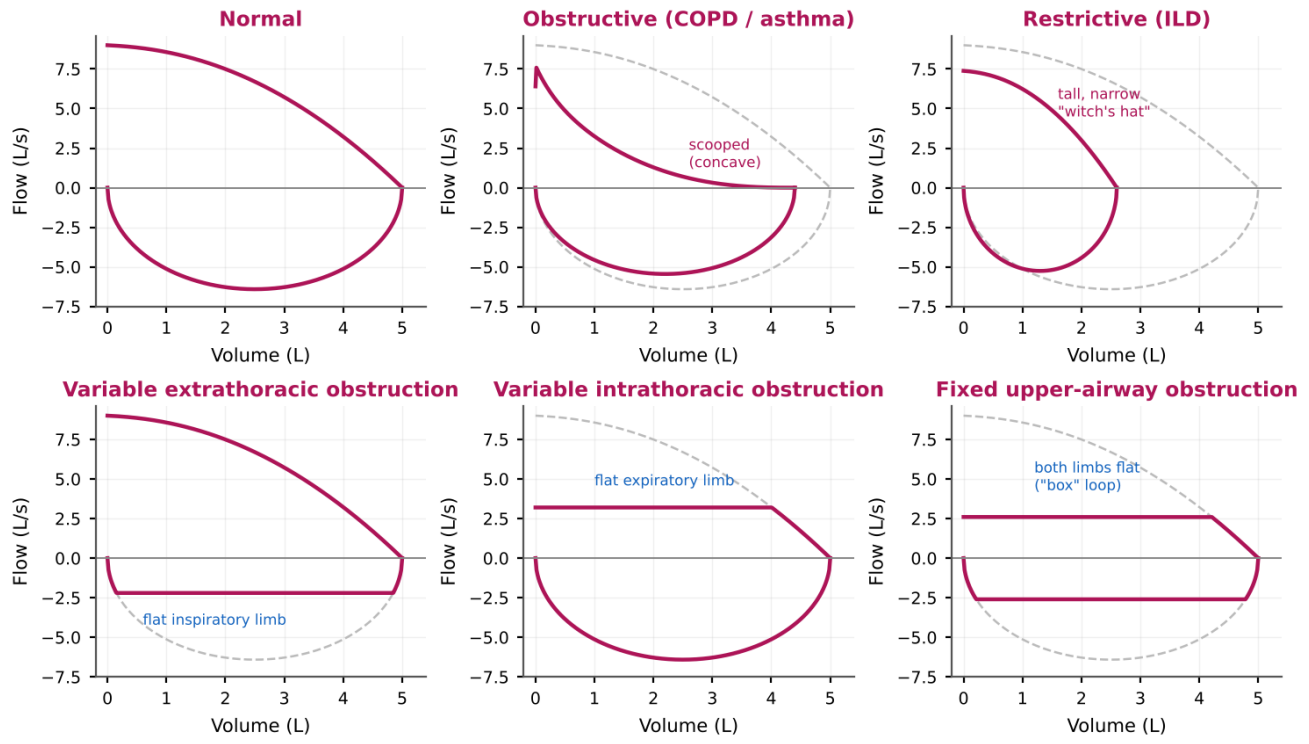


Figure 2.2 · The six flow–volume loops every postgraduate must recognise at a glance (grey dashed = normal reference)

MODEL ANSWER FRAMEWORK — *"Discuss the interpretation of pulmonary function tests." (10 marks)*

1. Definition and indications of PFT; equipment (spirometer, plethysmograph, DLCO).
2. Quality criteria — acceptability, repeatability, grading.
3. Reference equations (GLI Global), LLN and z-scores — why % predicted is abandoned.
4. Algorithm: obstruction → restriction → DLCO (draw the flowchart).
5. Bronchodilator response — old and new criteria; flow–volume loop patterns with sketches.
6. Severity grading; clinical correlation; common pitfalls (effort, obesity, fixed ratio in the elderly).

RADIOPAEDIA · IMAGE LINKS Flow volume loop cases Spirometry cases

AETCM YOUTUBE · VIDEO LECTURES ▶ spirometry ▶ pulmonary function test ▶ PFT interpretation

MERCK MANUAL PROFESSIONAL Pulmonary function testing

E2.2 Diffusing Capacity (DLCO) — Interpretation & Clinical Patterns

‹ Core topic 2.3 · p.26
Previous-year Qs ›

IN BRIEF DLCO (TLCO) measures the transfer of carbon monoxide from alveolar gas to haemoglobin. It is the most sensitive single test for interstitial and pulmonary vascular disease — and the most frequently misinterpreted.

The physiology behind the number

- Roughton–Forster: 1/DLCO = 1/DM + 1/(θ · Vc), where DM = membrane conductance, θ = CO uptake rate of blood, Vc = pulmonary capillary blood volume.
- **DLCO = KCO × VA** — the transfer coefficient (KCO, per litre of accessible alveolar volume) multiplied by the alveolar volume.
- Always correct DLCO for haemoglobin (and for carboxyhaemoglobin in smokers).

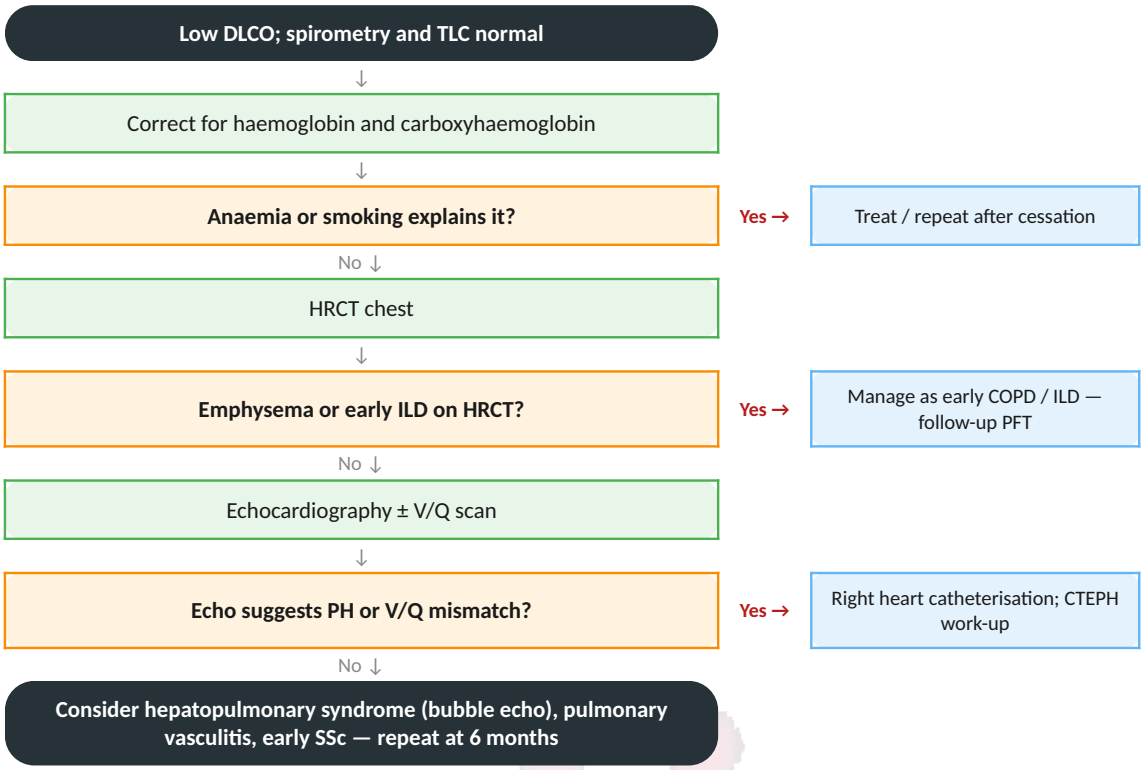
DLCO, KCO and VA — patterns and causes

Pattern	Mechanism	Examples
Low DLCO, low KCO, normal or low VA	Loss of alveolar–capillary surface	ILD, emphysema, pulmonary vascular disease (PAH, CTEPH, vasculitis)
Low DLCO, normal/high KCO, low VA	Incomplete lung expansion with healthy units	Neuromuscular weakness, chest wall disease, pneumonectomy, pleural disease
Low DLCO with normal spirometry and volumes	Pulmonary vascular disease or early ILD	PAH, CTEPH, early systemic sclerosis, anaemia, hepatopulmonary syndrome
High DLCO	Increased capillary blood volume or alveolar blood	Alveolar haemorrhage, polycythaemia, left-to-right shunt, asthma, obesity, supine, exercise

Clinical uses

- **Emphysema vs asthma:** both obstruct, but DLCO is low in emphysema and normal or high in asthma.
- **Screening for PAH in systemic sclerosis:** FVC% / DLCO% > 1.6 suggests pulmonary vascular disease (DETECT algorithm).
- **ILD progression:** a fall of ≥ 15% in DLCO (or ≥ 10% in FVC) over 6–12 months indicates progressive fibrosis.
- **Pre-operative risk:** predicted post-operative DLCO < 60% calls for exercise testing; < 30% indicates high risk.
- **Drug toxicity monitoring:** amiodarone, bleomycin, methotrexate — falls in DLCO precede symptoms.
- **Alveolar haemorrhage:** a rising DLCO in a patient with falling haemoglobin (KCO increase > 30%).

Algorithm E2.2.1 · Approach to an isolated low DLCO



★ CLINICAL PEARLS & EXAM FOCUS

- KCO is "corrected for volume" — a high KCO with a low VA means the lung is healthy but small (extraparenchymal restriction).
- Low DLCO is the commonest and earliest PFT abnormality in systemic sclerosis.
- DLCO is falsely low with poor breath-hold technique, high altitude correction errors and recent smoking.

MERCK MANUAL PROFESSIONAL | Diffusing capacity | Pulmonary hypertension

E2.3 Approach to Hypoxaemia — A–a Gradient Algorithm & Worked Examples

‹ Core topic 2.5 · p.29
Previous-year Qs ›

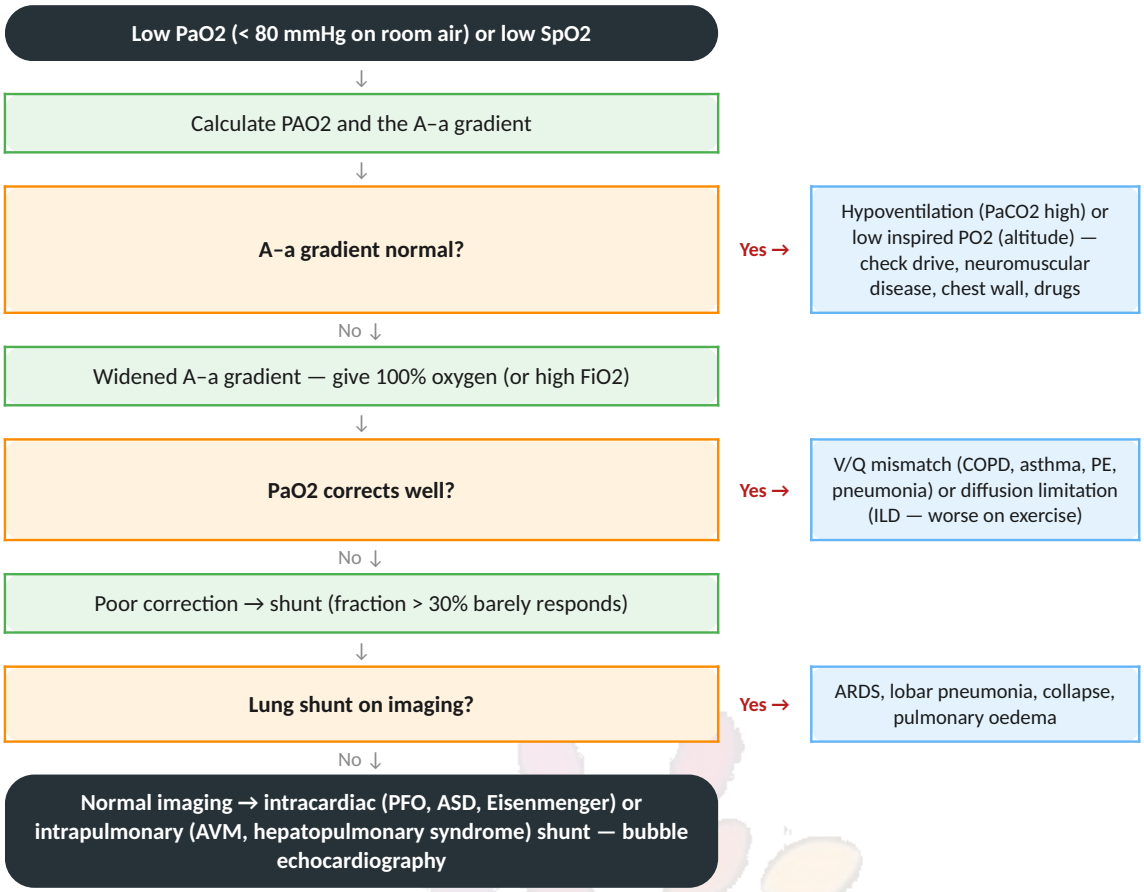
IN BRIEF Five mechanisms cause hypoxaemia. Two arithmetic steps — the alveolar gas equation and the response to 100% oxygen — sort them out at the bedside.

The alveolar gas equation

- PAO2 = FiO2 (Patm − PH2O) − PaCO2 / R = 0.21 × (760 − 47) − PaCO2/0.8 ≈ 150 − 1.25 × PaCO2 (room air, sea level).
- **Normal A–a gradient:** < 10–15 mmHg in the young; age/4 + 4 is a useful upper limit; it widens with FiO2 (so it is unreliable on high oxygen).
- On high FiO2 use the **PaO2/FiO2 ratio** (normal > 400) or the **oxygenation index** OI = (FiO2 × mean airway pressure × 100) / PaO2.

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Algorithm E2.3.1 · Mechanism of hypoxaemia



Worked examples

Calculating the gradient — three patients on room air

Patient	ABG	PAO2	A–a gradient	Interpretation
Opioid overdose, 25 years	PaO2 60, PaCO2 70	150 − 87.5 = 62.5	2.5	Pure hypoventilation — reverse the opioid, ventilate
COPD exacerbation	PaO2 50, PaCO2 60	150 − 75 = 75	25	Hypoventilation + V/Q mismatch
Suspected PE, 40 years	PaO2 65, PaCO2 30	150 − 37.5 = 112.5	47.5	V/Q mismatch — CTPA

Special syndromes

- **Platypnoea–orthodeoxia:** dyspnoea and desaturation on sitting up, relieved by lying down — hepatopulmonary syndrome (basal AVMs), PFO with raised right pressures after pneumonectomy.
- **Silent (happy) hypoxaemia:** profound hypoxaemia without dyspnoea — seen in COVID-19 pneumonia; the drive responds to CO2 rather than O2.
- **Pseudohypoxaemia:** leucocyte larceny in leukaemia with very high counts — falsely low PaO2 in a delayed sample; SpO2 is normal.
- **SpO2 pitfalls:** carbon monoxide (falsely normal), methaemoglobinaemia (reads ≈ 85%), dark skin pigmentation (overestimates saturation), poor perfusion and nail polish.

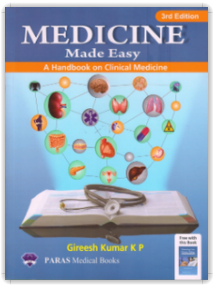
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★ CLINICAL PEARLS & EXAM FOCUS

- Every ABG answer should end with the A–a gradient — examiners expect it.
- Hypoxaemia that worsens on exercise with normal resting gas exchange = diffusion limitation (ILD) or pulmonary vascular disease.
- Shunt responds poorly to oxygen; V/Q mismatch responds well — this is why NIV/PEEP (recruiting shunt units) is needed in ARDS.

MERCK MANUAL PROFESSIONAL Hypoxemia Arterial blood gas

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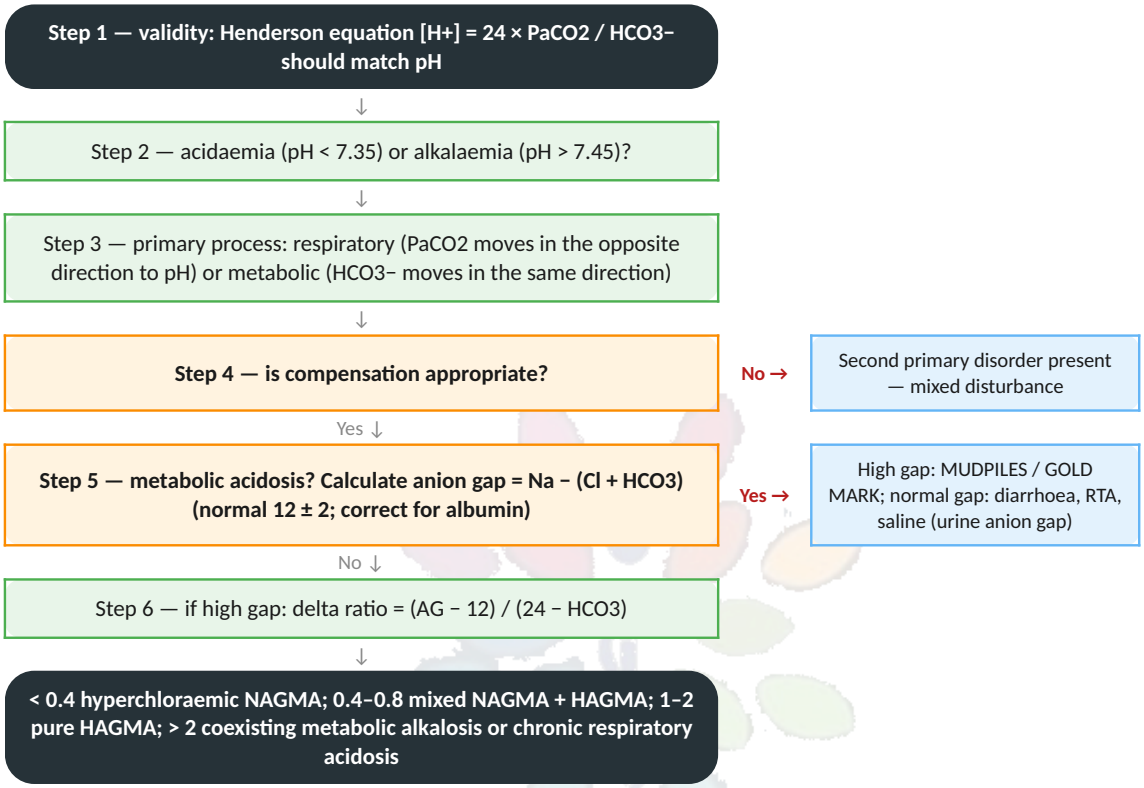
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E2.4 Arterial Blood Gas — Systematic Six-Step Analysis

‹ Core topic 2.8 · p.35
Previous-year Qs ›

IN BRIEF Use the same six steps every time: validity, primary disorder, compensation, anion gap, delta ratio, and the clinical story. The method is the answer.

Algorithm E2.4.1 · Six-step ABG interpretation



Expected compensation

Primary disorder	Expected compensation
Metabolic acidosis	PaCO2 = 1.5 × HCO3 + 8 ± 2 (Winter's formula)
Metabolic alkalosis	PaCO2 rises 0.7 per 1 mmol/L rise in HCO3 (rarely > 55)
Acute respiratory acidosis	HCO3 rises 1 per 10 mmHg rise in PaCO2; pH falls 0.08 per 10
Chronic respiratory acidosis	HCO3 rises 3.5–4 per 10 mmHg; pH falls 0.03 per 10
Acute respiratory alkalosis	HCO3 falls 2 per 10 mmHg fall in PaCO2
Chronic respiratory alkalosis	HCO3 falls 4–5 per 10 mmHg fall in PaCO2

Respiratory scenarios examiners love

Classic respiratory ABGs

Scenario	Typical ABG	Point to make
Acute severe asthma, tiring	pH 7.30, PaCO2 48, HCO3 24	"Normal" or rising PaCO2 in asthma is life-threatening — ICU
Acute-on-chronic COPD (type 2)	pH 7.28, PaCO2 75, HCO3 34	Chronic compensation + acute rise → NIV if pH < 7.35
Massive PE	pH 7.48, PaCO2 28, PaO2 55	Respiratory alkalosis with wide A–a gradient
Salicylate poisoning	pH 7.45, PaCO2 22, HCO3 15, AG 22	Mixed respiratory alkalosis + HAGMA
Diuretic-treated COPD	pH 7.48, PaCO2 58, HCO3 42	Metabolic alkalosis worsening hypoventilation — acetazolamide
Post-hypercapnic alkalosis	pH 7.52 after intubation of COPD	Over-ventilation of chronic hypercapnia — target the patient's baseline PaCO2

Venous blood gas and lactate

- VBG pH is about 0.03 lower and PCO2 about 4–5 mmHg higher than arterial; a venous PCO2 < 45 mmHg reliably excludes arterial hypercapnia.
- Lactate > 2 mmol/L in sepsis defines hypoperfusion; > 4 mmol/L with hypotension defines septic shock (Sepsis-3 uses > 2 with vasopressor need).

★ CLINICAL PEARLS & EXAM FOCUS

- Always calculate the anion gap — even when the pH is normal, a high gap reveals a hidden acidosis.
- Correct the anion gap for albumin: add 2.5 for every 1 g/dL fall in albumin below 4.
- A "normal" ABG in a breathless patient: think PE early, anxiety, anaemia, CO poisoning, or a mixed disorder.

AETCM YOUTUBE · VIDEO LECTURES ▶ ABG interpretation ▶ arterial blood gas ▶ acid base

MERCK MANUAL PROFESSIONAL Acid-base disorders | Metabolic acidosis | Respiratory acidosis

E2.5 Cardiopulmonary Exercise Testing (CPET) — Interpretation Algorithm

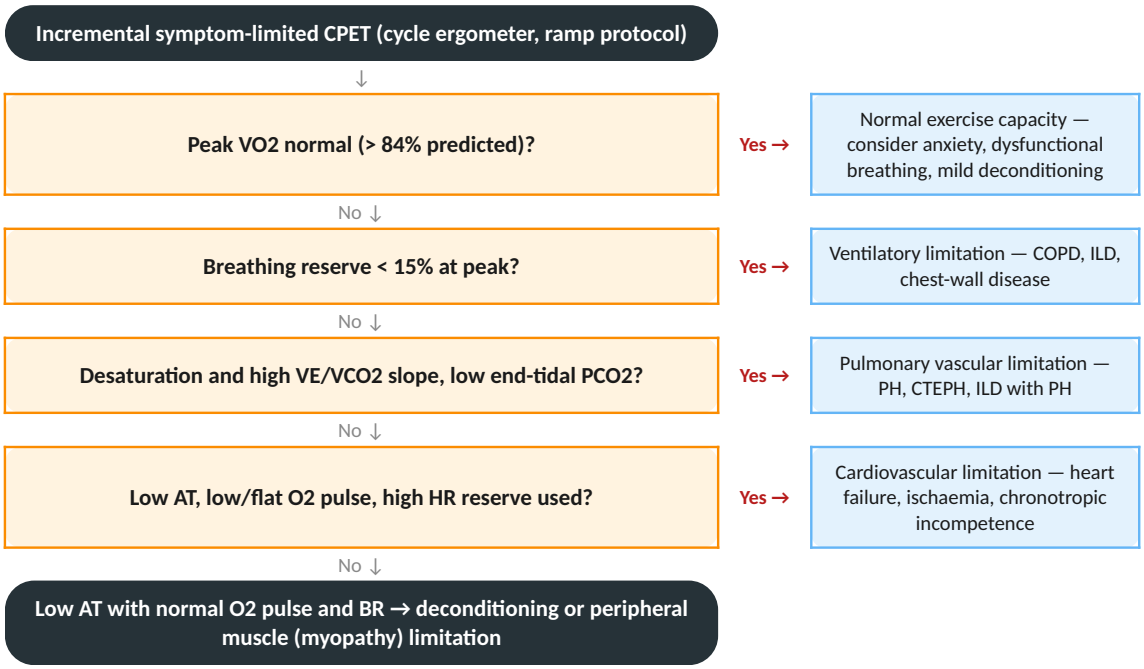
‹ Core topic 2.10 · p.39
Previous-year Qs ›

IN BRIEF CPET integrates the response of the lungs, heart, pulmonary circulation and muscles to incremental exercise. It is the gold standard to explain unexplained exertional dyspnoea and to quantify surgical risk.

Key variables

- **Peak VO2:** the highest oxygen uptake; < 84% predicted is reduced. Fick: VO2 = cardiac output × (CaO2 − CvO2).
- **Anaerobic (ventilatory) threshold (AT):** VO2 at which lactate accumulates; normally > 40% of predicted peak VO2. Low AT = deconditioning or cardiovascular limitation.
- **Breathing reserve (BR):** 1 − (peak VE / MVV); normal > 15–20%. Low BR = ventilatory limitation.
- **O2 pulse:** VO2/HR — a surrogate for stroke volume; flattening suggests cardiac limitation.
- **VE/VCO2 slope:** normal < 30; > 34 suggests inefficient ventilation (PH, heart failure, ILD); end-tidal PCO2 falls in pulmonary vascular disease.
- **Desaturation:** fall in SpO2 ≥ 4% indicates a gas-exchange limitation.

Algorithm E2.5.1 · CPET — what limits this patient's exercise?



Uses of CPET in pulmonary medicine

Use	Threshold to remember
Pre-operative risk (lung resection)	Peak VO2 > 20 mL/kg/min: low risk; < 10 mL/kg/min (or < 35% predicted): high risk — consider non-surgical options
Heart failure / transplant	Peak VO2 < 14 mL/kg/min (< 12 on β-blockers) supports listing
PAH prognosis	Peak VO2 < 11 mL/kg/min and VE/VCO2 > 44 = high risk (ESC/ERS 2022)
Unexplained dyspnoea	Pattern recognition as above; dysfunctional breathing shows erratic VE and early hyperventilation
Exercise-induced bronchoconstriction	Fall in FEV1 ≥ 10% after exercise challenge

Six-minute walk test — the field alternative

- Self-paced, 30-metre corridor; record distance, SpO2 nadir, Borg dyspnoea; minimal clinically important difference ≈ 30 m.
- Desaturation below 88% supports ambulatory oxygen; distance < 350 m signals poor prognosis in COPD (BODE), < 165 m high risk in PAH.
- Contraindications: unstable angina or MI within a month; relative — resting HR > 120, SBP > 180.

★ CLINICAL PEARLS & EXAM FOCUS

- "Ventilatory limitation" = low breathing reserve; "cardiac limitation" = low AT + flat O2 pulse; "pulmonary vascular" = high VE/VCO2 + desaturation.
- CPET separates the breathless obese patient who is simply deconditioned from the one with early PH.

MERCK MANUAL PROFESSIONAL Cardiopulmonary exercise testing

‹ Core topic 2.11 · p.40

E2.6 Respiratory Muscle Weakness — Physiology & Clinical Evaluation

Previous-year Qs ›

IN BRIEF Respiratory muscle weakness is easily missed: the chest radiograph may be normal, the patient may only complain of orthopnoea or poor sleep, and PFT shows a "mild restrictive" defect. Specific tests reveal it.

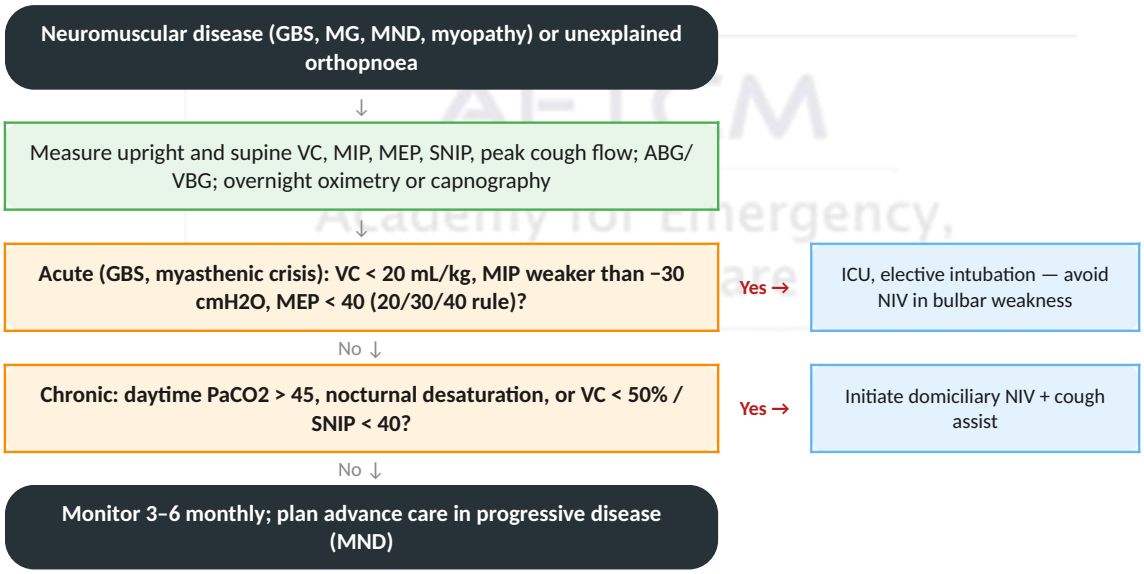
Physiology

- The diaphragm (C3–C5) provides ≈ 70% of tidal breathing; intercostals and accessory muscles take over in diaphragm weakness.
- Weakness reduces vital capacity disproportionately (TLC relatively preserved, RV may rise) and causes nocturnal hypoventilation first (REM sleep — when accessory muscles are atonic).
- **Bilateral diaphragm paralysis:** orthopnoea, paradoxical inward movement of the abdomen on inspiration (abdominal paradox), supine VC falls > 25–30%.

Tests of respiratory muscle strength

Test	Normal	Significance
Upright and supine VC	Fall on lying < 10%	Fall > 25% = bilateral diaphragm weakness
Maximal inspiratory pressure (MIP / PImax)	> 80 cmH2O men, > 70 women	< 60 cmH2O suggests weakness; < 30 → ventilatory failure risk
Maximal expiratory pressure (MEP)	> 100 cmH2O	< 60 → ineffective cough
Sniff nasal inspiratory pressure (SNIP)	> 70 men, > 60 women	Easy, reproducible; used in MND
Peak cough flow	> 270 L/min	< 160 L/min → ineffective cough; cough assist needed
Fluoroscopic sniff test / diaphragm ultrasound	Both hemidiaphragms descend	Paradoxical upward movement → paralysis; thickening fraction < 20% on ultrasound
Phrenic nerve conduction / transdiaphragmatic pressure	—	Gold standard (Pdi) — specialist centres

Algorithm E2.6.1 · Suspected neuromuscular respiratory failure



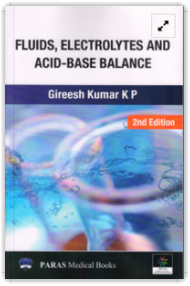
★ CLINICAL PEARLS & EXAM FOCUS

- In GBS and myasthenia, do not rely on SpO2 — oxygenation is preserved until late; follow VC and MIP serially.
- Single-breath count < 20 is a quick bedside surrogate for VC below about 1.5 L.
- Diaphragm ultrasound is now a bedside tool: excursion < 1 cm and thickening fraction < 20% indicate dysfunction (also predicts weaning failure).

RADIOPAEDIA · IMAGE LINKS Diaphragmatic paralysis cases Sniff test cases

MERCK MANUAL PROFESSIONAL Guillain-Barré syndrome Myasthenia gravis

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E2.7 Approach to Chronic Breathlessness — Physiology to Bedside

‹ Core topic 2.13 · p.43
Previous-year Qs ›

IN BRIEF Dyspnoea is a subjective sensation arising from a mismatch between central respiratory drive and the mechanical response of the respiratory system (neuromechanical dissociation). A structured approach separates the four big causes — lungs, heart, blood and deconditioning — in a few steps.

Mechanisms of dyspnoea

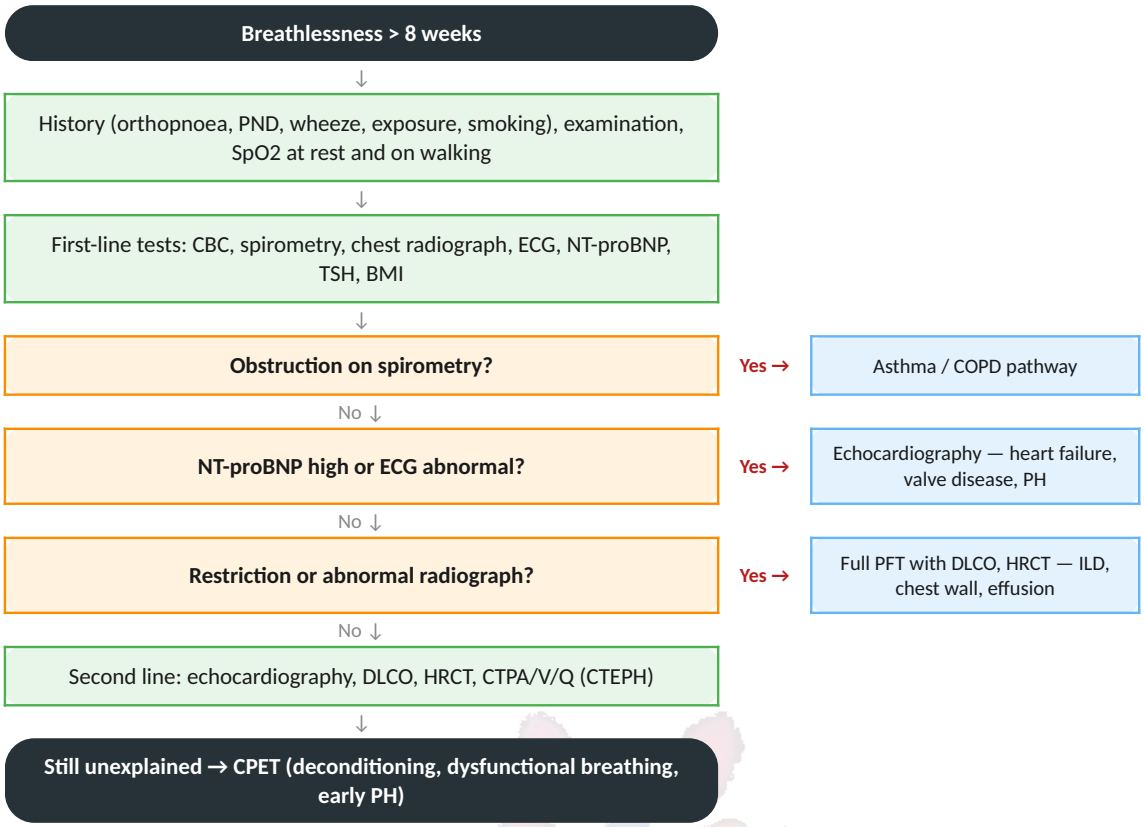
- **Increased drive:** hypoxaemia, hypercapnia, acidosis, J-receptor stimulation (oedema, ILD), pulmonary vascular receptors.
- **Increased load:** airway resistance (asthma, COPD), reduced compliance (ILD, oedema, obesity, chest wall).
- **Weak pump:** neuromuscular disease, hyperinflation shortening the diaphragm.
- **Afferent mismatch:** "air hunger", "work/effort", "chest tightness" are distinct qualities — tightness suggests bronchoconstriction.

Grading breathlessness

Scale	Use
mMRC (0–4)	Chronic dyspnoea in COPD; mMRC ≥ 2 = "more symptoms" in GOLD
NYHA (I–IV)	Heart failure, PAH (WHO functional class)
Borg CR-10	During exercise / 6MWT
CAT (0–40)	COPD health status; ≥ 10 = high symptom burden

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Algorithm E2.7.1 · Chronic breathlessness — first-line work-up



Chronic breathlessness syndrome — palliation

- Breathlessness persisting despite optimal treatment of the underlying disease.
- **Non-drug:** hand-held fan to the face (trigeminal stimulation), breathing control, pacing, pulmonary rehabilitation, "breathing–thinking–functioning" model.
- **Drugs:** low-dose oral morphine (5–10 mg/day sustained release) — evidence modest (BEAMS trial negative for routine use); benzodiazepines are not first line; oxygen only if hypoxaemic.

★ **CLINICAL PEARLS & EXAM FOCUS**

- Ask "What does your breathlessness feel like?" — tightness (asthma), cannot get a deep breath (dysfunctional breathing, restriction), air hunger (heart failure, PE).
- Dysfunctional breathing: frequent sighs, normal investigations, Nijmegen questionnaire > 23 — treat with breathing retraining.

MERCK MANUAL PROFESSIONAL Dyspnea

E2.8 Drug Chart — Drugs Used in Pulmonary Function & Physiological Testing

Previous-year Qs ›

IN BRIEF Drugs used in the PFT laboratory — for bronchodilator reversibility, bronchoprovocation, acid–base correction and respiratory stimulation — and the drugs that must be withheld before testing.

Drugs in physiological testing

Class	Drug	Dose	Indication	Caution
SABA	Salbutamol	400 µg (4 × 100 µg) via spacer; test 10–15 min later	Bronchodilator responsiveness test	Tremor, tachycardia
SAMA	Ipratropium	160 µg (4 × 40 µg); test at 30 min	Reversibility test when β-agonist contraindicated	Glaucoma, urinary retention
Bronchoprovocation agent	Methacholine	Doubling doses to PD20 FEV1; positive if PD20 ≤ 200 µg (PC20 < 4–8 mg/mL)	Suspected asthma with normal spirometry	Contraindicated if FEV1 < 60% or < 1.5 L, recent MI/stroke, uncontrolled HTN; have salbutamol ready
Indirect challenge	Mannitol (dry powder)	Cumulative to 635 mg; positive if 15% fall in FEV1	Exercise-induced bronchoconstriction, asthma	Cough; same contraindications
Indirect challenge	Hypertonic saline 4.5%	Nebulised, increasing durations	Asthma diagnosis	Bronchospasm
Carbonic anhydrase inhibitor	Acetazolamide	250–500 mg oral/IV 6–12 h	Metabolic alkalosis in ventilated COPD; AMS prophylaxis	Hypokalaemia, sulpha allergy, worsens acidosis in severe COPD
Buffer	Sodium bicarbonate 8.4%	1 mEq/kg slow IV (guided by pH < 7.1)	Severe metabolic acidosis, hyperkalaemia	Raises PaCO2 — hazardous in hypercapnic failure
Respiratory stimulant	Doxapram	1–4 mg/min IV	Rarely — hypercapnic failure when NIV not tolerated	Hypertension, agitation; obsolete in most settings
Respiratory stimulant	Medroxyprogesterone	20 mg TDS	Obesity hypoventilation (historical)	Thrombosis

Drug withholding before tests (ATS/ERS)

Withhold before bronchodilator or challenge testing

Drug	Withhold for
SABA (salbutamol)	4–6 h
SAMA (ipratropium)	12 h
LABA (salmeterol, formoterol)	24 h
Ultra-LABA (indacaterol, vilanterol, olodaterol)	36 h
LAMA (tiotropium, glycopyrronium)	36–48 h (≥ 7 days for methacholine challenge with tiotropium)
Theophylline	12–48 h
Leukotriene receptor antagonist	24 h (challenge tests)

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E2.9 Oxygen Transport, the Dissociation Curve & Tissue Delivery

‹ Core topic 2.6 · p.32
Previous-year Qs ›

IN BRIEF Oxygen content, delivery and extraction explain why a patient with a normal SpO2 can still be hypoxic. These equations recur in every viva.

The oxygen cascade — key equations

Quantity	Formula	Normal
Oxygen content (CaO2)	(1.34 × Hb × SaO2) + (0.003 × PaO2)	≈ 20 mL/dL
Oxygen delivery (DO2)	CaO2 × cardiac output × 10	≈ 1000 mL/min
Oxygen consumption (VO2)	(CaO2 − CvO2) × CO × 10	≈ 250 mL/min
Extraction ratio	VO2 / DO2	0.25 (25%)
Mixed venous saturation (SvO2)	—	65–75% (ScvO2 ≈ 70%)
P50	PO2 at 50% saturation	26.7 mmHg

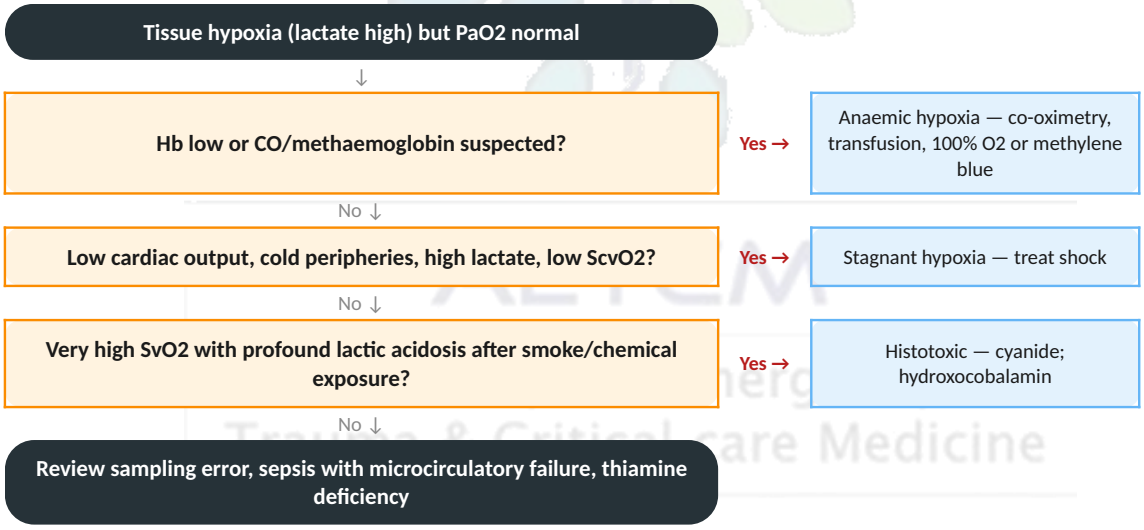
Shifts of the oxyhaemoglobin dissociation curve

Right shift (↓ affinity, easier unloading)	Left shift (↑ affinity, tighter binding)
↑ Temperature	↓ Temperature
↑ PaCO2, ↓ pH (Bohr effect)	↓ PaCO2, ↑ pH
↑ 2,3-DPG (anaemia, altitude, chronic hypoxia)	↓ 2,3-DPG (stored blood)
Abnormal Hb (HbS)	Fetal haemoglobin, carboxyhaemoglobin, methaemoglobin

Types of hypoxia — classification with examples

- **Hypoxaemic:** low PaO2 — altitude, hypoventilation, V/Q mismatch, shunt, diffusion limitation.
- **Anaemic:** low oxygen-carrying capacity — anaemia, CO poisoning, methaemoglobinaemia (PaO2 normal).
- **Stagnant (circulatory):** low cardiac output, shock, local vascular occlusion.
- **Histotoxic:** cells cannot use oxygen — cyanide, hydrogen sulphide (high SvO2).

Algorithm E2.9.1 · Hypoxia with a normal PaO2

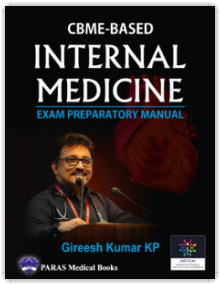


★ CLINICAL PEARLS & EXAM FOCUS

- Only 1.5% of oxygen is dissolved — but that fraction is what hyperbaric oxygen exploits in CO poisoning.
- The Haldane effect: deoxygenated haemoglobin carries more CO2 — part of the mechanism of oxygen-induced hypercapnia in COPD.

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PART II · SECTION 3

Microbiology, Pathology, Immunology & Pharmacology

Advanced theory & algorithms

E3.1 Reading TB Laboratory Reports — Smear, CBNAAT, Truenat, LPA, Culture & WGS 341
E3.2 Anti-TB Drug Adverse Events — Recognition & Management Algorithms 342
E3.3 Inhaler Devices, Aerosol Science & Device Selection 345
E3.4 Biologics in Airway & Lung Disease — Pharmacology Table 347
E3.5 Antifungal Pharmacology & Therapeutic Drug Monitoring 348
E3.6 Drug Chart — First-Line & Second-Line Anti-TB Drugs (NTEP Weight Bands) 350
E3.7 Corticosteroids & Immunosuppression in Respiratory Practice — Safe Prescribing 352

E3.1 Reading TB Laboratory Reports — Smear, CBNAAT, Truenat, LPA, Culture & WGS

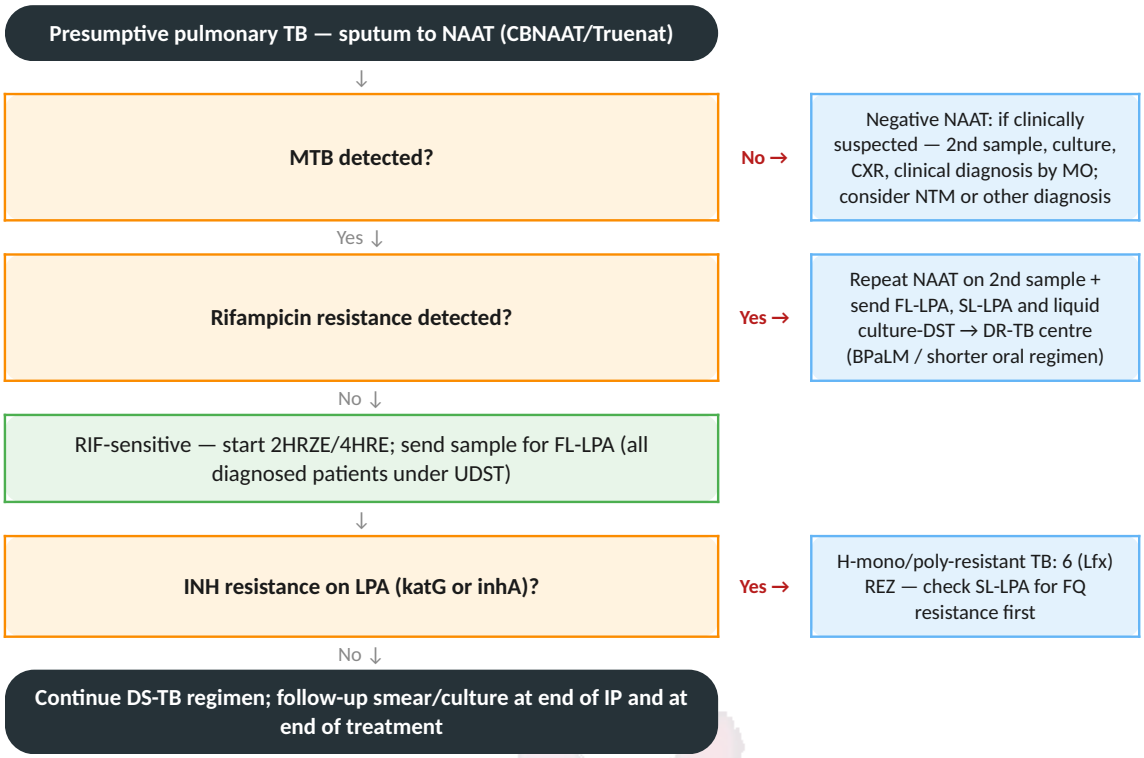
‹ Core topic 3.2 · p.51
Previous-year Qs ›

IN BRIEF Each TB test answers a different question — "Is it TB?", "Is it rifampicin-resistant?", "Which drugs work?". Knowing what a report means, and what it does not mean, prevents both missed diagnoses and wrong regimens.

TB diagnostic tests — what each one tells you

Test	Detects	Limit of detection / time	Key point
Ziehl–Neelsen / LED-FM smear	Acid-fast bacilli	5 000–10 000 bacilli/mL; same day	Graded scanty, 1+, 2+, 3+; cannot distinguish NTM or viability
CBNAAT (Xpert MTB/RIF Ultra)	MTB DNA + rpoB mutations (81-bp RRDR)	≈ 16 CFU/mL (Ultra); 2 h	"Trace" result = MTB detected, RIF indeterminate — valid diagnosis in children, PLHIV, EPTB
Truenat MTB / MTB Plus / MTB-RIF Dx	MTB DNA + rpoB (chip-based, battery operated)	≈ 100 CFU/mL; 1 h	Point-of-care in peripheral labs — NTEP's first-line NAAT along with CBNAAT
First-line LPA (GenoType MTBDRplus)	rpoB, katG (high-level INH), inhA (low-level INH, ethionamide cross-resistance)	Smear-positive samples or cultures; 2 days	inhA mutation → INH may still work at high dose; ethionamide resistant
Second-line LPA (MTBDRsl)	gyrA/gyrB (FQ), rrs/eis (aminoglycosides)	2 days	FQ resistance decides BPaLM vs BPaL / longer regimen
Liquid culture (MGIT 960)	Viable bacilli	10–100 CFU/mL; 10–42 days	Gold standard; needed for phenotypic DST of new drugs (Bdq, Lzd, Dlm, Pa)
Targeted NGS / WGS	Mutations across all drug targets	Days	Recommended by WHO (2023–24) for comprehensive resistance profiling
Urine LF-LAM	Lipoarabinomannan	30 min	PLHIV with CD4 ≤ 200 or seriously ill — positive result = start ATT

Algorithm E3.1.1 · From presumptive TB to regimen — reading the reports



Interpreting discordant results

- **NAAT RIF-resistant, LPA RIF-sensitive:** may be a "disputed" rpoB mutation (L430P, D435Y, H445N, L452P) missed by phenotypic MGIT DST — treat as resistant (WHO).
- **Smear-positive, NAAT-negative:** think NTM (M. avium, M. abscessus) — culture with species identification.
- **NAAT positive after treatment:** dead bacilli are detected for months — never use NAAT to monitor response; use smear and culture.
- **Culture contaminated:** repeat the sample; NALC-NaOH decontamination should keep contamination < 5–8%.

★ CLINICAL PEARLS & EXAM FOCUS

- Universal DST (UDST) under NTEP: every diagnosed TB patient gets at least rifampicin testing and, if RIF-sensitive, INH testing by FL-LPA.
- CBNAAT "trace" in adults with a history of TB may be a false positive — repeat and correlate.
- Bedaquiline and linezolid resistance can only be detected phenotypically or by sequencing — this is why WGS is expanding.

MERCK MANUAL PROFESSIONAL Tuberculosis

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E3.2 Anti-TB Drug Adverse Events — Recognition & Management Algorithms

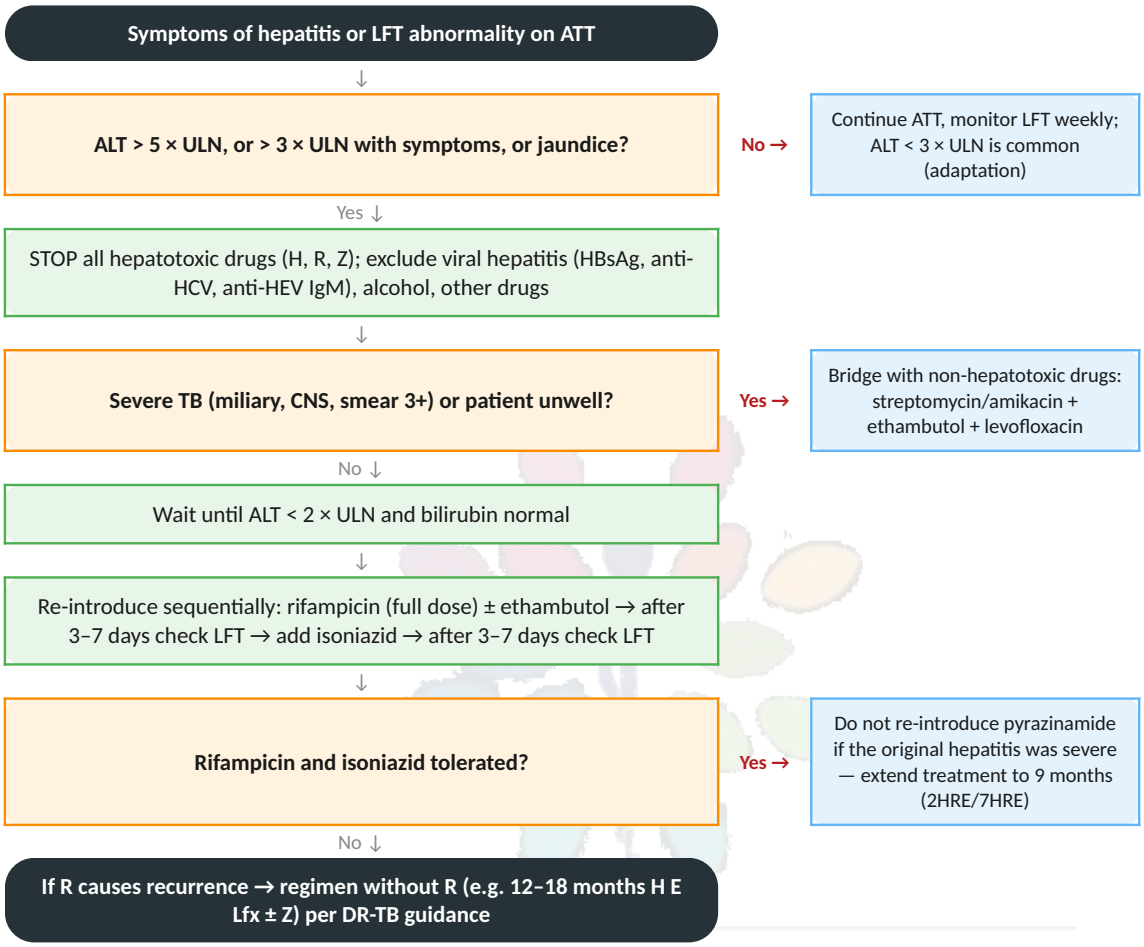
‹ Core topic 3.8 · p.58
Previous-year Qs ›

IN BRIEF Adverse drug reactions are the commonest reason for interrupting ATT. The examiner wants a clear algorithm for hepatotoxicity and an organised table for the rest.

Drug-induced liver injury (DILI) — definitions (ATS / NTEP)

- ALT (or AST) > 3 × ULN **with** symptoms (nausea, vomiting, anorexia, jaundice), or > 5 × ULN **without** symptoms, or bilirubin > 2 × ULN (> 3 mg/dL).
- Culprits: pyrazinamide (most hepatotoxic, dose-related) > isoniazid > rifampicin (cholestatic, hyperbilirubinaemia).
- Risk factors: age > 35, alcohol, HBV/HCV, HIV, malnutrition (hypoalbuminaemia), slow acetylators, concomitant hepatotoxic drugs, pregnancy/post-partum.

Algorithm E3.2.1 · Managing hepatotoxicity on ATT



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Other adverse effects of first-line drugs

Effect	Drug	Management
Peripheral neuropathy	Isoniazid (pyridoxine deficiency)	Pyridoxine 10 mg/day prophylaxis (NTEP gives with every regimen); treat with 100 mg/day
Optic neuritis (red–green colour loss)	Ethambutol	Stop permanently; baseline and monthly Ishihara in children > 5 and in renal failure
Hyperuricaemia / arthralgia	Pyrazinamide (inhibits urate excretion)	NSAIDs; allopurinol does not help; gout — stop Z
Orange urine, tears, sweat; contact lenses stained	Rifampicin	Counsel; harmless
Flu-like syndrome, thrombocytopenia, haemolysis, AKI	Rifampicin (intermittent dosing, immune)	Stop permanently in thrombocytopenia, haemolysis or AKI
Cutaneous reactions — rash to SJS/TEN/DRESS	Any (often R, Z, H)	Mild: antihistamine; severe: stop all, steroids, re-challenge one by one after recovery (never after SJS/TEN with the culprit)
Gastrointestinal intolerance	Z, R	Take with light food (not with antacids); antiemetic
Hepatitis	Z, H, R	Algorithm above

Second-line drugs — monitoring under the BPaLM era

Key toxicities in DR-TB regimens

Drug	Toxicity	Monitoring
Bedaquiline	QTc prolongation, hepatotoxicity	ECG baseline, 2, 12, 24 weeks; stop if QTcF > 500 ms; check K, Mg, Ca
Linezolid	Myelosuppression, peripheral and optic neuropathy, lactic acidosis	CBC weekly in first month; BPaLM uses 600 mg — reduce to 300 mg or stop for toxicity
Pretomanid	Hepatotoxicity, peripheral neuropathy	LFT monthly
Delamanid	QT prolongation (milder), psychiatric	ECG
Moxifloxacin / Levofloxacin	QT prolongation (Mfx > Lfx), tendinopathy, dysglycaemia	ECG; avoid in tendon disease
Clofazimine	Skin discolouration, QT prolongation, enteropathy	Counsel; ECG
Cycloserine	Psychosis, depression, seizures	Pyridoxine; psychiatric screening
Amikacin (now rarely used)	Ototoxicity, nephrotoxicity	Audiometry, creatinine

★ **CLINICAL PEARLS & EXAM FOCUS**

- Never add a single new drug to a failing regimen; never re-challenge after SJS/TEN with the culprit.
- Linezolid neuropathy is often irreversible — ask about tingling at every visit; optic neuropathy is usually reversible if detected early.
- Report every ADR through the PvPI / Nikshay ADR module (active drug safety monitoring — aDSM).

MERCK MANUAL PROFESSIONAL Antimycobacterial drugs

E3.3 Inhaler Devices, Aerosol Science & Device Selection

‹ Core topic 3.10 · p.61
Previous-year Qs ›

IN BRIEF The best inhaled drug fails in the wrong device. Up to 80% of patients make at least one critical error. Choose the device by inspiratory flow, coordination and cognition — then check technique at every visit.

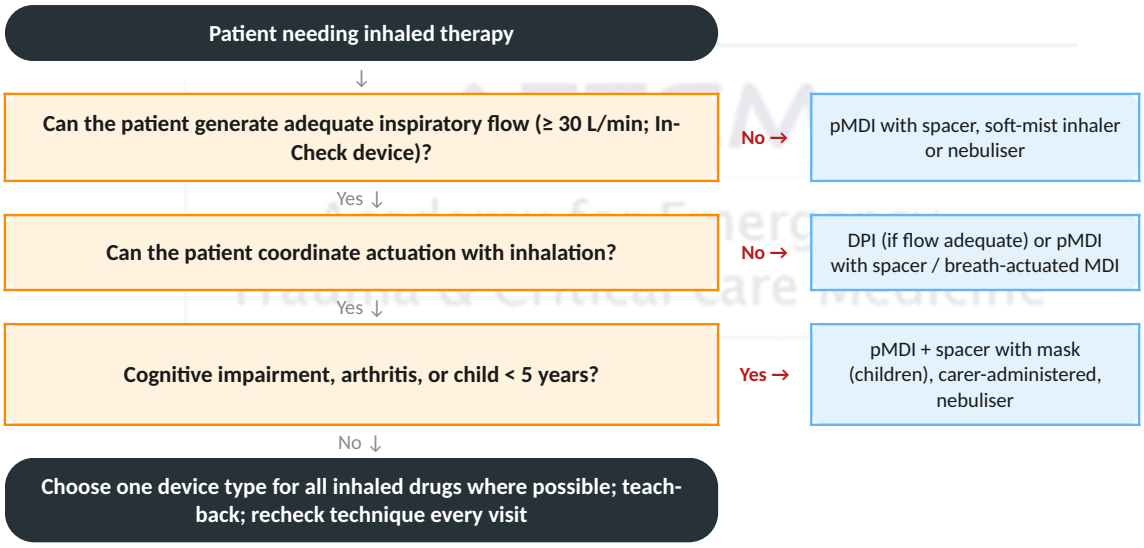
Comparison of inhaler devices

Device	Inspiratory effort needed	Advantages	Problems
pMDI	Slow, deep (30–60 L/min)	Cheap, portable, all drug classes	Coordination; cold-Freon effect; oropharyngeal deposition
pMDI + spacer ± mask	Tidal breathing acceptable	No coordination needed; ↓ oral deposition; best for children, elderly, acute attacks	Bulky; static charge (wash with detergent, air dry)
Breath-actuated MDI	Moderate	Solves coordination	Few drugs
Dry powder inhaler (Rotahaler, Accuhaler, Turbuhaler, Ellipta)	Fast, forceful (> 30–60 L/min; device resistance dependent)	No propellant, dose counters	Poor flow in severe COPD, children < 5, elderly; humidity
Soft-mist inhaler (Respimat)	Slow	Slow aerosol (≈ 1.5 s), high lung deposition	Assembly; cost
Nebuliser (jet, mesh)	Tidal breathing	Any patient, high doses, acute severe disease	Time, cost, infection risk; no better than spacer in acute asthma

Aerosol deposition — the physics

- Particles > 5 µm impact in the oropharynx and large airways (inertial impaction); 1–5 µm (respirable fraction) deposit in small airways by sedimentation; < 1 µm are exhaled or deposit by diffusion.
- Breath-hold of 5–10 seconds increases sedimentation; slow inhalation reduces impaction.

Algorithm E3.3.1 · Choosing an inhaler device



Critical errors to check

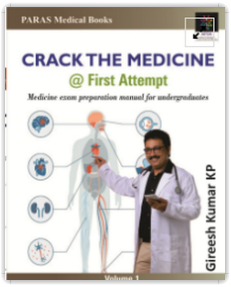
pMDI	DPI
Not shaking; not exhaling first	Not loading/priming correctly
Actuating before or after inhaling	Exhaling into the device
Inhaling too fast	Inhaling too slowly
No breath-hold	No breath-hold; device held wrongly (Turbuhaler upright when loading)

★ CLINICAL PEARLS & EXAM FOCUS

- "One patient, one device type" — mixing pMDI and DPI techniques doubles error rates.
- Spacer + pMDI is as effective as a nebuliser for acute asthma in the ED and children.
- Rinse and spit after inhaled corticosteroids to prevent oral candidiasis and dysphonia.

MERCK MANUAL PROFESSIONAL Asthma treatment

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E3.4 Biologics in Airway & Lung Disease — Pharmacology Table

‹ Core topic 3.12 · p.64
Previous-year Qs ›

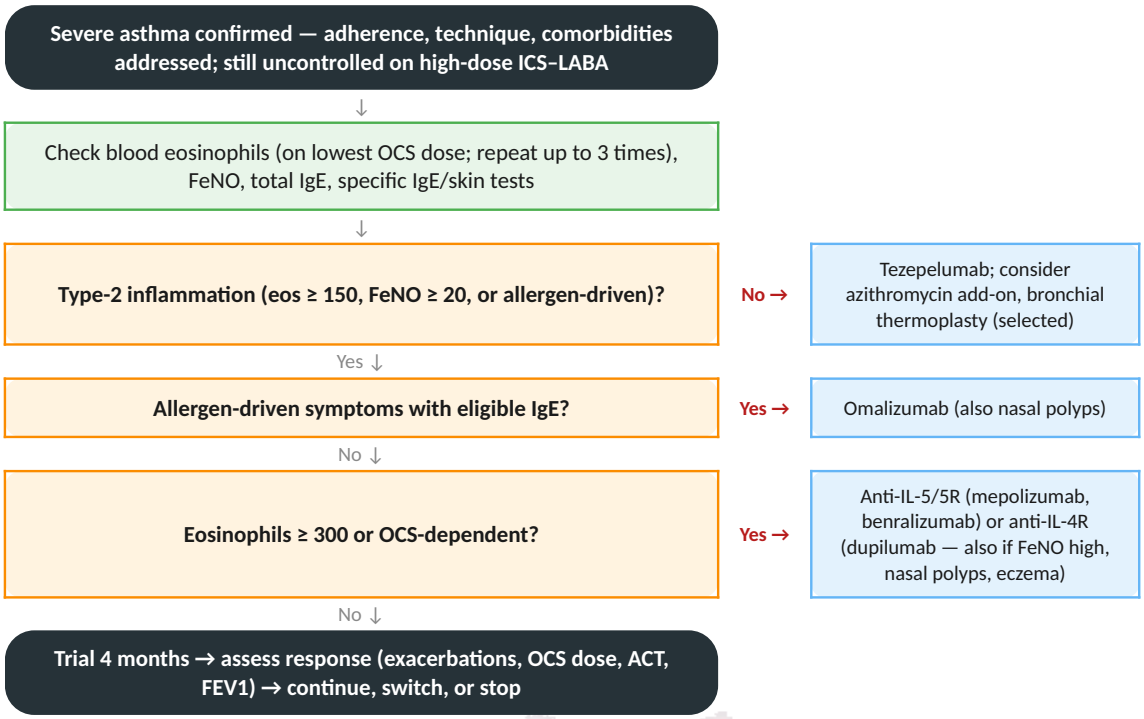
IN BRIEF Biologics have transformed severe asthma and are moving into COPD. Exam answers should link each biologic to its target, phenotype, biomarker and evidence.

Biologics used in respiratory medicine

Drug	Target	Indication / biomarker	Dose	Key trial / note
Omalizumab	IgE	Severe allergic asthma; IgE 30–1500 IU/mL with sensitisation; CSU, nasal polyps	SC every 2–4 weeks by IgE and weight	INNOVATE; reduces exacerbations ~25%
Mepolizumab	IL-5	Eosinophilic asthma (eos ≥ 150–300/µL); EGPA; HES; COPD with eos ≥ 300 (MATINEE 2025)	100 mg SC 4-weekly (300 mg in EGPA)	MENSA, MIRRA
Reslizumab	IL-5	Eosinophilic asthma (eos ≥ 400)	IV 3 mg/kg 4-weekly	Anaphylaxis risk
Benralizumab	IL-5Rα (eosinophil depletion by ADCC)	Eosinophilic asthma; EGPA (MANDARA)	30 mg SC 4-weekly × 3, then 8-weekly	SIROCCO, CALIMA, ZONDA (OCS sparing)
Dupilumab	IL-4Rα (blocks IL-4 and IL-13)	Type-2 asthma (eos ≥ 150 or FeNO ≥ 25), OCS-dependent, nasal polyps, atopic dermatitis; COPD with eos ≥ 300	200–300 mg SC 2-weekly	QUEST, VENTURE; BOREAS & NOTUS (COPD)
Tezepelumab	TSLP (upstream alarmin)	Severe asthma regardless of phenotype — including low eosinophil	210 mg SC 4-weekly	NAVIGATOR
Rituximab	CD20	ANCA vasculitis, refractory CTD-ILD, PAP (off-label)	IV	RAVE, RITUXILD
Tocilizumab	IL-6R	SSc-ILD (FDA), severe COVID-19	IV/SC	focuSSced
Nintedanib	Tyrosine kinases (VEGFR, FGFR, PDGFR)	IPF, progressive pulmonary fibrosis, SSc-ILD	150 mg BD	INPULSIS, INBUILD, SENSCIS
Pirfenidone	Antifibrotic (TGF-β)	IPF	801 mg TDS	CAPACITY, ASCEND

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Algorithm E3.4.1 · Choosing a biologic in severe asthma (GINA 2024–25)



★ **CLINICAL PEARLS & EXAM FOCUS**

- Dupilumab can raise blood eosinophils transiently — avoid in baseline eos > 1500/µL (risk of eosinophilic pneumonia/EGPA unmasking).
- Biologics are add-ons — never stop ICS–LABA on starting a biologic.
- First biologics approved for COPD: dupilumab (2024) and mepolizumab (2025) for eosinophilic COPD with exacerbations.

MERCK MANUAL PROFESSIONAL — Asthma — Idiopathic pulmonary fibrosis

E3.5 Antifungal Pharmacology & Therapeutic Drug Monitoring

‹ Core topic 3.11 · p.63
Previous-year Qs ›

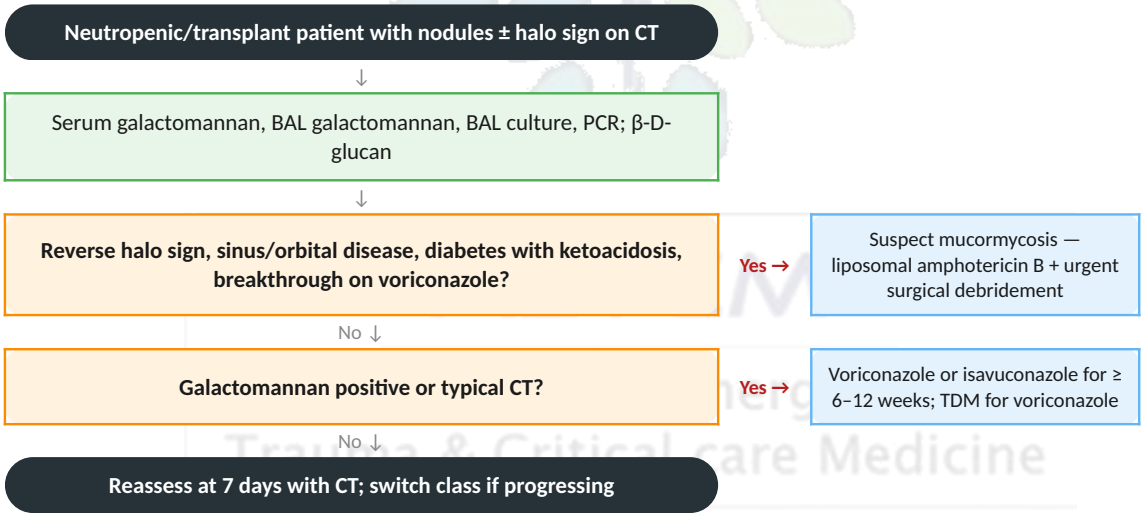
IN BRIEF Invasive fungal disease of the lung is rising — COVID-associated mucormycosis taught India this painfully. Know the classes, spectrum, toxicity and the drugs that need levels.

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Antifungal agents

Class / drug	Spectrum	Use in lung disease	Toxicity / interactions
Amphotericin B (deoxycholate, liposomal, lipid complex)	Broadest — Aspergillus, Mucorales, Cryptococcus, Candida, endemic fungi	Drug of choice for mucormycosis (L-AmB 5–10 mg/kg), severe endemic mycoses	Nephrotoxicity, hypokalaemia, hypomagnesaemia, infusion reactions (less with liposomal)
Voriconazole	Aspergillus (not Mucorales)	First-line invasive aspergillosis, CPA	Visual disturbance, hallucinations, photosensitivity, skin cancer, periostitis (fluoride); CYP interactions — TDM trough 1–5.5 mg/L
Isavuconazole	Aspergillus and Mucorales	Invasive aspergillosis; mucormycosis step-down	QT shortening; fewer interactions; no TDM routinely
Posaconazole	Aspergillus and Mucorales	Prophylaxis in neutropenia/GVHD; mucormycosis step-down	TDM trough > 0.7 (prophylaxis), > 1 mg/L (treatment)
Itraconazole	Aspergillus, endemic fungi	ABPA (steroid-sparing), CPA, histoplasmosis	Negative inotrope (avoid in heart failure); absorption — capsule with food, solution fasting; TDM > 0.5 mg/L
Fluconazole	Candida, Cryptococcus	Cryptococcosis consolidation	No mould activity
Echinocandins (caspofungin, micafungin, anidulafungin)	Candida, Aspergillus (fungistatic)	Salvage aspergillosis, candidaemia	Well tolerated; no oral form
Co-trimoxazole	Pneumocystis	PJP treatment (15–20 mg/kg TMP) and prophylaxis	Rash, hyperkalaemia, marrow suppression

Algorithm E3.5.1 · Empirical approach to a mould infection in the immunocompromised lung



★ CLINICAL PEARLS & EXAM FOCUS

- Galactomannan is falsely negative in mucormycosis and on mould-active prophylaxis; β-D-glucan is negative in Mucorales and Cryptococcus.
- Voriconazole + rifampicin is contraindicated (levels become undetectable) — important in TB with CPA.

MERCK MANUAL PROFESSIONAL Aspergillosis | Mucormycosis | Antifungal drugs

E3.6 Drug Chart — First-Line & Second-Line Anti-TB Drugs (NTEP Weight Bands)

Previous-year Qs ›

IN BRIEF Doses follow the NTEP / WHO weight-band approach. Fixed-dose combinations (FDC) are standard for drug-susceptible TB.

Anti-tuberculosis drugs

Class	Drug	Dose	Indication	Caution
Rifamycin	Rifampicin (R)	10 mg/kg (8–12; max 600 mg) daily; high-dose 35 mg/kg under study for TBM	All DS-TB regimens	Hepatitis, CYP3A4 induction (OCP, warfarin, ART, azoles), orange secretions, thrombocytopenia
Hydrazide	Isoniazid (H)	5 mg/kg (4–6; max 300 mg); high-dose 10–15 mg/kg	DS-TB, TPT (6H), inhA-mutant DR-TB	Hepatitis, neuropathy — give pyridoxine
Pyrazinoic acid	Pyrazinamide (Z)	25 mg/kg (20–30)	Intensive phase	Hepatitis, hyperuricaemia, arthralgia
Ethylenediamine	Ethambutol (E)	15 mg/kg (15–20)	Intensive and continuation (NTEP 4HRE)	Optic neuritis; reduce frequency in renal failure
Diarylquinoline	Bedaquiline (Bdq)	400 mg OD × 2 weeks, then 200 mg thrice weekly × 22 weeks (or 200 mg OD × 8 wk then 100 mg OD)	BPaLM, shorter and longer MDR regimens	QTc prolongation, hepatotoxicity
Nitroimidazole	Pretomanid (Pa)	200 mg OD × 26 weeks	BPaLM / BPaL	Hepatotoxicity, neuropathy; not in children/pregnancy
Oxazolidinone	Linezolid (Lzd)	600 mg OD (BPaLM); 600 mg OD long regimen	MDR/XDR-TB	Myelosuppression, neuropathy, optic neuritis, serotonin syndrome
Fluoroquinolone	Moxifloxacin (Mfx) / Levofloxacin (Lfx)	Mfx 400 mg OD; Lfx 750–1000 mg OD (15–20 mg/kg)	BPaLM (Mfx), Hr-TB (Lfx), shorter regimen	QT (Mfx > Lfx), tendinopathy, dysglycaemia
Riminophenazine	Clofazimine (Cfz)	100 mg OD	Longer / shorter MDR regimens	Skin pigmentation, QT
Nitro-dihydro-imidazooxazole	Delamanid (Dlm)	100 mg BD × 24 weeks	Longer MDR regimen, children	QT
Cycloserine / Terizidone	Cs / Trd	10–15 mg/kg (max 1 g)	Longer MDR regimen	Psychosis, seizures — pyridoxine 50 mg per 250 mg Cs
Aminoglycoside	Amikacin	15 mg/kg (≤ 1 g)	Only if susceptible and other options unavailable	Ototoxicity, nephrotoxicity — audiometry
Vitamin	Pyridoxine	10 mg/day prophylaxis (DS-TB); 50–100 mg in DR-TB	Prevent INH/Cs neuropathy	—

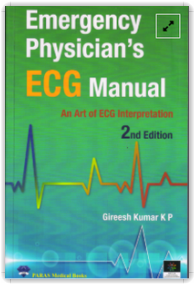
NTEP adult weight bands — daily FDC (4FDC = H75 R150 Z400 E275; 3FDC = H75 R150 E275)

Weight band	Intensive phase (4FDC tablets)	Continuation phase (3FDC tablets)
25–39 kg	2	2
40–54 kg	3	3
55–69 kg	4	4
≥ 70 kg	5	5

★ CLINICAL PEARLS & EXAM FOCUS

- NTEP continuation phase contains ethambutol (4HRE) — unlike the classical WHO 4HR — because of high background INH resistance in India.
- 4-month regimen 2HPZM/2HPM (rifapentine–moxifloxacin, Study 31) and 2-month bedaquiline-containing trials are on the horizon; know them as recent advances.

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E3.7 Corticosteroids & Immunosuppression in Respiratory Practice — Safe Prescribing

‹ Core topic 3.12 · p.64
Previous-year Qs ›

IN BRIEF Corticosteroids are the most-used and most-misused drugs in respiratory medicine. Knowing equivalence, tapering, and the harms prevents a great deal of iatrogenic disease.

Equivalent doses and relative potency

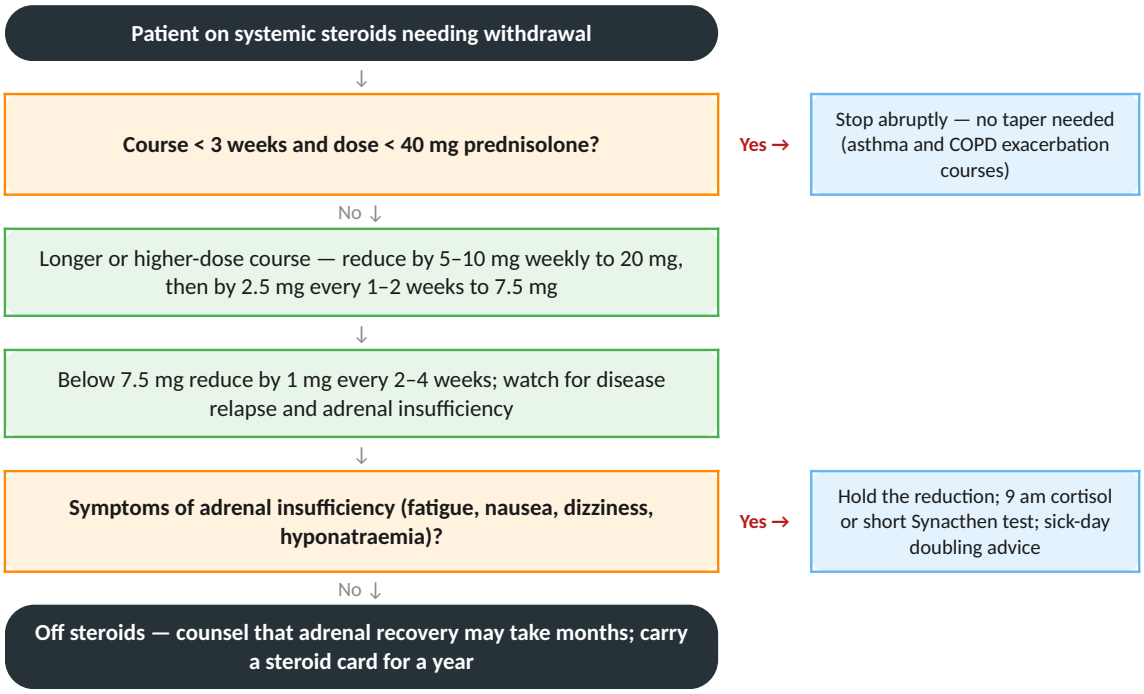
Drug	Equivalent dose	Glucocorticoid potency	Mineralocorticoid effect	Duration
Hydrocortisone	20 mg	1	1	Short (8–12 h)
Prednisolone	5 mg	4	0.8	Intermediate (12–36 h)
Methylprednisolone	4 mg	5	0.5	Intermediate
Deflazacort	6 mg	4	Low	Intermediate
Triamcinolone	4 mg	5	0	Intermediate
Dexamethasone	0.75 mg	25–30	0	Long (36–54 h)
Betamethasone	0.6 mg	25–30	0	Long

Harms of systemic steroids and their prevention

Harm	Prevention / monitoring
Hyperglycaemia, new diabetes	Capillary glucose on starting; treat; prefer morning dosing
Osteoporosis and fracture	Calcium 1000 mg + vitamin D; bisphosphonate if ≥ 7.5 mg prednisolone for ≥ 3 months or high FRAX; DEXA
Adrenal suppression	Taper if > 3 weeks of treatment or > 40 mg/day; sick-day rules; steroid card
Infection (TB reactivation, PJP, Strongyloides)	Screen IGRA/CXR; PJP prophylaxis with ≥ 20 mg/day for ≥ 4 weeks plus another immunosuppressant; ivermectin where Strongyloides is endemic
Peptic ulcer (with NSAIDs)	PPI if on NSAIDs or anticoagulants
Weight gain, Cushingoid change, myopathy	Diet, exercise, steroid-sparing agent
Cataract, glaucoma	Annual eye review on long-term therapy
Psychiatric effects, insomnia	Morning dosing; warn the patient

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Algorithm E3.7.1 · Tapering systemic corticosteroids



Screening before immunosuppression or biologics

Test	Reason
IGRA or TST + chest radiograph	Latent TB — treat before anti-TNF and before prolonged high-dose steroids
HBsAg, anti-HBc, anti-HCV	Reactivation risk with rituximab and steroids
HIV	Alters the differential and the risk
Immunoglobulins	Hypogammaglobulinaemia with rituximab
Vaccination status	Inactivated vaccines 2 weeks before; live vaccines contraindicated during treatment
Strongyloides serology (endemic areas)	Hyperinfection syndrome

★ **CLINICAL PEARLS & EXAM FOCUS**

- Rifampicin halves prednisolone levels — double the steroid dose when both are needed (TB pericarditis, IRIS).
- Dexamethasone has no mineralocorticoid action — it is the steroid of choice when fluid retention matters, and the wrong one for adrenal insufficiency.

MERCK MANUAL PROFESSIONAL Corticosteroids

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PART II · SECTION 4

Tuberculosis

Advanced theory & algorithms

E4.1 Diagnosis of TB in Special Groups — Children, EPTB & Smear-Negative TB 354
E4.2 DS-TB Treatment Monitoring, Outcomes & Programmatic Indicators 355
E4.3 Drug-Resistant TB — Regimen Selection Algorithm (NTEP / WHO 2024–25) 357
E4.4 TB Preventive Treatment & Contact Investigation — Algorithm 360
E4.5 Tuberculous Pleural Effusion — Diagnostic & Management Algorithm 361
E4.6 Post-TB Lung Disease, Haemoptysis & Surgery in TB 362
E4.7 Drug Chart — Drugs in TB Prevention, Adjunctive & Supportive Care 364

E4.1 Diagnosis of TB in Special Groups — Children, EPTB & Smear-Negative TB

‹ Core topic 4.3 · p.70
Previous-year Qs ›

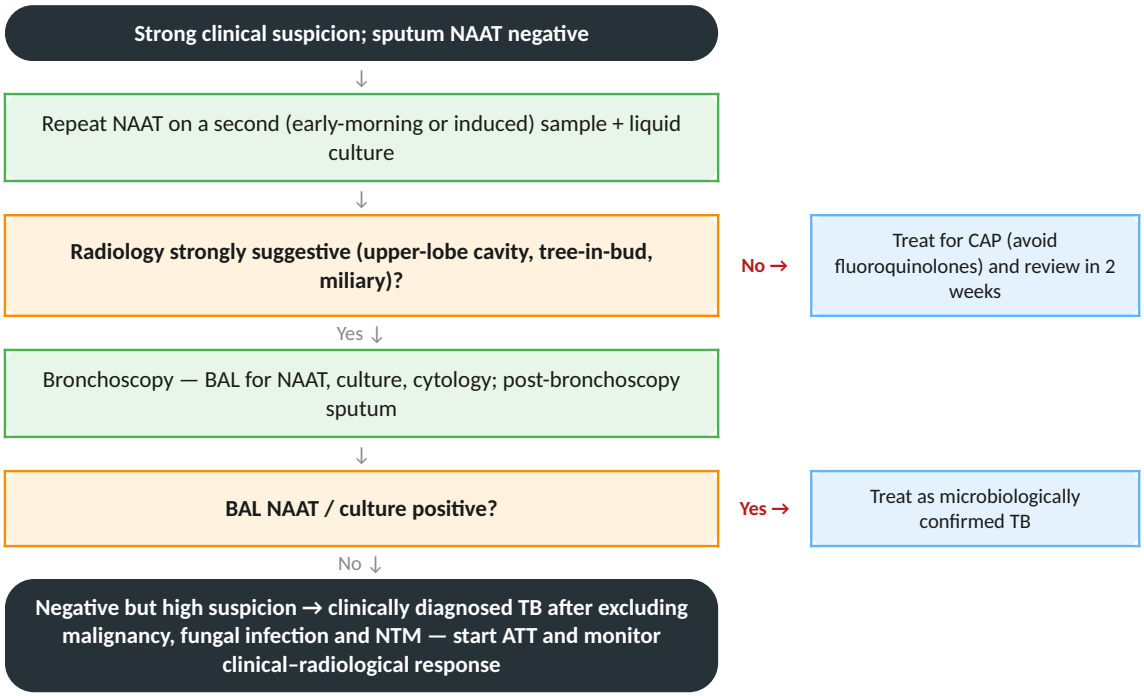
IN BRIEF The standard sputum-NAAT pathway fails where bacillary load is low — in children, in extrapulmonary disease and in paucibacillary pulmonary disease. Each group needs its own specimen strategy.

Specimen strategy by group

Group	Preferred specimens	Special tests
Children	Gastric aspirate (2 consecutive mornings), induced sputum, nasopharyngeal aspirate, stool NAAT (Ultra)	Stool Xpert Ultra is WHO-recommended; treatment-decision algorithms (WHO 2022)
TB lymphadenitis	FNAC with NAAT and culture; excision biopsy if FNA non-diagnostic	NAAT sensitivity ≈ 80%
Pleural TB	Pleural fluid ADA, lymphocyte-predominant exudate; thoracoscopic pleural biopsy — culture + histology	Fluid NAAT is insensitive (≈ 30–50%) — a negative result does not exclude
TB meningitis	CSF NAAT (Ultra; ≥ 6 mL, concentrated), culture	CSF lymphocytes, high protein, glucose ratio < 0.5; ADA > 10 supportive
Abdominal TB	Ascitic ADA (> 39 IU/L), colonoscopic or laparoscopic biopsy	SAAG < 1.1, lymphocytic
Spinal / bone	CT-guided biopsy for NAAT, culture, histology	MRI — subligamentous spread, skip lesions, paravertebral abscess
Pericardial	Pericardial fluid ADA, unstimulated IFN-γ, NAAT	ADA > 40 IU/L supportive

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Algorithm E4.1.1 · Smear-negative / NAAT-negative presumptive pulmonary TB



The WHO treatment-decision algorithm for children (2022)

- Integrated scoring of cough > 2 weeks, fever, lethargy, weight loss, haemoptysis, night sweats, swollen nodes, tachycardia, tachypnoea and CXR findings (cavity, nodes, opacities, miliary pattern, effusion).
- Score > 10 = treat for TB even if bacteriology is negative; allows primary-care decisions where CXR is unavailable (algorithm B).

★ **CLINICAL PEARLS & EXAM FOCUS**

- Pleural fluid with lymphocytes, ADA > 40 IU/L and few mesothelial cells (< 5%) in a young Indian adult is TB until proven otherwise — but ADA is also high in empyema, lymphoma and rheumatoid effusion.
- Never give fluoroquinolones as a "therapeutic trial" for pneumonia when TB is suspected — they mask TB and select for FQ resistance.

RADIOPAEDIA · IMAGE LINKS Miliary tuberculosis cases · Tuberculous lymphadenitis cases · Spinal tuberculosis cases

MERCK MANUAL PROFESSIONAL Tuberculosis · Extrapulmonary tuberculosis

E4.2 DS-TB Treatment Monitoring, Outcomes & Programmatic Indicators

‹ Core topic 4.5 · p.74
Previous-year Qs ›

IN BRIEF Treatment success depends as much on monitoring and support as on the regimen. Know the follow-up schedule, the outcome definitions and the NTEP indicators — they are asked as short notes.

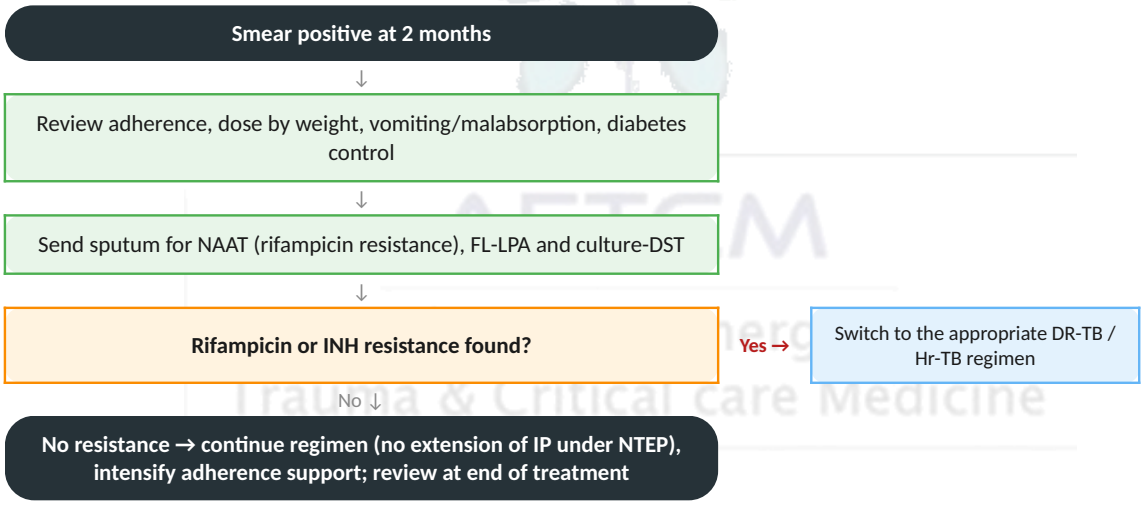
Follow-up schedule for drug-susceptible pulmonary TB (NTEP)

Time point	Clinical	Laboratory
Baseline	Weight, diabetes (RBS), CBC, LFT where indicated, CXR	Sputum smear + NAAT; FL-LPA (UDST)
End of intensive phase (2 months)	Weight, symptoms, adherence, ADRs	Sputum smear (culture where available)
Monthly in continuation phase	Adherence (99DOTS/MERM), ADR screen, weight-based dose revision	—
End of treatment (6 months)	Clinical	Sputum smear/culture; CXR if clinically indicated
Post-treatment	At 6, 12, 18 and 24 months	Symptom screen; sputum if symptomatic

WHO treatment outcome definitions (2021 revision)

Outcome	Definition
Cured	Bacteriologically confirmed at start; completed treatment with evidence of bacteriological response and no evidence of failure
Treatment completed	Completed treatment; no evidence of failure; does not meet "cured" criteria
Treatment failed	Regimen terminated or permanently changed for lack of response, ADR, or new resistance
Died	Died before or during treatment, any cause
Lost to follow-up	Did not start treatment or interrupted for ≥ 2 consecutive months
Not evaluated	No outcome assigned (includes transfer-out)
Treatment success	Cured + treatment completed
Sustained treatment success	Alive and TB-free 6 months (DS-TB) / 12 months (DR-TB) after successful treatment

Algorithm E4.2.1 · Persistent positive sputum at the end of the intensive phase



NTEP support systems

- **Ni-kshay** portal for notification (mandatory in public and private sectors); Ni-kshay Poshan Yojana nutrition support (₹1000 per month from 2024); Pradhan Mantri TB Mukt Bharat Abhiyaan (Ni-kshay Mitra food baskets).
- Adherence technologies: 99DOTS, MERM boxes, video-observed therapy; family treatment supporters.

- Indicators: presumptive TB examination rate, notification rate, UDST coverage, treatment success rate (target ≥ 90%), TPT coverage among contacts.

★ CLINICAL PEARLS & EXAM FOCUS

- The End TB Strategy targets an 80% fall in incidence and 90% fall in mortality by 2030 compared with 2015 — know its three pillars.
- "Lost to follow-up" is ≥ 2 consecutive months of interruption — not one missed dose.

MERCK MANUAL PROFESSIONAL Tuberculosis

E4.3 Drug-Resistant TB — Regimen Selection Algorithm (NTEP / WHO 2024–25)

‹ Core topic 4.7 · p.78
Previous-year Qs ›

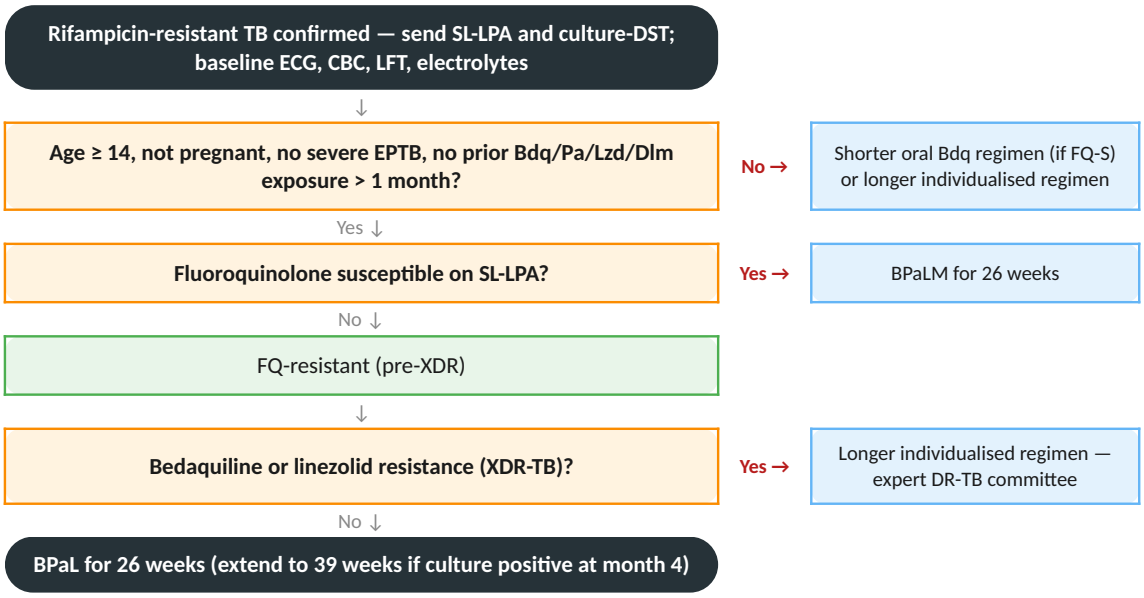
IN BRIEF The all-oral 6-month BPaLM regimen is the preferred treatment for MDR/RR-TB in India. Regimen choice depends on fluoroquinolone susceptibility, age, pregnancy, extent of disease and prior exposure to the newer drugs.

Regimens for MDR/RR-TB

Regimen	Composition	Eligibility
BPaLM (6 months)	Bedaquiline + Pretomanid + Linezolid (600 mg) + Moxifloxacin	Age ≥ 14 years, FQ-susceptible, not pregnant/breastfeeding, no extensive EPTB (CNS, osteoarticular, disseminated), < 1 month prior exposure to Bdq/Lzd/Pa/Dlm
BPaL (6 months)	As above without moxifloxacin	Same — but FQ-resistant (pre-XDR)
Shorter oral Bdq-containing regimen (9–11 months)	Bedaquiline-based all-oral combination with Lfx, Cfz, Z, E, high-dose H, Eto	Not eligible for BPaLM; FQ-susceptible; children, pregnancy
Modified 9-month all-oral regimens (endTB combinations, WHO 2025)	Bdq/Dlm/Lzd/Lfx/Cfz/Z-based	Including children and pregnancy
Longer individualised regimen (18–20 months)	≥ 4 effective drugs from Groups A, B and C	XDR-TB, failure of shorter regimens, extensive disease

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Algorithm E4.3.1 · Choosing the DR-TB regimen



Definitions (WHO 2021)

- **MDR-TB:** resistant to at least rifampicin and isoniazid. **RR-TB:** rifampicin resistance (treated as MDR).
- **Pre-XDR-TB:** MDR/RR + resistance to any fluoroquinolone.
- **XDR-TB:** pre-XDR + resistance to bedaquiline and/or linezolid.
- **Hr-TB:** isoniazid-resistant, rifampicin-susceptible — 6 (Lfx) REZ.

WHO grouping of medicines for longer regimens

Group	Drugs	Rule
A	Levofloxacin/moxifloxacin, bedaquiline, linezolid	Include all three
B	Clofazimine, cycloserine/terizidone	Add one or both
C	Ethambutol, delamanid, pyrazinamide, carbapenem (with clavulanate), amikacin, ethionamide, PAS	Use to complete the regimen when A/B cannot

LANDMARK TRIALS & GUIDELINES

- **TB-PRACTECAL** (2022): BPaLM for 6 months — 89% success vs 52% with standard care.
- **ZeNix:** linezolid 600 mg for 26 weeks as effective as 1200 mg with less toxicity.
- **endTB** (2024): three 9-month all-oral regimens non-inferior to standard care.
- **STREAM stage 2:** all-oral bedaquiline-containing shorter regimen superior to the injectable regimen.

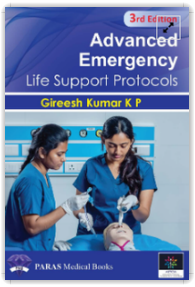
★ CLINICAL PEARLS & EXAM FOCUS

- Moxifloxacin in BPaLM is dropped if FQ resistance is found — start BPaLM and adjust when SL-LPA returns.
- Every DR-TB patient needs QTcF monitoring — bedaquiline, moxifloxacin and clofazimine add up.

AETCM YOUTUBE · VIDEO LECTURES ▶ MDR TB ▶ BPaLM regimen ▶ drug resistant tuberculosis

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E4.4 TB Preventive Treatment & Contact Investigation — Algorithm

‹ Core topic 4.4 · p.71

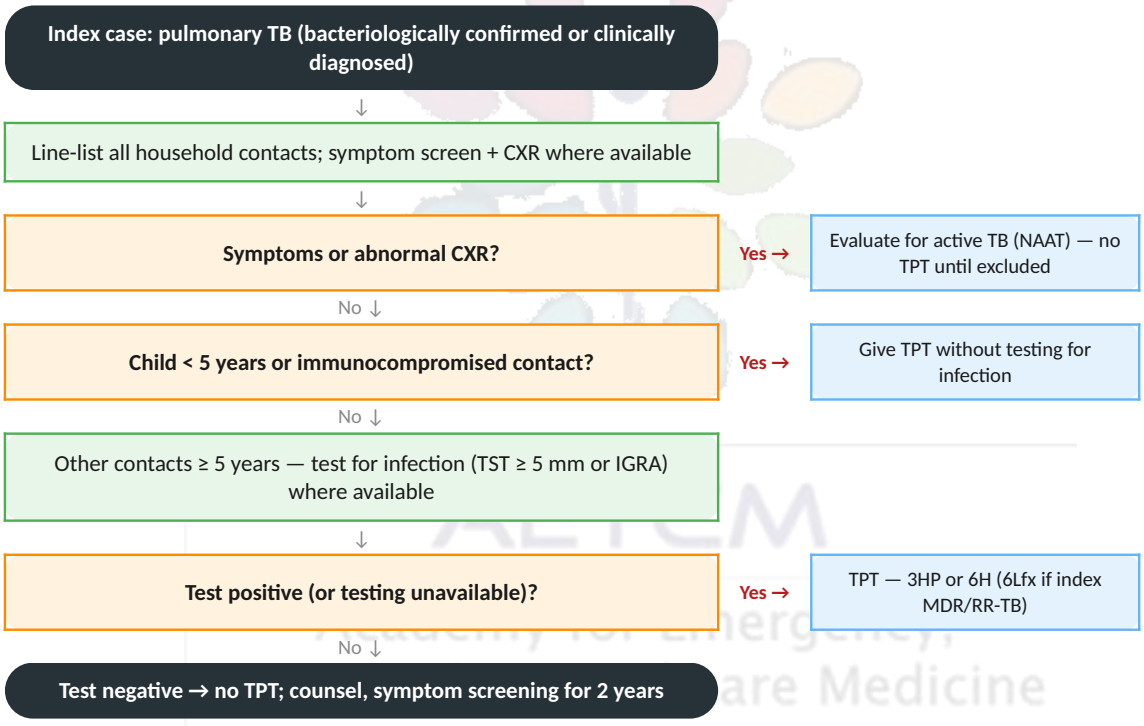
Previous-year Qs ›

IN BRIEF India's TPT policy extends preventive treatment to household contacts of pulmonary TB and other high-risk groups — always after active TB has been excluded.

TPT regimens (NTEP)

Regimen	Dose	Use
6H (6 months isoniazid)	10 mg/kg (< 30 kg), 5 mg/kg adults; max 300 mg	All ages; pregnancy
3HP (12 weekly doses isoniazid + rifapentine)	H 15 mg/kg (max 900 mg) + rifapentine by weight (900 mg ≥ 50 kg)	Age ≥ 2 years
1HP (28 daily doses)	H 300 mg + rifapentine 600 mg	≥ 13 years
4R (4 months rifampicin)	10 mg/kg	INH-intolerant
6Lfx (6 months levofloxacin)	15–20 mg/kg	Contacts of MDR/RR-TB (WHO 2024; V-QUIN and TB-CHAMP trials)

Algorithm E4.4.1 · Contact investigation and TPT



Interpreting TST and IGRA

- **TST (Mantoux):** 5 TU PPD-S or 2 TU RT23 intradermal; read at 48–72 h as induration. Positive ≥ 5 mm in contacts and immunosuppressed; ≥ 10 mm others. False positives — BCG, NTM; false negatives — malnutrition, steroids, measles, overwhelming TB.
- **IGRA (QuantiFERON-TB Gold Plus, T-SPOT.TB):** ESAT-6 and CFP-10 antigens absent from BCG and most NTM (except M. kansasii, marinum, szulgai). Single visit and more specific; cannot distinguish latent from active TB.
- **Antigen-based skin tests (Cy-Tb, C-Tb):** IGRA-like specificity with a skin test format (WHO 2022).

★ CLINICAL PEARLS & EXAM FOCUS

- Before anti-TNF biologics — screen with IGRA/TST and CXR; begin TPT at least one month before the biologic.
- TPT is never given until active TB is excluded — monotherapy for active TB breeds resistance.

MERCK MANUAL PROFESSIONAL Latent tuberculosis infection

E4.5 Tuberculous Pleural Effusion — Diagnostic & Management Algorithm

‹ Core topic 4.12 · p.86

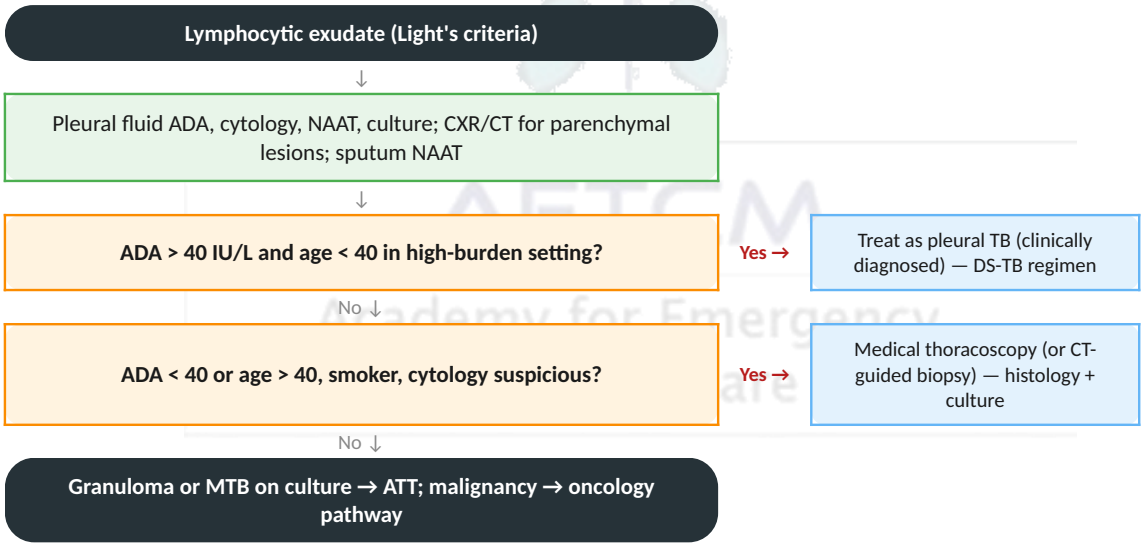
Previous-year Qs ›

IN BRIEF TB is the commonest cause of exudative pleural effusion in India. The diagnosis is usually made on fluid biochemistry; pleural biopsy settles the doubtful case.

Pleural fluid profile in TB vs its mimics

Feature	TB	Parapneumonic / empyema	Malignant
Cells	Lymphocytic (> 50%, often > 80%); early neutrophilic	Neutrophilic	Lymphocytic; malignant cells
ADA	> 40 IU/L (ADA2 isoform)	Can be very high in empyema	Usually < 40 (high in lymphoma)
Glucose / pH	Low–normal	Very low glucose, pH < 7.2	Low in heavy tumour burden
Mesothelial cells	< 5%	Variable	Variable
LDH	High	Very high	High
Yield of biopsy	Thoracoscopic granuloma > 95%	—	Thoracoscopy > 90%

Algorithm E4.5.1 · Lymphocytic exudate — is it TB?



Management points

- Standard 6-month DS-TB regimen; therapeutic thoracentesis for large symptomatic effusions.
- Corticosteroids do not reduce residual pleural thickening or improve lung function — not routinely recommended.

- Residual pleural thickening (> 10 mm) occurs in up to half — early drainage and physiotherapy may help; decortication for symptomatic trapped lung.
- Tuberculous empyema: drain, ATT for longer (≥ 9 months) and early surgical opinion — bronchopleural fistula is common.

★ CLINICAL PEARLS & EXAM FOCUS

- Paradoxical enlargement of a TB effusion during ATT occurs in ≈ 10% — not treatment failure.
- ADA > 250 IU/L should make you think of empyema or lymphoma rather than TB.

RADIOPAEDIA · IMAGE LINKS Tuberculous pleural effusion cases Medical thoracoscopy cases

MERCK MANUAL PROFESSIONAL Pleural effusion

E4.6 Post-TB Lung Disease, Haemoptysis & Surgery in TB

‹ Core topic 4.14 · p.89
Previous-year Qs ›

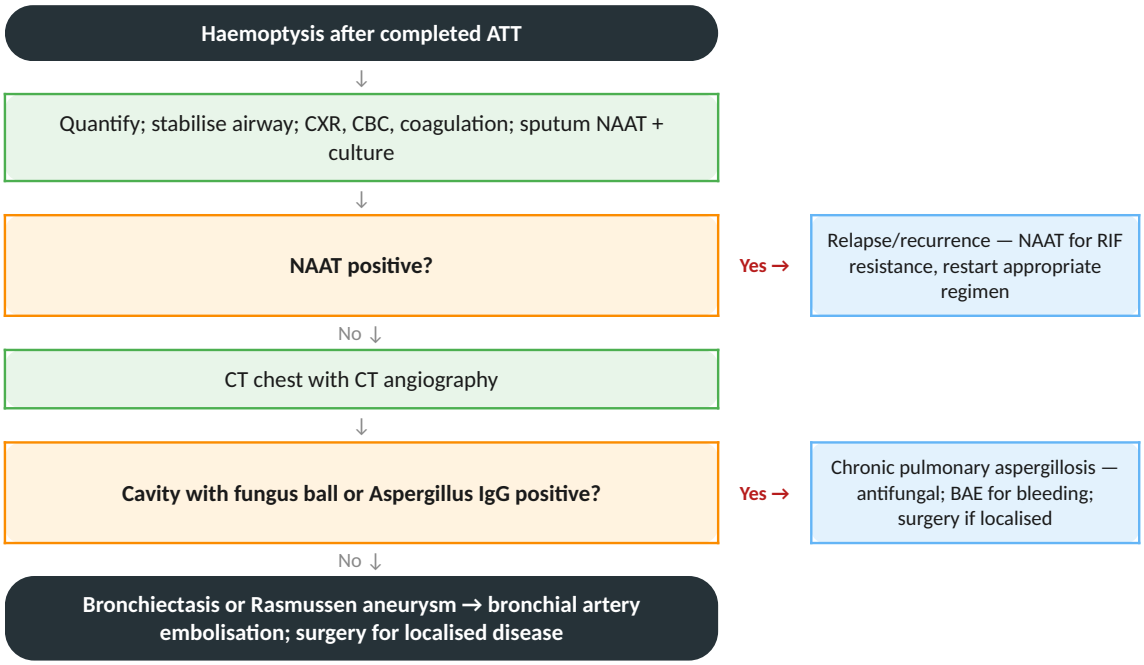
IN BRIEF Up to half of TB survivors have persistent lung damage — airflow obstruction, bronchiectasis, cavities with aspergilloma, fibrosis and pulmonary hypertension. Post-TB lung disease (PTLD) is now recognised as a chronic disease in its own right.

Patterns of post-TB lung disease

Pattern	Clinical problem	Management
Airflow obstruction ("TB-associated COPD")	Breathlessness, exacerbations	Inhaled bronchodilators (LAMA/LABA), rehabilitation; ICS only if eosinophilic
Bronchiectasis	Recurrent infections, haemoptysis	Airway clearance, treat exacerbations, vaccination
Residual cavity / CPA	Haemoptysis, weight loss	Aspergillus IgG; itraconazole/voriconazole ≥ 6 months; surgery for simple aspergilloma
Fibrosis and volume loss (destroyed lung)	Restriction, hypoxaemia, PH	LTOT, NIV in hypercapnia, pneumonectomy in selected
Broncholithiasis, tracheobronchial stenosis	Wheeze, collapse	Bronchoscopic dilatation, stenting

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Algorithm E4.6.1 · Haemoptysis in a TB survivor



Indications for surgery in TB

- Localised MDR/XDR-TB with persistent culture positivity despite optimal therapy (partial lung resection improves outcome).
- Massive or recurrent haemoptysis not controlled by embolisation.
- Destroyed lobe or lung with recurrent infection; aspergilloma; bronchopleural fistula; tuberculous empyema with trapped lung.
- Diagnostic doubt (nodule vs cancer); spinal TB with neurological deficit or instability.

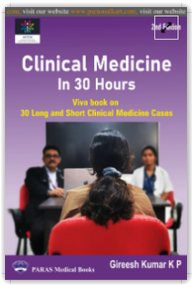
★ CLINICAL PEARLS & EXAM FOCUS

- Recurrent TB in a survivor must be confirmed bacteriologically — residual radiological shadows mislead.
- Pulmonary rehabilitation improves exercise capacity in PTLD as much as in COPD.

RADIOPAEDIA · IMAGE LINKS Rasmussen aneurysm cases Aspergilloma cases Destroyed lung cases

MERCK MANUAL PROFESSIONAL Bronchiectasis Aspergillosis

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E4.7 Drug Chart — Drugs in TB Prevention, Adjunctive & Supportive Care

Previous-year Qs ›

IN BRIEF Beyond the ATT backbone — drugs for preventive treatment, corticosteroid adjuncts, and supportive care in TB.

TB adjunctive and supportive drugs

Class	Drug	Dose	Indication	Caution
Rifamycin (long-acting)	Rifapentine	900 mg weekly (≥ 50 kg) in 3HP; 600 mg daily in 1HP	TB preventive treatment	Flu-like reactions, hepatitis, drug interactions
Hydrazide	Isoniazid	5 mg/kg (max 300 mg) daily × 6 months	6H TPT	Hepatitis; give pyridoxine
Fluoroquinolone	Levofloxacin	15–20 mg/kg daily × 6 months	TPT for contacts of MDR-TB	Tendinopathy, QT, children — joint concerns minimal
Corticosteroid	Dexamethasone	0.4 mg/kg/day IV, tapered over 6–8 weeks	TB meningitis (all grades)	Hyperglycaemia, GI bleed
Corticosteroid	Prednisolone	1 mg/kg (≈ 60 mg) tapered over 6–12 weeks	TB pericarditis (selected), IRIS, severe hypersensitivity	Rifampicin halves steroid levels — use higher dose
Corticosteroid	Prednisolone	0.5–1 mg/kg	Spinal TB with cord compression, airway compression by nodes in children	Monitor glucose
Vitamin	Pyridoxine	10 mg daily (25–50 mg in high-risk)	Prevention of INH neuropathy	—
Antiemetic	Ondansetron / domperidone	4–8 mg / 10 mg before ATT	Gastrointestinal intolerance	QT with Bdq/Mfx (ondansetron)
Antifibrinolytic	Tranexamic acid	1 g IV / 500 mg–1 g oral TDS	Mild-moderate haemoptysis	Thrombosis
Hepatoprotective	Not recommended routinely	—	—	No proven benefit in preventing DILI
Nutrition	Energy-protein supplementation	Food baskets / Ni-kshay Poshan	Undernourished TB patients	RATIONS trial — reduced TB incidence in contacts

LANDMARK TRIALS & GUIDELINES

- **RATIONS (2023, Jharkhand):** nutritional support to household contacts reduced TB incidence by about 40%.

PART II · SECTION 5

Non-Tuberculous Infections of the Lung

Advanced theory & algorithms

E5.1 Community-Acquired Pneumonia — Severity, Site of Care & Empirical Therapy 365
E5.2 HAP & VAP — Diagnosis, Risk-Based Empirical Therapy & De-escalation 367
E5.3 Non-Resolving Pneumonia — Systematic Approach 368
E5.4 Bronchiectasis — Evaluation, Exacerbation & Long-term Management 370
E5.5 Chronic Pulmonary Aspergillosis & Mucormycosis — Diagnostic Algorithms 371
E5.6 Pulmonary Infiltrates in the Immunocompromised Host — Algorithm 372
E5.7 Drug Chart — Antimicrobials for Respiratory Infections 375
E5.8 Viral Pneumonias, Influenza & COVID-19 — Management Algorithms 376
E5.9 Tropical & Zoonotic Pneumonias in India 377

E5.1 Community-Acquired Pneumonia — Severity, Site of Care & Empirical Therapy

‹ Core topic 5.2 · p.97
Previous-year Qs ›

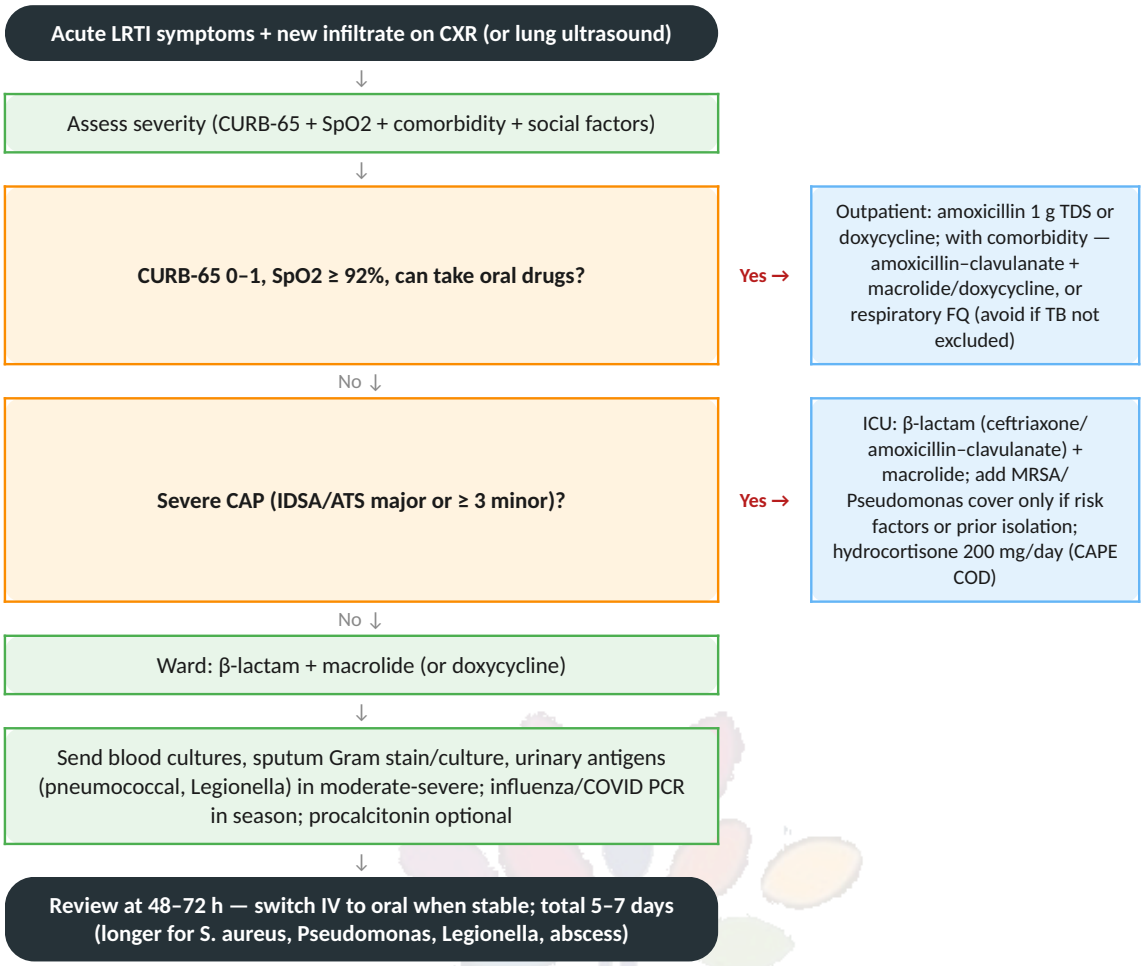
IN BRIEF Severity assessment decides where the patient is treated and how broad the antibiotics are. Use a score, but never let a score overrule clinical judgement — hypoxaemia alone mandates admission.

Severity scores

Score	Components	Action
CURB-65	Confusion, Urea > 7 mmol/L (BUN > 19 mg/dL), RR ≥ 30, BP < 90 systolic or ≤ 60 diastolic, age ≥ 65	0–1 home; 2 hospital; 3–5 severe — consider ICU
CRB-65	As above without urea	For primary care
PSI (PORT)	20 variables, classes I–V	Classes I–III outpatient; more sensitive for low risk
IDSA/ATS 2007 severe CAP	Major: septic shock on vasopressors, invasive ventilation. Minor (≥ 3): RR ≥ 30, PaO2/FiO2 ≤ 250, multilobar, confusion, BUN ≥ 20, WBC < 4000, platelets < 100 000, T < 36 °C, hypotension needing aggressive fluids	ICU admission
SMART-COP	Systolic BP, multilobar, albumin, RR, tachycardia, confusion, oxygen, pH	Predicts need for ventilatory or vasopressor support

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Algorithm E5.1.1 · CAP — from diagnosis to antibiotic



Indian context (ICS/NCCP guidelines)

- Pathogens: S. pneumoniae commonest; also H. influenzae, atypicals, S. aureus, Klebsiella; scrub typhus, leptospirosis, melioidosis and influenza in season.
- Always consider TB in subacute pneumonia and avoid empirical fluoroquinolone monotherapy.
- Doxycycline covers atypicals and scrub typhus — useful in monsoon-season fever with pneumonia.

LANDMARK TRIALS & GUIDELINES

- **CAPE COD (2023):** hydrocortisone in severe CAP reduced 28-day mortality (6.2% vs 11.9%).
- **CAP-START:** β-lactam monotherapy non-inferior to β-lactam–macrolide or FQ in non-ICU patients (Netherlands).

★ CLINICAL PEARLS & EXAM FOCUS

- Clinical stability criteria (Halm): T ≤ 37.8 °C, HR ≤ 100, RR ≤ 24, SBP ≥ 90, SpO2 ≥ 90%, normal mentation, able to eat.
- Radiological resolution lags behind clinical recovery by 6–12 weeks — repeat CXR at 6 weeks in smokers > 50 years to exclude cancer.

RADIOPAEDIA · IMAGE LINKS Lobar pneumonia cases Air bronchogram cases Round pneumonia cases

AETCM YOUTUBE · VIDEO LECTURES ▶ pneumonia ▶ CAP management ▶ sepsis

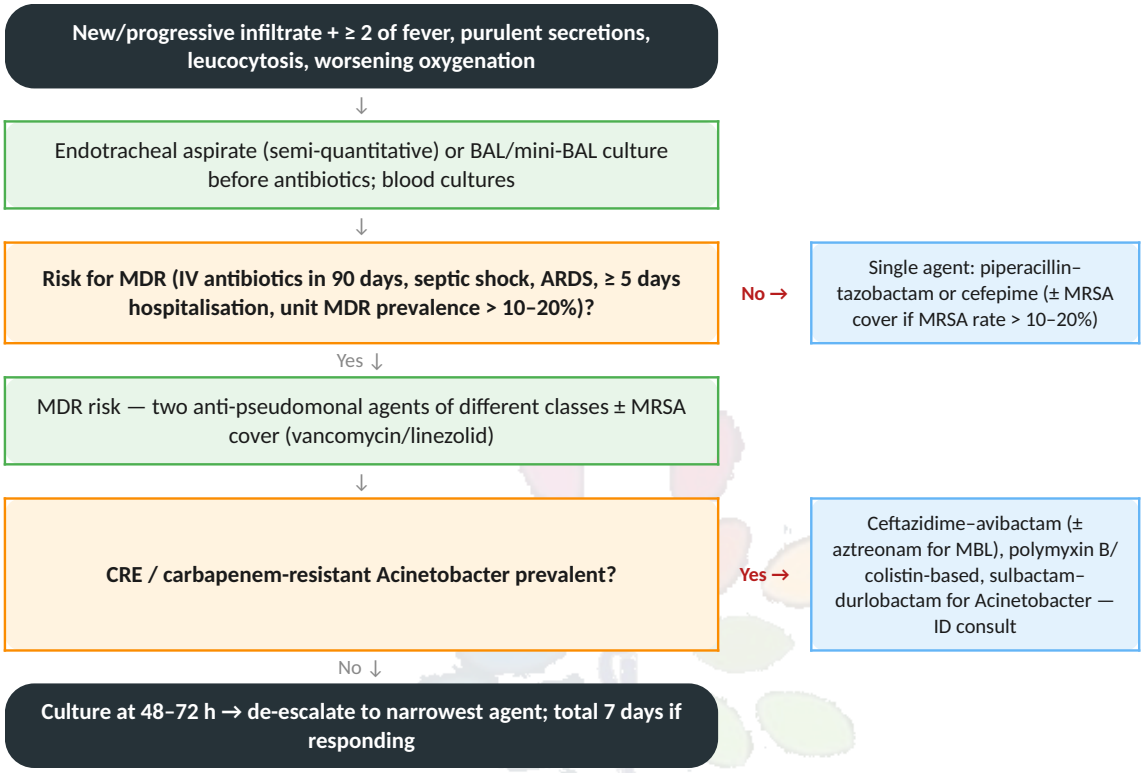
MERCK MANUAL PROFESSIONAL Community-acquired pneumonia

E5.2 HAP & VAP — Diagnosis, Risk-Based Empirical Therapy & De-escalation

‹ Core topic 5.3 · p.99
Previous-year Qs ›

IN BRIEF HAP occurs ≥ 48 h after admission; VAP ≥ 48 h after intubation. Multidrug-resistant gram-negatives (Acinetobacter, Klebsiella, Pseudomonas) dominate Indian ICUs — local antibiograms must drive choices.

Algorithm E5.2.1 · Suspected VAP



Prevention bundle for VAP

Element	Rationale
Head-of-bed elevation 30–45°	Reduces aspiration
Daily sedation interruption + SBT	Shortens ventilation
Subglottic secretion drainage tubes	Reduces micro-aspiration
Cuff pressure 20–30 cmH2O	Prevents leak without mucosal injury
Oral care (chlorhexidine debated) and toothbrushing	Reduces colonisation
Avoid unnecessary acid suppression and circuit changes	Reduce colonisation
Early mobilisation, early extubation to NIV/HFNC	Fewer ventilator days

★ **CLINICAL PEARLS & EXAM FOCUS**

- CPIS > 6 supports VAP but has poor accuracy — do not rely on it alone.
- Inhaled colistin/amikacin adjunct only for MDR organisms susceptible to these alone.
- Procalcitonin-guided stopping reduces antibiotic days without harm.

MERCK MANUAL PROFESSIONAL Hospital-acquired pneumonia | Ventilator-associated pneumonia

E5.3 Non-Resolving Pneumonia — Systematic Approach

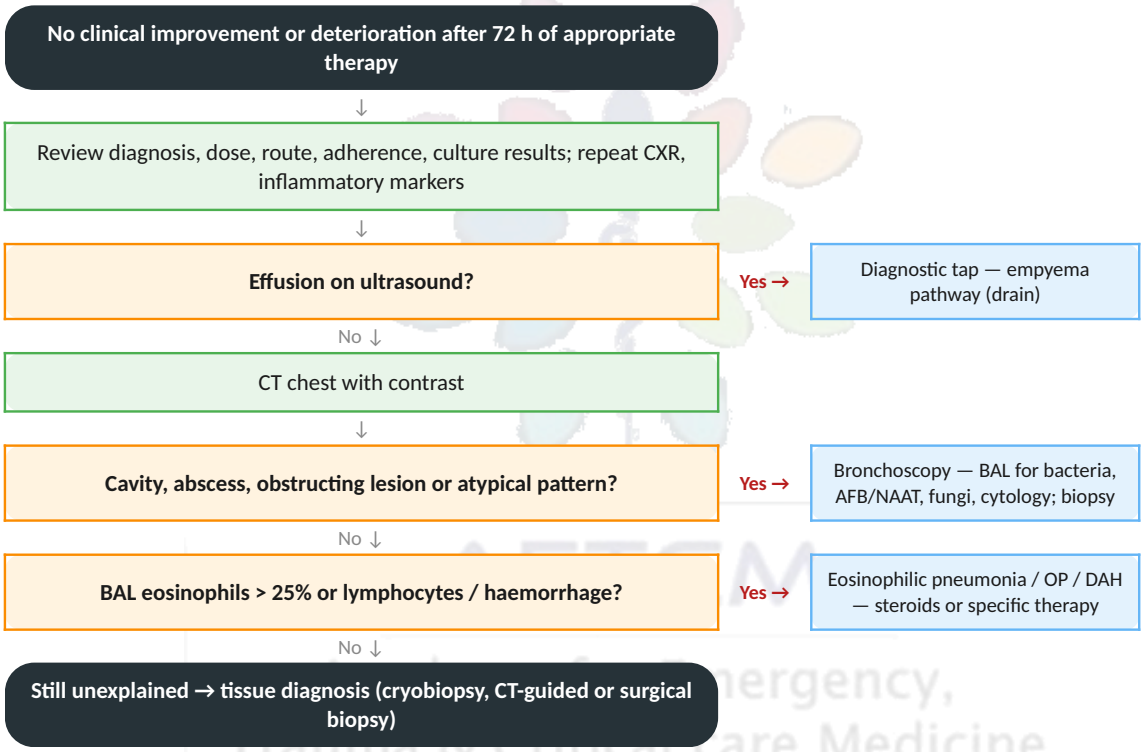
‹ Core topic 5.12 · p.113
Previous-year Qs ›

IN BRIEF A pneumonia that fails to improve clinically within 72 hours, or radiologically within the expected time, is either the wrong drug, the wrong bug, a complication, or not pneumonia at all.

Causes of non-resolving pneumonia

Category	Examples
Host factors	Age, COPD, diabetes, alcohol, immunosuppression — slow resolution
Wrong organism / resistant organism	TB, MRSA, MDR gram-negatives, melioidosis, fungi, Nocardia, Actinomyces, viruses, scrub typhus
Complications	Empyema, lung abscess, metastatic infection (endocarditis), drug fever, C. difficile
Obstruction	Endobronchial tumour, foreign body, broncholith
Non-infectious mimics	Organising pneumonia, eosinophilic pneumonia, vasculitis/DAH, lymphoma, adenocarcinoma (lepidic), pulmonary oedema, infarction, drug-induced, sarcoid

Algorithm E5.3.1 · Pneumonia not responding at 72 hours



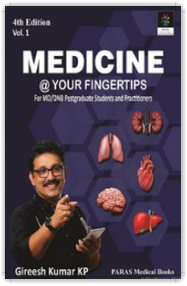
★ **CLINICAL PEARLS & EXAM FOCUS**

- Expected radiological clearance: 50% at 2 weeks, 64% at 4 weeks, 73% at 6 weeks — slower in elderly and multilobar disease.
- Recurrent pneumonia in the same lobe = local cause (obstruction, bronchiectasis, sequestration); in different lobes = host defect (immunodeficiency, aspiration, CF).

RADIOPAEDIA · IMAGE LINKS Organising pneumonia cases · Lung abscess cases

MERCK MANUAL PROFESSIONAL Pneumonia

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E5.4 Bronchiectasis — Evaluation, Exacerbation & Long-term Management

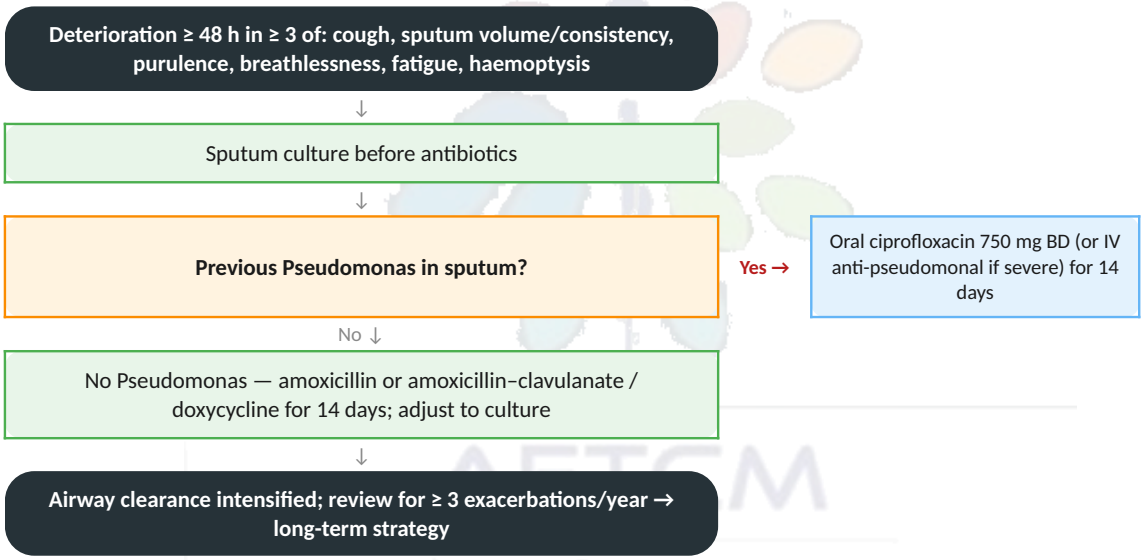
‹ Core topic 5.7 · p.104
Previous-year Qs ›

IN BRIEF Non-CF bronchiectasis is common in India — post-TB, post-infective and ABPA dominate. The aim is to find a treatable cause, clear the airways, and break the vicious vortex of infection, inflammation and damage.

Aetiological work-up (ERS 2017 / BTS 2019 minimum bundle)

Test	Detects
CBC with eosinophils, total IgE, Aspergillus-specific IgE/IgG	ABPA
Serum immunoglobulins (IgG, IgA, IgM) ± vaccine responses	Antibody deficiency (CVID)
Sputum culture including mycobacteria	Pseudomonas, NTM, TB
RF, anti-CCP, ANA	Rheumatoid arthritis, CTD
Sweat chloride / CFTR genetics (young, upper-lobe, pancreatic)	Cystic fibrosis
Nasal NO, ciliary studies (situs, sinusitis, otitis)	Primary ciliary dyskinesia
Alpha-1 antitrypsin (basal emphysema)	AATD

Algorithm E5.4.1 · Acute exacerbation of bronchiectasis



Long-term management

- **Airway clearance for all:** active cycle of breathing, oscillating PEP (Acapella, flutter), postural drainage; hypertonic saline 7% or isotonic saline before physiotherapy.
- **≥ 3 exacerbations per year:** long-term macrolide (azithromycin 250 mg daily or 500 mg thrice weekly — EMBRACE, BLESS) after excluding NTM; inhaled colistin/tobramycin if Pseudomonas.
- **Pseudomonas first isolation:** eradication — ciprofloxacin 2 weeks ± inhaled antibiotic for 3 months.
- **Brensocatib** (DPP-1 inhibitor, ASPEN 2025) — first approved anti-neutrophilic oral therapy reducing exacerbations.
- Pulmonary rehabilitation, vaccinations; surgery for localised disease or uncontrolled haemoptysis; transplant for end-stage.

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Bronchiectasis Severity Index (BSI) — elements

Element	Points (examples)
Age	≥ 80 years = 6
BMI < 18.5	2
FEV1 < 30% predicted	3
Hospital admission in previous 2 years	5
Exacerbations ≥ 3 in past year	2
mMRC 4	3
Pseudomonas colonisation	3
Radiological ≥ 3 lobes or cystic	1

LANDMARK TRIALS & GUIDELINES

- **ASPEN (2025):** brensocatib reduced pulmonary exacerbations.
- **EMBRACE, BLESS, BAT:** macrolides reduce exacerbations by about 40–60%.

★ CLINICAL PEARLS & EXAM FOCUS

- HRCT signs: bronchoarterial ratio > 1 (signet-ring), lack of tapering, airways visible within 1 cm of pleura.
- Always rule out NTM before starting long-term macrolide (resistance induction).

RADIOPAEDIA · IMAGE LINKS Bronchiectasis cases | Signet ring sign cases | Tram track sign cases

MERCK MANUAL PROFESSIONAL Bronchiectasis

E5.5 Chronic Pulmonary Aspergillosis & Mucormycosis — Diagnostic Algorithms

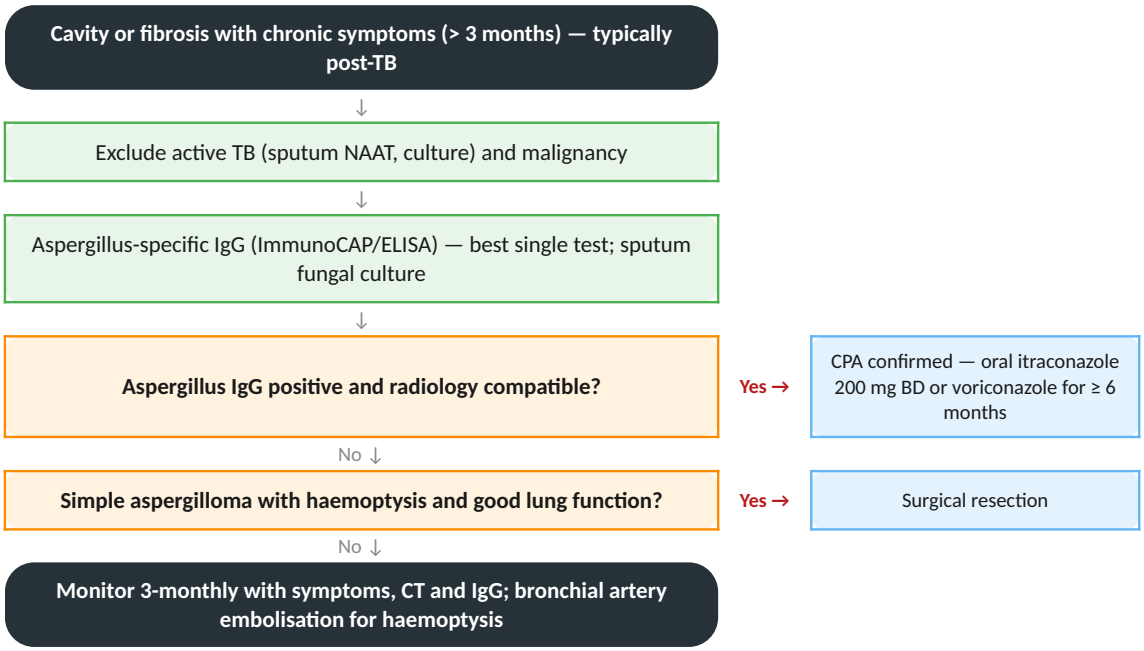
‹ Core topic 5.8 · p.106
Previous-year Qs ›

IN BRIEF India carries one of the world's largest burdens of CPA — mostly in TB survivors — and saw the 2021 epidemic of COVID-associated mucormycosis. Both need early recognition.

The aspergillosis spectrum

Entity	Host	Key features
Aspergilloma (simple)	Pre-existing cavity, immunocompetent	Mobile fungus ball, air crescent; stable for 3 months; haemoptysis
Chronic cavitary PA (CCPA)	TB survivors, COPD	≥ 1 cavity ± fungus ball, pericavitary fibrosis, symptoms ≥ 3 months
Chronic fibrosing PA	Untreated CCPA	Destruction of ≥ 2 lobes
Aspergillus nodule	Mild immune defect	Nodule mimicking cancer
Subacute invasive (CNPA)	Mild immunosuppression (diabetes, steroids, alcohol)	Progression over 1–3 months
Invasive PA	Neutropenia, transplant, ICU/influenza/COVID	Halo sign, angioinvasion, galactomannan
ABPA	Asthma, CF	Hypersensitivity — Section 6

Algorithm E5.5.1 · Diagnosing chronic pulmonary aspergillosis



Mucormycosis

- **Risk:** uncontrolled diabetes/DKA, corticosteroids, neutropenia, iron overload (deferoxamine), COVID-19.
- **Pulmonary:** reverse halo sign, vessel occlusion, multiple nodules, rapid cavitation; rhino-orbito-cerebral form commonest in India.
- **Treatment triad:** control the predisposition (glucose, stop steroids), surgical debridement, liposomal amphotericin B 5–10 mg/kg/day → step-down to posaconazole or isavuconazole.

★ CLINICAL PEARLS & EXAM FOCUS

- An air-crescent sign in a neutropenic patient signals recovery of neutrophils in invasive aspergillosis — different meaning from aspergilloma.
- Black necrotic eschar on the palate or turbinate in a diabetic = mucormycosis — emergency.

RADIOPAEDIA · IMAGE LINKS Aspergilloma cases | Air crescent sign cases | Reverse halo sign cases | Chronic pulmonary aspergillosis cases

MERCK MANUAL PROFESSIONAL Aspergillosis | Mucormycosis

E5.6 Pulmonary Infiltrates in the Immunocompromised Host — Algorithm

‹ Core topic 5.11 · p.110
Previous-year Qs ›

IN BRIEF Fever with lung infiltrates in a transplant recipient, a patient on chemotherapy or high-dose steroids is a race against time. The pattern on CT and the type of immune defect point to the likely organisms.

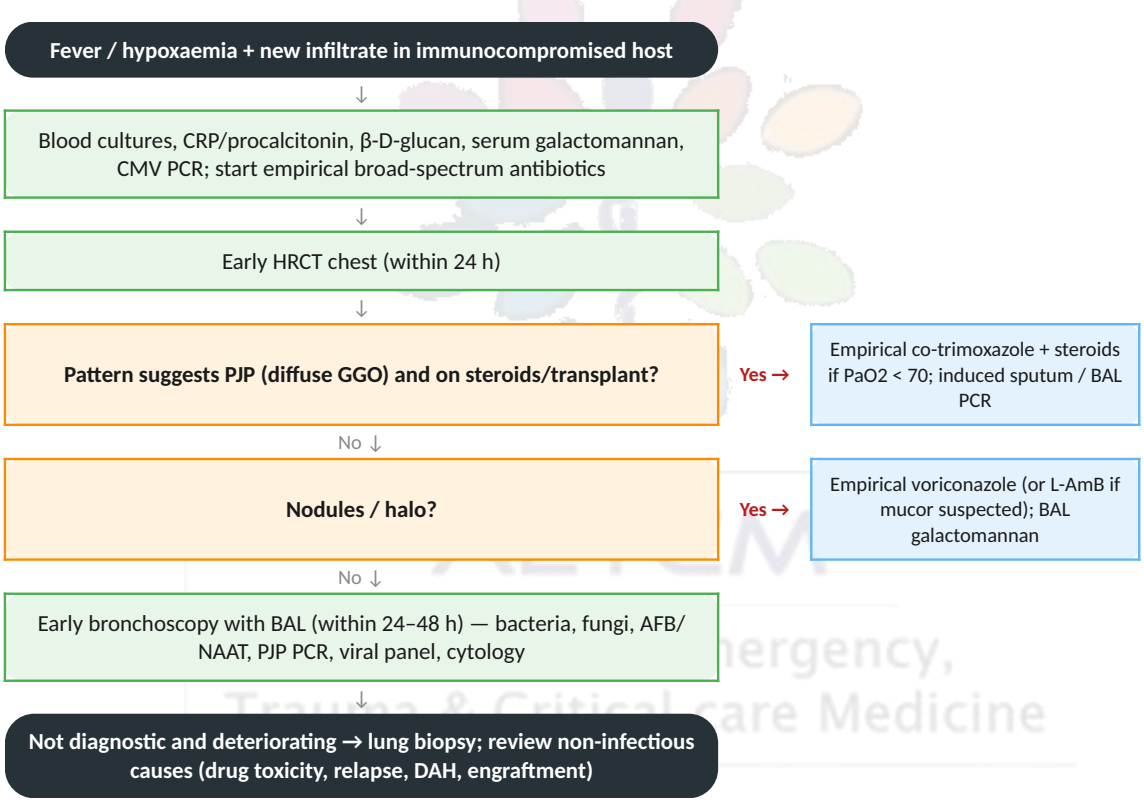
Immune defect and likely pathogens

Immune defect	Example host	Likely organisms
Neutropenia	Chemotherapy, aplastic anaemia	Gram-negatives, S. aureus, Aspergillus, Mucorales, Candida
T-cell defect	Transplant, high-dose steroids, lymphoma	Pneumocystis, CMV, TB, Nocardia, Cryptococcus, Legionella, endemic fungi
B-cell / humoral defect	CVID, myeloma, rituximab, splenectomy	Encapsulated bacteria — pneumococcus, H. influenzae
Anti-TNF therapy	Rheumatoid, IBD	TB reactivation, histoplasmosis, Listeria

CT pattern and differential

CT pattern	Think of
Nodules with halo / reverse halo	Invasive aspergillosis / mucormycosis
Diffuse ground-glass, upper zone, cysts	Pneumocystis (PJP)
Lobar consolidation	Bacterial pneumonia, Legionella
Tree-in-bud, cavity	TB, NTM, Nocardia
Ground-glass + septal thickening	CMV, drug toxicity, pulmonary oedema, DAH

Algorithm E5.6.1 · Febrile immunocompromised patient with infiltrates



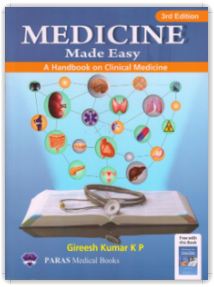
★ CLINICAL PEARLS & EXAM FOCUS

- β-D-glucan is very high in PJP — a negative result makes PJP unlikely.
- Prophylaxis with co-trimoxazole is indicated with prednisolone ≥ 20 mg/day for ≥ 4 weeks plus another immunosuppressant.

RADIOPAEDIA · IMAGE LINKS Pneumocystis pneumonia cases | Halo sign cases | Invasive pulmonary aspergillosis cases

MERCK MANUAL PROFESSIONAL | Pneumonia in immunocompromised patients | Pneumocystis pneumonia

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E5.7 Drug Chart — Antimicrobials for Respiratory Infections

Previous-year Qs ›

IN BRIEF Usual adult doses with normal renal function. Adjust for renal function and local antibiograms.

Antibacterials and antivirals in pulmonary infections

Class	Drug	Dose	Indication	Caution
Aminopenicillin	Amoxicillin	500 mg–1 g TDS oral	Outpatient CAP, bronchiectasis exacerbation	Penicillin allergy
β-lactam/β-lactamase inhibitor	Amoxicillin–clavulanate	625 mg TDS oral; 1.2 g IV TDS	CAP with comorbidity, aspiration, lung abscess	Cholestatic hepatitis, diarrhoea
β-lactam/β-lactamase inhibitor	Piperacillin–tazobactam	4.5 g IV 6–8-hourly (extended infusion)	HAP/VAP, Pseudomonas	Hypokalaemia, AKI with vancomycin
3rd-generation cephalosporin	Ceftriaxone	1–2 g IV OD	Inpatient CAP	Biliary sludge
4th-generation cephalosporin	Cefepime	2 g IV 8-hourly	HAP/VAP, Pseudomonas	Neurotoxicity in renal failure
Carbapenem	Meropenem	1 g IV 8-hourly (2 g in meningitis / extended infusion)	ESBL, severe nosocomial	Reserve; seizures (imipenem > meropenem)
Novel BL/BLI	Ceftazidime–avibactam (± aztreonam)	2.5 g IV 8-hourly	CRE (KPC, OXA-48); MBL with aztreonam	Stewardship
Macrolide	Azithromycin	500 mg OD × 3–5 days; 250 mg OD long-term	Atypical cover, bronchiectasis/COPD prophylaxis	QT, hearing loss, NTM resistance
Macrolide	Clarithromycin	500 mg BD	CAP, NTM (MAC)	CYP3A4 interactions
Tetracycline	Doxycycline	100 mg BD	CAP, scrub typhus, leptospirosis, melioidosis (maintenance)	Photosensitivity; avoid in pregnancy
Respiratory fluoroquinolone	Levofloxacin / Moxifloxacin	750 mg OD / 400 mg OD	Severe CAP with allergy; Legionella	Masks TB; QT; tendinopathy
Glycopeptide	Vancomycin	15–20 mg/kg IV 8–12-hourly; trough 15–20 or AUC 400–600	MRSA pneumonia	Nephrotoxicity
Oxazolidinone	Linezolid	600 mg BD IV/oral	MRSA pneumonia (good lung penetration)	Thrombocytopenia, serotonin syndrome
Polymyxin	Polymyxin B / Colistin	Polymyxin B 2.5–3 mg/kg/day; colistin 9 MU loading then 4.5 MU BD	CRAB, CRE	Nephro- and neurotoxicity
Lincosamide	Clindamycin	600 mg IV 8-hourly	Aspiration, lung abscess, toxin suppression (PVL S. aureus)	C. difficile
Folate antagonist	Co-trimoxazole	15–20 mg/kg/day TMP in 3–4 doses (PJP); 960 mg OD/thrice weekly prophylaxis	PJP, Nocardia, melioidosis eradication	Hyperkalaemia, rash, marrow

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Class	Drug	Dose	Indication	Caution
Carbapenem / cephalosporin	Ceftazidime / Meropenem	Ceftazidime 2 g IV 6–8-hourly	Melioidosis intensive phase (≥ 2 weeks)	—
Neuraminidase inhibitor	Oseltamivir	75 mg BD × 5 days	Influenza (start within 48 h; any time if severe)	Renal dose adjustment
Antiviral	Remdesivir	200 mg IV day 1, then 100 mg × 4 days	COVID-19 pneumonia needing oxygen	LFT, renal
Corticosteroid	Hydrocortisone	200 mg/day infusion × 4–7 days, tapered	Severe CAP in ICU (CAPE COD)	Not in influenza / aspergillosis

E5.8 Viral Pneumonias, Influenza & COVID-19 — Management Algorithms

‹ Core topic 5.5 · p.101
Previous-year Qs ›

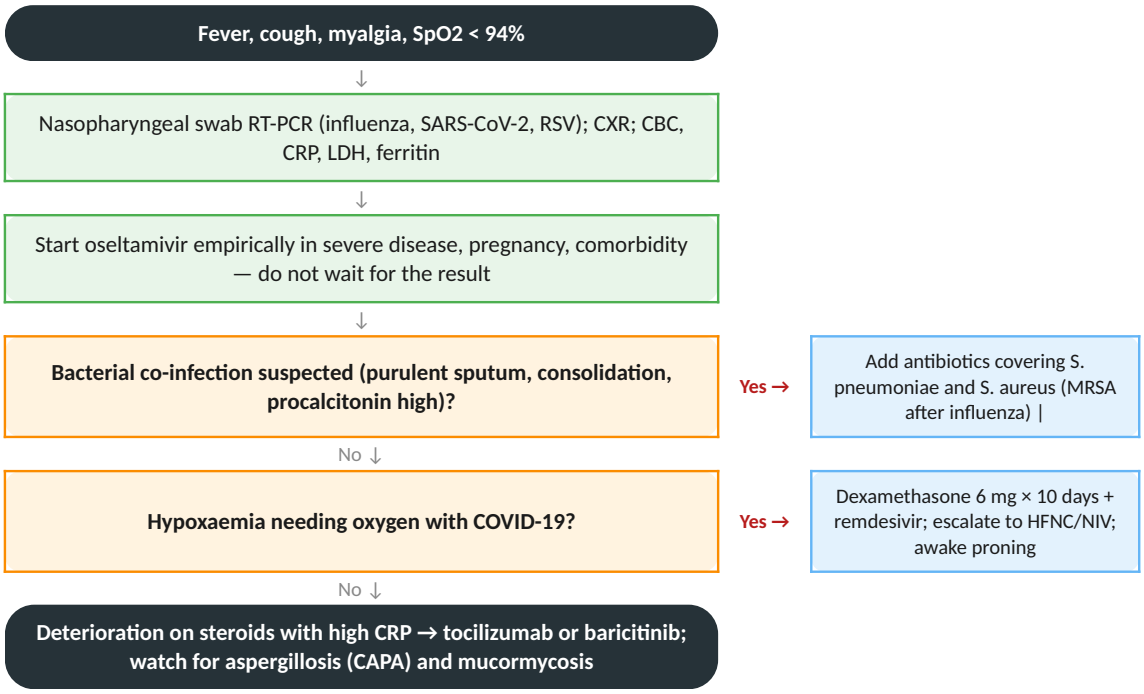
IN BRIEF Seasonal influenza and SARS-CoV-2 remain the commonest viral causes of adult pneumonia. Early antivirals, correct oxygen escalation and steroids only where proven are the four decisions that matter.

Viral pneumonias at a glance

Virus	Clues	Treatment
Influenza A/ H1N1, H3N2	Abrupt fever, myalgia, seasonal (monsoon and winter in India); rapid progression to ARDS	Oseltamivir 75 mg BD × 5 days (start within 48 h; any time if severe); baloxavir alternative
SARS-CoV-2	Anosmia, delayed hypoxaemia, peripheral GGO	Oxygen; dexamethasone 6 mg if hypoxaemic; remdesivir; tocilizumab/baricitinib if progressive inflammation
RSV	Wheeze, elderly and COPD patients	Supportive; vaccine for ≥ 60 years
Adenovirus	Conjunctivitis, pharyngitis, outbreaks	Supportive; cidofovir in immunocompromised
Varicella pneumonia	Rash, adult smokers, pregnancy; miliary calcification later	Acyclovir IV 10 mg/kg 8-hourly
CMV	Transplant, HIV; GGO with nodules	Ganciclovir / valganciclovir
Hantavirus, Nipah	Rodent / bat exposure, outbreaks (Kerala Nipah)	Supportive; ribavirin (Nipah, unproven); strict isolation

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Algorithm E5.8.1 · Influenza-like illness with hypoxaemia



★ **CLINICAL PEARLS & EXAM FOCUS**

- Steroids are harmful in influenza pneumonia (prolonged shedding, more infections) but beneficial in COVID-19 hypoxaemia — know the difference.
- Post-influenza S. aureus necrotising pneumonia in a young patient is a recognised rapid killer.

RADIOPAEDIA · IMAGE LINKS COVID-19 cases · Influenza pneumonia cases · Varicella pneumonia cases

MERCK MANUAL PROFESSIONAL Influenza · COVID-19

E5.9 Tropical & Zoonotic Pneumonias in India

‹ Core topic 5.4 · p.100
Previous-year Qs ›

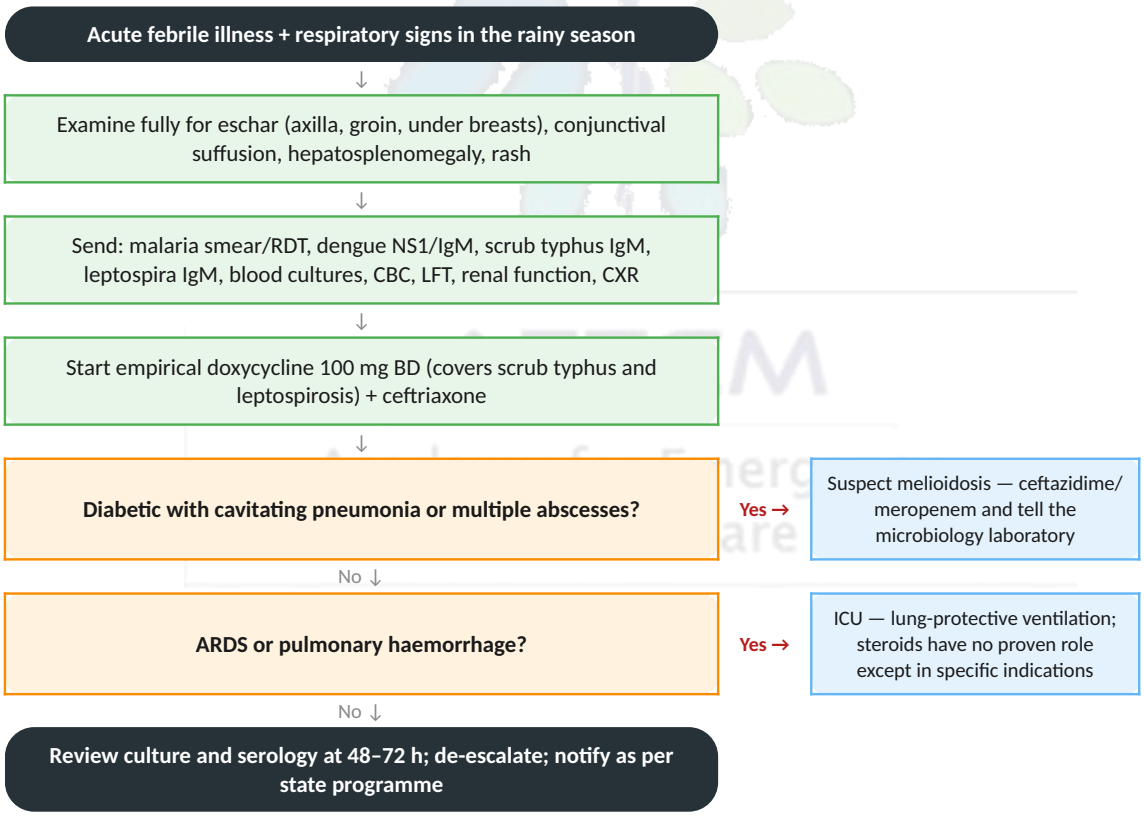
IN BRIEF Monsoon fever with pneumonia in India has a short differential that no textbook from the West will cover. Empirical doxycycline saves lives.

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Tropical infections with pulmonary involvement

Disease	Exposure / clue	Diagnosis	Treatment
Scrub typhus	Rural, mites; eschar, lymphadenopathy, ARDS	IgM ELISA, Weil–Felix (poor), PCR of eschar	Doxycycline 100 mg BD (azithromycin in pregnancy)
Leptospirosis	Flood water, rodents; conjunctival suffusion, jaundice, AKI, pulmonary haemorrhage	IgM ELISA, MAT, PCR	Penicillin G or ceftriaxone; doxycycline in mild
Melioidosis	Diabetics, soil/water exposure (coastal, NE India); cavitary pneumonia, abscesses	Culture (Burkholderia pseudomallei) — alert the lab	Ceftazidime or meropenem ≥ 2 weeks → co-trimoxazole 3–6 months
Malaria (falciparum, vivax)	Travel/endemic; ARDS, severe anaemia	Smear, rapid antigen	Artesunate IV
Dengue	Thrombocytopenia, plasma leak, pleural effusion	NS1, IgM	Fluid management; no NSAIDs
Hydatid disease	Sheep/dog contact; cyst with water-lily sign	Imaging + serology	Albendazole + surgery/PAIR; avoid rupture
Paragonimiasis	Raw crab/crayfish (NE India); haemoptysis mimicking TB	Sputum/stool ova, serology	Praziquantel
Tropical pulmonary eosinophilia	Filarial endemic; nocturnal cough, eosinophils > 3000, high IgE	Clinical + filarial antibody	Diethylcarbamazine 6 mg/kg/day × 21 days
Amoebic liver abscess rupture	Right basal effusion, anchovy-sauce pus	Serology, imaging	Metronidazole + drainage

Algorithm E5.9.1 · Monsoon fever with pneumonia and multi-organ involvement



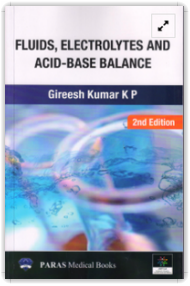
★ CLINICAL PEARLS & EXAM FOCUS

- Doxycycline given empirically within the first week dramatically reduces mortality in scrub typhus — do not wait for serology.
- Melioidosis is "the great mimicker" of TB in diabetics; culture is the only reliable diagnosis.

RADIOPAEDIA · IMAGE LINKS Melioidosis cases Hydatid disease of the lung cases Scrub typhus cases

MERCK MANUAL PROFESSIONAL Scrub typhus Leptospirosis Melioidosis

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PART II · SECTION 6

Obstructive Airway Diseases

Advanced theory & algorithms

E6.1 Diagnosis of Asthma — GINA 2024–25 Algorithm 380
E6.2 Stepwise Management of Chronic Asthma — GINA Track 1 & Track 2 382
E6.3 Acute Severe Asthma — Emergency Department Algorithm 383
E6.4 COPD — GOLD 2025 Assessment & Pharmacological Algorithm 386
E6.5 Acute Exacerbation of COPD — Hospital Algorithm & NIV Decisions 387
E6.6 ABPA — ISHAM-AWG 2024 Criteria & Management 389
E6.7 Long-Term Oxygen Therapy & Pulmonary Rehabilitation — Prescribing Algorithm 391
E6.8 Drug Chart — Inhaled & Oral Drugs for Asthma and COPD 392
E6.9 Severe Asthma — Phenotyping, Comorbidities & the Difficult-Asthma Clinic 393
E6.10 Asthma–COPD Overlap, Small-Airway Disease & Bronchiolitis 395

E6.1 Diagnosis of Asthma — GINA 2024–25 Algorithm

‹ Core topic 6.1 · p.115
Previous-year Qs ›

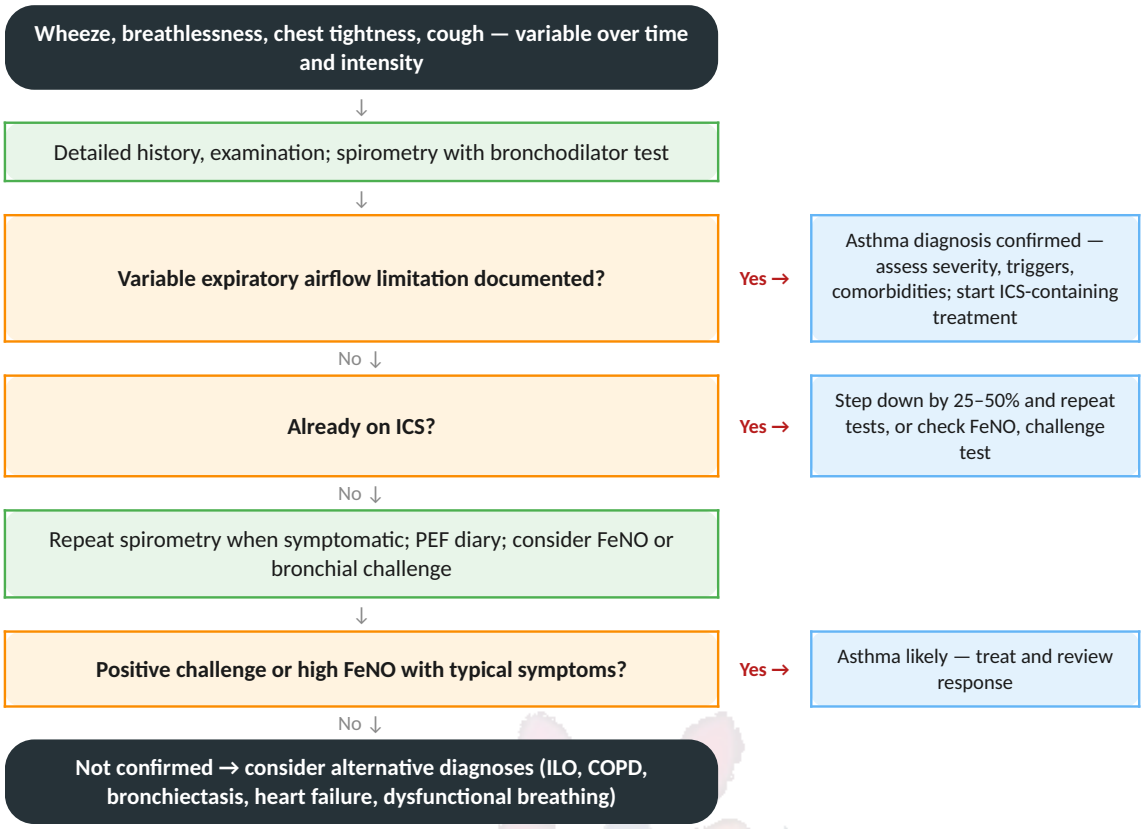
IN BRIEF Asthma is a clinical diagnosis confirmed by documented variable expiratory airflow limitation. Confirm the diagnosis before starting controller therapy whenever possible — it becomes harder once treatment has begun.

Criteria for variable airflow limitation (GINA)

Test	Positive in adults
Bronchodilator responsiveness	FEV1 increase > 12% and > 200 mL from baseline (ATS/ERS 2022: > 10% of predicted)
PEF variability	Average daily diurnal variability > 10% (twice-daily readings over 1–2 weeks)
Response to 4 weeks of anti-inflammatory treatment	FEV1 increase > 12% and > 200 mL
Exercise challenge	Fall in FEV1 > 10% and > 200 mL
Bronchial challenge (methacholine, mannitol)	Fall in FEV1 ≥ 20% (methacholine) / ≥ 15% (mannitol, hypertonic saline)
Variation between visits	FEV1 variation > 12% and > 200 mL (outside infections)
FeNO (supportive)	≥ 50 ppb in adults — supports type-2 inflammation, not diagnostic alone

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Algorithm E6.1.1 · Diagnosing asthma in a patient with respiratory symptoms



Conditions that mimic asthma

Mimic	Distinguishing clue
Inducible laryngeal obstruction (vocal cord dysfunction)	Inspiratory stridor, flattened inspiratory loop, laryngoscopy during attack
COPD	Smoking > 10 pack-years, persistent obstruction, low DLCO
Central airway obstruction	Monophonic wheeze, box-shaped flow–volume loop
Bronchiectasis	Daily purulent sputum, crackles, HRCT
Heart failure ("cardiac asthma")	Orthopnoea, BNP, echo
Eosinophilic granulomatosis with polyangiitis	Eosinophilia > 10%, neuropathy, sinusitis
ABPA	Very high IgE, fleeting infiltrates, central bronchiectasis

★ CLINICAL PEARLS & EXAM FOCUS

- A normal spirometry between attacks does not exclude asthma — repeat when symptomatic or use challenge testing.
- Cough-variant asthma presents with cough alone; the methacholine challenge is positive.
- The single best predictor of future exacerbation is a previous exacerbation in the past year.

MERCK MANUAL PROFESSIONAL Asthma

E6.2 Stepwise Management of Chronic Asthma — GINA Track 1 & Track 2

‹ Core topic 6.2 · p.117
Previous-year Qs ›

IN BRIEF GINA no longer recommends SABA-only treatment for any adult or adolescent. The preferred Track 1 uses as-needed ICS–formoterol as the reliever (anti-inflammatory reliever, AIR) at every step.

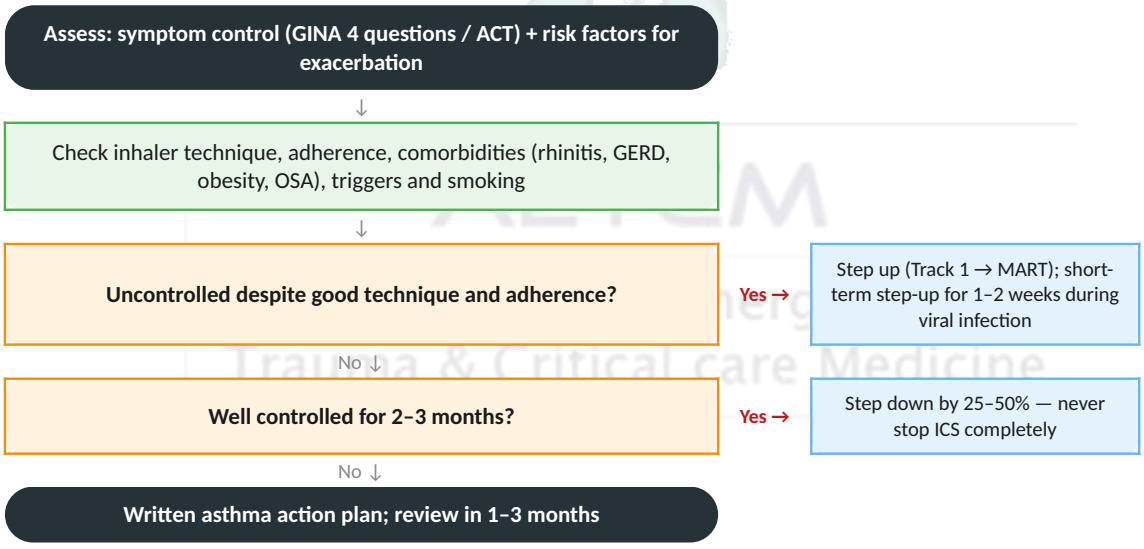
GINA adults and adolescents (≥ 12 years)

Step	Track 1 (preferred) — reliever: as-needed low-dose ICS–formoterol	Track 2 (alternative) — reliever: SABA (or ICS–SABA)
1–2	As-needed low-dose budesonide–formoterol only	Step 1: ICS taken whenever SABA is taken; Step 2: daily low-dose ICS
3	Low-dose maintenance ICS–formoterol (MART)	Low-dose ICS–LABA
4	Medium-dose maintenance ICS–formoterol (MART)	Medium/high-dose ICS–LABA
5	Add-on LAMA; refer for phenotypic assessment; consider high-dose ICS–formoterol, anti-IgE, anti-IL-5/5R, anti-IL-4R, anti-TSLP; azithromycin	Same add-ons

ICS dose equivalents (adults, daily)

ICS	Low	Medium	High
Beclometasone (pMDI, extra-fine)	100–200 µg	> 200–400 µg	> 400 µg
Budesonide (DPI)	200–400 µg	> 400–800 µg	> 800 µg
Fluticasone propionate	100–250 µg	> 250–500 µg	> 500 µg
Fluticasone furoate	100 µg	100 µg	200 µg
Ciclesonide	80–160 µg	> 160–320 µg	> 320 µg

Algorithm E6.2.1 · Review cycle for asthma control



GINA symptom control — in the past 4 weeks

Question	Answer yes = point
Daytime symptoms more than twice a week?	1
Any night waking due to asthma?	1
SABA reliever needed more than twice a week?	1
Any activity limitation due to asthma?	1
0 = well controlled; 1–2 = partly controlled; 3–4 = uncontrolled	—

LANDMARK TRIALS & GUIDELINES

- **SYGMA 1 & 2, Novel START, PRACTICAL:** as-needed budesonide–formoterol reduced severe exacerbations vs SABA alone, non-inferior to daily ICS.
- **MANDALA (2022):** as-needed albuterol–budesonide reduced severe exacerbations — basis for ICS–SABA reliever in Track 2.

★ CLINICAL PEARLS & EXAM FOCUS

- Dispensing ≥ 3 SABA canisters a year is linked to higher exacerbation risk; ≥ 12 a year to higher death risk.
- MART: maximum budesonide–formoterol 72 µg formoterol per day (12 inhalations of 200/6) in adults.

MERCK MANUAL PROFESSIONAL — Asthma treatment

E6.3 Acute Severe Asthma — Emergency Department Algorithm

‹ Core topic 6.3 · p.118
Previous-year Qs ›

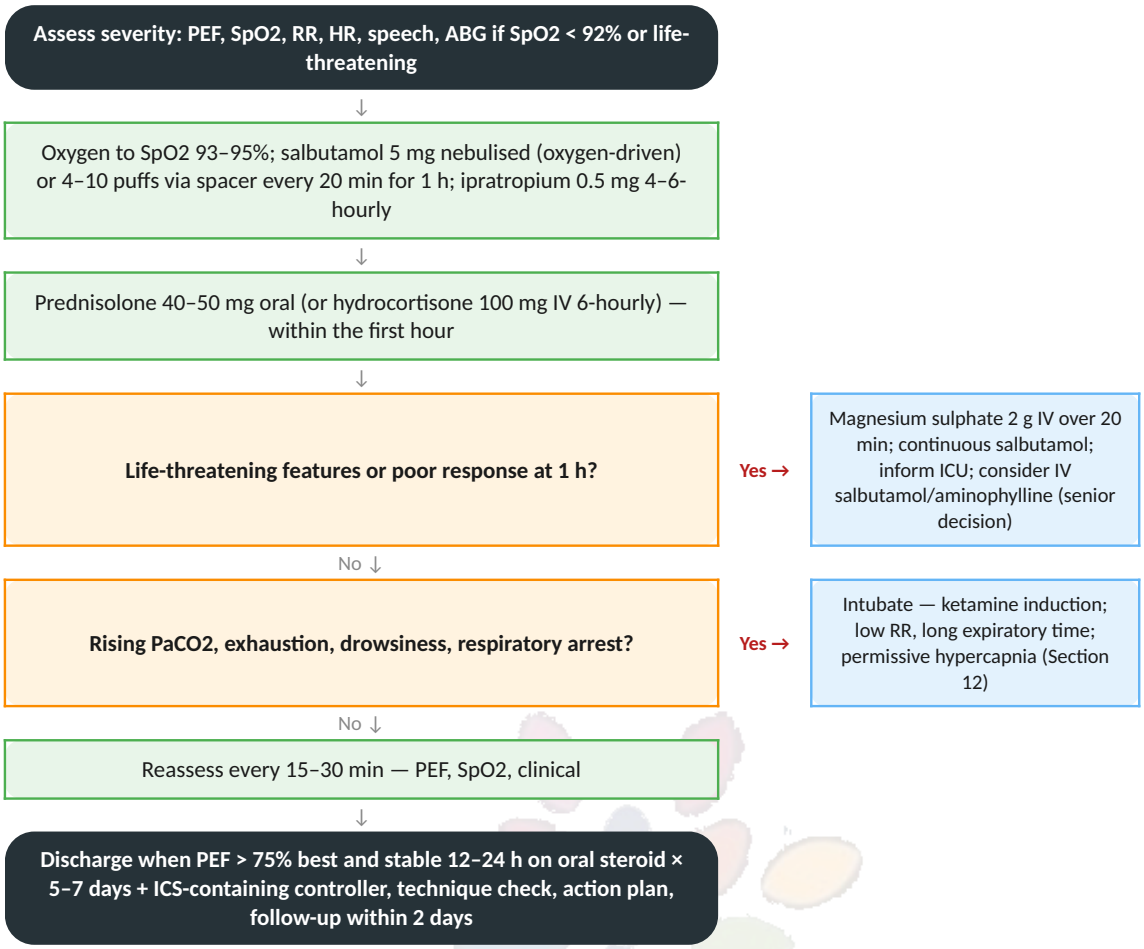
IN BRIEF Most asthma deaths are preventable and follow under-assessment. Measure severity objectively (PEF, SpO2, ABG) and treat aggressively; a normal PaCO2 in a breathless asthmatic is a danger sign.

Severity of acute asthma (BTS/SIGN, adults)

Category	Features
Moderate	PEF > 50–75% best/predicted; increasing symptoms; no features of severe
Acute severe	Any one: PEF 33–50%, RR ≥ 25/min, HR ≥ 110/min, inability to complete sentences
Life-threatening	Any one: PEF < 33%, SpO2 < 92%, PaO2 < 60 mmHg, normal PaCO2 (35–45), silent chest, cyanosis, poor effort, arrhythmia, exhaustion, altered consciousness, hypotension
Near-fatal	Raised PaCO2 and/or need for mechanical ventilation with raised inflation pressures

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Algorithm E6.3.1 · Management of acute severe asthma in the ED



Pitfalls

- Heliox and IV aminophylline add little and aminophylline causes toxicity; no role for antibiotics unless infection is evident.
- Sedation outside the ICU is contraindicated.
- Chest radiograph only if pneumothorax, pneumonia, or life-threatening features are suspected.
- Lactic acidosis from high-dose salbutamol may drive tachypnoea — do not mistake it for worsening asthma.

★ CLINICAL PEARLS & EXAM FOCUS

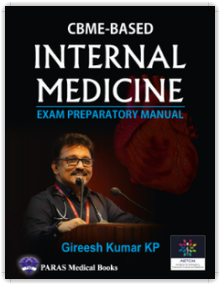
- "Silent chest" = so little airflow that no wheeze is heard — pre-arrest.
- Pulsus paradoxus > 25 mmHg indicates severe asthma, but its absence does not exclude it.

RADIOPAEDIA · IMAGE LINKS Pneumomediastinum cases · Status asthmaticus cases

AETCM YOUTUBE · VIDEO LECTURES ▶ acute severe asthma · ▶ status asthmaticus · ▶ nebulisation

MERCK MANUAL PROFESSIONAL Acute asthma exacerbation

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E6.4 COPD — GOLD 2025 Assessment & Pharmacological Algorithm

‹ Core topic 6.8 · p.127
Previous-year Qs ›

IN BRIEF GOLD combines spirometric grade (1–4) with the ABE assessment of symptoms and exacerbation risk. Blood eosinophils guide ICS use; LABA + LAMA is the backbone of maintenance therapy.

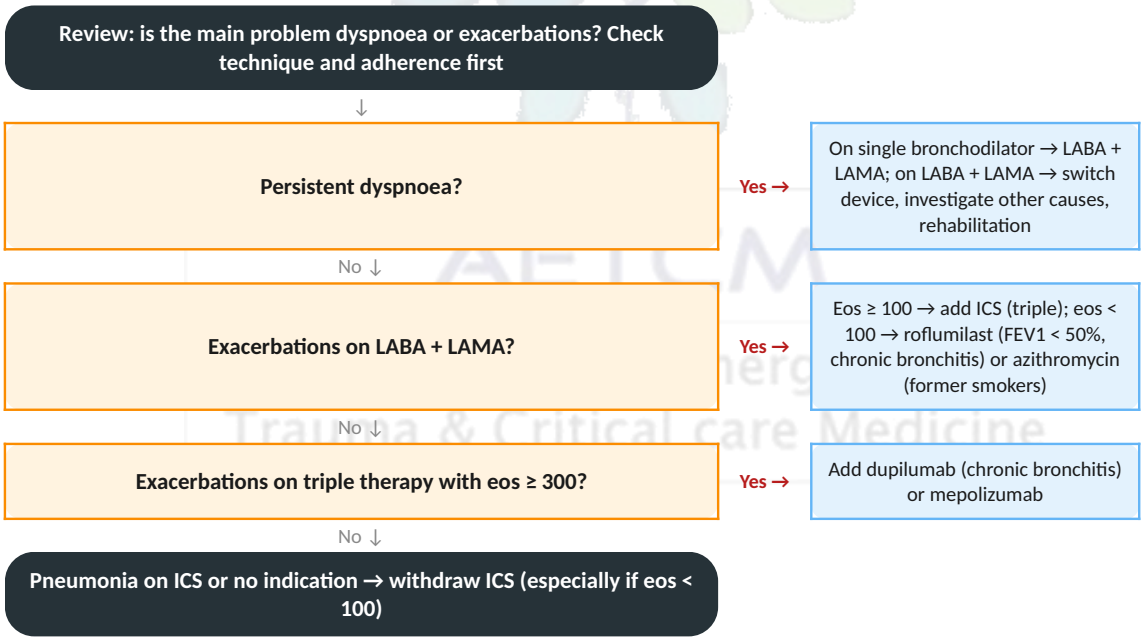
GOLD ABE assessment

Group	Exacerbations in past year	Symptoms	Initial therapy
A	0–1 (not leading to admission)	mMRC 0–1, CAT < 10	A bronchodilator (long-acting preferred)
B	0–1 (not leading to admission)	mMRC ≥ 2, CAT ≥ 10	LABA + LAMA (single inhaler)
E	≥ 2 moderate or ≥ 1 leading to hospitalisation	Any	LABA + LAMA; consider LABA + LAMA + ICS if eosinophils ≥ 300/µL

Spirometric grade (post-bronchodilator FEV1/FVC < 0.70)

GOLD grade	FEV1 % predicted
1 Mild	≥ 80
2 Moderate	50–79
3 Severe	30–49
4 Very severe	< 30

Algorithm E6.4.1 · Follow-up pharmacotherapy (GOLD 2025)



Blood eosinophils and ICS

Eosinophils	ICS
≥ 300 cells/µL	Strongly favours use
100–300 cells/µL	Favours use
< 100 cells/µL	Against use

LANDMARK TRIALS & GUIDELINES

- **IMPACT, ETHOS:** triple therapy reduced exacerbations and all-cause mortality compared with dual therapy.
- **BOREAS / NOTUS (dupilumab), MATINEE (mepolizumab):** biologics reduced exacerbations in eosinophilic COPD.
- **UPLIFT, TORCH, SUMMIT:** long-acting bronchodilator milestones.

★ CLINICAL PEARLS & EXAM FOCUS

- Smoking cessation and LTOT (in resting hypoxaemia) are the only interventions proven to prolong survival — plus triple therapy in frequent exacerbators.
- Pre-COPD and PRISm (GOLD 2023 onwards) describe people at risk with normal ratio.

RADIOPAEDIA · IMAGE LINKS Chronic obstructive pulmonary disease cases | Centrilobular emphysema cases

MERCK MANUAL PROFESSIONAL Chronic obstructive pulmonary disease

E6.5 Acute Exacerbation of COPD — Hospital Algorithm & NIV Decisions

‹ Core topic 6.9 · p.129
Previous-year Qs ›

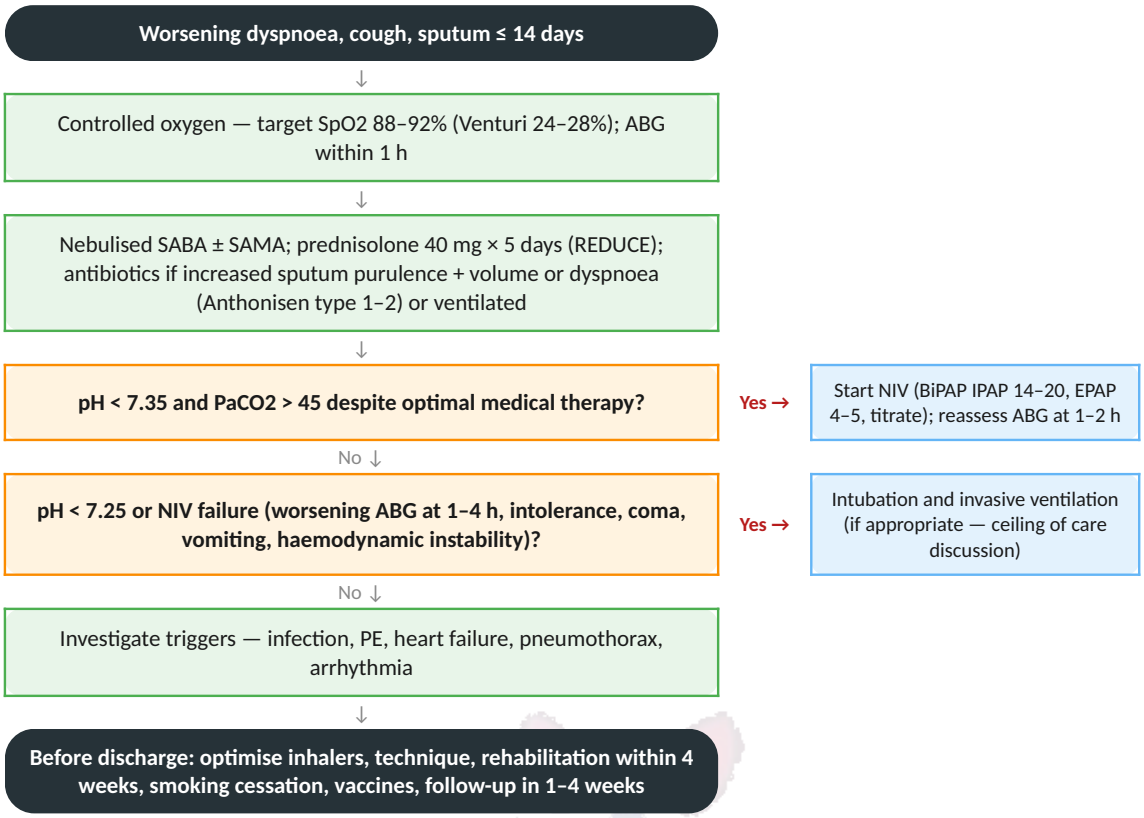
IN BRIEF The ROME proposal (2021) grades exacerbation severity objectively using dyspnoea, respiratory rate, heart rate, SpO2, CRP and ABG. Controlled oxygen, bronchodilators, steroids, antibiotics when indicated and NIV for acidotic hypercapnia save lives.

ROME classification of AECOPD severity

Severity	Criteria
Mild	Dyspnoea VAS < 5, RR < 24, HR < 95, SpO2 ≥ 92% at rest, CRP < 10 mg/L
Moderate	≥ 3 of: VAS ≥ 5, RR ≥ 24, HR ≥ 95, SpO2 < 92% (or fall > 3%), CRP ≥ 10; ABG may show hypoxaemia or hypercapnia without acidosis
Severe	Hypercapnia with acidosis (PaCO2 > 45, pH < 7.35)

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Algorithm E6.5.1 · Managing AECOPD in hospital



NIV in AECOPD — practical settings

Parameter	Typical setting
Mode	Spontaneous/timed (S/T) BiPAP
IPAP	Start 10–12, increase by 2–5 every 10 min to 20 (max ≈ 25–30)
EPAP	4–5 cmH2O (offsets intrinsic PEEP)
Backup rate	12–16/min
Interface	Full-face mask initially
Oxygen	Entrained to SpO2 88–92%

LANDMARK TRIALS & GUIDELINES

- **REDUCE (2013):** 5 days of prednisolone non-inferior to 14 days.
- **Plant et al. (2000):** NIV on general wards reduced intubation and mortality in acidotic AECOPD.
- **HOT-HMV (2017):** home NIV + oxygen after hypercapnic exacerbation reduced readmission or death.

★ CLINICAL PEARLS & EXAM FOCUS

- Over-oxygenation in COPD worsens hypercapnia (V/Q mismatch, Haldane effect — not only "loss of hypoxic drive").
- Eosinophil-guided steroid therapy (eos ≥ 300) can reduce steroid exposure.

AETCM YOUTUBE · VIDEO LECTURES ▶ COPD exacerbation ▶ NIV in COPD

MERCK MANUAL PROFESSIONAL Chronic obstructive pulmonary disease

E6.6 ABPA — ISHAM-AWG 2024 Criteria & Management

‹ Core topic 6.5 · p.122
Previous-year Qs ›

IN BRIEF Allergic bronchopulmonary aspergillosis complicates 5–10% of asthma in India — one of the highest prevalences worldwide. Early diagnosis before bronchiectasis develops preserves lung function.

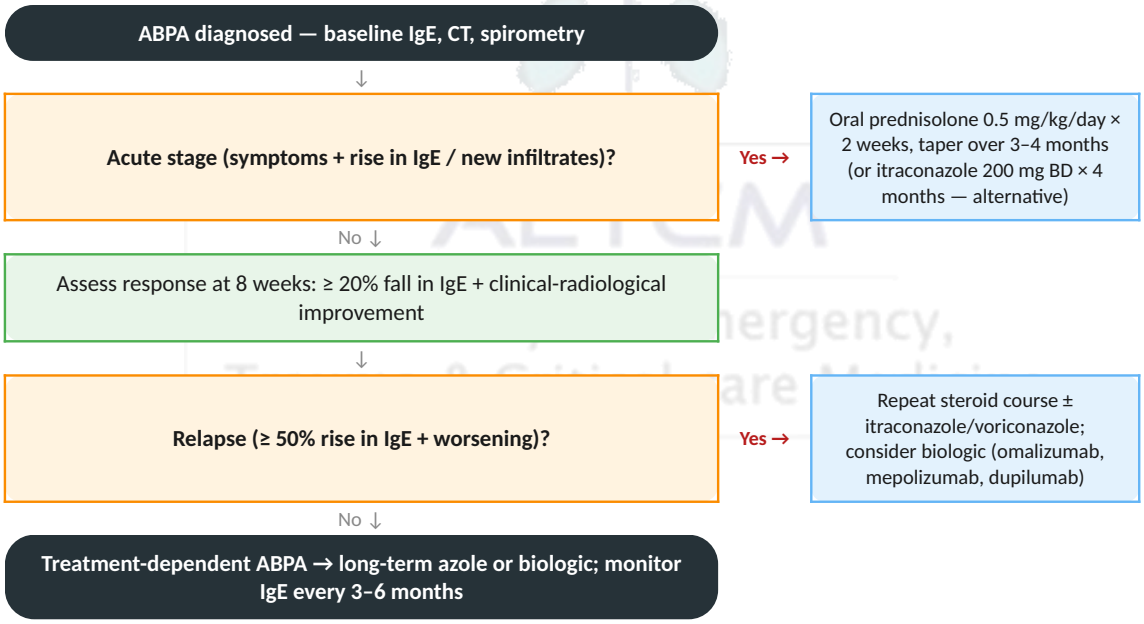
Revised ISHAM-AWG diagnostic criteria (2024)

Component	Criteria
Predisposing condition	Asthma, cystic fibrosis, bronchiectasis, COPD (or compatible clinical-radiological presentation)
Essential (both)	A. fumigatus-specific IgE ≥ 0.35 kUA/L **and** total serum IgE ≥ 500 IU/mL
Other (≥ 2)	Positive Aspergillus-specific IgG (or precipitins); blood eosinophils ≥ 500/µL (ever); imaging consistent — transient infiltrates, mucus plugging, central bronchiectasis, high-attenuation mucus (HAM)
Note	If IgE < 500 but all other criteria present — ABPA may still be diagnosed; HAM or mucus plugs on CT are highly specific

Clinical-radiological staging and treatment

Stage	Description
Asthma with Aspergillus sensitisation	Specific IgE positive; criteria not met — treat asthma
ABPA-S (serological)	No bronchiectasis
ABPA-B	Bronchiectasis
ABPA-HAM	High-attenuation mucus — more relapses
ABPA-CPF	Chronic pleuropulmonary fibrosis

Algorithm E6.6.1 · Management of ABPA



LANDMARK TRIALS & GUIDELINES

- **Agarwal et al. (PGIMER Chandigarh):** medium-dose prednisolone as effective as high-dose with fewer adverse effects; itraconazole monotherapy effective in acute ABPA.

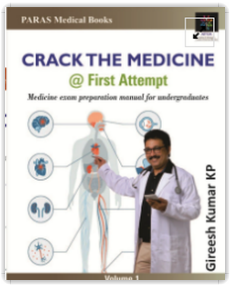
★ CLINICAL PEARLS & EXAM FOCUS

- Every asthmatic should be screened for Aspergillus sensitisation (specific IgE) in high-prevalence countries like India.
- "Finger-in-glove" opacities on CXR = mucoid impaction in dilated central bronchi.

RADIOPAEDIA · IMAGE LINKS Allergic bronchopulmonary aspergillosis cases · Finger in glove sign cases · High attenuation mucus cases

MERCK MANUAL PROFESSIONAL Allergic bronchopulmonary aspergillosis

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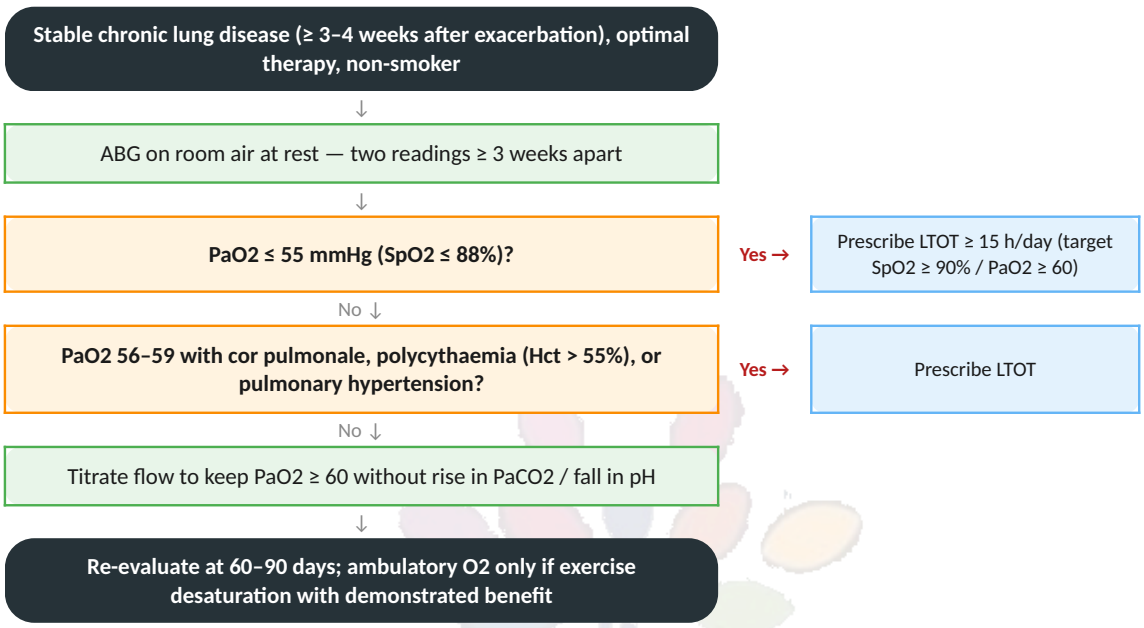
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E6.7 Long-Term Oxygen Therapy & Pulmonary Rehabilitation — Prescribing Algorithm

‹ Core topic 6.12 · p.133
Previous-year Qs ›

IN BRIEF LTOT prolongs life in COPD with severe resting hypoxaemia; it does not help moderate hypoxaemia (LOTT). Pulmonary rehabilitation is the most effective non-pharmacological therapy in chronic lung disease.

Algorithm E6.7.1 · Prescribing long-term oxygen therapy



Pulmonary rehabilitation — components

Component	Details
Patient selection	COPD with mMRC ≥ 2, post-exacerbation (within 4 weeks), ILD, bronchiectasis, PH, pre/post lung surgery
Exercise training	Endurance (cycling/walking 20–30 min at 60–80% peak work) + resistance training, 2–3 sessions/week for 6–12 weeks
Education & self-management	Inhaler technique, action plans, energy conservation
Breathing techniques	Pursed-lip breathing, diaphragmatic breathing, airway clearance
Nutrition & psychosocial	BMI, muscle mass; anxiety and depression
Outcomes	6MWD (MCID ≈ 30 m), CAT, CRQ, SGRQ (MCID 4 units)

LANDMARK TRIALS & GUIDELINES

- **NOTT (1980) and MRC (1981):** continuous oxygen reduced mortality in severe hypoxaemia.
- **LOTT (2016):** no benefit of LTOT in moderate resting or exercise desaturation.

★ CLINICAL PEARLS & EXAM FOCUS

- Smoking with oxygen is a fire hazard — burns are a real risk; counsel strongly.
- Rehabilitation started within 4 weeks of hospital discharge reduces readmissions and mortality.

E6.8 Drug Chart — Inhaled & Oral Drugs for Asthma and COPD

Previous-year Qs ›

IN BRIEF Adult doses. Combination inhalers are preferred for adherence; match the device to the patient (E3.3).

Airway pharmacotherapy

Class	Drug	Dose	Indication	Caution
SABA	Salbutamol	100–200 µg as needed; 2.5–5 mg nebulised	Reliever (Track 2), acute attacks	Tremor, tachycardia, hypokalaemia, lactic acidosis
SAMA	Ipratropium	20–40 µg QID; 0.5 mg nebulised	AECOPD, acute asthma add-on	Glaucoma, urinary retention
LABA	Formoterol / Salmeterol	12 µg BD / 50 µg BD	With ICS (asthma) or LAMA (COPD)	Never as monotherapy in asthma
Ultra-LABA	Indacaterol / Olodaterol / Vilanterol	150–300 µg OD / 5 µg OD / 25 µg OD	COPD maintenance	Cough
LAMA	Tiotropium / Glycopyrronium / Umeclidinium	18 µg OD (2.5 µg × 2 SMI) / 50 µg OD / 62.5 µg OD	COPD, severe asthma add-on	Dry mouth, urinary retention
ICS	Budesonide / Fluticasone / Beclometasone / Ciclesonide	See dose-equivalence table	Asthma controller; COPD with eos ≥ 100	Oral candidiasis, pneumonia (COPD), bruising
ICS–formoterol	Budesonide–formoterol 200/6	1–2 puffs as needed (AIR) or 1–2 BD + as needed (MART)	GINA Track 1	Max 12 inhalations/day
Triple single inhaler	FF/UMEC/VI; BUD/GLY/FOR; BDP/FOR/GB	Once or twice daily	COPD group E with eos ≥ 100	Pneumonia
Systemic corticosteroid	Prednisolone	40–50 mg × 5–7 days (asthma); 40 mg × 5 days (AECOPD)	Exacerbations	Hyperglycaemia; cumulative OCS harm
Methylxanthine	Theophylline (SR) / Doxofylline	200–300 mg BD (level 5–15 mg/L) / 400 mg BD	Add-on where cost limits access	Narrow therapeutic index, arrhythmia, seizures, interactions
PDE4 inhibitor	Roflumilast	250 µg OD × 4 weeks then 500 µg OD	COPD with chronic bronchitis, FEV1 < 50%, exacerbations	Weight loss, diarrhoea, psychiatric
Macrolide	Azithromycin	250 mg OD or 500 mg thrice weekly	Frequent exacerbators (asthma/COPD)	QT, hearing, NTM
LTRA	Montelukast	10 mg at night	Asthma with rhinitis, EIB	Neuropsychiatric effects (boxed warning)
Mucolytic / antioxidant	N-acetylcysteine / Carbocisteine / Erdosteine	600 mg BD / 750 mg BD / 300 mg BD	COPD exacerbation prevention (not on ICS)	GI upset
Magnesium	Magnesium sulphate	2 g IV over 20 min	Acute severe asthma	Hypotension, flushing

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Class	Drug	Dose	Indication	Caution
Anti-IgE / anti-IL-5 / anti-IL-4R / anti-TSLP	Omalizumab, mepolizumab, benralizumab, dupilumab, tezepelumab	See E3.4	Severe asthma; eosinophilic COPD (dupilumab, mepolizumab)	Anaphylaxis (rare), cost
Alpha-1 antitrypsin augmentation	Plasma-derived AAT	60 mg/kg IV weekly	Severe AATD with emphysema	Cost; IgA deficiency

E6.9 Severe Asthma — Phenotyping, Comorbidities & the Difficult-Asthma Clinic

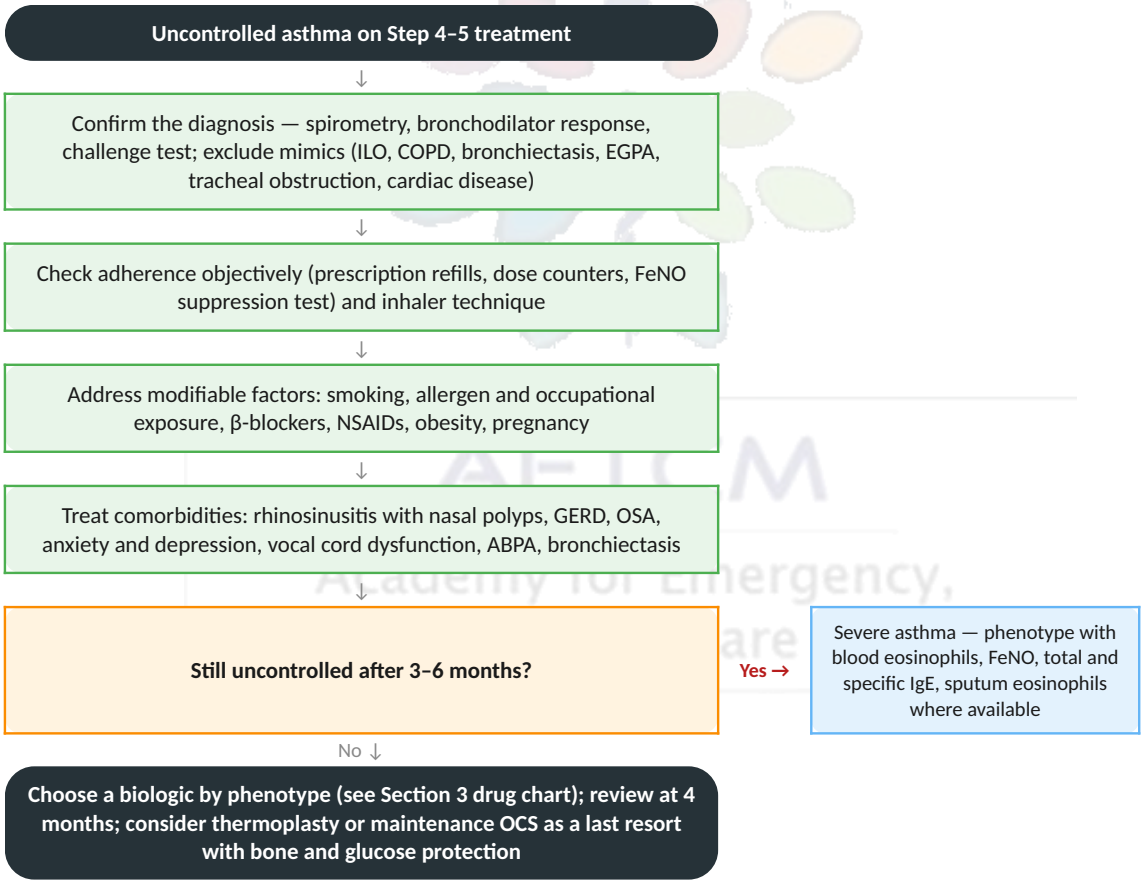
‹ Core topic 6.4 · p.120
Previous-year Qs ›

IN BRIEF Before calling asthma "severe", prove that it is not simply difficult-to-treat. Most patients referred as severe have poor adherence, wrong technique, ongoing exposure or an untreated comorbidity.

Difficult-to-treat versus severe asthma

Difficult-to-treat	Severe
Uncontrolled despite medium/high-dose ICS–LABA because of modifiable factors	Uncontrolled despite optimised high-dose ICS–LABA and correct management of comorbidities, or worsens when treatment is reduced

Algorithm E6.9.1 · The severe-asthma assessment pathway (GINA)



Recognising aspirin-exacerbated respiratory disease (AERD / Samter triad)

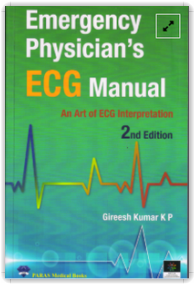
Component	Detail
Asthma	Usually adult-onset, severe
Chronic rhinosinusitis with nasal polyps	Anosmia, recurrent polypectomy
NSAID sensitivity	Reaction within 30–180 min of aspirin or any COX-1 inhibitor
Management	Avoid COX-1 NSAIDs (coxibs usually tolerated), leukotriene modifiers, dupilumab, aspirin desensitisation in specialist centres

★ CLINICAL PEARLS & EXAM FOCUS

- An FeNO suppression test (directly observed ICS for 7 days with FeNO monitoring) distinguishes non-adherence from true steroid-refractory disease.
- Maintenance oral steroids cause cumulative harm from as little as 0.5–1 g of prednisolone per year — count every rescue course.

MERCK MANUAL PROFESSIONAL Asthma

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E6.10 Asthma–COPD Overlap, Small-Airway Disease & Bronchiolitis

‹ Core topic 6.11 · p.132

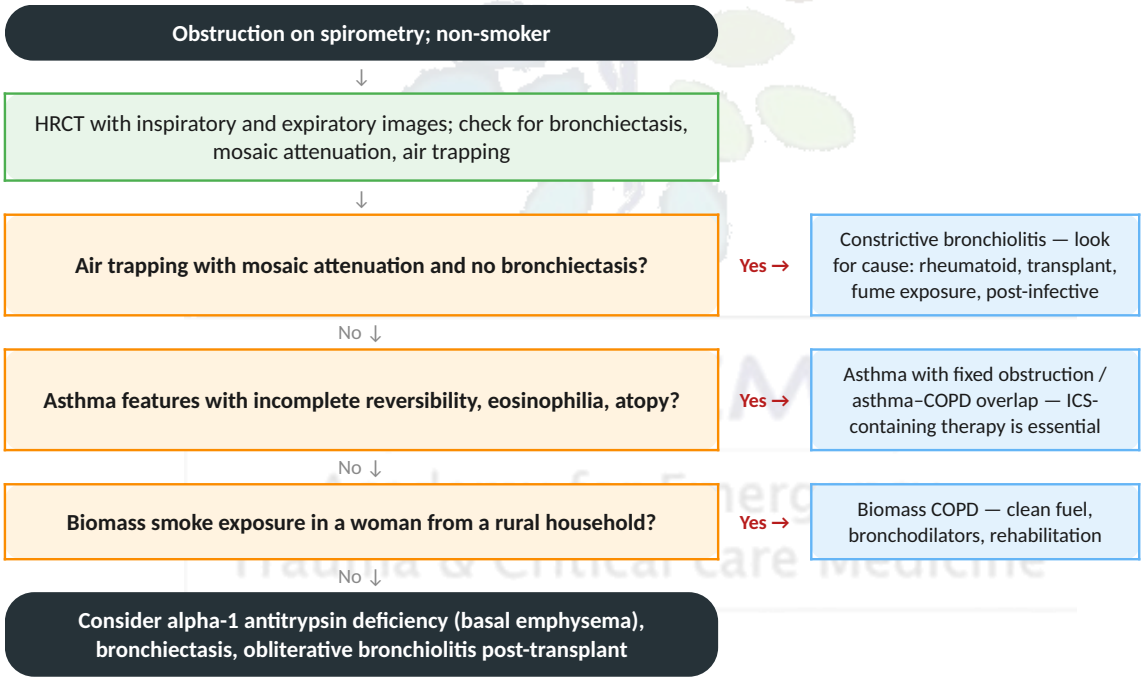
Previous-year Qs ›

IN BRIEF Bronchiolitis is airway disease too small to be seen directly: suspect it when there is airflow obstruction with mosaic attenuation and air trapping but normal large airways.

Classification of bronchiolitis

Type	Cause	Features
Constrictive (obliterative) bronchiolitis	Post-infective (adenovirus — Swyer-James), post-transplant (BOS), rheumatoid, fumes (diacetyl, "popcorn lung"), drugs (penicillamine)	Fixed obstruction, mosaic attenuation, air trapping; poorly reversible
Cellular / follicular bronchiolitis	Connective tissue disease, immunodeficiency	Centrilobular nodules
Respiratory bronchiolitis (RB / RB-ILD)	Smoking	Upper-zone centrilobular ground-glass nodules; stop smoking
Diffuse panbronchiolitis	East Asian; chronic sinusitis	Tree-in-bud; dramatic response to low-dose macrolide
Organising pneumonia (BOOP)	Idiopathic or secondary	Consolidation rather than obstruction; steroid-responsive

Algorithm E6.10.1 · Fixed airflow obstruction in a non-smoker



★ **CLINICAL PEARLS & EXAM FOCUS**

- "Asthma–COPD overlap" is a description, not a diagnosis — but these patients must always receive an inhaled corticosteroid.
- Biomass-related COPD in Indian women has more chronic bronchitis and less emphysema than tobacco COPD.

RADIOPAEDIA · IMAGE LINKS Constrictive bronchiolitis cases · Swyer-James syndrome cases · Air trapping cases

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PART II · SECTION 7

Interstitial Lung Diseases & Immunological Disorders

Advanced theory & algorithms

E7.1 ILD — Diagnostic Algorithm & the Multidisciplinary Discussion 397
E7.2 HRCT Patterns in Fibrosing ILD — UIP Categories 399
E7.3 IPF & Progressive Pulmonary Fibrosis — Treatment & Acute Exacerbation Algorithm 400
E7.4 Hypersensitivity Pneumonitis — ATS/JRS/ALAT 2020 Algorithm 402
E7.5 Sarcoidosis — Organ Assessment & Treatment Algorithm (ERS 2021) 403
E7.6 CTD-ILD, Diffuse Alveolar Haemorrhage & Cystic Lung Disease — Algorithms 404
E7.7 Drug Chart — Immunosuppressants & Antifibrotics in ILD 407
E7.8 Eosinophilic Lung Disease & Pulmonary Vasculitis — Approach 408

E7.1 ILD — Diagnostic Algorithm & the Multidisciplinary Discussion

‹ Core topic 7.1 · p.139
Previous-year Qs ›

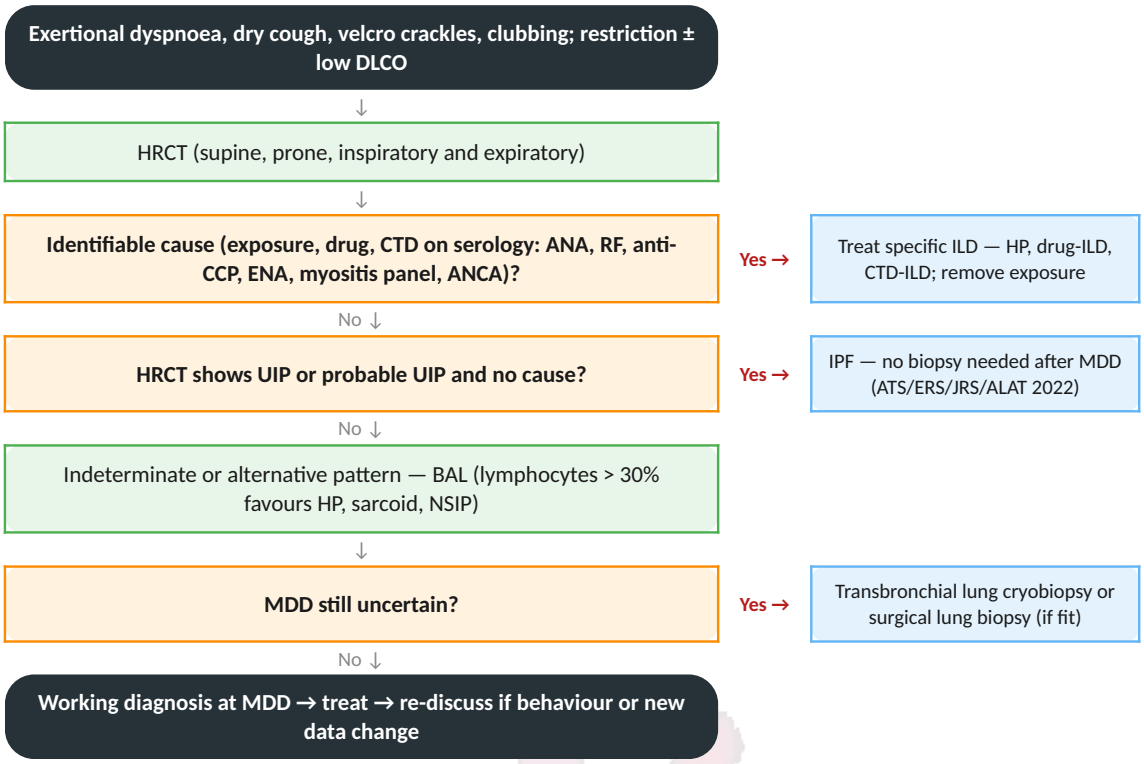
IN BRIEF The diagnosis of interstitial lung disease is reached by multidisciplinary discussion (MDD) between pulmonologist, radiologist and pathologist. The first job is to find an exposure, a connective tissue disease or a drug; only then is a disease called "idiopathic".

History checklist in suspected ILD

Domain	Ask about
Exposures	Birds, feathers, moulds, humidifiers, air-conditioners (HP); silica, asbestos, metal dust (occupational); smoking (RB-ILD, DIP, PLCH)
Drugs	Amiodarone, methotrexate, nitrofurantoin, bleomycin, immune checkpoint inhibitors, chemotherapy, radiation
CTD features	Raynaud's, arthritis, morning stiffness, sicca, rash, mechanic's hands, dysphagia, myalgia
Family history	Familial pulmonary fibrosis (telomere genes), early greying, cirrhosis, marrow failure
Systemic	Fever, weight loss, uveitis, skin nodules (sarcoid)

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Algorithm E7.1.1 · Approach to suspected ILD



BAL differential cell counts in ILD

BAL finding	Suggests
Lymphocytes > 30% (> 50% strongly)	Hypersensitivity pneumonitis, sarcoid, NSIP, drug-ILD
CD4/CD8 > 3.5	Sarcoidosis
Eosinophils > 25%	Eosinophilic pneumonia
Neutrophils > 50%	Aspiration, infection, AIP, IPF exacerbation
Progressively bloody aliquots, haemosiderin-laden macrophages > 20%	Diffuse alveolar haemorrhage
Milky fluid, PAS-positive material	Pulmonary alveolar proteinosis
CD1a > 5%	Pulmonary Langerhans cell histiocytosis

★ CLINICAL PEARLS & EXAM FOCUS

- Prone images separate dependent atelectasis from true fibrosis at the bases.
- Expiratory images show air trapping (mosaic attenuation) — classic for fibrotic HP ("three-density/headcheese sign").
- MDD diagnosis is a working diagnosis — revisit at follow-up.

RADIOPAEDIA · IMAGE LINKS Usual interstitial pneumonia cases | Headcheese sign cases | Honeycombing cases

AETCM YOUTUBE · VIDEO LECTURES ▶ interstitial lung disease | ▶ HRCT chest

MERCK MANUAL PROFESSIONAL Interstitial lung disease

E7.2 HRCT Patterns in Fibrosing ILD — UIP Categories

‹ Core topic 7.2 · p.141
Previous-year Qs ›

IN BRIEF The 2022 IPF guideline describes four HRCT categories. Learn the features of each — this table is a guaranteed short-note question.

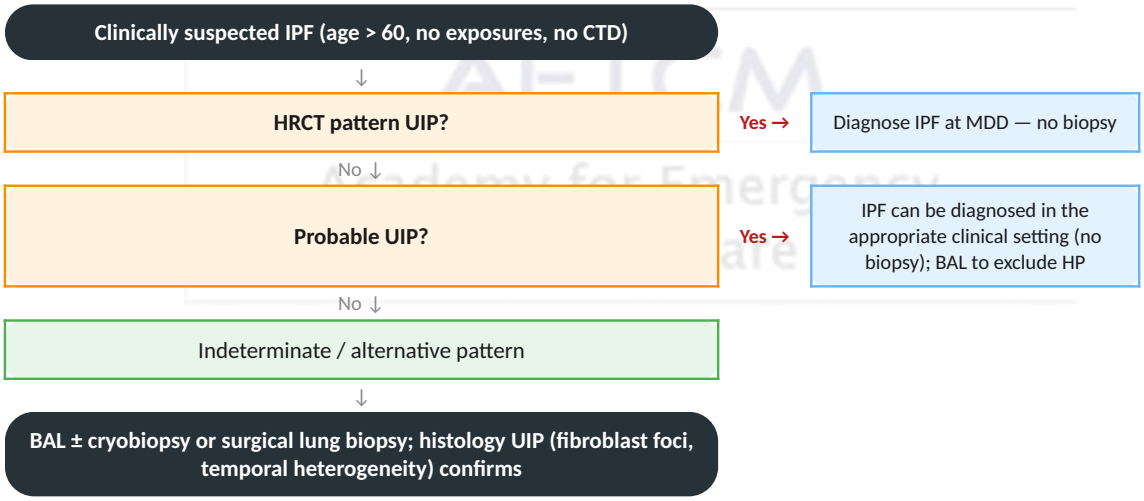
HRCT categories of UIP (ATS/ERS/JRS/ALAT 2018/2022)

Category	Distribution	Features
UIP	Subpleural, basal predominant; often heterogeneous	Honeycombing ± peripheral traction bronchiectasis/bronchiolectasis
Probable UIP	Subpleural, basal predominant	Reticulation with peripheral traction bronchiectasis; **no** honeycombing
Indeterminate for UIP	Variable or diffuse	Fibrosis with some features not suggestive of any specific aetiology
Alternative diagnosis	Upper/mid-lung, peribronchovascular, subpleural sparing	Predominant consolidation, extensive pure GGO, profuse micronodules, centrilobular nodules, cysts, marked mosaic attenuation with three-density sign, pleural plaques, oesophageal dilatation

Features suggesting an alternative to IPF

Feature	Think of
Subpleural sparing, GGO, lower zones	NSIP (CTD, drug)
Upper-lobe fibrosis, perilymphatic nodules	Sarcoidosis, silicosis
Centrilobular nodules, mosaic attenuation, three-density sign	Hypersensitivity pneumonitis
Pleural plaques	Asbestosis
Patulous oesophagus	Systemic sclerosis
Peribronchovascular consolidation, reverse halo	Organising pneumonia
Upper-lobe pleuroparenchymal thickening	PPFE

Algorithm E7.2.1 · Using HRCT to decide about biopsy in suspected IPF



★ CLINICAL PEARLS & EXAM FOCUS

- Honeycombing = clustered cystic airspaces (3–10 mm, up to 2.5 cm) with shared walls in subpleural layers.
- "Straight-edge sign" and "exuberant honeycombing" suggest CTD-associated UIP (rheumatoid).

RADIOPAEDIA · IMAGE LINKS Usual interstitial pneumonia cases | Non-specific interstitial pneumonia cases | Straight edge sign cases | Traction bronchiectasis cases

MERCK MANUAL PROFESSIONAL Idiopathic pulmonary fibrosis

E7.3 IPF & Progressive Pulmonary Fibrosis — Treatment & Acute Exacerbation Algorithm

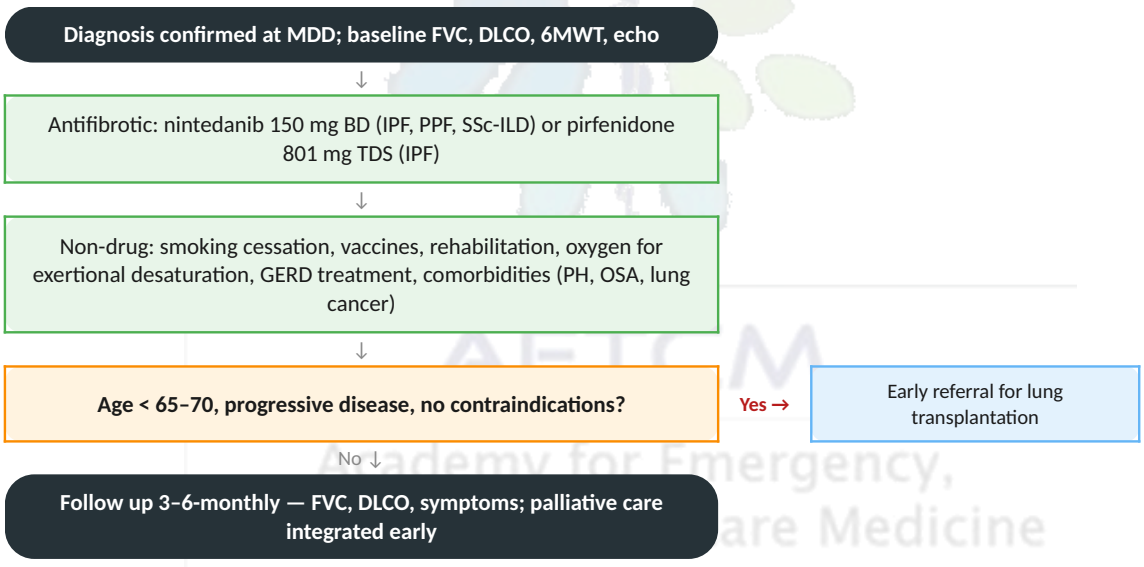
‹ Core topic 7.2 · p.141
Previous-year Qs ›

IN BRIEF Antifibrotics slow FVC decline by about half in IPF and in progressive pulmonary fibrosis (PPF) of other causes. Acute exacerbation carries a 50% in-hospital mortality.

Progressive pulmonary fibrosis (ATS/ERS/JRS/ALAT 2022) — any 2 of 3 within 1 year, no alternative explanation

Domain	Criterion
Symptoms	Worsening respiratory symptoms
Physiology	Absolute FVC decline ≥ 5% or DLCO decline ≥ 10% predicted within 1 year
Radiology	Increased extent or severity of fibrosis on HRCT

Algorithm E7.3.1 · Managing IPF / PPF



Algorithm E7.3.2 · Acute exacerbation of IPF (AE-IPF)

Acute worsening of dyspnoea < 1 month + new bilateral GGO/ consolidation on HRCT

↓

Exclude heart failure, fluid overload, PE, pneumothorax; screen infection (cultures, viral PCR, procalcitonin)

↓

Infection found? — Yes → Treat infection (still counts as AE-IPF if criteria met)

No ↓

Supportive: oxygen/HFNC, broad-spectrum antibiotics till cultures, continue antifibrotic

↓

Respiratory failure needing invasive ventilation? — Yes → Usually futile unless bridge to transplant — discuss ceiling of care

No ↓

Steroids are commonly given (pulse methylprednisolone) — weak evidence; cyclophosphamide harmful (EXAFIP)

LANDMARK TRIALS & GUIDELINES

- **INPULSIS (nintedanib), CAPACITY/ASCEND (pirfenidone):** slowed FVC decline in IPF.
- **INBUILD:** nintedanib in progressive fibrosing ILD of other causes.
- **PANTHER-IPF:** prednisolone + azathioprine + NAC increased mortality — never use in IPF.
- **FIBRONEER-IPF (2025):** nerandomilast (PDE4B inhibitor) slowed FVC decline — newest antifibrotic.

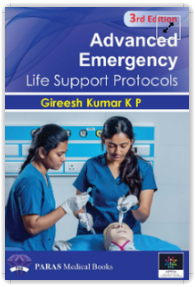
★ CLINICAL PEARLS & EXAM FOCUS

- Nintedanib — diarrhoea (manage with loperamide, dose reduction to 100 mg BD); pirfenidone — photosensitivity, nausea, rash.
- GAP index (Gender, Age, Physiology) stages IPF prognosis.

RADIOPAEDIA · IMAGE LINKS — Idiopathic pulmonary fibrosis · cases · Acute exacerbation of idiopathic pulmonary fibrosis · cases

MERCK MANUAL PROFESSIONAL — Idiopathic pulmonary fibrosis

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E7.4 Hypersensitivity Pneumonitis — ATS/JRS/ALAT 2020 Algorithm

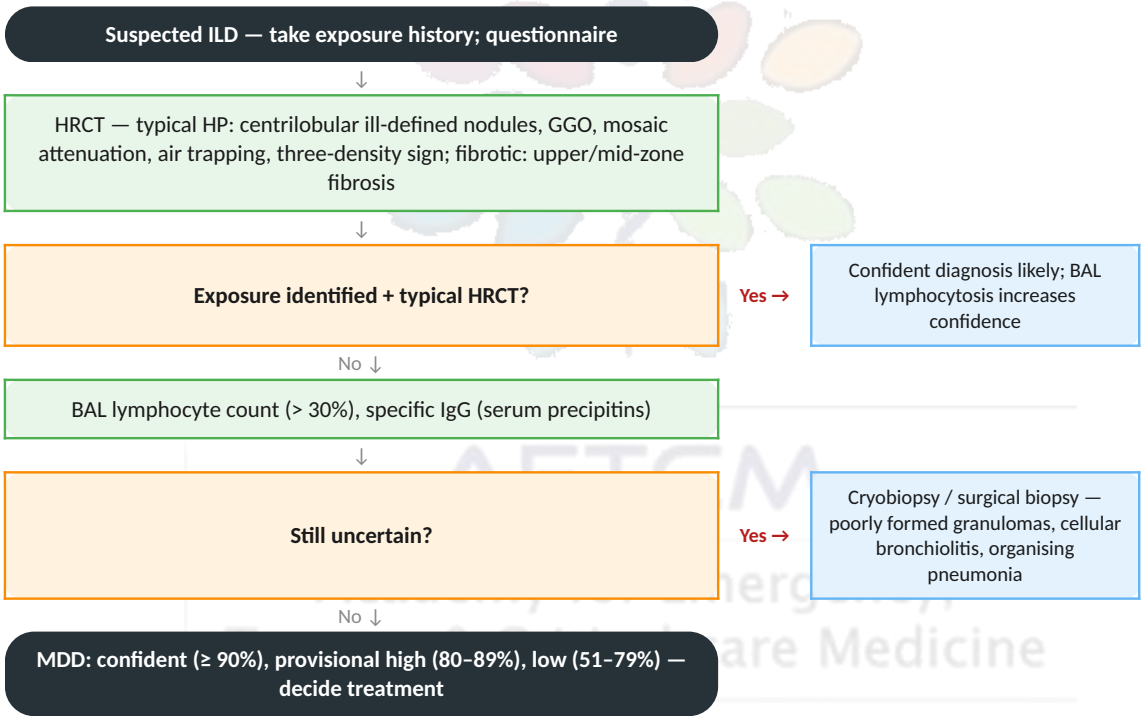
‹ Core topic 7.4 · p.145
Previous-year Qs ›

IN BRIEF HP is classified as non-fibrotic or fibrotic (not acute/subacute/chronic). Antigen identification and avoidance is the most important treatment; fibrotic HP may behave like IPF.

Common antigens in India

Exposure	Antigen	Disease name
Pigeons, parrots, poultry	Avian proteins	Bird fancier's lung
Mouldy hay, grain	Thermophilic actinomycetes	Farmer's lung
Air-conditioners, humidifiers, coolers	Bacteria, fungi	Humidifier / air-conditioner lung
Feather pillows, duvets	Avian	Feather duvet lung
Sugarcane (bagasse)	Thermoactinomyces sacchari	Bagassosis
Mushrooms	Spores	Mushroom worker's lung
Isocyanates, paints	Chemicals	Chemical worker's lung

Algorithm E7.4.1 · Diagnosis of HP (ATS/JRS/ALAT 2020)



Treatment

- **Antigen avoidance** — complete removal of birds, cleaning of coolers; may need change of home or job.
- **Non-fibrotic HP with symptoms:** prednisolone 0.5 mg/kg/day tapered over 3–6 months.
- **Fibrotic HP:** mycophenolate or azathioprine as steroid-sparing; nintedanib if progressive (PPF — INBUILD).
- Transplant for end-stage disease.

★ CLINICAL PEARLS & EXAM FOCUS

- Smokers are less likely to develop HP (nicotine suppresses macrophage activation).
- Outcome in fibrotic HP is worse when no antigen is found (≈ 50% of cases).

RADIOPAEDIA · IMAGE LINKS Hypersensitivity pneumonitis cases Mosaic attenuation cases

MERCK MANUAL PROFESSIONAL Hypersensitivity pneumonitis

E7.5 Sarcoidosis — Organ Assessment & Treatment Algorithm (ERS 2021)

‹ Core topic 7.5 · p.147
Previous-year Qs ›

IN BRIEF Sarcoidosis is a diagnosis of exclusion — in India especially of tuberculosis. Not every patient needs treatment; treat to protect an organ or relieve major symptoms.

Initial organ screening in every patient

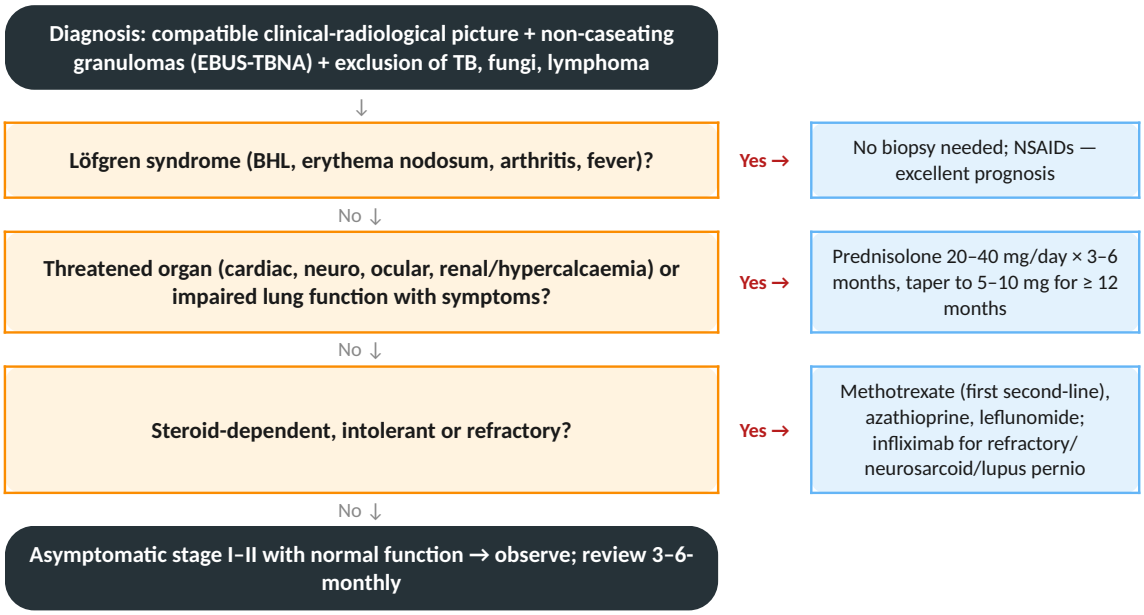
Organ	Screen
Lungs	CXR (Scadding stage), PFT with DLCO, HRCT
Eye	Slit-lamp examination (uveitis may be silent)
Heart	ECG ± Holter, echo; cardiac MRI/FDG-PET if abnormal
Kidney / calcium	Serum creatinine, calcium, 24-h urine calcium
Liver	LFT
Skin	Examination — lupus pernio, erythema nodosum
Others	CBC, vitamin D (25-OH and 1,25), ACE (supportive only)

Scadding radiographic stages

Stage	CXR	Spontaneous remission
0	Normal	—
I	Bilateral hilar lymphadenopathy (BHL)	60–90%
II	BHL + parenchymal infiltrates	40–70%
III	Infiltrates without BHL	10–20%
IV	Fibrosis	0%

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Algorithm E7.5.1 · Whom to treat in sarcoidosis



LANDMARK TRIALS & GUIDELINES

- ERS clinical practice guideline 2021 — glucocorticoids as first-line for pulmonary sarcoid with risk of mortality or impairment; methotrexate as steroid-sparing.

★ CLINICAL PEARLS & EXAM FOCUS

- Serum ACE is raised in ≈ 60% — neither sensitive nor specific; not for diagnosis.
- Granulomas without caseation do not exclude TB — send tissue for NAAT and culture always in India.
- Heerfordt syndrome: uveitis, parotitis, facial palsy, fever.

RADIOPAEDIA · IMAGE LINKS Sarcoidosis cases | Bilateral hilar lymphadenopathy cases | Perilymphatic nodules cases

MERCK MANUAL PROFESSIONAL Sarcoidosis

E7.6 CTD-ILD, Diffuse Alveolar Haemorrhage & Cystic Lung Disease — Algorithms

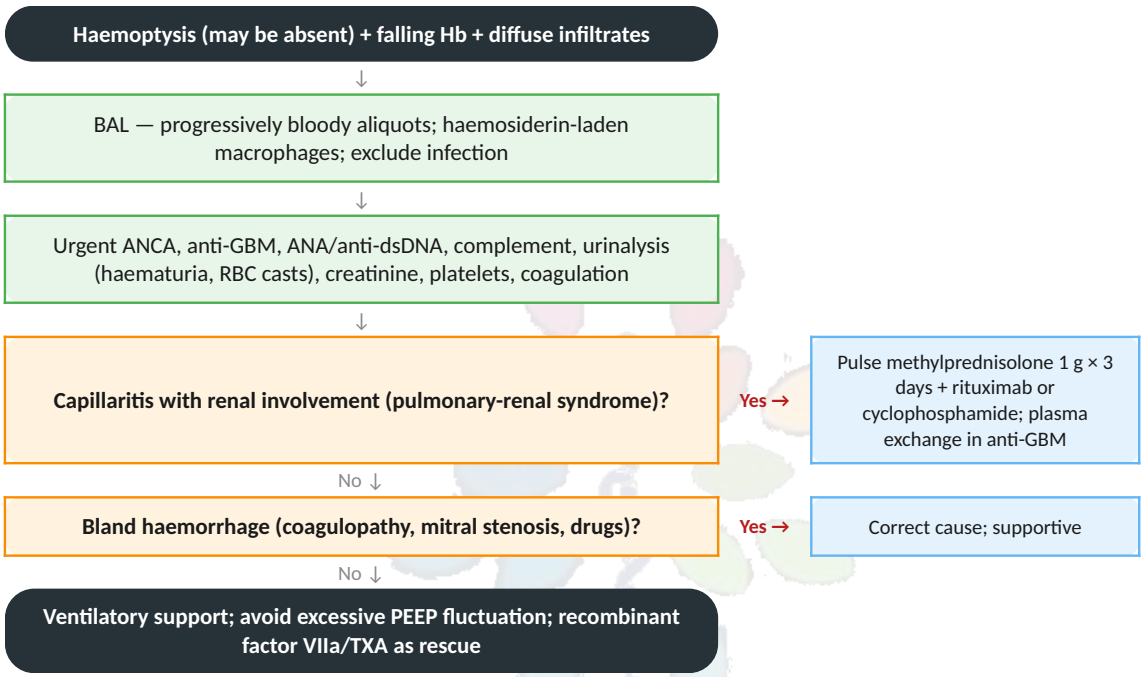
‹ Core topic 7.6 · p.149
Previous-year Qs ›

IN BRIEF Connective tissue diseases account for a quarter of ILD. DAH is a pulmonary emergency, and diffuse cystic lung disease has a short differential that HRCT usually resolves.

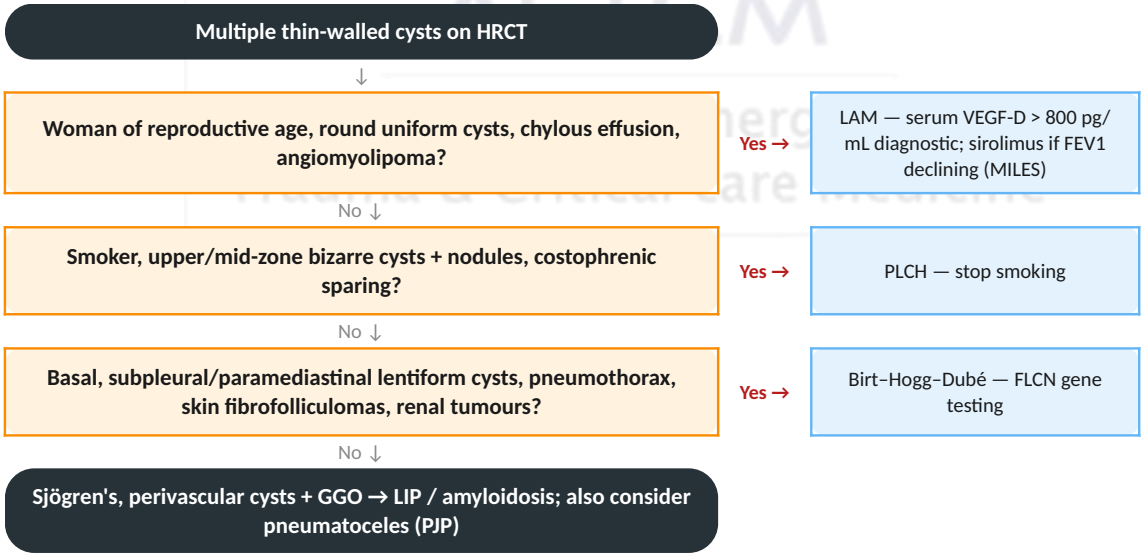
CTD-ILD at a glance

CTD	Usual ILD pattern	Treatment notes
Systemic sclerosis	NSIP (≈ 75%), UIP	Mycophenolate (SLS II), tocilizumab, nintedanib (SENSCIS); avoid high-dose steroids (renal crisis)
Rheumatoid arthritis	UIP (commonest), NSIP	Antifibrotic if UIP progressive; methotrexate generally safe
Myositis (anti-synthetase, anti-MDA5)	NSIP, OP; rapidly progressive in MDA5	Steroids + tacrolimus/cyclophosphamide; triple therapy for MDA5
Sjögren's	LIP, NSIP, cysts	Steroids, mycophenolate
SLE	Pleuritis common; acute lupus pneumonitis, DAH, shrinking lung	Steroids, cyclophosphamide

Algorithm E7.6.1 · Diffuse alveolar haemorrhage



Algorithm E7.6.2 · Diffuse cystic lung disease



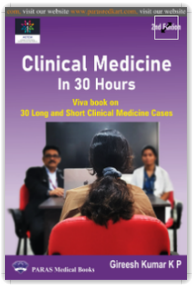
★ **CLINICAL PEARLS & EXAM FOCUS**

- KCO increase > 30% suggests active alveolar haemorrhage.
- Anti-MDA5 dermatomyositis: palmar papules, skin ulcers, rapidly progressive ILD with high mortality.

RADIOPAEDIA · IMAGE LINKS Lymphangioleiomyomatosis cases · Pulmonary Langerhans cell histiocytosis cases · Birt-Hogg-Dube syndrome cases · Diffuse alveolar haemorrhage cases

MERCK MANUAL PROFESSIONAL Systemic sclerosis · Diffuse alveolar hemorrhage · Lymphangioleiomyomatosis

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E7.7 Drug Chart — Immunosuppressants & Antifibrotics in ILD

Previous-year Qs ›

IN BRIEF Screen for TB (IGRA/CXR), hepatitis B/C and vaccinate before immunosuppression. Give PJP prophylaxis with combined immunosuppression.

Drugs in interstitial and immunological lung disease

Class	Drug	Dose	Indication	Caution
Corticosteroid	Prednisolone	0.5–1 mg/kg/day, taper over months	Sarcoid, HP, OP, CTD-ILD, AEP, CEP	Diabetes, osteoporosis (calcium, vitamin D, bisphosphonate), infections
Corticosteroid (pulse)	Methylprednisolone	500–1000 mg IV × 3 days	DAH, vasculitis, AE-IPF, rapidly progressive ILD	Hyperglycaemia, psychosis
Antimetabolite	Mycophenolate mofetil	500 mg BD, increase to 1–1.5 g BD	SSc-ILD (first-line), CTD-ILD, fibrotic HP	Leucopenia, diarrhoea, teratogenic
Purine analogue	Azathioprine	1–2 mg/kg/day (check TPMT)	Steroid-sparing in CTD-ILD, sarcoid, HP	Myelosuppression, hepatitis, pancreatitis; never in IPF
Antifolate	Methotrexate	7.5–25 mg weekly + folic acid	Sarcoidosis (steroid-sparing)	Hepatotoxicity, pneumonitis, cytopenias
Alkylating agent	Cyclophosphamide	500–750 mg/m² IV monthly (or 15 mg/kg)	Severe CTD-ILD, vasculitis, DAH	Haemorrhagic cystitis (mesna), infertility, malignancy
Calcineurin inhibitor	Tacrolimus	0.05–0.1 mg/kg/day (trough 5–10)	Anti-synthetase / MDA5 ILD	Nephrotoxicity, hypertension, diabetes
Anti-CD20	Rituximab	1 g IV × 2 doses 2 weeks apart	ANCA vasculitis, refractory CTD-ILD, autoimmune PAP	Infusion reaction, hypogammaglobulinaemia, HBV reactivation
IL-6R antagonist	Tocilizumab	162 mg SC weekly	Early SSc-ILD	Infection, raised lipids, bowel perforation
Anti-TNF	Infliximab	3–5 mg/kg IV at 0, 2, 6 weeks then 8-weekly	Refractory sarcoid (neuro, cardiac, lupus pernio)	TB reactivation, heart failure
Tyrosine kinase inhibitor	Nintedanib	150 mg BD (100 mg BD if intolerant)	IPF, PPF, SSc-ILD	Diarrhoea, hepatotoxicity, bleeding risk with anticoagulants
Antifibrotic	Pirfenidone	267 mg TDS, titrate to 801 mg TDS over 2 weeks	IPF	Photosensitivity, nausea, LFT
mTOR inhibitor	Sirolimus	1–2 mg/day (trough 5–15 ng/mL)	LAM with declining lung function	Mouth ulcers, hyperlipidaemia, wound healing
Inhaled GM-CSF	Molgramostim / Sargramostim	300 µg inhaled OD (cyclical)	Autoimmune PAP (IMPALA-2)	Cost
Antibiotic prophylaxis	Co-trimoxazole	960 mg thrice weekly or 480 mg OD	PJP prophylaxis on combined immunosuppression	Rash, hyperkalaemia

E7.8 Eosinophilic Lung Disease & Pulmonary Vasculitis — Approach

‹ Core topic 7.8 · p.152
Previous-year Qs ›

IN BRIEF Pulmonary eosinophilia in India means parasites and drugs before it means idiopathic disease. The ANCA-associated vasculitides are the other group not to miss, because untreated they kill.

Causes of pulmonary eosinophilia

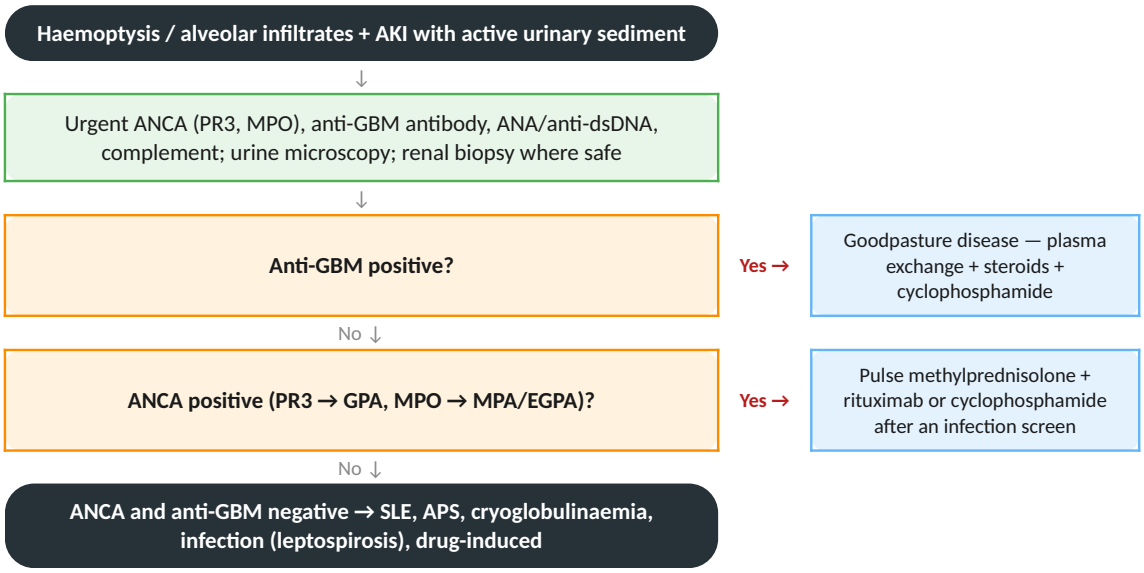
Group	Conditions
Idiopathic	Acute eosinophilic pneumonia (AEP), chronic eosinophilic pneumonia (CEP), hypereosinophilic syndrome
Parasitic	Tropical pulmonary eosinophilia (filaria), Ascaris/Strongyloides/hookworm (Löffler), Paragonimus, Toxocara
Fungal	ABPA
Drug-induced	Nitrofurantoin, sulfasalazine, NSAIDs, daptomycin, minocycline, checkpoint inhibitors
Vasculitic	Eosinophilic granulomatosis with polyangiitis (EGPA)
Other	Asthma, malignancy, radiation, e-cigarette (EVALI)

Distinguishing the main syndromes

Feature	AEP	CEP	EGPA
Onset	Days (often new smokers)	Weeks–months	Asthma for years, then systemic
Blood eosinophils	Often normal initially	High	Very high (> 10%)
Imaging	Diffuse GGO + effusions	"Photographic negative of pulmonary oedema" — peripheral upper-zone consolidation	Transient infiltrates, nodules
BAL eosinophils	> 25%	> 25%	> 25%
Other organs	None	Rare	Neuropathy, sinusitis, skin, cardiac, renal; ANCA (MPO) in 40%
Treatment	Steroids — rapid response, no relapse; stop smoking	Steroids ≥ 6 months; relapses common	Steroids + cyclophosphamide/ rituximab; mepolizumab

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Algorithm E7.8.1 · Pulmonary–renal syndrome



ANCA-associated vasculitis — lung features

Disease	Lung	Other
Granulomatosis with polyangiitis (GPA)	Nodules with cavitation, subglottic stenosis, DAH	Sinusitis, saddle nose, glomerulonephritis; PR3-ANCA
Microscopic polyangiitis (MPA)	DAH, pulmonary fibrosis	Glomerulonephritis; MPO-ANCA
EGPA	Asthma, transient infiltrates	Eosinophilia, neuropathy, cardiac involvement

★ CLINICAL PEARLS & EXAM FOCUS

- Always screen for Strongyloides (serology, stool) before steroids in an endemic area — steroids can precipitate fatal hyperinfection.
- CEP relapses in more than half when steroids are stopped; taper slowly over 6–12 months.

RADIOPAEDIA · IMAGE LINKS Chronic eosinophilic pneumonia cases Granulomatosis with polyangiitis cases Eosinophilic granulomatosis with polyangiitis cases

MERCK MANUAL PROFESSIONAL Eosinophilic pneumonia Granulomatosis with polyangiitis

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PART II · SECTION 8

Occupational & Environmental Lung Disease, Smoking & Inhalational Injury

Advanced theory & algorithms

E8.1 Occupational History, ILO Classification & Silicosis in India 410
E8.2 Asbestos-Related Diseases & Other Pneumoconioses 411
E8.3 Smoking Cessation — 5A/5R Model & Pharmacotherapy 413
E8.4 Toxic Inhalational Injury, Smoke Inhalation & Air Pollution 415
E8.5 Drug Chart — Smoking Cessation & Inhalational Injury Antidotes 416

E8.1 Occupational History, ILO Classification & Silicosis in India

‹ Core topic 8.1 · p.159
Previous-year Qs ›

IN BRIEF Silicosis is India's commonest and most neglected occupational lung disease — stone quarrying, crushing, sandstone carving, agate grinding, foundries and engineered stone. It is notifiable in several states and compensable.

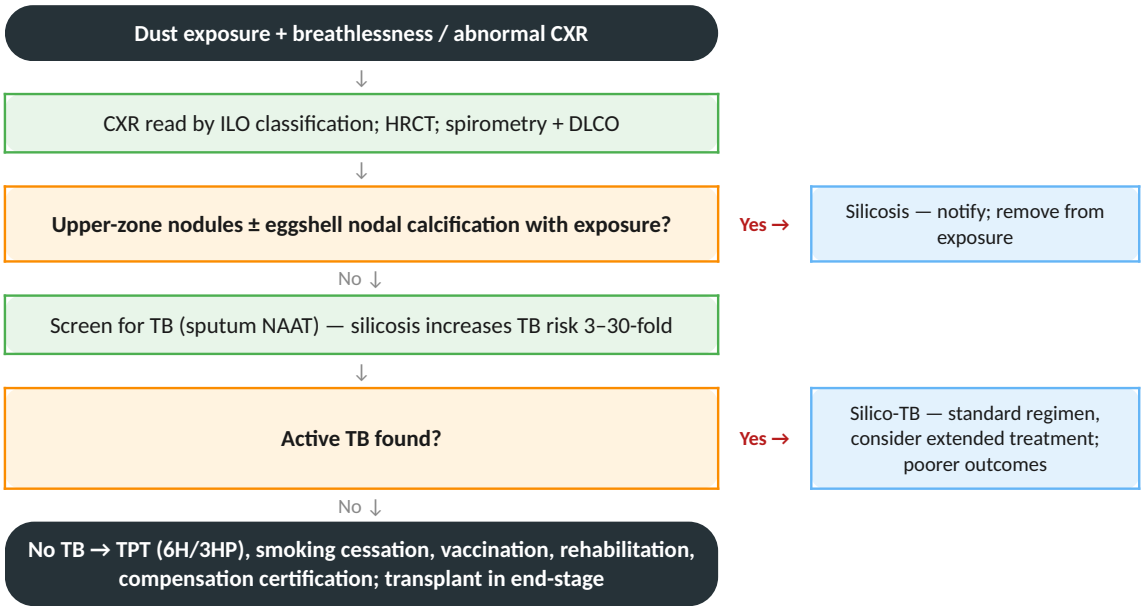
Taking an occupational history

- List every job held with dates, tasks, dust and fume exposure, ventilation, masks, and co-workers with similar illness.
- Ask about home exposures (birds, biomass smoke) and hobbies.
- **Latency:** chronic silicosis > 10 years; accelerated 5–10 years; acute silicoproteinosis within weeks to 5 years of very heavy exposure (sandblasting, engineered stone).

ILO International Classification of Radiographs of Pneumoconioses (2022 revision) — essentials

Element	Description
Small opacities — shape	Rounded: p (< 1.5 mm), q (1.5–3 mm), r (3–10 mm); irregular: s, t, u
Profusion	12-point scale 0/- to 3/+ (major category/alternative considered)
Zones	Upper, middle, lower — right and left
Large opacities	A (> 10 mm up to 50 mm combined), B (> A but < RUZ area), C (> RUZ area) — progressive massive fibrosis
Pleural	Plaques (site, width, extent), diffuse thickening, calcification, costophrenic obliteration
Symbols	e.g. "es" eggshell calcification, "tb" tuberculosis, "hi" hilar enlargement

Algorithm E8.1.1 · Suspected silicosis



Diseases associated with silica exposure

Association	Comment
Tuberculosis and NTM	Impaired macrophage function
Lung cancer	IARC Group 1 carcinogen
COPD, emphysema	Independent of smoking
Autoimmune disease	Erasmus syndrome (silicosis + systemic sclerosis), RA (Caplan-like), ANCA vasculitis
Chronic kidney disease	Silica nephropathy

★ CLINICAL PEARLS & EXAM FOCUS

- Eggshell calcification of hilar nodes: silicosis, sarcoidosis, coal workers' pneumoconiosis, post-radiation lymphoma.
- Rajasthan's Pneumoconiosis Policy (2019) — model for diagnosis, compensation and rehabilitation of silicosis.

RADIOPAEDIA · IMAGE LINKS Silicosis cases | Eggshell calcification cases | Progressive massive fibrosis cases

MERCK MANUAL PROFESSIONAL Silicosis | Pneumoconioses

E8.2 Asbestos-Related Diseases & Other Pneumoconioses

‹ Core topic 8.2 · p.161
Previous-year Qs ›

IN BRIEF Asbestos causes a spectrum from benign pleural plaques to mesothelioma, typically after long latency. India still uses chrysotile asbestos in roofing sheets.

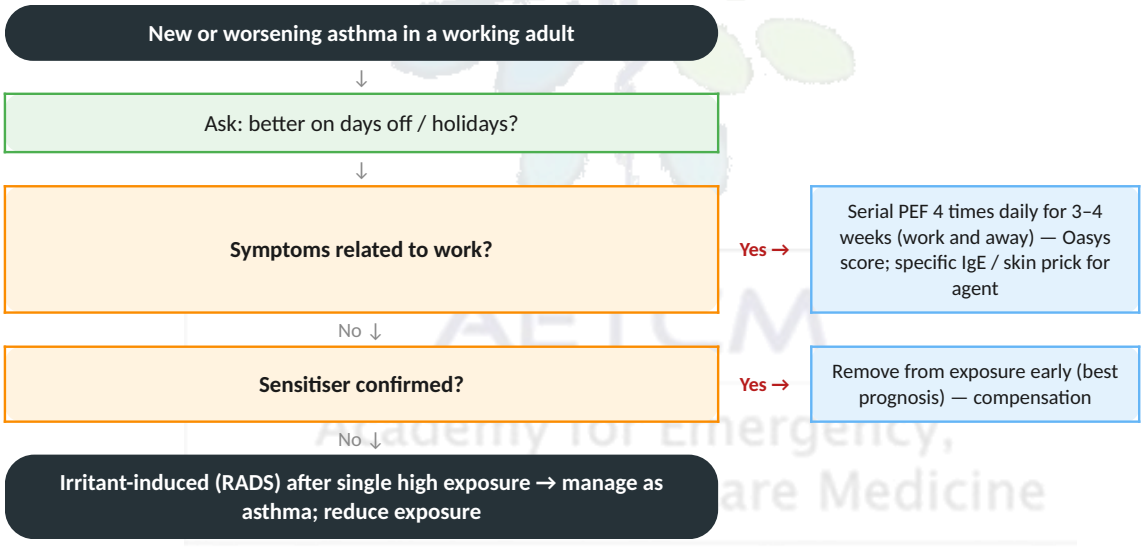
Asbestos-related diseases

Disease	Latency	Features
Pleural plaques	20–30 years	Parietal pleura, calcified "holly-leaf" plaques, spare apices and costophrenic angles — marker of exposure, not premalignant
Benign asbestos pleural effusion	< 10–20 years (earliest)	Exudative, often bloody; diagnosis of exclusion
Diffuse pleural thickening	Variable	Restriction; obliterated costophrenic angle
Rounded atelectasis	Variable	Comet-tail sign — mass mimicking cancer
Asbestosis	20–40 years	Basal UIP-like fibrosis + pleural plaques; asbestos bodies in BAL/tissue
Lung cancer	20–40 years	Synergy with smoking (multiplicative risk ≈ 50×)
Malignant mesothelioma	30–40 years	Dose-related; amphiboles (crocidolite) most potent

Other pneumoconioses and occupational disorders

Disease	Exposure	Hallmark
Coal workers' pneumoconiosis	Coal dust	Upper-zone nodules; PMF; Caplan syndrome with RA
Berylliosis	Aerospace, electronics	Sarcoid-like granulomas; beryllium lymphocyte proliferation test
Hard-metal lung disease	Cobalt, tungsten carbide	Giant-cell interstitial pneumonia
Byssinosis	Cotton, flax, hemp	"Monday morning chest tightness"
Siderosis	Iron fumes (welders)	Benign radiological nodules
Occupational asthma	Isocyanates, flour, latex, wood dust	Work-related symptoms; serial PEF

Algorithm E8.2.1 · Suspected occupational asthma



★ CLINICAL PEARLS & EXAM FOCUS

- Mesothelioma has no safe threshold of exposure — even household contacts of workers are at risk.
- Asbestos bodies (ferruginous bodies) on BAL indicate exposure, not disease.

RADIOPAEDIA · IMAGE LINKS Pleural plaques cases Asbestosis cases Rounded atelectasis cases

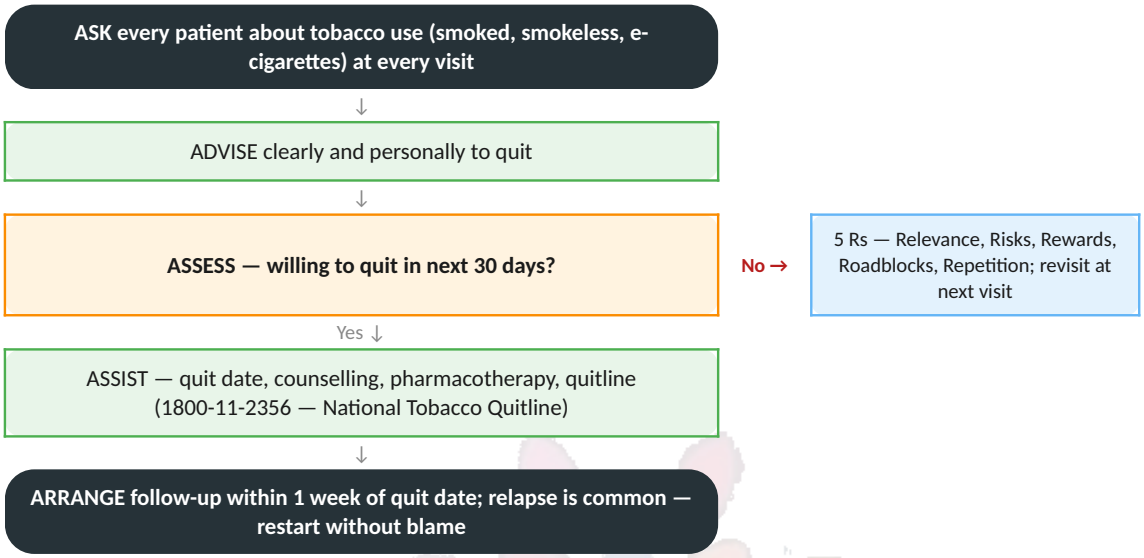
MERCK MANUAL PROFESSIONAL Asbestos-related disease Occupational asthma

E8.3 Smoking Cessation — 5A/5R Model & Pharmacotherapy

‹ Core topic 8.4 · p.164
Previous-year Qs ›

IN BRIEF Tobacco use is the single most important preventable cause of lung disease. Brief advice by a doctor doubles quit rates; combined counselling and pharmacotherapy quadruples them.

Algorithm E8.3.1 · Tobacco cessation in the clinic



Measuring dependence

Tool	Use
Fagerström Test for Nicotine Dependence (FTND, 0–10)	≥ 6 = high dependence
Time to first cigarette	< 5 min after waking = high dependence
Pack-years	(Cigarettes per day / 20) × years smoked; bidi counts as cigarette
Exhaled CO / urinary cotinine	Objective verification

LANDMARK TRIALS & GUIDELINES

- **EAGLES (2016):** varenicline more effective than bupropion and NRT without increase in neuropsychiatric events.
- **Cytisine (ORCA-2, 2023):** effective and low-cost alternative.

E-cigarettes and heated tobacco

- Banned in India (Prohibition of Electronic Cigarettes Act, 2019).
- EVALI — vaping-associated lung injury (vitamin E acetate) — GGO with subpleural sparing, lipid-laden macrophages.

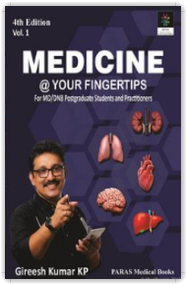
★ CLINICAL PEARLS & EXAM FOCUS

- COPD patients who quit slow FEV1 decline to that of non-smokers (Lung Health Study).
- Smoking induces CYP1A2 — theophylline, clozapine and olanzapine levels rise on quitting.

MERCK MANUAL PROFESSIONAL Smoking cessation

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E8.4 Toxic Inhalational Injury, Smoke Inhalation & Air Pollution

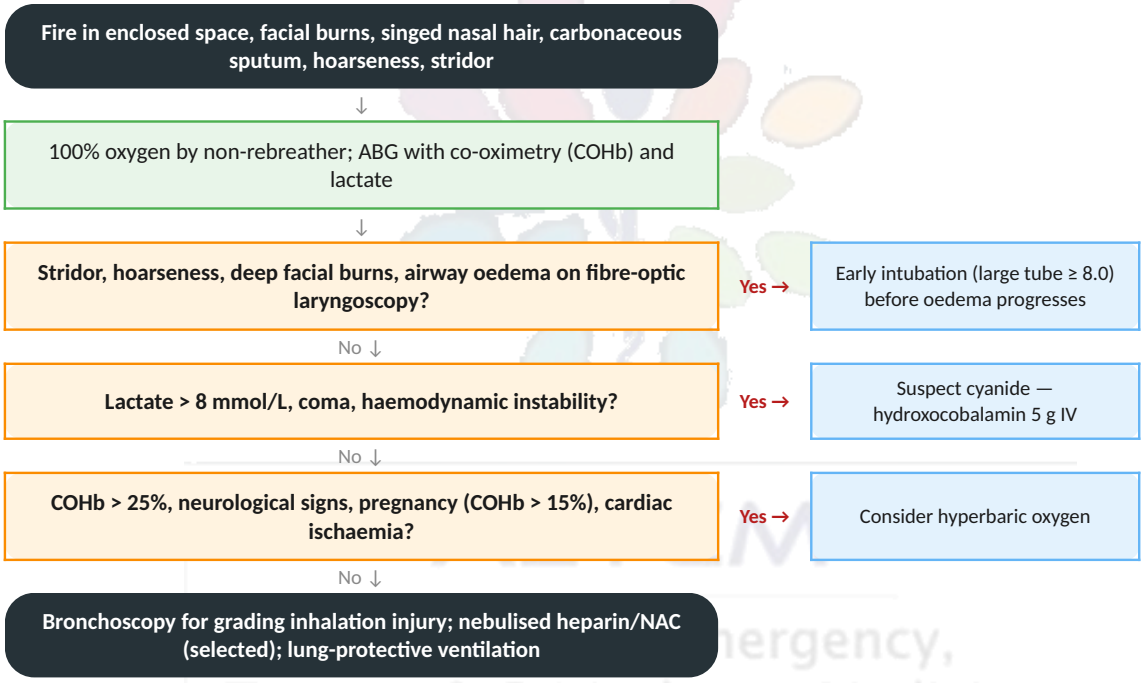
‹ Core topic 8.5 · p.165
Previous-year Qs ›

IN BRIEF Water solubility of a gas decides where it injures — highly soluble gases burn the upper airway; poorly soluble gases reach the alveoli and cause delayed pulmonary oedema.

Irritant gases by water solubility

Solubility	Gases	Site and features
High	Ammonia, chlorine (intermediate), sulphur dioxide, hydrogen chloride	Eyes, nose, throat, laryngeal oedema — immediate symptoms
Intermediate	Chlorine	Upper and lower airway; RADS
Low	Phosgene, nitrogen dioxide (silo filler's), ozone	Delayed (6–24 h) non-cardiogenic pulmonary oedema; bronchiolitis obliterans later
Asphyxiants (chemical)	Carbon monoxide, cyanide, hydrogen sulphide	Tissue hypoxia; normal SpO2 with CO

Algorithm E8.4.1 · Smoke inhalation injury



Air pollution and the lung

- PM2.5 (≤ 2.5 µm) penetrates alveoli; India's NAAQS 24-h limit 60 µg/m³ (annual 40), WHO 2021 guideline 15 µg/m³ (24 h).
- Effects: asthma and COPD exacerbations, lung cancer, low birth weight, cardiovascular deaths; household biomass smoke causes COPD in non-smoking women.
- AQI bands (CPCB): Good 0–50, Satisfactory 51–100, Moderate 101–200, Poor 201–300, Very poor 301–400, Severe 401–500.
- Advice on severe AQI days: stay indoors, HEPA purifiers, N95 masks outdoors, keep reliever handy.

★ CLINICAL PEARLS & EXAM FOCUS

- Pulse oximeters read COHb as oxyhaemoglobin — SpO2 is falsely normal in CO poisoning.
- Silo filler's disease (NO2) can produce bronchiolitis obliterans 2–6 weeks after apparent recovery.

MERCK MANUAL PROFESSIONAL Smoke inhalation | Carbon monoxide poisoning | Air pollution

E8.5 Drug Chart — Smoking Cessation & Inhalational Injury Antidotes

Previous-year Qs ›

Pharmacotherapy for tobacco dependence and toxic inhalations

Class	Drug	Dose	Indication	Caution
Nicotine replacement — patch	Nicotine patch	21 mg/24 h (> 10 cig/day) × 6 weeks → 14 mg × 2 weeks → 7 mg × 2 weeks	Tobacco cessation	Skin irritation, vivid dreams (remove at night)
Nicotine replacement — short-acting	Gum 2/4 mg; lozenge	1 piece every 1–2 h (max 24 gum/day); "chew and park"	Cravings; combine with patch	Jaw pain, dyspepsia
Partial nicotinic agonist	Varenicline	0.5 mg OD × 3 days, 0.5 mg BD × 4 days, then 1 mg BD for 12 weeks	Most effective single agent	Nausea; dose-reduce in renal failure
Partial nicotinic agonist	Cytisine	1.5 mg tablets — tapering schedule over 25 days	Low-cost alternative	Nausea, sleep disturbance
Aminoketone antidepressant	Bupropion SR	150 mg OD × 3 days, then 150 mg BD for 7–12 weeks; start 1–2 weeks before quit date	Tobacco cessation, especially with depression	Contraindicated in seizures, eating disorders, MAOI use
Oxygen	Oxygen 100% / hyperbaric	100% via non-rebreather; HBO 2.5–3 ATA	CO poisoning	Pneumothorax before HBO
Cyanide antidote	Hydroxocobalamin	5 g IV over 15 min (repeat once)	Cyanide toxicity in smoke inhalation	Red urine/skin, interferes with colorimetric tests
Cyanide antidote	Sodium nitrite + sodium thiosulphate	300 mg IV + 12.5 g IV	Cyanide (if hydroxocobalamin unavailable)	Methaemoglobinaemia — avoid in CO co-poisoning
Methaemoglobin reducer	Methylene blue	1–2 mg/kg IV over 5 min	Methaemoglobinaemia > 20–30% (nitrates, dapsone)	Contraindicated in G6PD deficiency, serotonin syndrome
Nebulised anticoagulant	Heparin 5000–10 000 U + N-acetylcysteine	4-hourly nebulisation	Severe smoke inhalation (selected centres)	Bleeding
Bronchodilator	Salbutamol	2.5–5 mg nebulised	Bronchospasm after irritant exposure	—
Neutralising nebulisation	Sodium bicarbonate 4% (3–4 mL)	Nebulised	Chlorine inhalation (weak evidence)	—

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PART II · SECTION 9

Pulmonary Circulation — Hypertension, Embolism, Oedema & Haemoptysis

Advanced theory & algorithms

E9.1 Pulmonary Hypertension — ESC/ERS 2022 Diagnostic Algorithm 417
E9.2 PAH — Risk Stratification & Treatment Algorithm 418
E9.3 Pulmonary Embolism — Diagnostic Algorithm 420
E9.4 PE — Risk Stratification, Reperfusion & Anticoagulation Duration 422
E9.5 Massive Haemoptysis — Emergency Algorithm 423
E9.6 Drug Chart — Anticoagulants, Thrombolytics & PAH-Specific Therapy 424
E9.7 CTEPH & Pulmonary Oedema — Diagnostic and Management Pathways 426

E9.1 Pulmonary Hypertension — ESC/ERS 2022 Diagnostic Algorithm

‹ Core topic 9.1 · p.168
Previous-year Qs ›

IN BRIEF PH is now defined as mean pulmonary artery pressure (mPAP) > 20 mmHg at rest on right heart catheterisation (RHC). The echocardiogram estimates probability; RHC confirms and classifies.

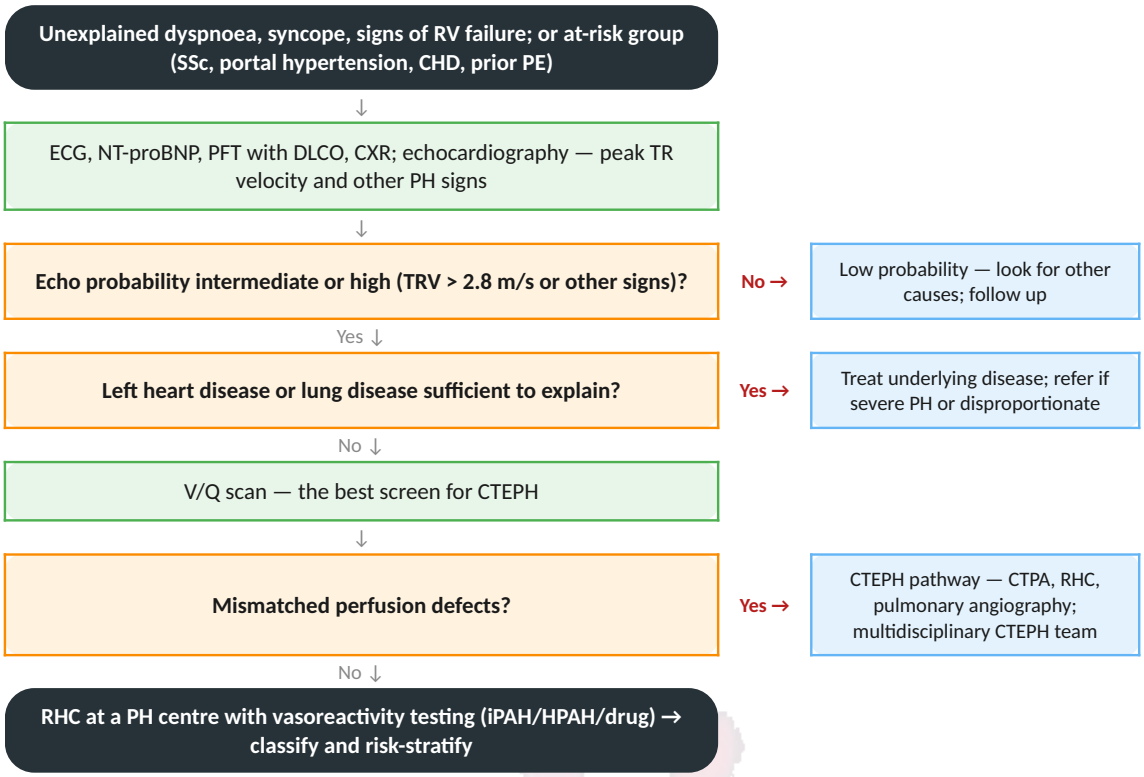
Haemodynamic definitions (ESC/ERS 2022)

Definition	Haemodynamics
Pulmonary hypertension	mPAP > 20 mmHg
Pre-capillary PH	mPAP > 20, PAWP ≤ 15, PVR > 2 WU
Isolated post-capillary PH	mPAP > 20, PAWP > 15, PVR ≤ 2 WU
Combined pre- and post-capillary PH	mPAP > 20, PAWP > 15, PVR > 2 WU
Exercise PH	mPAP/CO slope > 3 mmHg/L/min between rest and exercise

Clinical classification

Group	Examples
1 PAH	Idiopathic, heritable (BMPR2), drug/toxin-induced (methamphetamine, dasatinib), CTD (SSc), HIV, portal hypertension, congenital heart disease, schistosomiasis; PVOD/PCH
2 Left heart disease	HFpEF, HFrEF, valve disease
3 Lung disease / hypoxia	COPD, ILD, OHS, high altitude
4 Pulmonary artery obstruction	CTEPH, other obstructions (tumours, arteritis)
5 Unclear / multifactorial	Sarcoidosis, haematological, metabolic disorders, chronic renal failure on dialysis

Algorithm E9.1.1 · Suspected pulmonary hypertension



★ CLINICAL PEARLS & EXAM FOCUS

- An echo TRV > 3.4 m/s indicates high probability; 2.9–3.4 intermediate (with other signs).
- V/Q scanning is more sensitive than CTPA for CTEPH — never skip it.
- DLCO < 45% predicted with mild obstruction in a smoker suggests PH out of proportion or PVOD.

RADIOPAEDIA · IMAGE LINKS Pulmonary arterial hypertension cases | Chronic thromboembolic pulmonary hypertension cases

MERCK MANUAL PROFESSIONAL Pulmonary hypertension

E9.2 PAH — Risk Stratification & Treatment Algorithm

‹ Core topic 9.2 · p.170
Previous-year Qs ›

IN BRIEF PAH therapy targets three pathways — endothelin, nitric oxide and prostacyclin — and now the activin pathway (sotatercept). Initial combination therapy is standard for most patients.

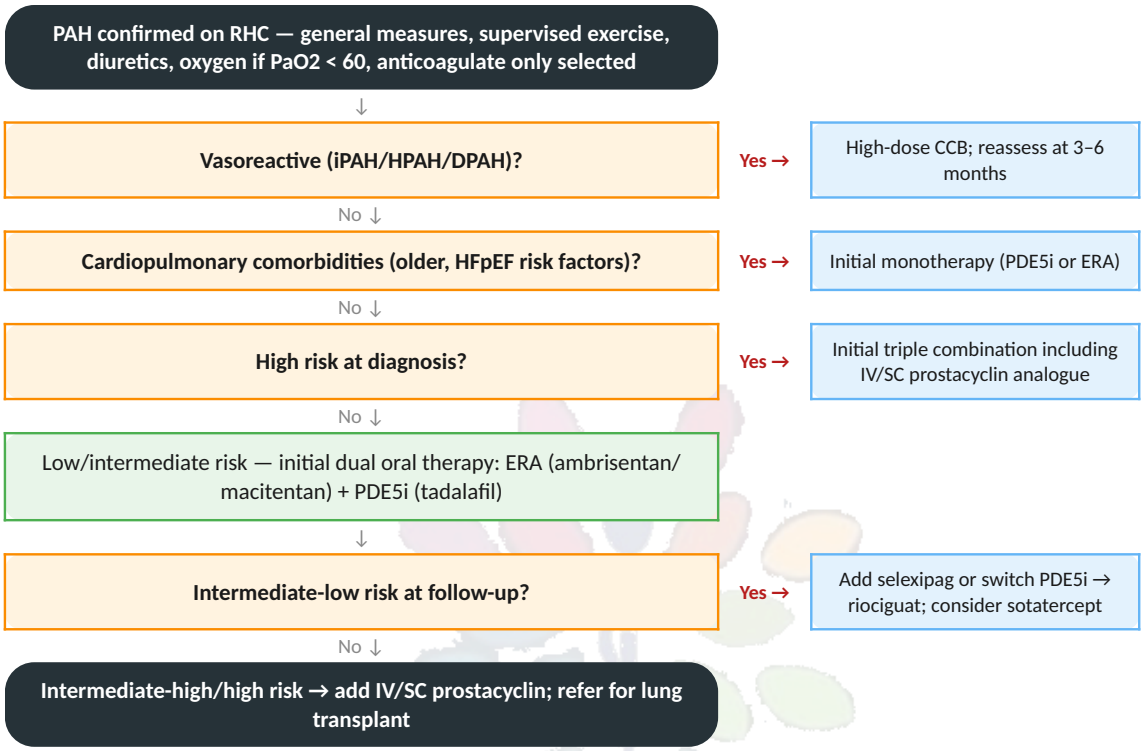
Four-strata follow-up risk tool (ESC/ERS 2022)

Variable	Low (1)	Intermediate-low (2)	Intermediate-high (3)	High (4)
WHO functional class	I or II	—	III	IV
6MWD	> 440 m	320–440 m	165–319 m	< 165 m
NT-proBNP	< 300 ng/L	300–649	650–1100	> 1100

Vasoreactivity testing

Feature	Detail
Who	Idiopathic, heritable and drug-induced PAH
Agent	Inhaled NO 20–40 ppm, IV epoprostenol, IV adenosine
Positive	Fall in mPAP ≥ 10 mmHg to ≤ 40 mmHg with unchanged or increased cardiac output
Implication	High-dose calcium channel blocker (nifedipine, diltiazem, amlodipine) — only ≈ 10% respond

Algorithm E9.2.1 · PAH treatment (ESC/ERS 2022)



LANDMARK TRIALS & GUIDELINES

- **AMBITION:** initial ambrisentan + tadalafil reduced clinical failure by 50%.
- **GRIPHON (selexipag), SERAPHIN (macitentan).**
- **STELLAR (2023), ZENITH (2025):** sotatercept (activin signalling inhibitor) improved 6MWD and reduced morbidity/mortality.

★ CLINICAL PEARLS & EXAM FOCUS

- Riociguat must not be combined with PDE5 inhibitors (hypotension).
- Endothelin receptor antagonists are teratogenic — pregnancy test monthly; pregnancy is contraindicated in PAH.

MERCK MANUAL PROFESSIONAL Pulmonary arterial hypertension

E9.3 Pulmonary Embolism — Diagnostic Algorithm

‹ Core topic 9.4 · p.173
Previous-year Qs ›

IN BRIEF Clinical probability first, then D-dimer or imaging. In haemodynamically unstable patients, bedside echocardiography and immediate reperfusion take priority over CTPA.

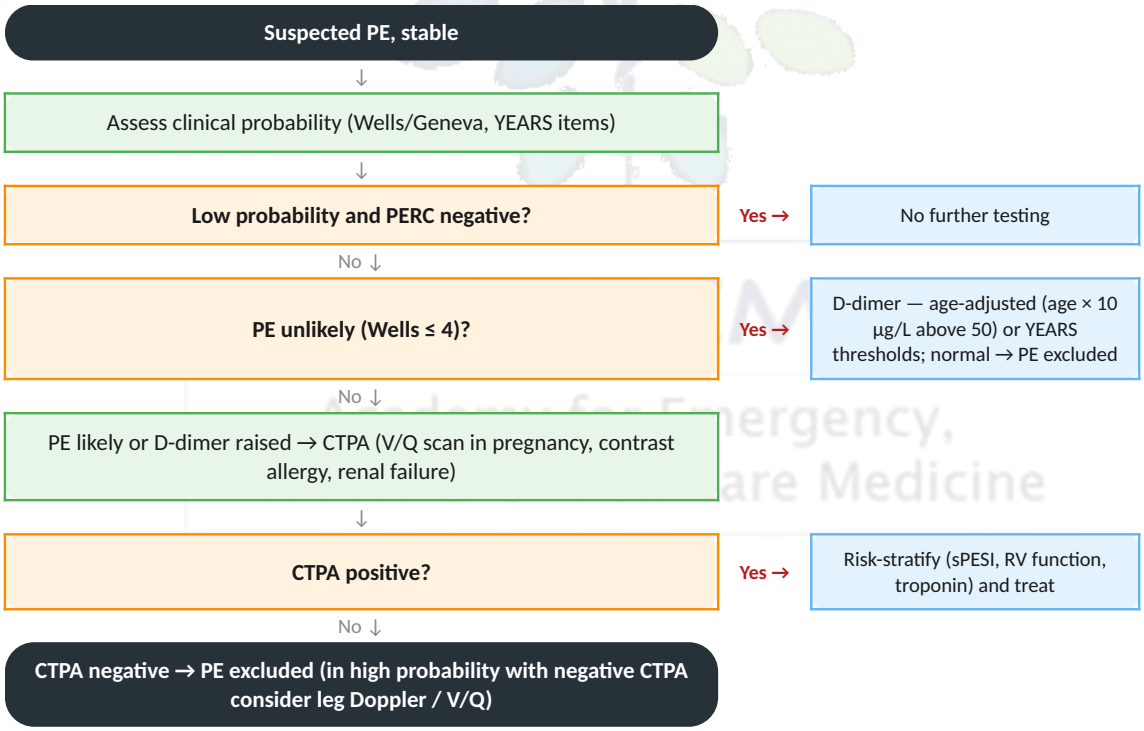
Clinical probability scores

Wells score item	Points
Clinical signs of DVT	3
PE most likely diagnosis	3
HR > 100	1.5
Immobilisation ≥ 3 days or surgery in 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Active cancer	1
≤ 4 = PE unlikely; > 4 = PE likely	—

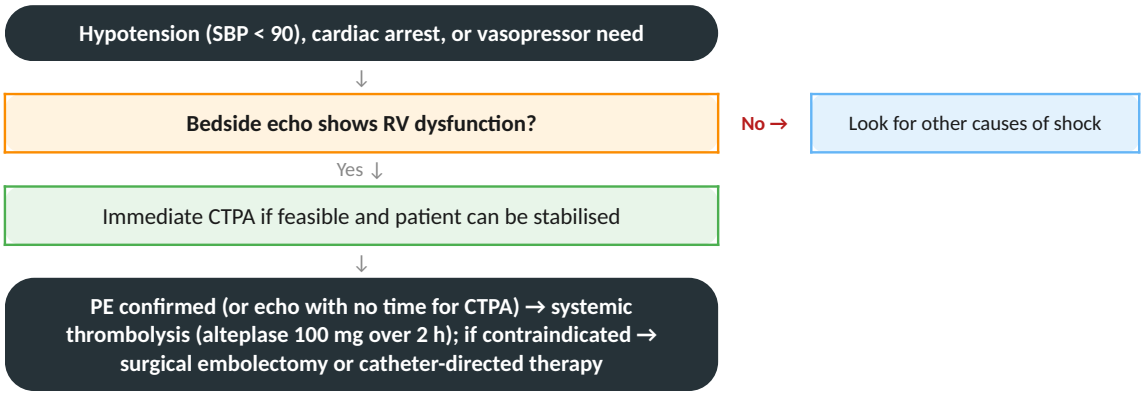
PERC rule — in low pre-test probability, PE excluded if all are negative

- Age < 50, HR < 100, SpO2 ≥ 95%, no haemoptysis, no oestrogen use, no prior VTE, no unilateral leg swelling, no recent surgery/trauma.

Algorithm E9.3.1 · Suspected PE — haemodynamically stable



Algorithm E9.3.2 · Suspected high-risk PE (shock or hypotension)



★ CLINICAL PEARLS & EXAM FOCUS

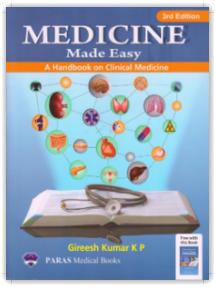
- YEARS algorithm: no items → D-dimer threshold 1000; ≥ 1 item → 500. Reduces CTPA use by ≈ 14%.
- In pregnancy: Doppler of the legs first if symptoms; the pregnancy-adapted YEARS algorithm is validated.

RADIOPAEDIA · IMAGE LINKS Pulmonary embolism cases · Hampton hump cases · Westermark sign cases · Right ventricular strain cases

AETCM YOUTUBE · VIDEO LECTURES ▶ pulmonary embolism · ▶ thrombolysis · ▶ DVT

MERCK MANUAL PROFESSIONAL Pulmonary embolism

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Gireesh Kumar K P

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E9.4 PE — Risk Stratification, Reperfusion & Anticoagulation Duration

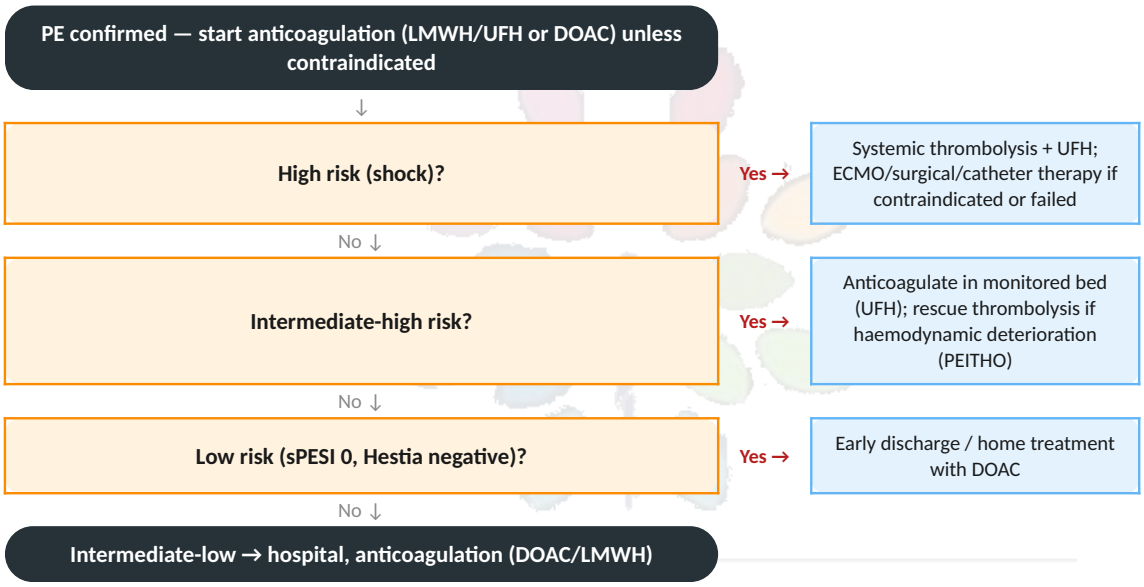
‹ Core topic 9.5 · p.175
Previous-year Qs ›

IN BRIEF Treatment is guided by early mortality risk (ESC 2019): high, intermediate-high, intermediate-low and low. Most patients need only anticoagulation; low-risk patients can be treated at home.

ESC risk classes

Risk	Shock	PESI III–V / sPESI ≥ 1	RV dysfunction (echo/CT)	Troponin raised
High	+	(+)	+	(+)
Intermediate-high	–	+	+	+
Intermediate-low	–	+	One or none positive	One or none positive
Low	–	–	– (optional)	– (optional)

Algorithm E9.4.1 · Management by risk class



Duration of anticoagulation

Situation	Duration
Provoked by major transient risk factor (surgery, trauma)	3 months
Minor transient factor (OCP, travel, minor injury)	3 months, consider extended
Unprovoked or persistent risk factor	Extended (indefinite) — reduced-dose apixaban 2.5 mg BD / rivaroxaban 10 mg OD after 6 months
Active cancer	LMWH or DOAC (edoxaban, rivaroxaban, apixaban) for ≥ 6 months / while cancer active
Antiphospholipid syndrome (triple positive)	Warfarin indefinitely — avoid DOACs
Pregnancy	LMWH through pregnancy and ≥ 6 weeks postpartum (total ≥ 3 months)

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LANDMARK TRIALS & GUIDELINES

- **PEITHO (2014):** tenecteplase in intermediate-high risk reduced decompensation but increased major and intracranial bleeding.
- **HI-PEITHO, PEERLESS (2024–25):** catheter-directed therapy trials.

★ CLINICAL PEARLS & EXAM FOCUS

- IVC filter only when anticoagulation is absolutely contraindicated in acute VTE — retrieve when possible.
- Follow-up at 3–6 months for persistent dyspnoea — screen for CTEPH (echo, V/Q).

MERCK MANUAL PROFESSIONAL Pulmonary embolism

E9.5 Massive Haemoptysis — Emergency Algorithm

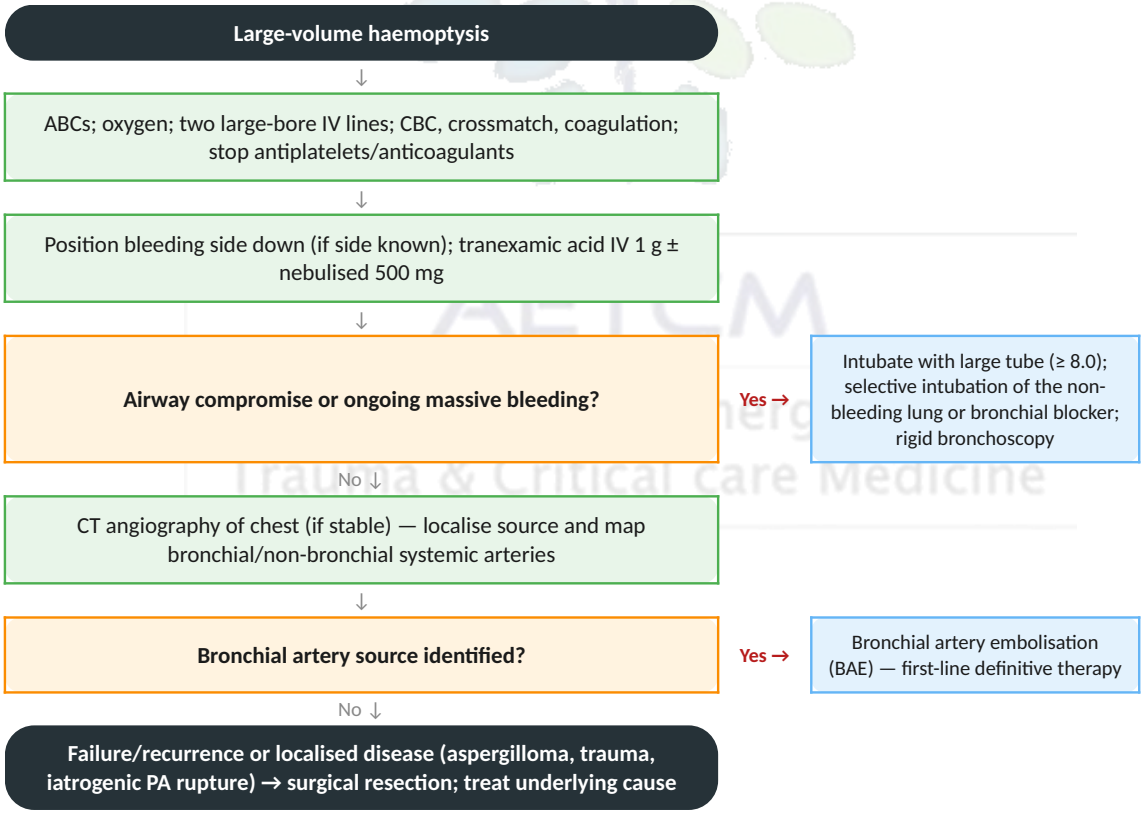
‹ Core topic 9.7 · p.178
Previous-year Qs ›

IN BRIEF Massive (life-threatening) haemoptysis kills by asphyxia, not exsanguination. Protect the good lung, then localise and stop the bleeding. 90% of bleeding arises from the bronchial (systemic) circulation.

Definitions and common causes

Item	Detail
Life-threatening haemoptysis	> 100–600 mL/24 h, or any bleeding causing airway obstruction, hypoxaemia or haemodynamic instability
Commonest causes in India	Active or post-TB (bronchiectasis, cavity, Rasmussen aneurysm), aspergilloma, bronchiectasis, lung cancer
Others	Mitral stenosis, AVM, vasculitis/DAH, coagulopathy, PE, trauma, iatrogenic

Algorithm E9.5.1 · Massive haemoptysis



Bronchoscopic measures

- Cold saline lavage, topical adrenaline (1:20 000) or tranexamic acid, balloon tamponade (Fogarty), endobronchial blockers, argon plasma coagulation for visible tumours.
- Rigid bronchoscopy allows better suction and airway control.

★ CLINICAL PEARLS & EXAM FOCUS

- BAE complication: spinal cord ischaemia (anterior spinal artery from intercostobronchial trunk) — rare but feared.
- Rasmussen aneurysm = pseudoaneurysm of a pulmonary artery branch in a TB cavity — needs pulmonary angiography.

RADIOPAEDIA · IMAGE LINKS Bronchial artery embolisation cases Rasmussen aneurysm cases Haemoptysis cases

AETCM YOUTUBE · VIDEO LECTURES ▶ haemoptysis ▶ bronchial artery embolisation

MERCK MANUAL PROFESSIONAL Hemoptysis

E9.6 Drug Chart — Anticoagulants, Thrombolytics & PAH-Specific Therapy

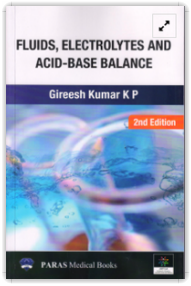
Previous-year Qs ›

Drugs in pulmonary vascular disease

Class	Drug	Dose	Indication	Caution
Unfractionated heparin	Heparin	80 U/kg bolus then 18 U/kg/h (aPTT 1.5–2.5×)	High-risk PE, before thrombolysis, renal failure	HIT, bleeding — reversible with protamine
LMWH	Enoxaparin	1 mg/kg SC BD (or 1.5 mg/kg OD)	VTE treatment, pregnancy, cancer	Reduce dose if CrCl < 30; anti-Xa monitoring in pregnancy/obesity
Factor Xa inhibitor (DOAC)	Rivaroxaban	15 mg BD × 21 days then 20 mg OD	VTE treatment	Avoid in triple-positive APS, pregnancy, CrCl < 15
Factor Xa inhibitor (DOAC)	Apixaban	10 mg BD × 7 days then 5 mg BD; 2.5 mg BD extended	VTE treatment and extended prophylaxis	Same
Factor Xa inhibitor (DOAC)	Edoxaban	60 mg OD after ≥ 5 days of parenteral heparin	VTE, cancer VTE	30 mg if CrCl 15–50 or ≤ 60 kg
Direct thrombin inhibitor	Dabigatran	150 mg BD after ≥ 5 days of heparin	VTE	Renal excretion; reversal idarucizumab
Vitamin K antagonist	Warfarin	Start 5 mg, INR 2–3; overlap heparin ≥ 5 days and INR ≥ 2 × 24 h	APS, mechanical valves, CTEPH	Diet/drug interactions; teratogenic
Fibrinolytic	Alteplase	100 mg IV over 2 h (0.6 mg/kg over 15 min in arrest)	High-risk PE	Bleeding, ICH — check contraindications
Fibrinolytic	Tenecteplase	Weight-based single bolus (30–50 mg)	High-risk PE (off-label)	Bleeding
Fibrinolytic	Streptokinase	250 000 IU over 30 min then 100 000 IU/h × 24 h	High-risk PE where alteplase unavailable	Hypotension, allergy
Antifibrinolytic	Tranexamic acid	1 g IV 8-hourly; 500 mg nebulised	Haemoptysis	Thrombosis
PDE5 inhibitor	Sildenafil / Tadalafil	20 mg TDS / 40 mg OD	PAH	Never with nitrates or riociguat

Class	Drug	Dose	Indication	Caution
sGC stimulator	Riociguat	1 mg TDS titrated to 2.5 mg TDS	CTEPH (inoperable/ residual), PAH	Hypotension; teratogenic
Endothelin receptor antagonist	Ambrisentan / Bosentan / Macitentan	5–10 mg OD / 62.5–125 mg BD / 10 mg OD	PAH	Teratogenic; LFT (bosentan); anaemia, oedema
Prostacyclin receptor agonist	Selexipag	200 µg BD titrated to 1600 µg BD	PAH add-on	Headache, jaw pain, diarrhoea
Prostacyclin analogue	Epoprostenol / Treprostinil / Iloprost	IV infusion / SC or inhaled / inhaled 6–9 times daily	High-risk PAH	Line sepsis, rebound PH if interrupted
Activin signalling inhibitor	Sotatercept	0.3 → 0.7 mg/kg SC every 3 weeks	PAH add-on	Raised Hb, thrombocytopenia, telangiectasia
Calcium channel blocker	Nifedipine / Diltiazem / Amlodipine	Up to 120–240 / 240–720 / 20 mg daily	Vasoreactive PAH only	Hypotension; harmful in non-responders
Loop diuretic	Furosemide	20–80 mg/day	RV failure	Hypovolaemia worsens RV preload

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Fluids, Electrolytes & Acid-Base Balance

Clinical reference · 2nd Edition

Gireesh Kumar K P

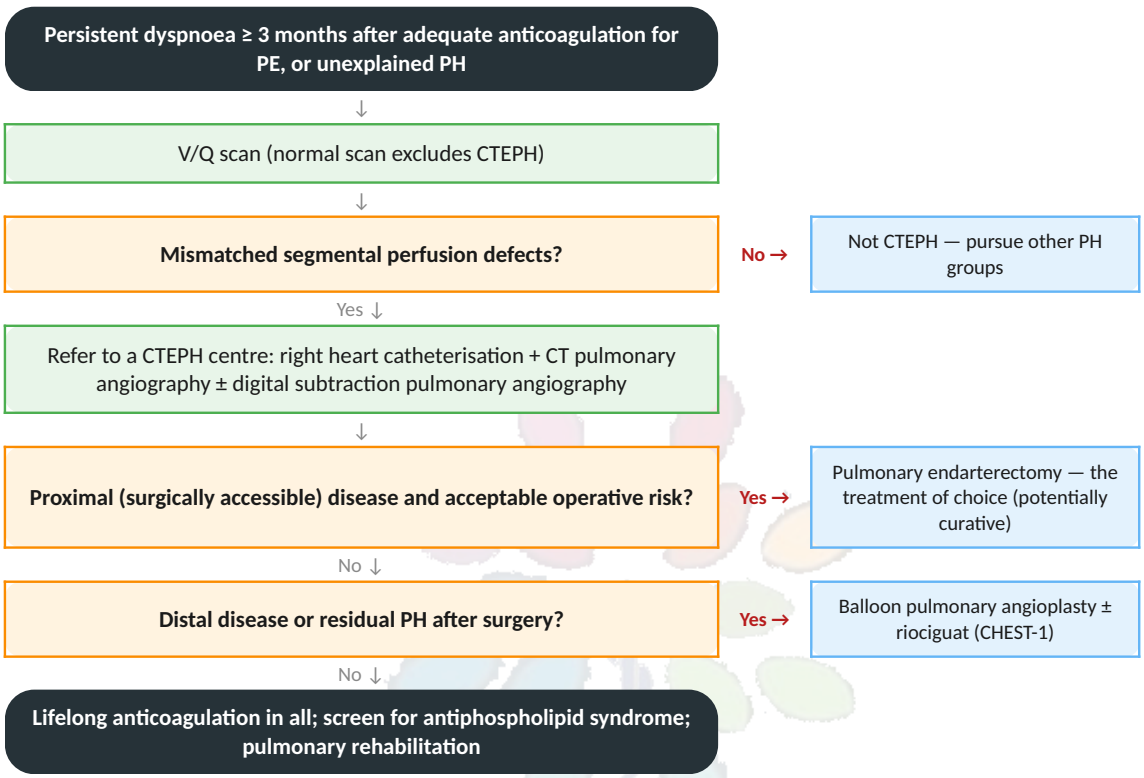
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E9.7 CTEPH & Pulmonary Oedema — Diagnostic and Management Pathways

‹ Core topic 9.3 · p.172
Previous-year Qs ›

IN BRIEF CTEPH is the only form of pulmonary hypertension that is potentially curable by surgery. Pulmonary oedema needs separation into cardiogenic and non-cardiogenic at the bedside.

Algorithm E9.7.1 · CTEPH pathway



Cardiogenic vs non-cardiogenic pulmonary oedema

Feature	Cardiogenic	Non-cardiogenic (ARDS)
History	Cardiac disease, fluid overload	Sepsis, aspiration, pancreatitis, trauma
Onset	Minutes–hours	Hours–days
JVP, gallop, peripheral oedema	Present	Usually absent
CXR	Central "bat-wing", septal lines, effusions, cardiomegaly	Peripheral patchy, air bronchograms, normal heart size
Echo / BNP	Reduced EF or raised filling pressures; NT-proBNP high	Normal function; NT-proBNP lower
Lung ultrasound	Diffuse smooth B-lines, smooth pleura	B-lines with spared areas, irregular pleura, subpleural consolidations
Oedema fluid protein / plasma protein	< 0.65	> 0.75
Management	Diuretics, nitrates, NIV/CPAP, treat ischaemia or arrhythmia	Treat cause, lung-protective ventilation, conservative fluids

Other forms of pulmonary oedema

Type	Mechanism
Neurogenic	Massive sympathetic surge after head injury, SAH, seizures
Re-expansion	Rapid drainage of a large effusion or long-standing pneumothorax
High-altitude (HAPE)	Uneven hypoxic vasoconstriction, high capillary pressure
Negative-pressure (post-obstructive)	Forceful inspiration against a closed glottis (laryngospasm)
Immersion / swimming-induced	Central blood volume shift with cold and exertion
Transfusion-related (TACO vs TRALI)	Volume overload vs antibody-mediated lung injury

★ **CLINICAL PEARLS & EXAM FOCUS**

- A normal V/Q scan effectively excludes CTEPH; a normal CTPA does not.
- Re-expansion pulmonary oedema: drain no more than 1–1.5 L at a time and stop for chest tightness or cough.

RADIOPAEDIA · IMAGE LINKS Chronic thromboembolic pulmonary hypertension cases · Pulmonary oedema cases · Bat wing opacities cases

MERCK MANUAL PROFESSIONAL Pulmonary edema · Chronic thromboembolic pulmonary hypertension

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PART II · SECTION 10

Lung Cancer & Other Thoracic Tumours

Advanced theory & algorithms

E10.1 Solitary Pulmonary Nodule — Fleischner 2017 & BTS Management Algorithm 428
E10.2 Lung Cancer — Diagnosis & Staging Algorithm (TNM 9th Edition) 429
E10.3 NSCLC & SCLC — Stage-Wise Treatment Algorithm 431
E10.4 Thoracic Oncological Emergencies — SVCO, Central Airway Obstruction & Paraneoplastic Crises 433
E10.5 Lung Cancer Screening & Prevention 434
E10.6 Drug Chart — Systemic Therapy in Thoracic Oncology 435
E10.7 Immunotherapy Toxicity & Thoracic Radiation Injury 437

E10.1 Solitary Pulmonary Nodule — Fleischner 2017 & BTS Management Algorithm

‹ Core topic 10.7 · p.190
Previous-year Qs ›

IN BRIEF A nodule is a rounded opacity ≤ 3 cm; larger lesions are masses. Size, attenuation (solid, part-solid, ground-glass), growth and patient risk decide surveillance, PET-CT or biopsy.

Fleischner 2017 — incidental solid nodules (adults ≥ 35 years, not for screening or cancer patients)

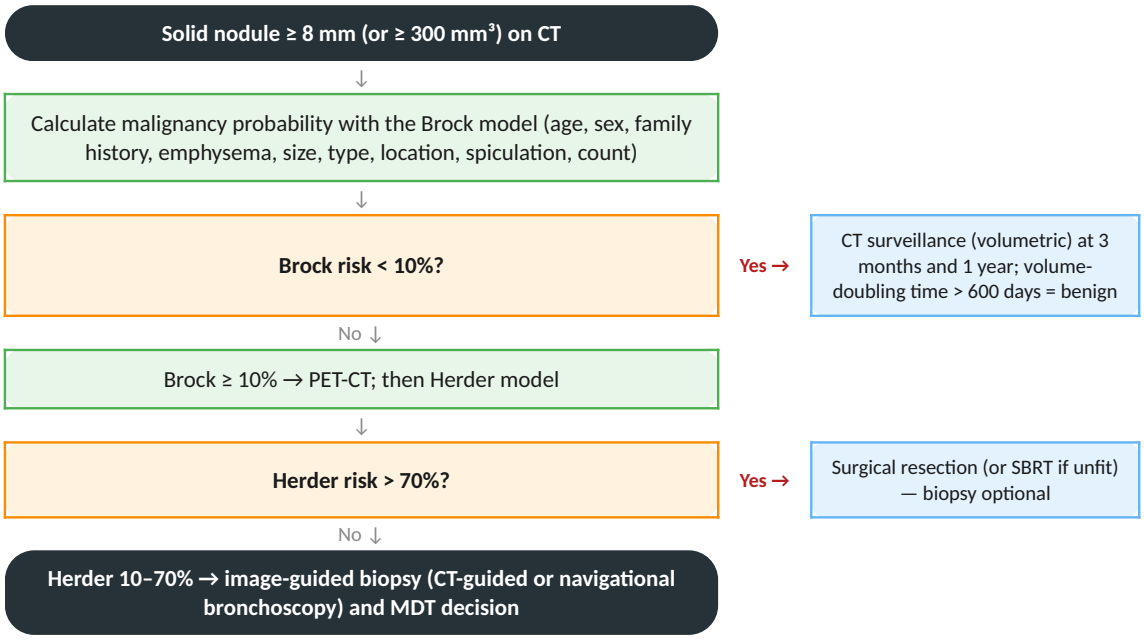
Size	Low-risk patient	High-risk patient
< 6 mm (< 100 mm^3)	No routine follow-up	Optional CT at 12 months
6–8 mm (100–250 mm^3)	CT at 6–12 months, then consider 18–24 months	CT at 6–12 months, then 18–24 months
> 8 mm (> 250 mm^3)	Consider CT at 3 months, PET-CT, or tissue sampling	Same

Subsolid nodules (Fleischner)

Type	Recommendation
Pure GGN < 6 mm	No routine follow-up
Pure GGN ≥ 6 mm	CT at 6–12 months, then every 2 years until 5 years
Part-solid < 6 mm	No routine follow-up
Part-solid ≥ 6 mm, solid part < 6 mm	CT at 3–6 months; annual for 5 years if persistent
Part-solid with solid component ≥ 6 mm	Highly suspicious — PET-CT, biopsy or resection

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Algorithm E10.1.1 · Managing a solid nodule ≥ 8 mm (BTS 2015 approach)



Features of benign vs malignant nodules

Benign	Malignant
Calcification: central, laminated, diffuse, popcorn (hamartoma)	Spiculation, lobulation, pleural tag
Intranodular fat (hamartoma)	Upper-lobe location, size > 8 mm
Stable for 2 years (solid)	Growth — volume-doubling time 20–400 days
Perifissural nodule with triangular shape	Part-solid with growing solid part, cavity with thick wall > 15 mm

★ CLINICAL PEARLS & EXAM FOCUS

- In India, granulomas (TB) are a common cause of PET-avid nodules — PET-CT has lower specificity here; tissue diagnosis is often needed.
- Volume-doubling time < 20 days suggests infection; > 400–600 days suggests benign (except GGN adenocarcinoma, which grows slowly).

RADIOPAEDIA · IMAGE LINKS Solitary pulmonary nodule cases | Fleischner society pulmonary nodule recommendations cases | Popcorn calcification cases

MERCK MANUAL PROFESSIONAL Solitary pulmonary nodule

E10.2 Lung Cancer — Diagnosis & Staging Algorithm (TNM 9th Edition)

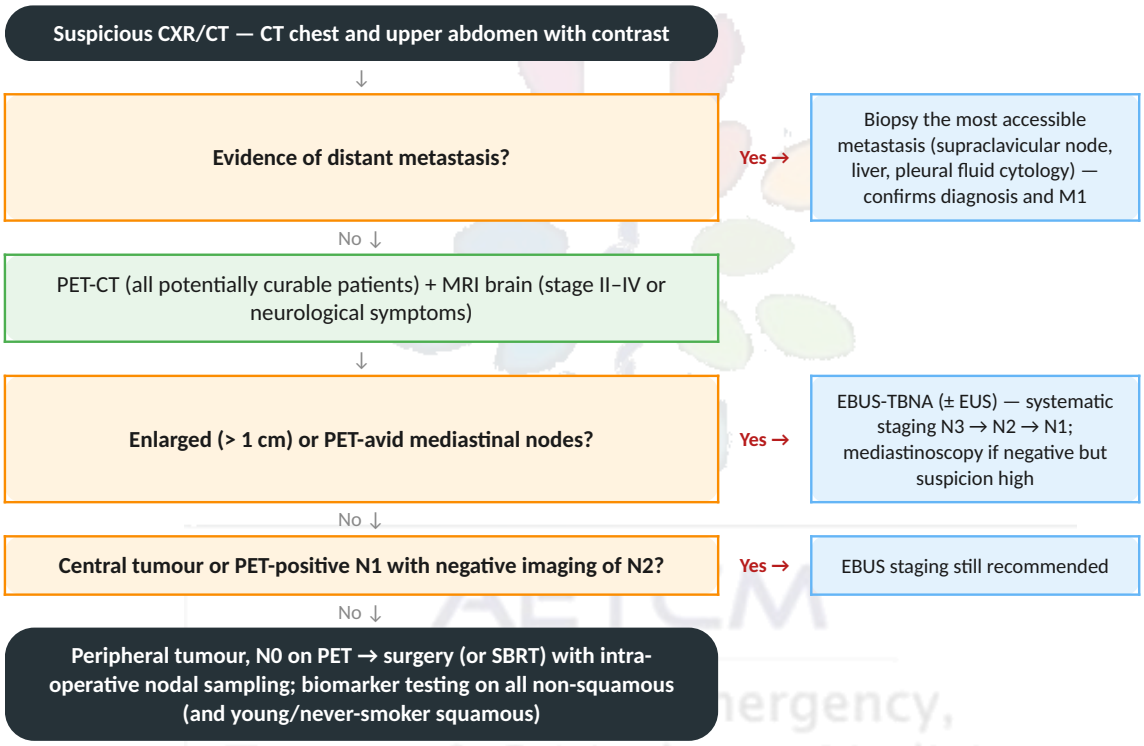
‹ Core topic 10.4 · p.186
Previous-year Qs ›

IN BRIEF Diagnose and stage with a single procedure whenever possible — sample the site that gives the highest stage. EBUS-TBNA of a mediastinal node often does both.

TNM 9th edition (IASLC, 2024) — key changes

Element	Definition
T1	≤ 3 cm: T1a ≤ 1, T1b > 1–2, T1c > 2–3 cm
T2	> 3–5 cm, or main bronchus involvement, visceral pleural invasion, atelectasis to hilum (T2a > 3–4, T2b > 4–5)
T3	> 5–7 cm, separate nodule same lobe, chest wall/phrenic/parietal pericardium
T4	> 7 cm, separate nodule different ipsilateral lobe, mediastinum, heart, great vessels, trachea, carina, oesophagus, vertebra, diaphragm
N1	Ipsilateral peribronchial/hilar
N2a / N2b	Single-station / multiple-station ipsilateral mediastinal (new in 9th edition)
N3	Contralateral mediastinal/hilar, any scalene/supraclavicular
M1a	Pleural/pericardial effusion or nodules, contralateral lung nodule
M1b	Single extrathoracic metastasis
M1c1 / M1c2	Multiple extrathoracic metastases in one organ system / in multiple organ systems (new)

Algorithm E10.2.1 · Suspected lung cancer — diagnosis and staging



Biomarker testing (every non-squamous NSCLC)

- EGFR (exon 19 deletion, L858R; T790M at progression; exon 20 insertions), ALK, ROS1, BRAF V600E, KRAS G12C, MET exon 14 skipping, RET, NTRK, HER2 — by NGS preferably.
- PD-L1 IHC (TPS) for immunotherapy decisions.
- Liquid biopsy (ctDNA) when tissue is insufficient — a negative result needs tissue confirmation.

★ CLINICAL PEARLS & EXAM FOCUS

- In India, EGFR mutations occur in ≈ 25–30% of adenocarcinomas — higher in non-smoking women.
- Always obtain enough tissue for NGS — cryobiopsy or core biopsy rather than small FNA where possible.

RADIOPAEDIA · IMAGE LINKS Lung cancer staging cases | Pancoast tumour cases | Golden S sign cases

AETCM YOUTUBE · VIDEO LECTURES ▶ lung cancer | ▶ thoracic oncology

MERCK MANUAL PROFESSIONAL Lung carcinoma

E10.3 NSCLC & SCLC — Stage-Wise Treatment Algorithm

‹ Core topic 10.5 · p.187
Previous-year Qs ›

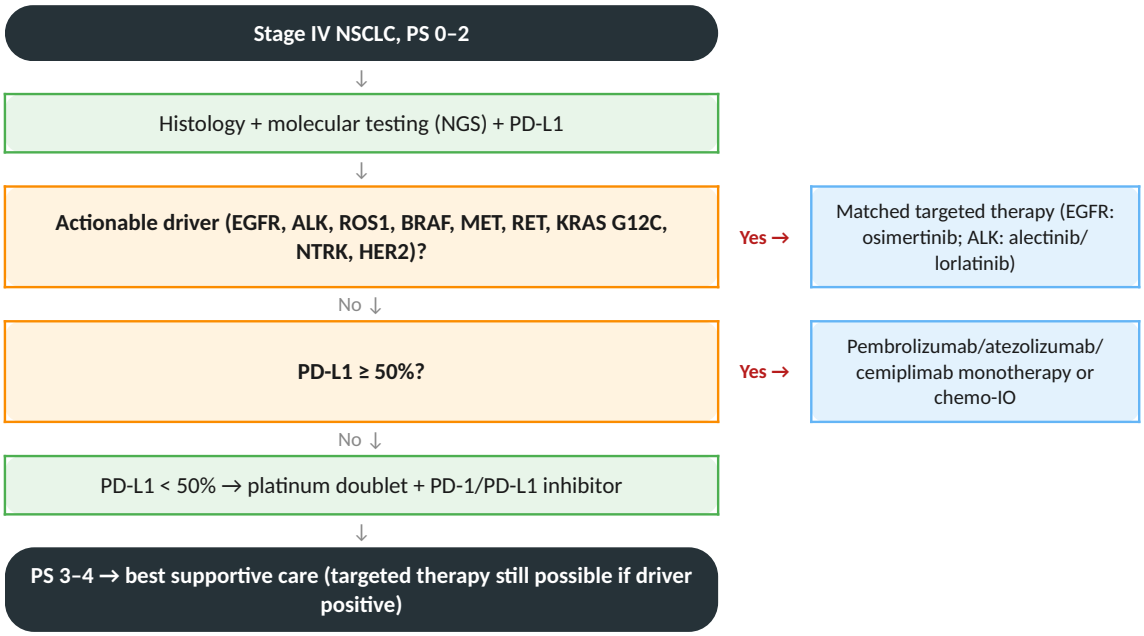
IN BRIEF Treatment depends on stage, histology, biomarkers, PD-L1 and fitness. Curative options exist for stage I–III; targeted therapy and immunotherapy have transformed stage IV survival.

Stage-wise treatment of NSCLC

Stage	Treatment
IA–IB	Lobectomy (segmentectomy for ≤ 2 cm peripheral — JCOG0802, CALGB 140503); SBRT if inoperable
II–IIIA resectable	Perioperative chemo-immunotherapy (KEYNOTE-671) or neoadjuvant nivolumab–chemo (CheckMate 816) → surgery; adjuvant osimertinib if EGFR+ (ADAURA); alectinib if ALK+ (ALINA)
III unresectable	Concurrent chemoradiation → durvalumab consolidation × 12 months (PACIFIC); osimertinib if EGFR+ (LAURA)
IV driver-positive	Targeted therapy — osimertinib (EGFR; ± chemotherapy FLAURA2; amivantamab + lazertinib MARIPOSA); alectinib/lorlatinib (ALK — CROWN); others by driver
IV no driver, PD-L1 ≥ 50%	Pembrolizumab monotherapy (KEYNOTE-024) or chemo-immunotherapy
IV no driver, PD-L1 < 50%	Platinum doublet + pembrolizumab (KEYNOTE-189 non-squamous, -407 squamous)
Oligometastatic	Systemic therapy + local consolidative therapy (SBRT/surgery)

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Algorithm E10.3.1 · Advanced NSCLC — first-line decision



Small-cell lung cancer

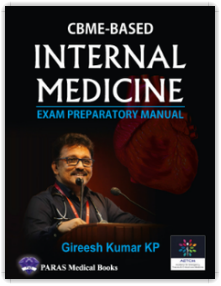
- **Limited stage (T any N any M0, encompassed by one radiation field):** concurrent cisplatin–etoposide + thoracic RT (45 Gy BD); consolidation durvalumab (ADRIATIC 2024); PCI or MRI surveillance.
- **Extensive stage:** carboplatin–etoposide + atezolizumab (IMpower133) or durvalumab (CASPIAN); tarlatamab (DLL3 BiTE) at relapse (DeLLphi-304, 2025).
- Very chemosensitive; relapse is common; median survival ≈ 12–15 months in extensive stage.

★ CLINICAL PEARLS & EXAM FOCUS

- Immune-related adverse events: pneumonitis (grade ≥ 2 → hold, prednisolone 1–2 mg/kg), colitis, hepatitis, thyroiditis, hypophysitis.
- EGFR-mutant tumours respond poorly to immunotherapy; avoid IO before osimertinib (pneumonitis risk).

MERCK MANUAL PROFESSIONAL · Lung carcinoma · Small cell lung cancer

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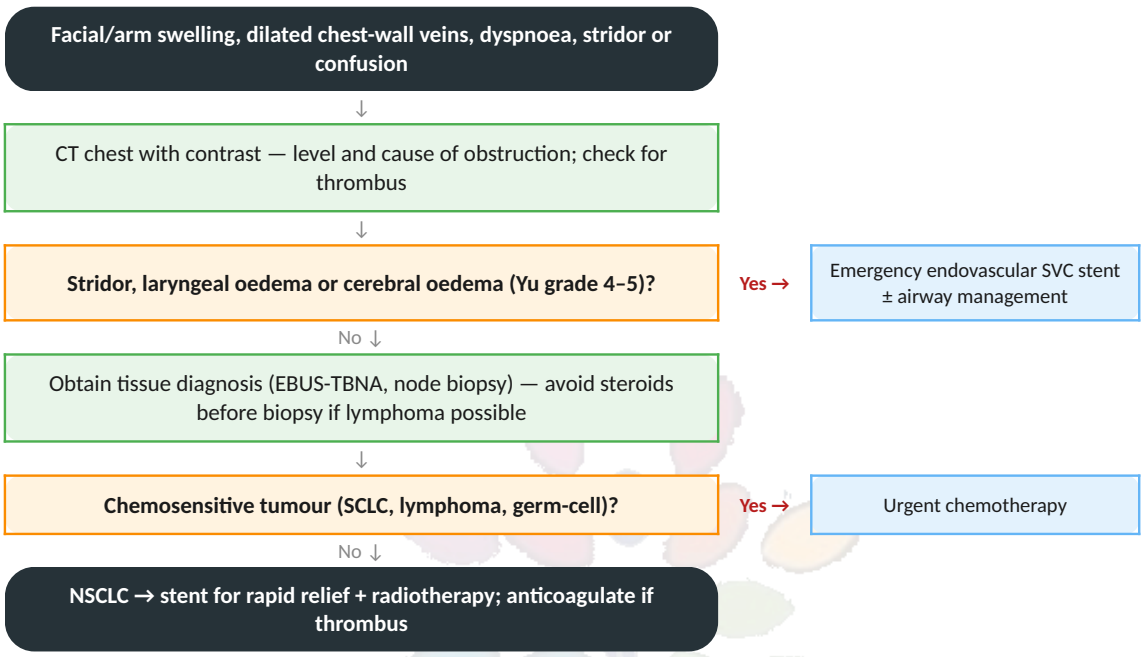
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E10.4 Thoracic Oncological Emergencies — SVCO, Central Airway Obstruction & Paraneoplastic Crises

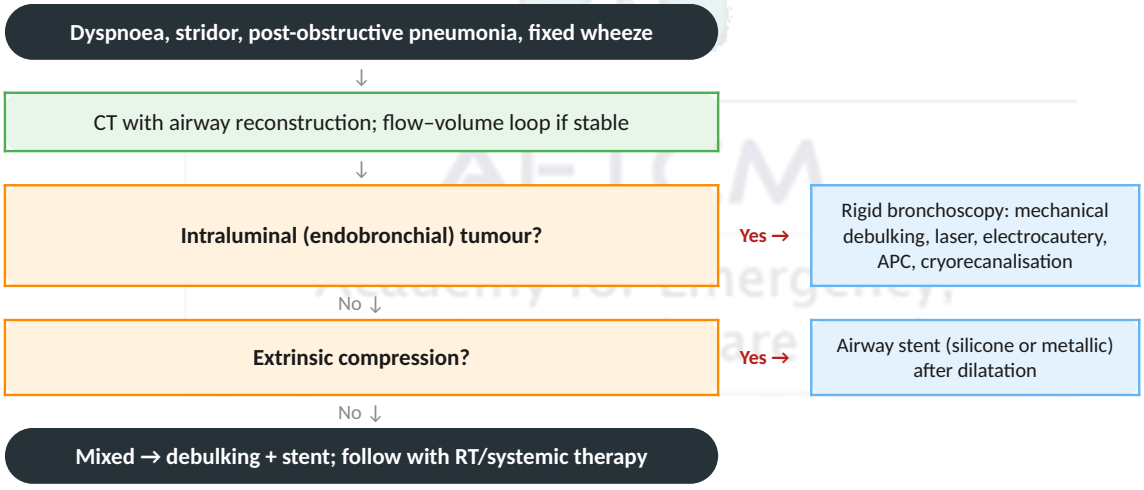
‹ Core topic 10.9 · p.193
Previous-year Qs ›

IN BRIEF Oncological emergencies are frequently the first presentation of lung cancer. Rapid recognition and a tissue diagnosis before steroids or radiotherapy (when safe) give the best outcomes.

Algorithm E10.4.1 · Superior vena cava obstruction



Algorithm E10.4.2 · Malignant central airway obstruction



Paraneoplastic syndromes of lung cancer

Syndrome	Tumour	Clue
SIADH (hyponatraemia)	SCLC	Euvolaemic hyponatraemia, low serum osmolality
Hypercalcaemia (PTHrP)	Squamous cell	Confusion, constipation, AKI
Ectopic ACTH	SCLC, carcinoid	Hypokalaemic alkalosis, pigmentation, weakness
Lambert–Eaton syndrome	SCLC	Proximal weakness improving with exercise; VGCC antibodies
Hypertrophic pulmonary osteoarthropathy	Adenocarcinoma, squamous	Clubbing, painful periostitis
Paraneoplastic cerebellar degeneration, encephalitis	SCLC	Anti-Hu, anti-Yo antibodies
Trousseau syndrome	Adenocarcinoma	Migratory thrombophlebitis

★ CLINICAL PEARLS & EXAM FOCUS

- Hypercalcaemia treatment: IV saline 3–4 L/day, zoledronic acid 4 mg IV, calcitonin for rapid effect, denosumab in renal failure.
- Correct chronic SIADH hyponatraemia no faster than 8–10 mmol/L in 24 h (osmotic demyelination).

RADIOPAEDIA · IMAGE LINKS Superior vena cava obstruction cases Endobronchial stent cases

MERCK MANUAL PROFESSIONAL Superior vena cava syndrome Paraneoplastic syndromes

E10.5 Lung Cancer Screening & Prevention

‹ Core topic 10.1 · p.181
Previous-year Qs ›

IN BRIEF Low-dose CT (LDCT) screening reduces lung cancer mortality by 20–24% in high-risk smokers. India has not yet adopted population screening; high TB prevalence complicates interpretation.

Screening evidence and eligibility

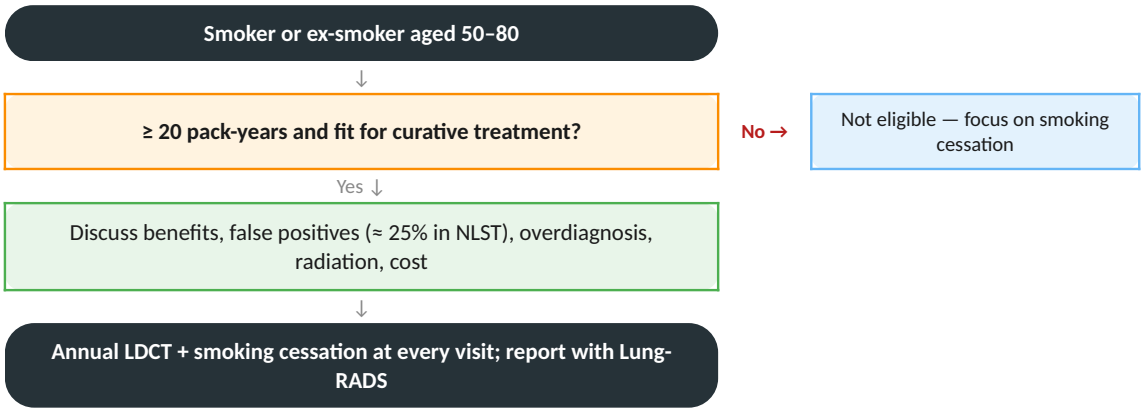
Item	Detail
NLST (2011)	LDCT vs CXR — 20% reduction in lung cancer mortality
NELSON (2020)	24% reduction in men at 10 years
USPSTF 2021	Age 50–80, ≥ 20 pack-years, current smoker or quit within 15 years — annual LDCT
ACS 2023	Removed years-since-quitting criterion
Lung-RADS v2022	Categories 0–4X standardise reporting and follow-up

Lung-RADS categories (simplified)

Category	Meaning	Management
1	Negative	Annual LDCT
2	Benign appearance	Annual LDCT
3	Probably benign	LDCT in 6 months
4A	Suspicious	LDCT in 3 months; PET-CT if solid component ≥ 8 mm
4B / 4X	Very suspicious	Chest CT ± PET-CT, tissue sampling

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Algorithm E10.5.1 · Shared decision-making for screening



★ CLINICAL PEARLS & EXAM FOCUS

- Smoking cessation intervention at the screening visit is mandatory — it is the most cost-effective element.
- Screening in India would need to account for TB nodules and non-smoker (biomass, air pollution) risk.

RADIOPAEDIA · IMAGE LINKS Lung-RADS cases Low dose CT lung cancer screening cases

MERCK MANUAL PROFESSIONAL Lung cancer screening

E10.6 Drug Chart — Systemic Therapy in Thoracic Oncology

Previous-year Qs ›

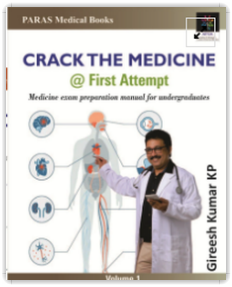
IN BRIEF For orientation in exams and clinics — regimens are prescribed by oncologists after MDT discussion.

Common thoracic oncology drugs

Class	Drug	Dose (usual)	Indication	Caution
Platinum	Cisplatin / Carboplatin	75 mg/m² / AUC 5 every 3 weeks	Backbone for NSCLC and SCLC doublets	Cisplatin: nephro-, oto-, neurotoxicity, emesis; carboplatin: thrombocytopenia
Antifolate	Pemetrexed	500 mg/m² every 3 weeks + folic acid + B12	Non-squamous NSCLC, mesothelioma	Myelosuppression; avoid if CrCl < 45
Taxane	Paclitaxel / Docetaxel	175–200 / 75 mg/m² every 3 weeks	NSCLC	Neuropathy, hypersensitivity (premedicate), neutropenia
Topoisomerase II inhibitor	Etoposide	100 mg/m² days 1–3	SCLC	Myelosuppression
PD-1 inhibitor	Pembrolizumab / Nivolumab	200 mg 3-weekly (400 mg 6-weekly) / 240 mg 2-weekly	NSCLC (PD-L1), perioperative	Immune-related pneumonitis, colitis, endocrinopathies
PD-L1 inhibitor	Atezolizumab / Durvalumab	1200 mg 3-weekly / 10 mg/kg 2-weekly (1500 mg 4-weekly)	NSCLC, SCLC, PACIFIC consolidation	Same
EGFR TKI (3rd generation)	Osimertinib	80 mg OD	EGFR-mutant NSCLC (adjuvant, stage III, IV)	ILD (stop), QT, cardiomyopathy

Class	Drug	Dose (usual)	Indication	Caution
EGFR TKI (1st/2nd generation)	Gefitinib / Erlotinib / Afatinib	250 mg / 150 mg / 40 mg OD	EGFR-mutant (cost-effective in India)	Rash, diarrhoea, paronychia
ALK inhibitor	Alectinib / Lorlatinib	600 mg BD / 100 mg OD	ALK-positive NSCLC	Lorlatinib: hyperlipidaemia, CNS effects
Anti-VEGF	Bevacizumab	15 mg/kg 3-weekly	Non-squamous NSCLC	Haemoptysis (avoid in squamous, cavities), hypertension
KRAS G12C inhibitor	Sotorasib	960 mg OD	KRAS G12C NSCLC (2nd line)	Hepatotoxicity
Bispecific antibody	Amivantamab	IV weight-based	EGFR exon 20 insertion, with lazertinib	Infusion reactions, rash
Bispecific T-cell engager	Tarlatamab	IV step-up	Relapsed SCLC	Cytokine release syndrome, ICANS
Bisphosphonate	Zoledronic acid	4 mg IV	Hypercalcaemia, bone metastases	Renal, osteonecrosis of jaw
RANKL antibody	Denosumab	120 mg SC 4-weekly	Bone metastases	Hypocalcaemia
Vasopressin antagonist	Tolvaptan	15 mg OD	SIADH hyponatraemia	Overly rapid correction, liver injury
Corticosteroid	Dexamethasone	8–16 mg/day	Brain metastases oedema, SVCO (after biopsy), cord compression	Hyperglycaemia

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E10.7 Immunotherapy Toxicity & Thoracic Radiation Injury

‹ Core topic 10.5 · p.187
Previous-year Qs ›

IN BRIEF Checkpoint inhibitors and thoracic radiotherapy have created a new group of iatrogenic lung diseases. Pneumonitis is the one that kills.

Grading and management of checkpoint-inhibitor pneumonitis (CTCAE / ASCO)

Grade	Features	Management
1	Radiographic changes only, asymptomatic	Hold immunotherapy; repeat imaging in 3–4 weeks; monitor
2	Symptomatic, limits instrumental activities	Hold drug; prednisolone 1–2 mg/kg/day, taper over 4–6 weeks; bronchoscopy to exclude infection
3	Severe symptoms, oxygen needed, limits self-care	Admit; permanently discontinue; methylprednisolone 2 mg/kg/day IV
4	Life-threatening, ventilatory support	ICU; pulse steroids; add infliximab, mycophenolate or IVIG if no improvement at 48 h

Other immune-related adverse events relevant to the chest

Event	Clue
Thyroiditis / hypothyroidism	Fatigue, abnormal TSH — commonest irAE
Hypophysitis	Headache, hyponatraemia, low cortisol
Colitis	Diarrhoea — steroids, infliximab
Myocarditis	Troponin rise, arrhythmia — high mortality, pulse steroids
Sarcoid-like reaction	Hilar nodes and nodules — may mimic progression (pseudoprogression)

Radiation-induced lung injury

Phase	Timing	Features	Management
Radiation pneumonitis	4–12 weeks after RT	Cough, dyspnoea, fever; GGO/consolidation conforming to the radiation port (straight edges crossing fissures)	Prednisolone 0.5–1 mg/kg tapered over 4–8 weeks in symptomatic patients
Radiation fibrosis	6–24 months	Volume loss, traction bronchiectasis within the port	Supportive; rehabilitation, oxygen
Radiation recall	After chemotherapy given later	Pneumonitis reappears in the old field	Steroids; stop the trigger drug
Out-of-field ("sporadic" organising pneumonia)	Months	Migratory consolidation beyond the port, often after breast RT	Steroids

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Algorithm E10.7.1 · New infiltrates in a patient on cancer therapy

New breathlessness and infiltrates during cancer treatment

↓

CT chest; oxygen saturation at rest and on walking; CBC, CRP, procalcitonin; blood and sputum cultures; consider COVID/influenza PCR

↓

Features of infection (fever, purulent sputum, neutropenia, focal consolidation)?

Yes → Treat as febrile neutropenia / pneumonia; bronchoscopy with BAL if immunocompromised

No ↓

On a checkpoint inhibitor with bilateral GGO/organising pattern and negative microbiology?

Yes → Immune-related pneumonitis — grade and treat as above

No ↓

Infiltrate conforms to the radiation field?

Yes → Radiation pneumonitis — steroids if symptomatic

No ↓

Also consider lymphangitic carcinomatosis, drug toxicity (bleomycin, gemcitabine), cardiac failure, PE, diffuse alveolar haemorrhage

★ CLINICAL PEARLS & EXAM FOCUS

- Always exclude infection before (or alongside) starting steroids for pneumonitis — BAL is usually justified.
- Lymphangitic carcinomatosis shows nodular interlobular septal thickening with preserved lung architecture — distinguishes it from pneumonitis.

RADIOPAEDIA · IMAGE LINKS Radiation pneumonitis cases · Immune checkpoint inhibitor pneumonitis cases · Lymphangitic carcinomatosis cases

MERCK MANUAL PROFESSIONAL Lung carcinoma

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PART II · SECTION 11

Pleura, Mediastinum, Diaphragm & Chest Wall

Advanced theory & algorithms

E11.1 Pleural Effusion — Diagnostic Algorithm Beyond Light's Criteria 439
E11.2 Pleural Infection & Empyema — BTS 2023 Algorithm & MIST-2 440
E11.3 Malignant Pleural Effusion & Pneumothorax — Management Algorithms 442
E11.4 Chylothorax, Pseudochylothorax, Haemothorax & Mesothelioma 444
E11.5 Diaphragm, Mediastinitis & Chest Wall — Clinical Algorithms 445
E11.6 Drug Chart — Drugs Used in Pleural Disease 447
E11.7 Pneumothorax — Physiology, Special Situations & Recurrence Prevention 448

E11.1 Pleural Effusion — Diagnostic Algorithm Beyond Light's Criteria

‹ Core topic 11.1 · p.196
Previous-year Qs ›

IN BRIEF Light's criteria separate exudates from transudates with ≈ 98% sensitivity but misclassify about a quarter of transudates on diuretics as exudates. Use corrective tests, then let cell counts and biochemistry guide the next step.

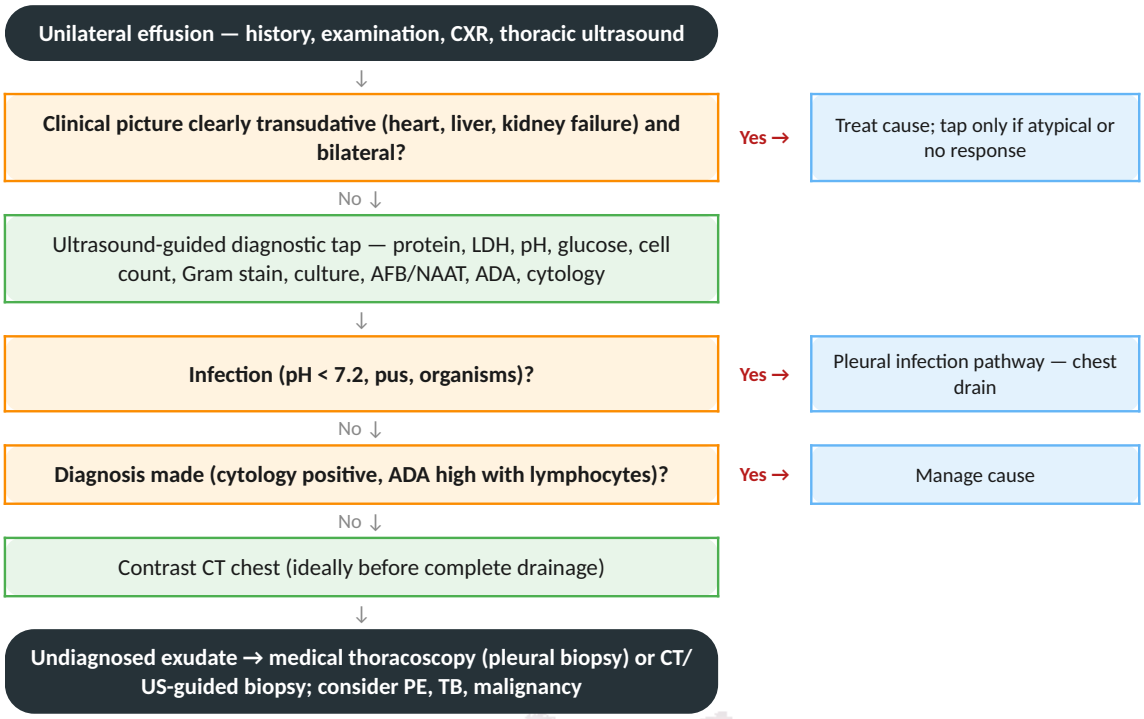
Light's criteria — exudate if any one is present

Criterion	Threshold
Pleural fluid protein / serum protein	> 0.5
Pleural fluid LDH / serum LDH	> 0.6
Pleural fluid LDH	> 2/3 of the upper limit of normal serum LDH
On diuretics with "borderline exudate"	Serum–pleural albumin gradient > 1.2 g/dL or protein gradient > 3.1 g/dL → transudate; NT-proBNP > 1500 pg/mL supports heart failure

Pleural fluid tests and what they mean

Test	Interpretation
Appearance	Pus (empyema), milky (chylothorax — TG > 110 mg/dL; pseudochylothorax — cholesterol > 200, crystals), bloody (malignancy, trauma, PE; haematocrit > 50% of blood = haemothorax)
pH	< 7.2 in parapneumonic effusion → drain; also low in TB, malignancy, RA, oesophageal rupture
Glucose	< 60 mg/dL: empyema, RA (< 30), TB, malignancy, lupus
ADA	> 40 IU/L: TB (also empyema, lymphoma, RA)
Amylase	Pancreatitis, oesophageal rupture (salivary), malignancy
Cytology	Positive in ≈ 60% of malignant effusions (higher with two samples)
Cell count	Neutrophils: parapneumonic, PE, pancreatitis; lymphocytes > 50%: TB, malignancy, lymphoma, cardiac surgery; eosinophils > 10%: air/blood in pleura, drugs, parasites, asbestos

Algorithm E11.1.1 · Investigating a unilateral pleural effusion (BTS 2023)



★ CLINICAL PEARLS & EXAM FOCUS

- Always do thoracic ultrasound before tapping — reduces pneumothorax and improves yield.
- Septations on ultrasound suggest exudate; homogeneous echogenic fluid suggests haemorrhage or empyema.

RADIOPAEDIA · IMAGE LINKS Pleural effusion cases · Meniscus sign cases · Subpulmonic effusion cases

AETCM YOUTUBE · VIDEO LECTURES ▶ pleural effusion · ▶ thoracentesis · ▶ pleural tap

MERCK MANUAL PROFESSIONAL Pleural effusion

E11.2 Pleural Infection & Empyema — BTS 2023 Algorithm & MIST-2

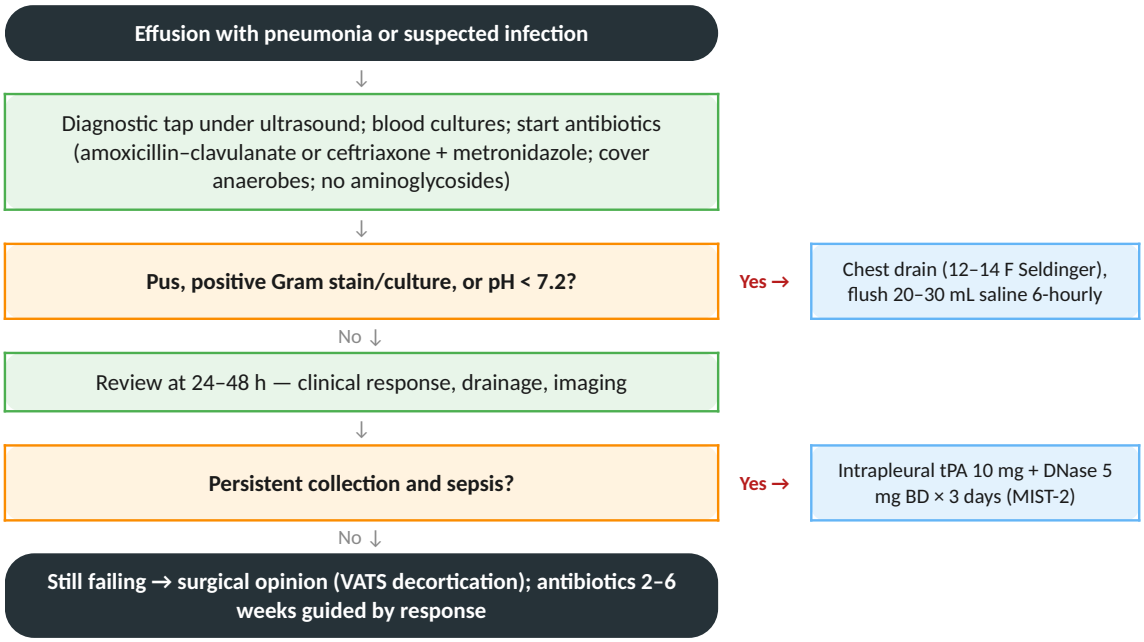
‹ Core topic 11.2 · p.198
Previous-year Qs ›

IN BRIEF Pleural infection needs early drainage, appropriate antibiotics and — for non-draining loculated collections — intrapleural tPA–DNase. The RAPID score predicts mortality.

Light's classification of parapneumonic effusion (simplified)

Type	Features	Action
Simple parapneumonic	pH > 7.2, glucose > 60, LDH < 1000, no organisms	Antibiotics; tap if large
Complicated parapneumonic	pH < 7.2 or glucose < 60 or positive Gram stain/culture; loculations	Chest drain
Empyema	Frank pus	Chest drain ± fibrinolytics/surgery

Algorithm E11.2.1 · Management of pleural infection



RAPID score

Variable	0	1	2
Renal — urea (mmol/L)	< 5	5–8	> 8
Age	< 50	50–70	> 70
Purulence of fluid	Purulent	Non-purulent	—
Infection source	Community	Hospital	—
Dietary — albumin (g/L)	≥ 27	< 27	—
0–2 low risk; 3–4 medium; 5–7 high (3-month mortality ≈ 30%)	—	—	—

LANDMARK TRIALS & GUIDELINES

- **MIST-1:** streptokinase alone no benefit. **MIST-2 (2011):** tPA + DNase improved drainage and reduced surgical referral; either alone ineffective.
- **MIST-3 (2024):** early VATS vs early fibrinolytics feasible; no clear winner.

★ CLINICAL PEARLS & EXAM FOCUS

- Anaerobes and Streptococcus anginosus group dominate community empyema; S. aureus and gram-negatives in hospital-acquired.
- "Split pleura sign" on contrast CT = enhancing visceral and parietal pleura separated by fluid — empyema.

RADIOPAEDIA · IMAGE LINKS Empyema cases Split pleura sign cases

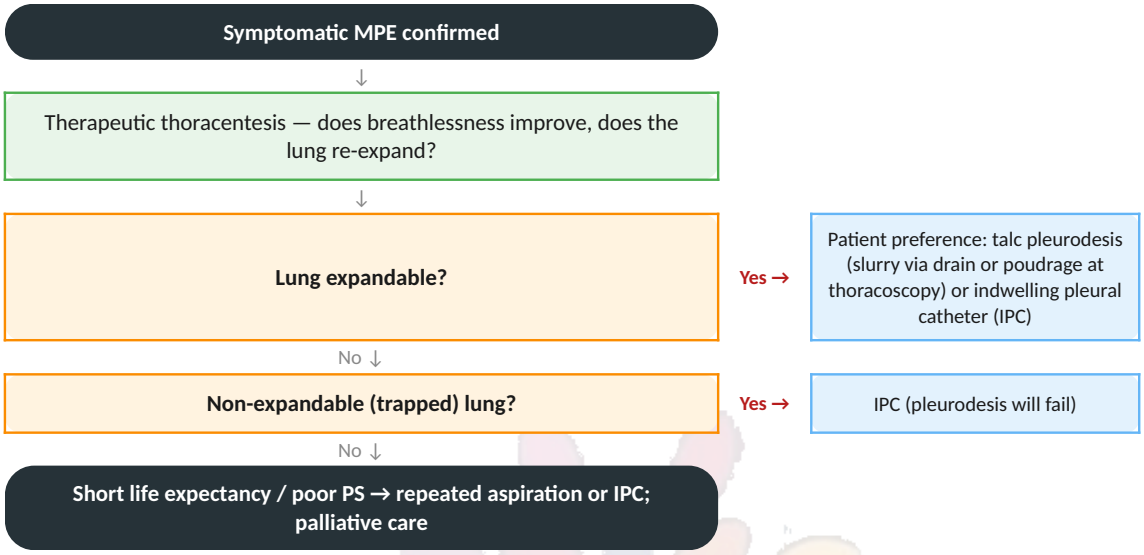
MERCK MANUAL PROFESSIONAL Empyema

E11.3 Malignant Pleural Effusion & Pneumothorax — Management Algorithms

‹ Core topic 11.3 · p.200
Previous-year Qs ›

IN BRIEF MPE indicates advanced disease; the goals are symptom relief and fewer procedures. Pneumothorax management has moved towards conservative and ambulatory care for stable patients (BTS 2023).

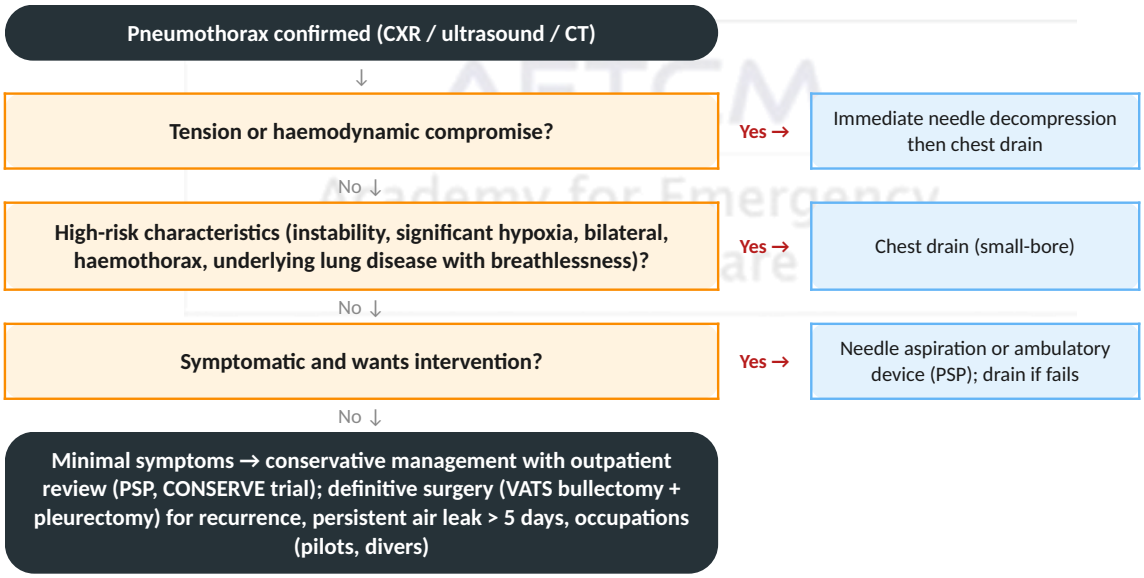
Algorithm E11.3.1 · Malignant pleural effusion



LANDMARK TRIALS & GUIDELINES

- **TIME2:** IPC vs talc — similar breathlessness relief; IPC fewer days in hospital.
- **AMPLE-2:** daily IPC drainage gives higher spontaneous pleurodesis.
- **IPC-PLUS:** talc through IPC improved pleurodesis rate.

Algorithm E11.3.2 · Spontaneous pneumothorax (BTS 2023)



★ CLINICAL PEARLS & EXAM FOCUS

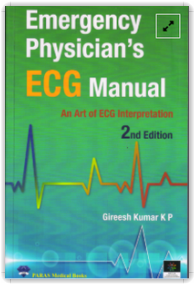
- Secondary pneumothorax (COPD, ILD, TB) is more dangerous — usually needs a drain and admission.
- No flying for 1 week after full resolution; diving is barred for life unless bilateral surgical pleurectomy.

RADIOPAEDIA · IMAGE LINKS Pneumothorax cases | Deep sulcus sign cases | Tension pneumothorax cases

AETCM YOUTUBE · VIDEO LECTURES ▶ pneumothorax | ▶ intercostal drain | ▶ chest tube

MERCK MANUAL PROFESSIONAL Pneumothorax | Malignant pleural effusion

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E11.4 Chylothorax, Pseudochylothorax, Haemothorax & Mesothelioma

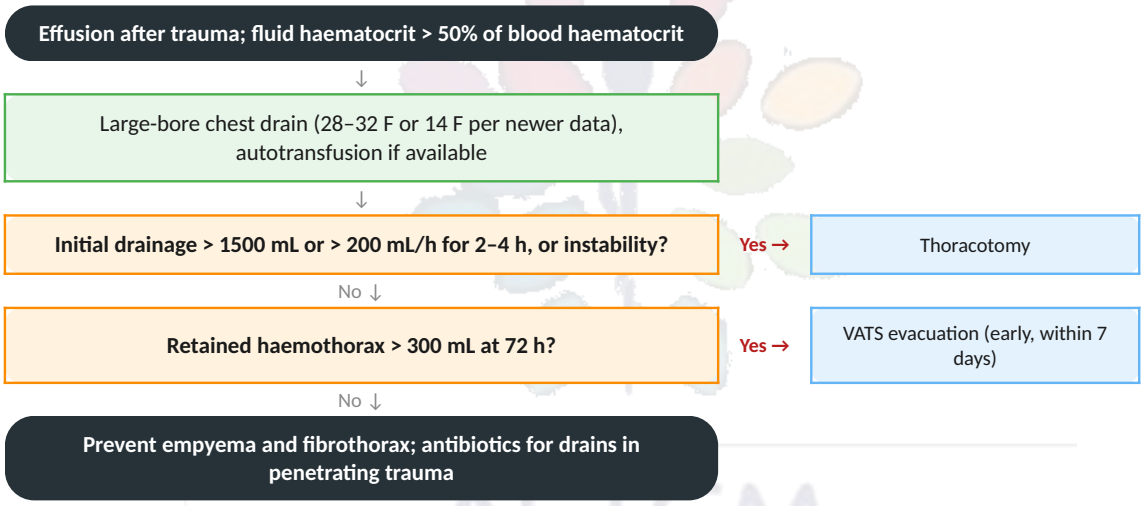
‹ Core topic 11.5 · p.203
Previous-year Qs ›

IN BRIEF Milky fluid is not always chyle — measure triglycerides and cholesterol. Haemothorax is defined by haematocrit. Mesothelioma is diagnosed by thoracoscopic biopsy.

Chylothorax vs pseudochylothorax

Feature	Chylothorax	Pseudochylothorax (cholesterol effusion)
Cause	Thoracic duct injury — surgery, trauma, lymphoma, LAM, TB	Long-standing effusion — TB, rheumatoid, trapped lung
Onset	Acute	Chronic (years), thickened pleura
Triglycerides	> 110 mg/dL; chylomicrons present	< 50 mg/dL
Cholesterol	< 200 mg/dL	> 200 mg/dL; cholesterol crystals
Treatment	Drain, low-fat/MCT diet or TPN, octreotide; thoracic duct ligation/ embolisation	Treat cause; decortication if symptomatic

Algorithm E11.4.1 · Traumatic haemothorax



Malignant pleural mesothelioma

- **Presentation:** chest pain, breathlessness, unilateral effusion, nodular circumferential pleural thickening, volume loss; asbestos exposure in 80%.
- **Diagnosis:** thoracoscopic pleural biopsy (cytology insensitive); IHC — calretinin, WT-1, D2-40 positive; BAP1 loss, CDKN2A deletion.
- **Subtypes:** epithelioid (best prognosis), sarcomatoid, biphasic.
- **Treatment:** nivolumab + ipilimumab first-line (CheckMate 743 — greater benefit in non-epithelioid); pemetrexed–platinum ± bevacizumab; pleurectomy/decortication in selected (MARS-2 showed harm from extended surgery).

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★ CLINICAL PEARLS & EXAM FOCUS

- Port-site metastasis after thoracoscopy is rare; prophylactic radiotherapy is not recommended (SMART, PIT).
- Circumferential pleural thickening, mediastinal pleural involvement, nodularity and thickness > 1 cm = Leung's criteria for malignancy on CT.

RADIOPAEDIA · IMAGE LINKS Chylothorax cases Mesothelioma cases Haemothorax cases

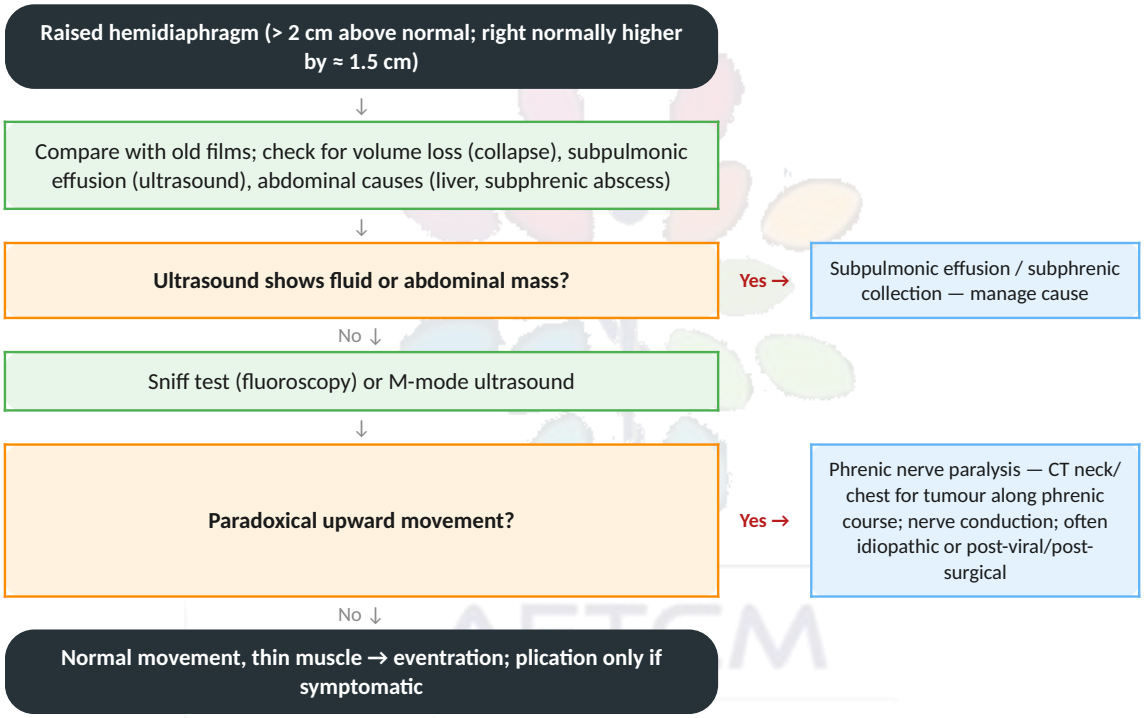
MERCK MANUAL PROFESSIONAL Chylothorax Mesothelioma

E11.5 Diaphragm, Mediastinitis & Chest Wall — Clinical Algorithms

‹ Core topic 11.8 · p.207
Previous-year Qs ›

IN BRIEF An elevated hemidiaphragm, a widened mediastinum and a deformed chest wall each have a short differential. A structured approach saves needless investigations.

Algorithm E11.5.1 · Elevated hemidiaphragm on CXR



Acute mediastinitis

Feature	Detail
Causes	Oesophageal perforation (Boerhaave, endoscopy), post-sternotomy, descending necrotising infection from neck/dental
Signs	Sepsis, chest pain, Hamman's crunch, subcutaneous emphysema
Imaging	CT — fluid collections, gas, pleural effusion
Treatment	Broad-spectrum antibiotics, surgical drainage/debridement, repair of perforation

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Chest wall disorders and ventilatory failure

Disorder	Key point
Kyphoscoliosis	Cobb angle > 100° → respiratory failure; nocturnal NIV
Thoracoplasty (post-TB)	Late hypercapnic failure; NIV
Flail chest	≥ 3 adjacent ribs fractured at 2 places; analgesia (epidural/serratus block), ventilation if needed, surgical fixation in selected
Ankylosing spondylitis	Upper-lobe fibrobullous disease; restriction
Pectus excavatum	Usually cosmetic; Haller index > 3.25 for surgery

★ CLINICAL PEARLS & EXAM FOCUS

- Bilateral diaphragm paralysis: orthopnoea in a patient with clear lungs — check supine VC.
- Boerhaave: left-sided effusion with high amylase and low pH after vomiting — surgical emergency.

RADIOPAEDIA · IMAGE LINKS Diaphragmatic paralysis cases | Eventration of the diaphragm cases | Pneumomediastinum cases

MERCK MANUAL PROFESSIONAL Mediastinitis | Diaphragmatic paralysis

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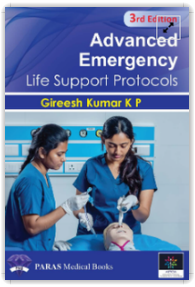
E11.6 Drug Chart — Drugs Used in Pleural Disease

Previous-year Qs ›

Pleural pharmacology

Class	Drug	Dose	Indication	Caution
Sclerosant	Sterile graded talc	4–5 g slurry via drain or poudrage at thoracoscopy	Pleurodesis in MPE, recurrent pneumothorax	ARDS with ungraded small-particle talc; pain, fever
Sclerosant (alternative)	Doxycycline / Bleomycin / Povidone-iodine	500 mg / 60 IU / 20 mL of 10%	Pleurodesis where talc unavailable	Pain; bleomycin systemic absorption
Autologous blood patch	Patient's own blood	50–100 mL via drain	Persistent air leak	Infection, clot obstructing drain
Fibrinolytic	Alteplase (tPA)	10 mg intrapleural BD (5 mg in some protocols)	Pleural infection, with DNase	Bleeding (pleural) — rare
Mucolytic enzyme	Dornase alfa (DNase)	5 mg intrapleural BD × 3 days	Pleural infection with tPA	Not alone
β-lactam/β-lactamase inhibitor	Amoxicillin–clavulanate	1.2 g IV TDS → 625 mg TDS oral	Community pleural infection	—
Nitroimidazole	Metronidazole	500 mg IV/oral TDS	Anaerobic cover with cephalosporin	Disulfiram reaction
Somatostatin analogue	Octreotide	50–100 µg SC TDS	Chylothorax	Gallstones, hyperglycaemia
Local anaesthetic (intrapleural)	Lidocaine 1%	3 mg/kg before talc	Pain at pleurodesis	Toxicity
Analgesic	NSAIDs	Ibuprofen 400 mg TDS	Pleuritic pain; do not reduce pleurodesis success (TIME1)	GI bleeding, renal
Analgesic	Opioids (morphine)	2.5–5 mg oral 4-hourly	Pleural pain, MPE dyspnoea	Constipation, sedation
Diuretic	Furosemide / Spironolactone	40 mg / 100 mg (ratio 40:100)	Hepatic hydrothorax, cardiac effusions	Electrolytes, renal function
Checkpoint inhibitors	Nivolumab + Ipilimumab	3 mg/kg 2-weekly + 1 mg/kg 6-weekly	Mesothelioma	Immune-related adverse events

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E11.7 Pneumothorax — Physiology, Special Situations & Recurrence Prevention

‹ Core topic 11.6 · p.204
Previous-year Qs ›

IN BRIEF Beyond the acute algorithm, examiners ask about catamenial, iatrogenic and traumatic pneumothorax, persistent air leak and the rules for flying and diving.

Classification

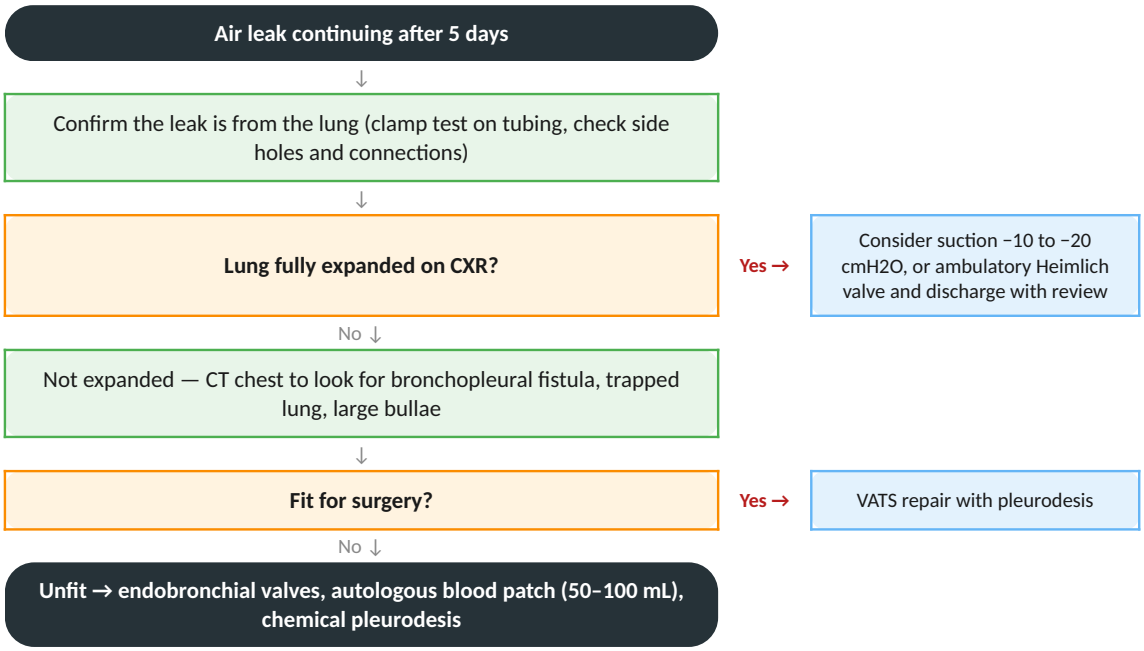
Type	Features
Primary spontaneous (PSP)	Tall thin young men, smokers; apical blebs; recurrence ≈ 30% at 1 year, 50% after a second episode
Secondary spontaneous (SSP)	COPD, TB, ILD, cystic fibrosis, PJP, LAM, PLCH, Marfan; more symptomatic, needs admission and usually a drain
Traumatic	Blunt or penetrating; often with haemothorax
Iatrogenic	Subclavian line, pleural tap, transbronchial or transthoracic biopsy, barotrauma on ventilation
Catamenial	Within 72 h of menstruation, right-sided, thoracic endometriosis; hormonal therapy + VATS
Tension	Any type — a clinical diagnosis requiring immediate decompression

Preventing recurrence

Intervention	Comment
VATS bullectomy + pleurectomy or pleural abrasion	Lowest recurrence (≈ 1–5%) — procedure of choice
Chemical pleurodesis (talc) through the drain	For patients unfit for surgery; recurrence ≈ 10–20%
Smoking cessation	Reduces recurrence substantially — the single most important advice
Indications for definitive intervention	Second ipsilateral or first contralateral pneumothorax, bilateral, persistent air leak > 5 days, tension, haemopneumothorax, high-risk occupation (pilot, diver), lack of access to care

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Algorithm E11.7.1 · Persistent air leak (bubbling beyond day 5)



Advice after pneumothorax

- **Flying:** wait at least 7 days after radiological resolution (1 week after drain removal and confirmed expansion); airlines may require longer.
- **Diving:** permanently prohibited after spontaneous pneumothorax unless bilateral surgical pleurectomy has been performed and lung function and CT are normal.
- **Smoking:** lifetime risk of recurrence falls markedly on stopping — document the advice.

★ **CLINICAL PEARLS & EXAM FOCUS**

- Tension pneumothorax is treated before imaging; in a ventilated patient the first sign is a sudden rise in airway pressure with hypotension.
- In secondary pneumothorax, even a small rim of air can cause severe symptoms — admit and usually drain.

RADIOPAEDIA · IMAGE LINKS Primary spontaneous pneumothorax cases · Catamenial pneumothorax cases · Bronchopleural fistula cases

MERCK MANUAL PROFESSIONAL Pneumothorax

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PART II · SECTION 12

Critical Care Pulmonary Medicine

Advanced theory & algorithms

E12.1 Acute Hypoxaemic Respiratory Failure — Oxygen, HFNC & NIV Decision Algorithm 450
E12.2 Mechanical Ventilation — Initial Settings & Waveform Troubleshooting 451
E12.3 ARDS — Berlin to Global Definition & Management Algorithm 454
E12.4 Weaning, Spontaneous Breathing Trials & Extubation Failure 457
E12.5 Sepsis & Septic Shock — Surviving Sepsis Campaign Bundle 458
E12.6 Difficult Airway — Prediction & Unanticipated Difficult Intubation Algorithm 459
E12.7 Respiratory ICU Monitoring — Capnography, Lung Ultrasound & Haemodynamics 461
E12.8 Drug Chart — Respiratory ICU Drugs 462
E12.9 Ventilation in Obstructive Airway Disease — Auto-PEEP & Permissive Hypercapnia 463
E12.10 Extracorporeal Support & Rescue Therapies in Respiratory Failure 465
E12.11 Non-Invasive Ventilation — Modes, Interfaces & Setting Up at the Bedside 466

E12.1 Acute Hypoxaemic Respiratory Failure — Oxygen, HFNC & NIV Decision Algorithm

‹ Core topic 12.1 · p.211
Previous-year Qs ›

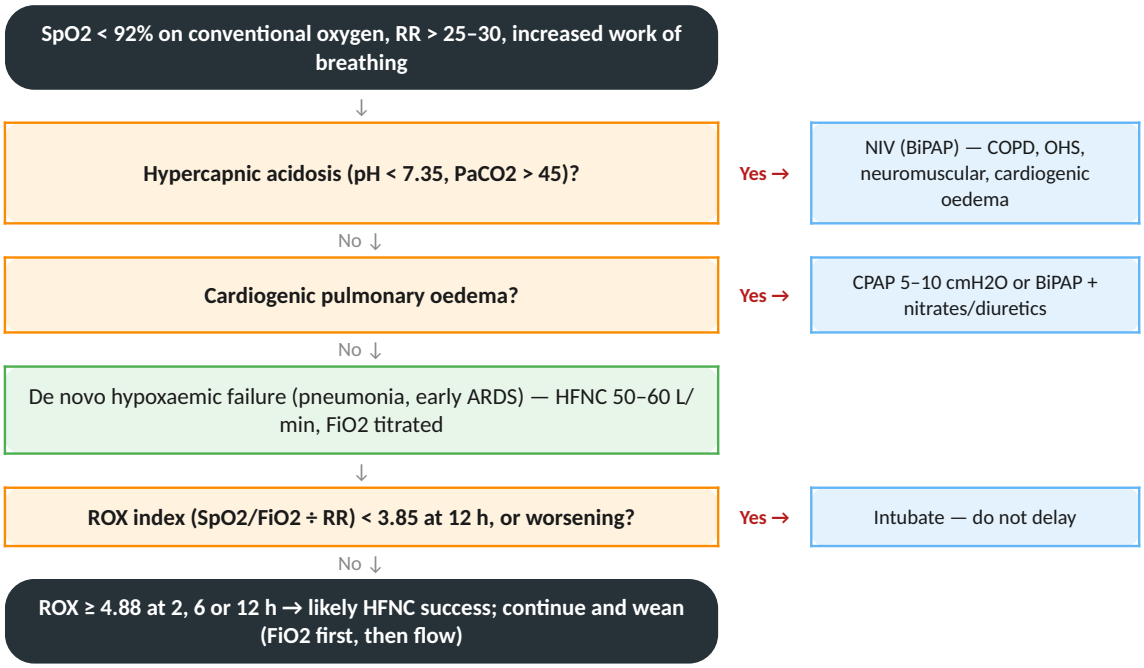
IN BRIEF Choose the least invasive support that corrects the problem — but recognise failure early. Delayed intubation after failed HFNC or NIV increases mortality.

Oxygen targets

Patient	Target SpO2
Most acutely ill adults	94–98% (BTS); avoid hyperoxia (> 96% liberal harm in IOTA meta-analysis)
Risk of hypercapnia (COPD, OHS, neuromuscular, chest wall)	88–92%
Critically ill on ventilator	92–96% (88–92% acceptable in ARDS)

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Algorithm E12.1.1 · Choosing respiratory support in acute hypoxaemic failure



High-flow nasal cannula — physiology

Effect	Mechanism
Precise high FiO2	Flow exceeds peak inspiratory flow
Dead-space washout	Flushes nasopharyngeal CO2
Low-level PEEP	≈ 1 cmH2O per 10 L/min with mouth closed
Reduced work of breathing	Warm humidified gas, better mucociliary clearance, comfort

LANDMARK TRIALS & GUIDELINES

- **FLORALI (2015):** HFNC reduced 90-day mortality vs NIV/standard O2 in hypoxaemic failure (PaO2/FiO2 ≤ 200 subgroup).
- **HENIVOT, RECOVERY-RS:** helmet NIV / CPAP in COVID-19 reduced intubation.
- **HIGH-WEAN, Hernández:** HFNC post-extubation reduces reintubation in low-risk patients.

★ CLINICAL PEARLS & EXAM FOCUS

- An expired tidal volume > 9.5 mL/kg on NIV in de novo hypoxaemic failure predicts failure (patient self-inflicted lung injury — P-SILI).
- HACOR score (heart rate, acidosis, consciousness, oxygenation, RR) > 5 at 1 h predicts NIV failure.

Trauma & Critical care Medicine

MERCK MANUAL PROFESSIONAL — Acute hypoxemic respiratory failure | Oxygen therapy

E12.2 Mechanical Ventilation — Initial Settings & Waveform Troubleshooting

‹ Core topic 12.4 · p.216
Previous-year Qs ›

IN BRIEF Start with lung-protective settings for everyone, adjust to the disease, and read the waveforms — they tell you whether the patient and the ventilator are breathing together.

AETCM ONLINE RESOURCE AETCM Mechanical Ventilation module ↗

Initial ventilator settings by condition

Setting	ARDS / normal lungs	Obstructive (asthma/COPD)	Neuromuscular
Mode	Volume control (AC)	Volume control	Volume or pressure control
Tidal volume	6 mL/kg PBW (4–8)	6–8 mL/kg PBW	6–8 mL/kg PBW
Rate	16–24 (up to 35)	8–12 (long expiration)	12–16
PEEP	5–15+ per ARDSNet table	3–5 (≤ 80% of intrinsic PEEP in COPD)	5
Flow / I:E	60 L/min, I:E 1:2	80–100 L/min, I:E 1:4–1:5	Standard
Plateau target	≤ 30 cmH2O; driving pressure ≤ 15	≤ 30; monitor auto-PEEP	≤ 30
FiO2	To SpO2 88–95%	To SpO2 88–92%	Lowest

- **Predicted body weight:** men 50 + 0.91 (height cm − 152.4); women 45.5 + 0.91 (height cm − 152.4).

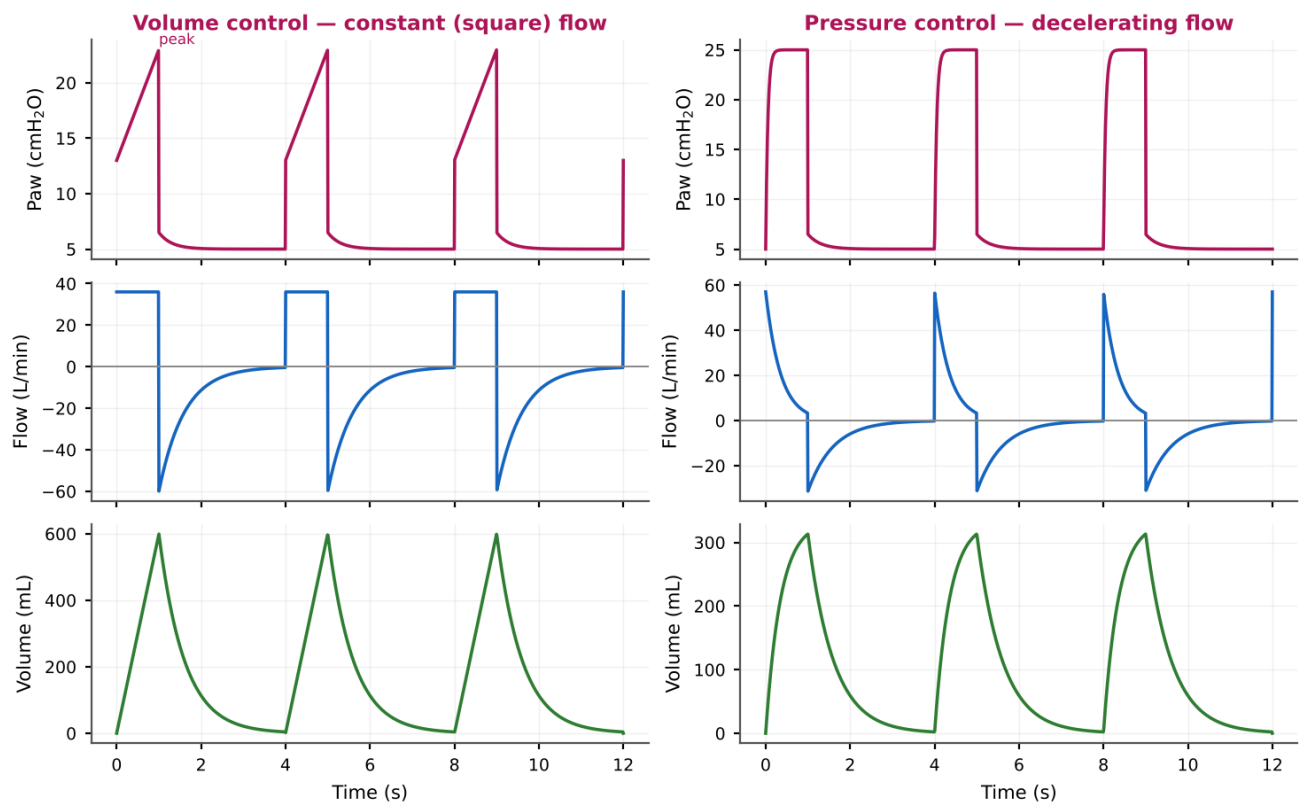
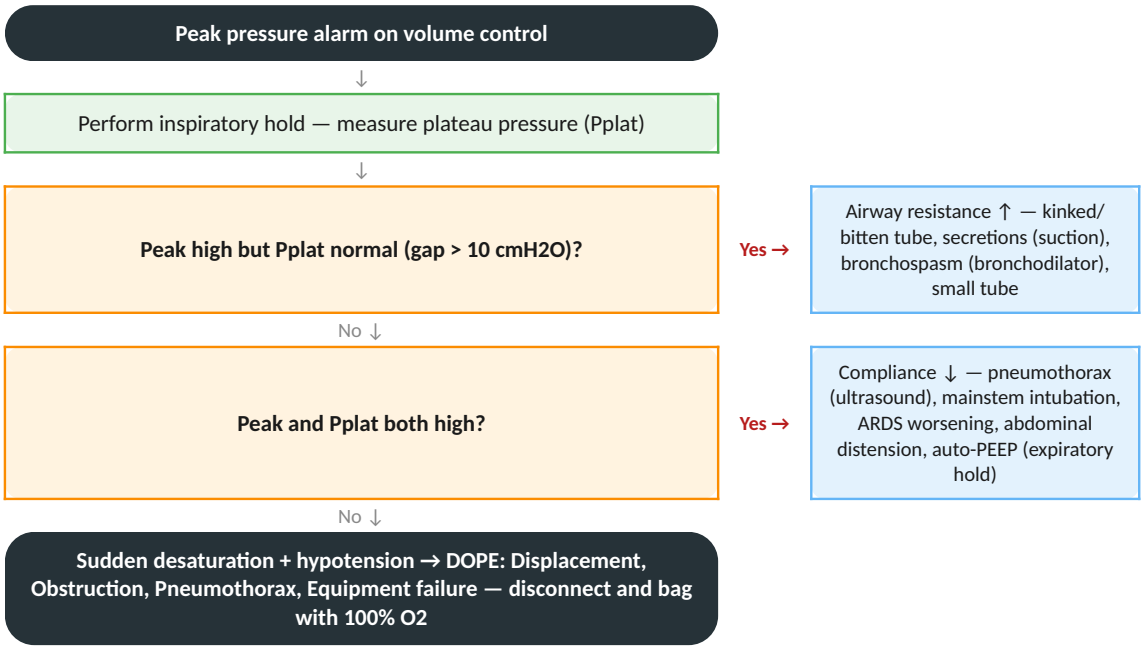


Figure 12.1 · Normal ventilator waveforms — volume control (square flow) and pressure control (decelerating flow)

Trauma & Critical care Medicine

Algorithm E12.2.1 · High airway pressure alarm



Patient–ventilator asynchrony on waveforms

Asynchrony	Waveform clue	Fix
Ineffective triggering	Negative pressure/flow dips without breath delivered	Reduce auto-PEEP, increase trigger sensitivity, reduce sedation/PS
Double triggering	Two breaths stacked	Increase Ti or tidal volume, more sedation in ARDS
Flow starvation	"Scooped" concave pressure curve in VC	Increase flow or switch to pressure mode
Premature / delayed cycling	Pressure spike at end (delayed) or short breath	Adjust expiratory trigger (cycling %) in PSV
Auto-PEEP	Expiratory flow does not return to zero	Lower rate, shorter Ti, bronchodilators
Reverse triggering	Patient effort triggered by mandatory breath	Adjust sedation, rate

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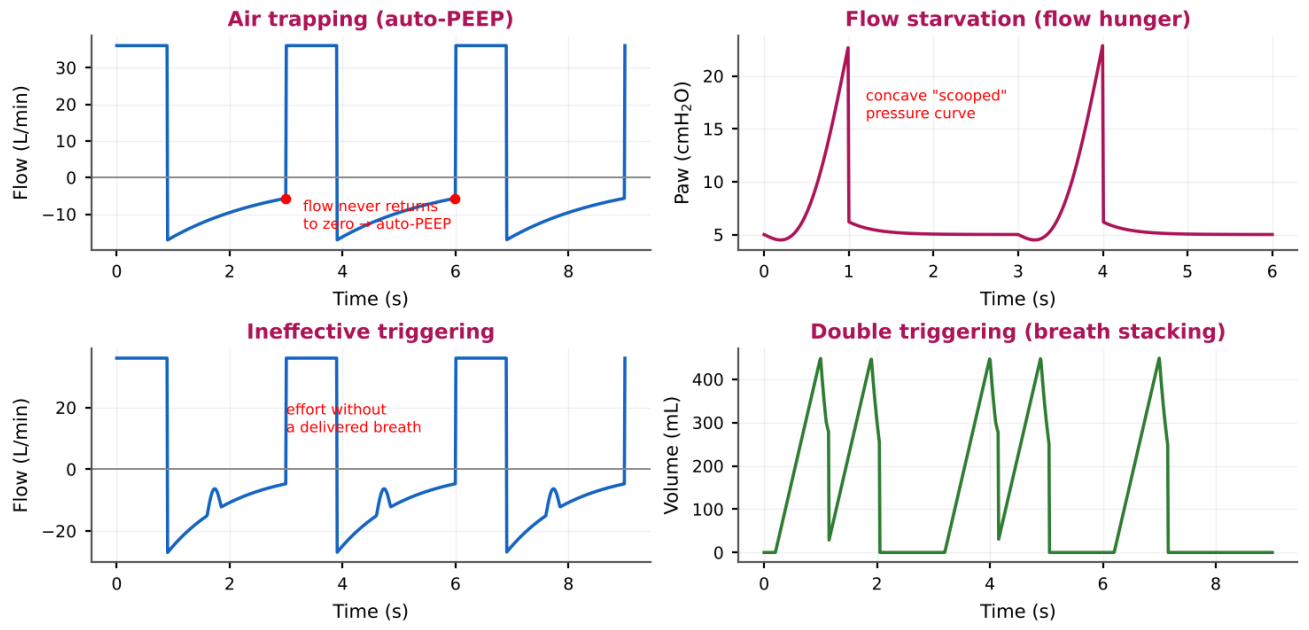


Figure 12.2 · Abnormal waveforms — air trapping, flow starvation, ineffective triggering and double triggering

★ CLINICAL PEARLS & EXAM FOCUS

- Driving pressure (Pplat − PEEP) is the ventilator variable most strongly associated with ARDS mortality (Amato 2015).
- Mechanical power combines pressure, volume, flow and rate — a newer integrated concept of VILI.

AETCM YOUTUBE · VIDEO LECTURES ▶ mechanical ventilation ▶ ventilator settings ▶ ventilator graphics

MERCK MANUAL PROFESSIONAL Mechanical ventilation | Overview of mechanical ventilation

E12.3 ARDS — Berlin to Global Definition & Management Algorithm

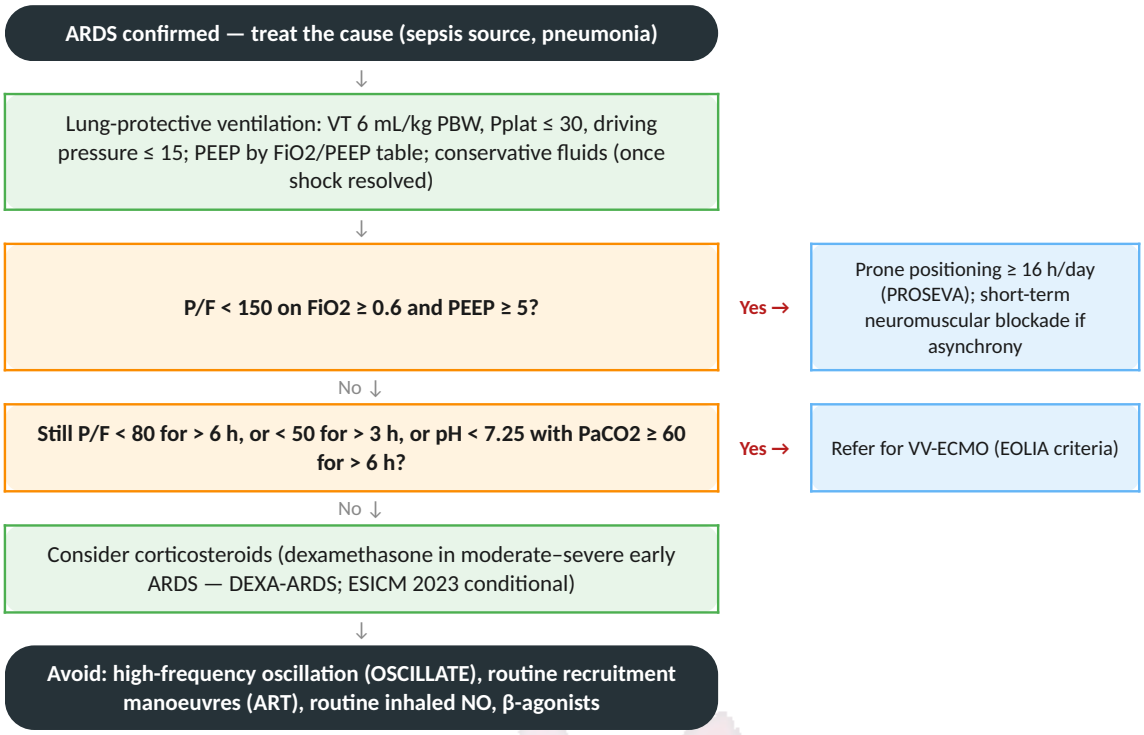
‹ Core topic 12.5 · p.219
Previous-year Qs ›

IN BRIEF The 2023 global definition of ARDS includes patients on HFNC (≥ 30 L/min), allows SpO2/FiO2 instead of PaO2/FiO2, accepts lung ultrasound for bilateral opacities and provides a modification for resource-limited settings.

Global definition of ARDS (2023)

Element	Criterion
Risk factor and timing	Acute onset (within 1 week) of new or worsening symptoms from a known risk factor; not fully explained by cardiac failure or fluid overload
Chest imaging	Bilateral opacities on CXR or CT, or bilateral B-lines/consolidation on lung ultrasound
Non-intubated	PaO2/FiO2 ≤ 300 or SpO2/FiO2 ≤ 315 (SpO2 ≤ 97%) on HFNC ≥ 30 L/min or NIV/CPAP ≥ 5 cmH2O
Intubated — severity	Mild: P/F 200–300 (S/F 235–315); moderate: 100–200 (S/F 148–235); severe: ≤ 100 (S/F ≤ 148)
Resource-limited	SpO2/FiO2 ≤ 315 without PEEP or flow requirement

Algorithm E12.3.1 · Stepwise management of ARDS



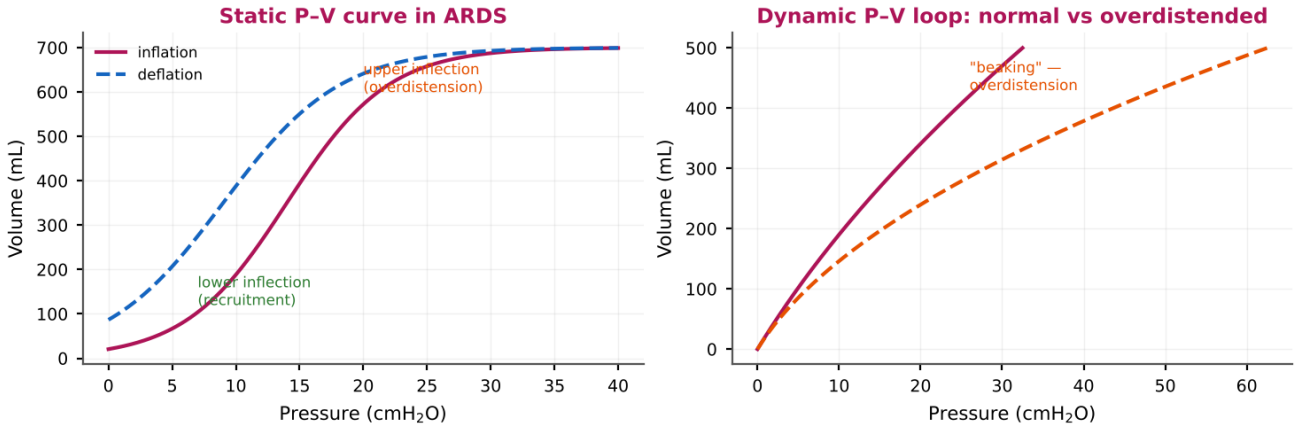


Figure 12.3 · Pressure–volume relationships in ARDS — inflection points and the "beaking" of overdistension

LANDMARK TRIALS & GUIDELINES

- **ARMA/ARDSNet (2000):** 6 vs 12 mL/kg — mortality 31% vs 40%.
- **PROSEVA (2013):** prone ≥ 16 h — 28-day mortality 16% vs 33% in P/F < 150.
- **EOLIA (2018):** VV-ECMO — mortality 35% vs 46% (not significant; crossover), Bayesian re-analysis favours ECMO.
- **FACTT:** conservative fluid strategy increased ventilator-free days.

★ CLINICAL PEARLS & EXAM FOCUS

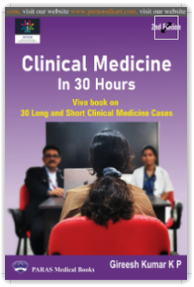
- ARDS phenotypes: hyperinflammatory (high IL-6, low bicarbonate, shock) vs hypoinflammatory — respond differently to fluids and PEEP in post-hoc analyses.
- Oxygenation index > 16 and ventilatory ratio > 2 indicate severe disease.

RADIOPAEDIA · IMAGE LINKS Acute respiratory distress syndrome cases

ULTRASOUND VIDEO LINKS ▶ ARDS — AETCM lecture

MERCK MANUAL PROFESSIONAL Acute respiratory distress syndrome

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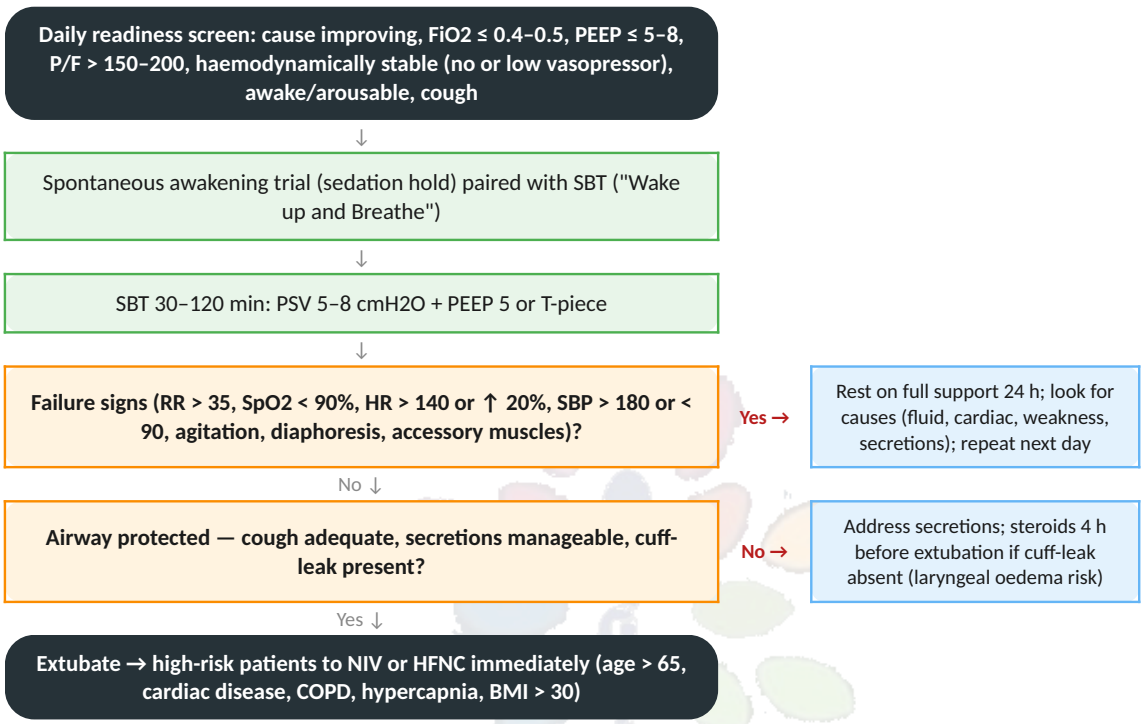
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E12.4 Weaning, Spontaneous Breathing Trials & Extubation Failure

‹ Core topic 12.7 · p.222
Previous-year Qs ›

IN BRIEF About 40% of ventilator time is spent weaning. A daily screen, a spontaneous breathing trial (SBT) and a clear extubation plan shorten it.

Algorithm E12.4.1 · Daily weaning protocol



Weaning predictors

Index	Threshold suggesting success
Rapid shallow breathing index (RSBI = RR/VT in L)	< 105 (Yang–Tobin)
Negative inspiratory force (MIP)	More negative than −20 to −30 cmH2O
Minute ventilation	< 10 L/min
P0.1	< 3.5–4 cmH2O (drive)
Diaphragm thickening fraction (US)	> 30%
Peak cough flow	> 60 L/min

Weaning classification (WIND)

Group	Definition
Short weaning	Extubated within 24 h of first separation attempt
Difficult weaning	Within 7 days
Prolonged weaning	> 7 days — consider tracheostomy, rehabilitation, weaning unit

LANDMARK TRIALS & GUIDELINES

- **ABC trial (2008):** paired SAT + SBT reduced mortality.
- **Subirà (2019):** 30-min PSV SBT led to higher extubation success than 2-h T-piece.

★ CLINICAL PEARLS & EXAM FOCUS

- Weaning-induced pulmonary oedema: rise in BNP or extravascular lung water during SBT — diuretics and nitrates.
- Early tracheostomy (< 7 days) does not reduce mortality (TracMan) but may aid comfort and reduce sedation.

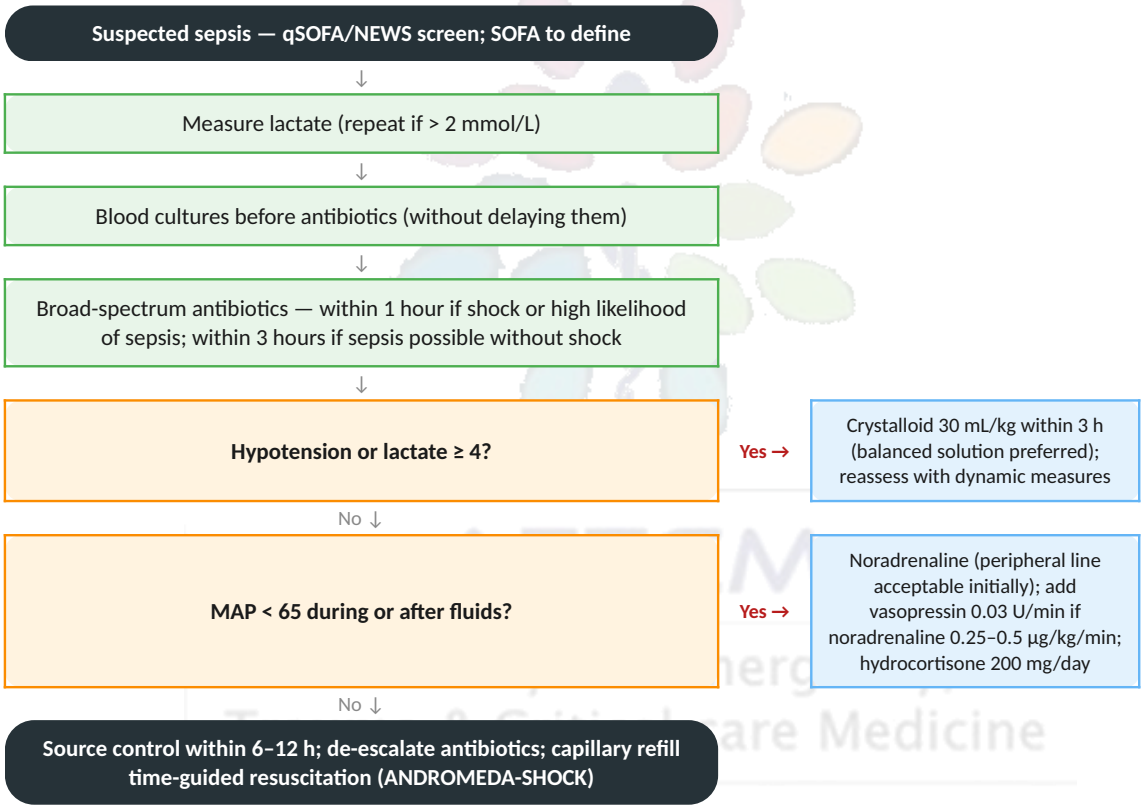
MERCK MANUAL PROFESSIONAL — Liberation from mechanical ventilation

E12.5 Sepsis & Septic Shock — Surviving Sepsis Campaign Bundle

‹ Core topic 12.8 · p.224
Previous-year Qs ›

IN BRIEF Sepsis is life-threatening organ dysfunction (SOFA rise ≥ 2) caused by a dysregulated host response to infection. Septic shock adds vasopressor need to keep MAP ≥ 65 and lactate > 2 mmol/L despite adequate fluids.

Algorithm E12.5.1 · The hour-1 bundle (SSC 2021)



SOFA score — organ systems

System	Variable
Respiration	PaO2/FiO2
Coagulation	Platelets
Liver	Bilirubin
Cardiovascular	MAP / vasopressors
CNS	Glasgow Coma Scale
Renal	Creatinine / urine output

LANDMARK TRIALS & GUIDELINES

- **ADRENAL / APROCCHSS:** hydrocortisone speeds shock reversal; APROCCHSS (with fludrocortisone) reduced mortality.
- **CLOVERS, CLASSIC:** restrictive vs liberal fluid — no mortality difference; avoid fluid overload.
- **BaSICS, PLUS:** balanced crystalloids vs saline — no large mortality difference.

★ CLINICAL PEARLS & EXAM FOCUS

- qSOFA (RR ≥ 22, altered mentation, SBP ≤ 100) is a prognostic tool — SSC 2021 advises against using it alone for screening.
- Respiratory sources (pneumonia) are the commonest cause of sepsis in the ICU.

AETCM YOUTUBE · VIDEO LECTURES ▶ septic shock ▶ surviving sepsis ▶ vasopressors

MERCK MANUAL PROFESSIONAL Sepsis and septic shock

E12.6 Difficult Airway — Prediction & Unanticipated Difficult Intubation Algorithm

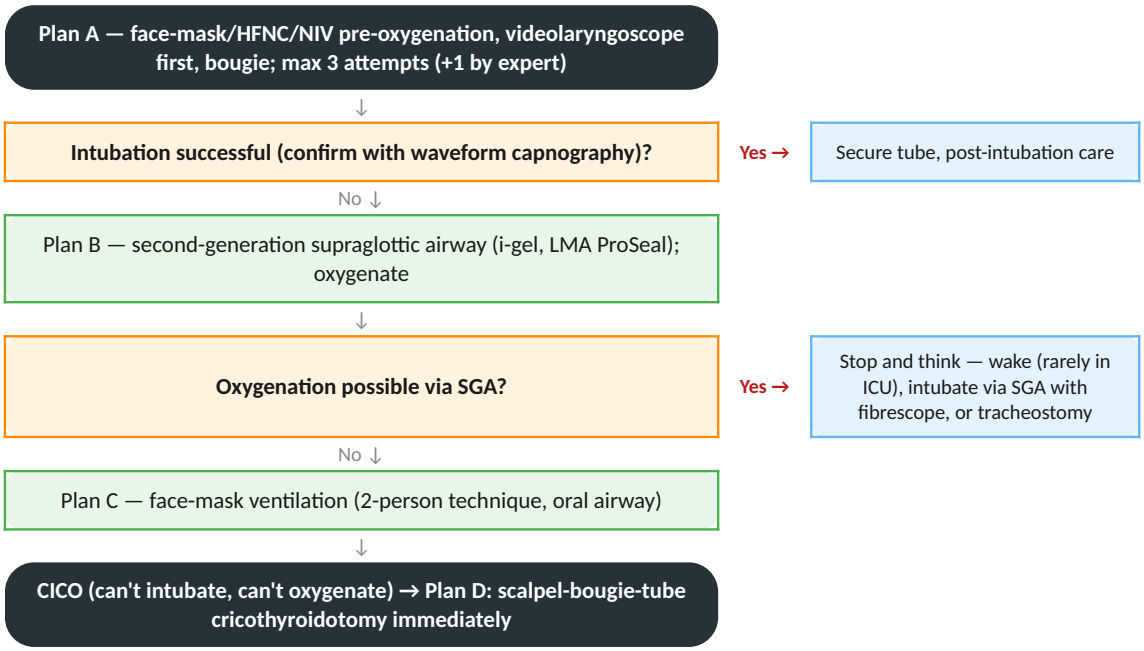
‹ Core topic 12.10 · p.227
Previous-year Qs ›

IN BRIEF The respiratory ICU airway is physiologically difficult — hypoxaemia, acidosis and hypotension. Pre-oxygenate, optimise haemodynamics and follow a plan with a clear rescue (front-of-neck access).

Predicting a difficult airway

Tool	Elements
LEMON	Look externally, Evaluate 3-3-2 rule, Mallampati, Obstruction/Obesity, Neck mobility
MACOCHA (ICU)	Mallampati III/IV, OSA, reduced cervical mobility, mouth opening < 3 cm, coma, severe hypoxaemia, non-anaesthetist operator
HOp killers (physiological)	Hypotension, Oxygenation (hypoxaemia), pH (metabolic acidosis)

Algorithm E12.6.1 · Unanticipated difficult intubation (DAS ICU 2018 framework)



Peri-intubation physiology checklist

- Pre-oxygenate with NIV or HFNC (PREOXI 2024 — NIV reduced hypoxaemia); continue HFNC during apnoea (apnoeic oxygenation).
- Fluid bolus and vasopressor ready (push-dose); choose ketamine or etomidate for induction in shock; rocuronium 1.2 mg/kg or succinylcholine 1.5 mg/kg.
- Waveform capnography is mandatory — "no trace = wrong place".

LANDMARK TRIALS & GUIDELINES

- **DEVICE (2023):** videolaryngoscopy improved first-pass success vs direct laryngoscopy in critically ill adults.
- **BOUGIE trial:** bougie did not improve first-pass success vs stylet overall.

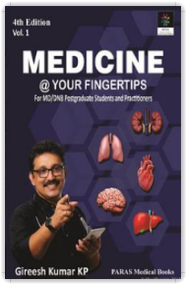
★ CLINICAL PEARLS & EXAM FOCUS

- Delayed sequence intubation (ketamine for agitated hypoxic patients to permit pre-oxygenation).
- Never give up on oxygenation while pursuing intubation.

AETCM YOUTUBE · VIDEO LECTURES ▶ rapid sequence intubation ▶ difficult airway ▶ cricothyroidotomy

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E12.7 Respiratory ICU Monitoring — Capnography, Lung Ultrasound & Haemodynamics

‹ Core topic 12.9 · p.225
Previous-year Qs ›

IN BRIEF Monitoring in respiratory critical care integrates gas exchange, lung mechanics and circulation. Capnography and point-of-care ultrasound are now standard of care.

Capnography waveforms

Finding	Meaning
Normal square waveform, EtCO2 35–45	Normal ventilation; PaCO2–EtCO2 gap 2–5 mmHg
"Shark-fin" sloping phase III	Bronchospasm — asthma, COPD
Sudden loss of waveform	Oesophageal intubation, disconnection, tube displacement, cardiac arrest
Gradual fall in EtCO2	Hyperventilation, falling cardiac output, PE (dead space ↑)
Rising baseline	Rebreathing — exhausted absorber, valve fault
EtCO2 < 10 mmHg during CPR	Poor-quality compressions; sudden rise > 40 suggests ROSC

Lung ultrasound signs in the ICU

Sign	Interpretation
A-lines with sliding	Normal aerated lung (or COPD/asthma, PE)
≥ 3 B-lines per intercostal space, bilateral	Interstitial syndrome — pulmonary oedema, ARDS
Absent sliding, stratosphere (barcode) sign on M-mode	Pneumothorax possible
Lung point	Pneumothorax — 100% specific
Tissue-like pattern, dynamic air bronchogram	Consolidation (pneumonia)
Anechoic space with quad and sinusoid sign	Pleural effusion
Diaphragm excursion < 1 cm	Diaphragm dysfunction

Fluid responsiveness — dynamic indices

Index	Threshold	Requirement
Pulse pressure variation	> 12–13%	Controlled ventilation, VT ≥ 8 mL/kg, sinus rhythm
Passive leg raising	↑ stroke volume/VTI > 10%	Works in spontaneously breathing patients
IVC distensibility	> 18% (ventilated)	Limited reliability
End-expiratory occlusion test	↑ CO > 5%	Ventilated

★ **CLINICAL PEARLS & EXAM FOCUS**

- A widened PaCO2–EtCO2 gap suggests increased dead space — PE, low cardiac output, hyperinflation.
- Lung ultrasound score (LUS 0–36) tracks aeration and predicts weaning success.

RADIOPAEDIA · IMAGE LINKS Lung ultrasound cases · A lines cases · B lines cases · Lung point sign cases

MERCK MANUAL PROFESSIONAL Monitoring the critically ill patient

E12.8 Drug Chart — Respiratory ICU Drugs

Previous-year Qs ›

Sedation, analgesia, paralysis and vasoactive drugs

Class	Drug	Dose	Indication	Caution
Induction agent	Ketamine	1–2 mg/kg IV; 0.5–2 mg/kg/h infusion	Intubation in shock/bronchospasm, sedation in asthma	Hypersalivation, emergence reactions
Induction agent	Etomidate	0.3 mg/kg IV	Haemodynamically unstable intubation	Adrenal suppression (single dose)
Induction / sedative	Propofol	1–2 mg/kg induction; 5–50 µg/kg/min infusion	Sedation	Hypotension, propofol infusion syndrome
α2-agonist	Dexmedetomidine	0.2–1.4 µg/kg/h	Light sedation, delirium, NIV tolerance	Bradycardia, hypotension
Opioid	Fentanyl	25–100 µg bolus; 0.5–2 µg/kg/h	Analgosedation (analgesia first)	Accumulation, chest-wall rigidity
Depolarising NMB	Succinylcholine	1.5 mg/kg IV	Rapid sequence intubation	Hyperkalaemia (burns, crush, neuromuscular disease, > 72 h immobility)
Non-depolarising NMB	Rocuronium	1.2 mg/kg RSI; reversal sugammadex 16 mg/kg	RSI, ventilation	Anaphylaxis
Non-depolarising NMB	Cisatracurium	0.15–0.2 mg/kg bolus; 1–3 µg/kg/min	Severe ARDS (short-term)	ICU-acquired weakness
Vasopressor	Noradrenaline	0.05–0.5+ µg/kg/min	First-line in septic shock	Extravasation, ischaemia
Vasopressor	Vasopressin	0.03 U/min (fixed)	Add-on in septic shock	Digital/mesenteric ischaemia
Inotrope / vasopressor	Adrenaline	0.05–0.5 µg/kg/min; 0.5 mg IM in anaphylaxis	Refractory shock, anaphylaxis	Lactate rise, arrhythmia
Inotrope	Dobutamine	2.5–20 µg/kg/min	Low cardiac output, RV failure	Tachyarrhythmia, hypotension
Corticosteroid	Hydrocortisone	200 mg/day (50 mg 6-hourly or infusion)	Septic shock on vasopressors, severe CAP	Hyperglycaemia
Corticosteroid	Dexamethasone	20 mg/day × 5 days then 10 mg × 5 days (DEXA-ARDS); 6 mg × 10 days in COVID	Moderate–severe ARDS	Infections
Pulmonary vasodilator (inhaled)	Nitric oxide	5–20 ppm	Rescue hypoxaemia, RV failure	Methaemoglobinaemia, rebound; no mortality benefit
Stress ulcer prophylaxis	Pantoprazole	40 mg IV OD	Ventilated with coagulopathy/shock	VAP, C. difficile
VTE prophylaxis	Enoxaparin / UFH	40 mg SC OD / 5000 U SC 8–12-hourly	All ICU patients unless contraindicated	Bleeding, HIT
Antipsychotic	Haloperidol	0.5–5 mg IV	Agitated delirium (short-term)	QT, extrapyramidal

Academy for Emergency,

E12.9 Ventilation in Obstructive Airway Disease — Auto-PEEP & Permissive Hypercapnia

‹ Core topic 12.6 · p.221
Previous-year Qs ›

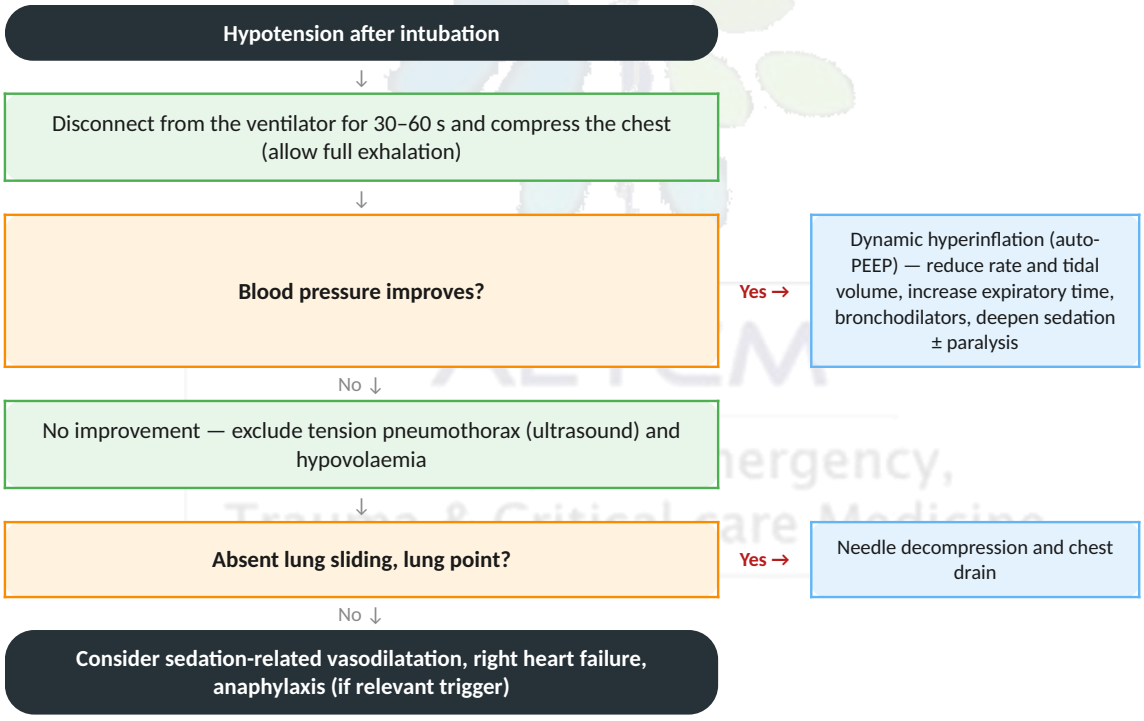
IN BRIEF The ventilated asthmatic dies of dynamic hyperinflation, not of hypercapnia. Give the lungs time to empty: slow rate, high flow, small tidal volume, and accept a high PaCO2.

AETCM ONLINE RESOURCE AETCM Mechanical Ventilation module ↗

Initial settings in severe asthma / COPD

Parameter	Setting	Reason
Mode	Volume control	Guaranteed volume with high resistance
Tidal volume	6–8 mL/kg PBW	Limits hyperinflation
Rate	8–12/min	Long expiratory time
Inspiratory flow	80–100 L/min (square wave)	Shortens inspiration
I:E ratio	1:4 to 1:5	Allows emptying
PEEP	0–5 (COPD: up to 80% of intrinsic PEEP)	Counterbalances auto-PEEP in COPD; little benefit in asthma
Targets	Pplat < 30 cmH2O, Vei < 20 mL/kg; accept pH ≥ 7.15–7.20	Permissive hypercapnia

Algorithm E12.9.1 · Sudden hypotension in a ventilated asthmatic



Measuring auto-PEEP and hyperinflation

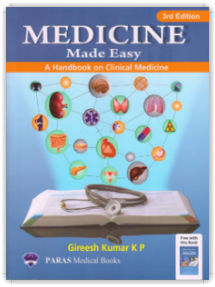
Measurement	Method	Threshold
Intrinsic PEEP	End-expiratory hold in a passive patient	> 5 cmH2O significant
Expiratory flow waveform	Flow does not return to zero before next breath	Qualitative
Plateau pressure	Inspiratory hold	≤ 30 cmH2O
End-inspiratory lung volume (Vei)	Volume exhaled over 60 s apnoea	< 20 mL/kg

★ CLINICAL PEARLS & EXAM FOCUS

- Ketamine is the induction agent of choice in severe asthma (bronchodilator); avoid morphine (histamine release).
- Heavy sedation ± short-term neuromuscular blockade reduces oxygen consumption and allows controlled hypoventilation; monitor for myopathy with steroids.

MERCK MANUAL PROFESSIONAL · Status asthmaticus · Mechanical ventilation

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E12.10 Extracorporeal Support & Rescue Therapies in Respiratory Failure

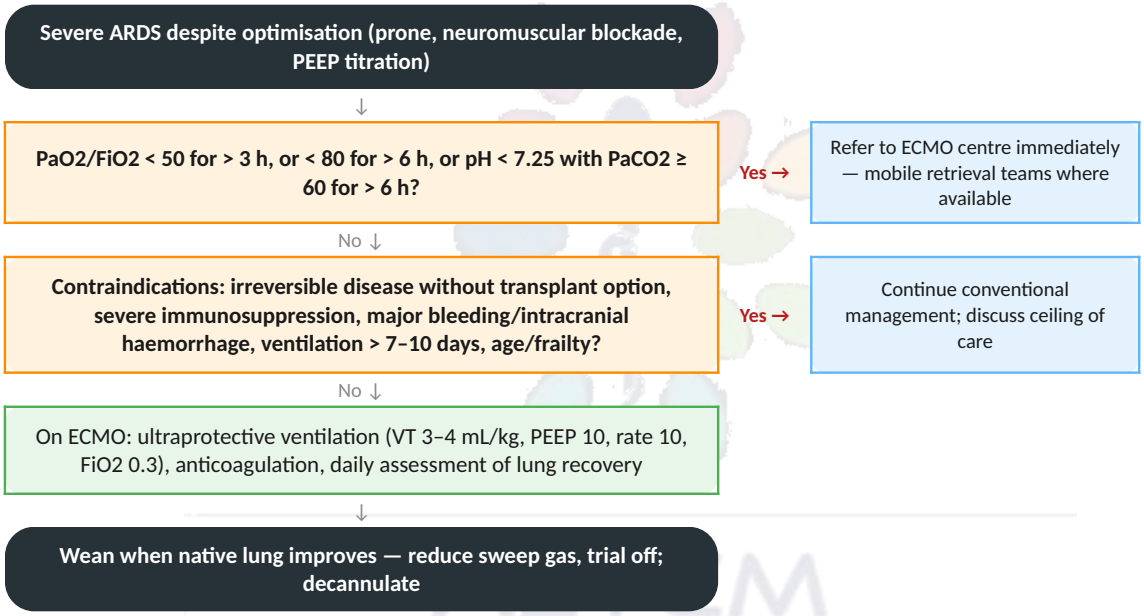
‹ Core topic 12.5 · p.219
Previous-year Qs ›

IN BRIEF ECMO is a bridge, not a treatment: it buys time for the lung to recover, for a transplant or for a decision. Patient selection and timely referral matter more than the circuit.

ECMO modes

Mode	Cannulation	Supports	Used for
Veno-venous (VV)	Femoral drainage + internal jugular return (or dual-lumen)	Gas exchange only	Severe ARDS, status asthmaticus, bridge to transplant
Veno-arterial (VA)	Femoral vein + femoral/axillary artery	Gas exchange + circulation	Cardiogenic shock, massive PE with arrest
ECCO2R	Low-flow veno-venous	CO2 removal only	Hypercapnic failure, ultraprotective ventilation (REST trial negative)

Algorithm E12.10.1 · Referral for VV-ECMO in ARDS (EOLIA-type criteria)



Other rescue therapies in refractory hypoxaemia

Therapy	Comment
Prone positioning	Proven mortality benefit — use before considering ECMO
Neuromuscular blockade	Short course in severe ARDS with asynchrony (ACURASYS; ROSE neutral)
Inhaled nitric oxide / prostacyclin	Transient oxygenation improvement, no mortality benefit; useful in RV failure
Recruitment manoeuvres	Not routine (ART trial harm with aggressive recruitment)
High-frequency oscillation	Harmful (OSCILLATE) — abandoned
Corticosteroids	Conditional recommendation in early moderate–severe ARDS

★ CLINICAL PEARLS & EXAM FOCUS

- Awake ECMO is used as a bridge to lung transplantation, allowing rehabilitation before surgery.
- The commonest ECMO complications are bleeding, cannula-site infection, haemolysis and circuit thrombosis.

MERCK MANUAL PROFESSIONAL Extracorporeal membrane oxygenation

E12.11 Non-Invasive Ventilation — Modes, Interfaces & Setting Up at the Bedside

‹ Core topic 12.3 · p.215
Previous-year Qs ›

IN BRIEF NIV succeeds or fails in the first two hours. Choose the right indication, the right interface and the right settings, then reassess with an arterial blood gas.

AETCM ONLINE RESOURCE AETCM Mechanical Ventilation module ↗

Evidence for NIV by indication

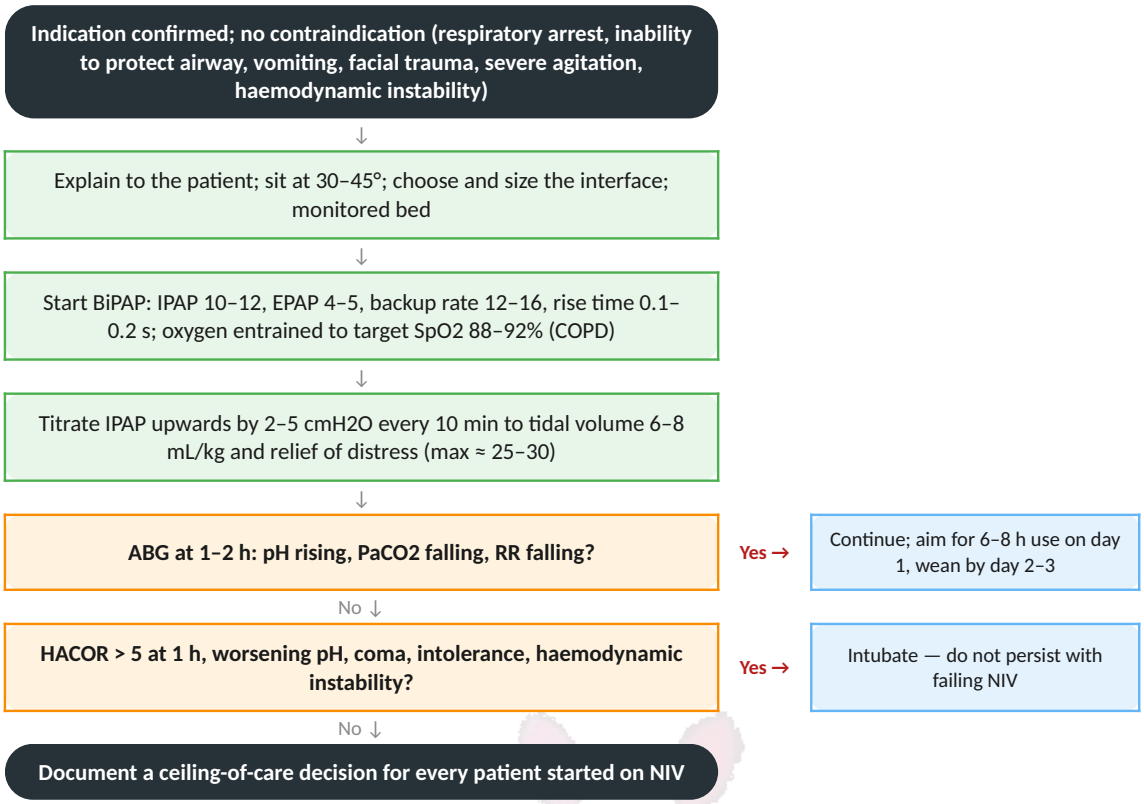
Indication	Strength of evidence
AECOPD with pH < 7.35 and PaCO2 > 45	Strong — reduces intubation and mortality
Cardiogenic pulmonary oedema	Strong — CPAP or BiPAP reduces intubation (3CPO: no mortality difference)
Immunocompromised with hypoxaemic failure	Moderate — early NIV or HFNC reduces intubation
Obesity hypoventilation, neuromuscular, chest wall	Strong — domiciliary and acute use
Post-extubation respiratory failure (established)	Harmful if used to delay re-intubation; useful prophylactically in high-risk patients
De novo hypoxaemic failure (pneumonia, ARDS)	Weak — HFNC preferred; helmet NIV an option
Asthma	Weak — use with caution in a monitored setting

Interfaces

Interface	Advantage	Problem
Oronasal (full face)	First choice in acute failure — mouth leak avoided	Claustrophobia, aspiration risk, pressure sores on the nose bridge
Nasal mask	Comfortable, eating and speech possible	Mouth leak
Total face mask	Even pressure distribution, fewer sores	Bulky
Helmet	High pressures tolerated, fewer sores; good for hypoxaemic failure	CO2 rebreathing at low flows, noise, poor triggering

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Algorithm E12.11.1 · Setting up acute NIV



Troubleshooting NIV

Problem	Solution
Mask leak	Refit, resize, chin strap; avoid overtightening
Nasal bridge pressure sore	Hydrocolloid dressing, alternate interfaces
Asynchrony / auto-triggering	Reduce leak, adjust trigger and cycling, rise time
Gastric distension	Reduce IPAP, nasogastric tube if severe
Intolerance/agitation	Coaching, low-dose dexmedetomidine or morphine in selected monitored patients
Persistent hypercapnia	Increase pressure support (IPAP − EPAP), check leak, treat metabolic alkalosis

★ CLINICAL PEARLS & EXAM FOCUS

- The pressure support is the difference IPAP − EPAP — raising both equally does not improve ventilation, only oxygenation.
- Avoid NIV in bulbar weakness and in patients who cannot clear secretions — use cough assist and consider early tracheostomy.

AETCM YOUTUBE · VIDEO LECTURES ▶ non invasive ventilation ▶ BiPAP ▶ CPAP

MERCK MANUAL PROFESSIONAL Noninvasive ventilation

PART II · SECTION 13

Sleep-Related Breathing Disorders

Advanced theory & algorithms

E13.1 Obstructive Sleep Apnoea — Diagnostic Algorithm (HSAT vs PSG) 468
E13.2 PAP Therapy, Alternatives & Troubleshooting 469
E13.3 Obesity Hypoventilation, Central Sleep Apnoea & Hypersomnolence 470
E13.4 Drug Chart — Drugs in Sleep-Disordered Breathing 473
E13.5 Sleep Physiology & the Polysomnogram — Reading the Report 473

E13.1 Obstructive Sleep Apnoea — Diagnostic Algorithm (HSAT vs PSG)

‹ Core topic 13.2 · p.232
Previous-year Qs ›

IN BRIEF OSA affects about 1 in 10 Indian adults, mostly undiagnosed. Screening questionnaires identify risk; home sleep apnoea testing (HSAT) confirms uncomplicated cases; full polysomnography is needed when comorbidities or other sleep disorders are suspected.

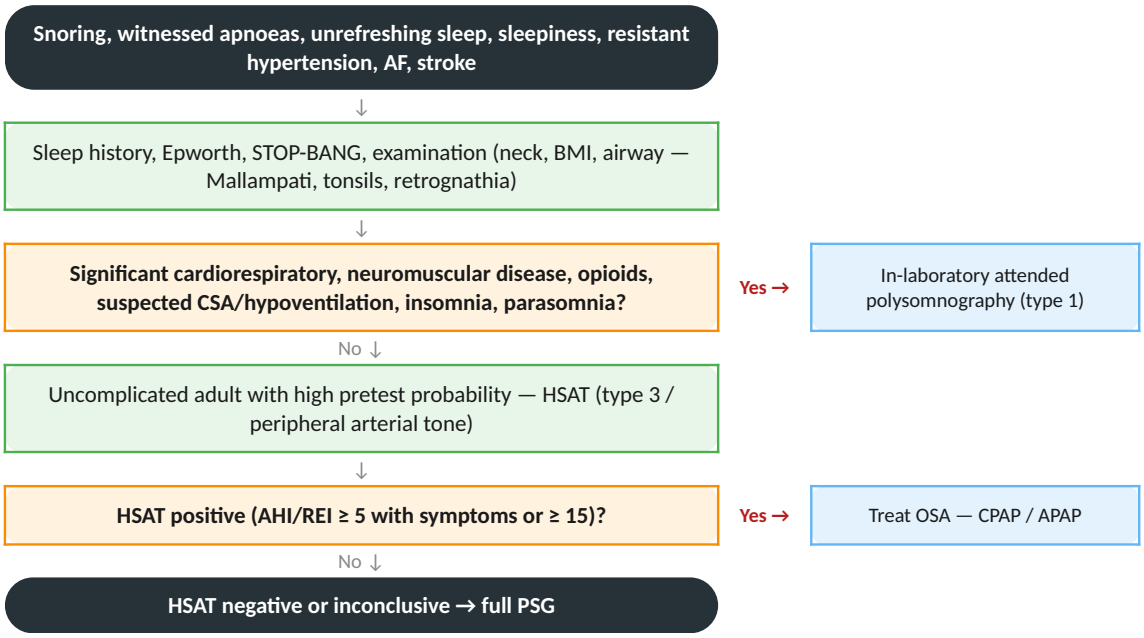
Screening tools

Tool	Items	Interpretation
STOP-BANG	Snoring, Tiredness, Observed apnoea, Pressure (HTN), BMI > 35, Age > 50, Neck > 40 cm, Gender male	≥ 3 intermediate; ≥ 5 high risk
Epworth Sleepiness Scale	8 situations, 0–3 each	> 10 = excessive daytime sleepiness
Berlin questionnaire	Snoring, sleepiness, HTN/BMI	2 of 3 categories positive = high risk

Polysomnographic definitions (AASM)

Term	Definition
Apnoea	≥ 90% drop in airflow for ≥ 10 s
Hypopnoea	≥ 30% drop in airflow for ≥ 10 s with ≥ 3% desaturation or arousal (AASM recommended)
AHI	Apnoeas + hypopnoeas per hour of sleep — mild 5–14, moderate 15–29, severe ≥ 30
RERA	Respiratory effort-related arousal (flow limitation without apnoea/hypopnoea) — included in RDI
Obstructive vs central	Effort present (obstructive) vs absent (central)

Algorithm E13.1.1 · Suspected OSA



★ **CLINICAL PEARLS & EXAM FOCUS**

- HSAT underestimates AHI (uses recording time, not sleep time) — a negative HSAT never excludes OSA.
- Drowsy driving: advise against driving until treated; ask about near-miss accidents.

MERCK MANUAL PROFESSIONAL Obstructive sleep apnea

E13.2 PAP Therapy, Alternatives & Troubleshooting

‹ Core topic 13.3 · p.234
Previous-year Qs ›

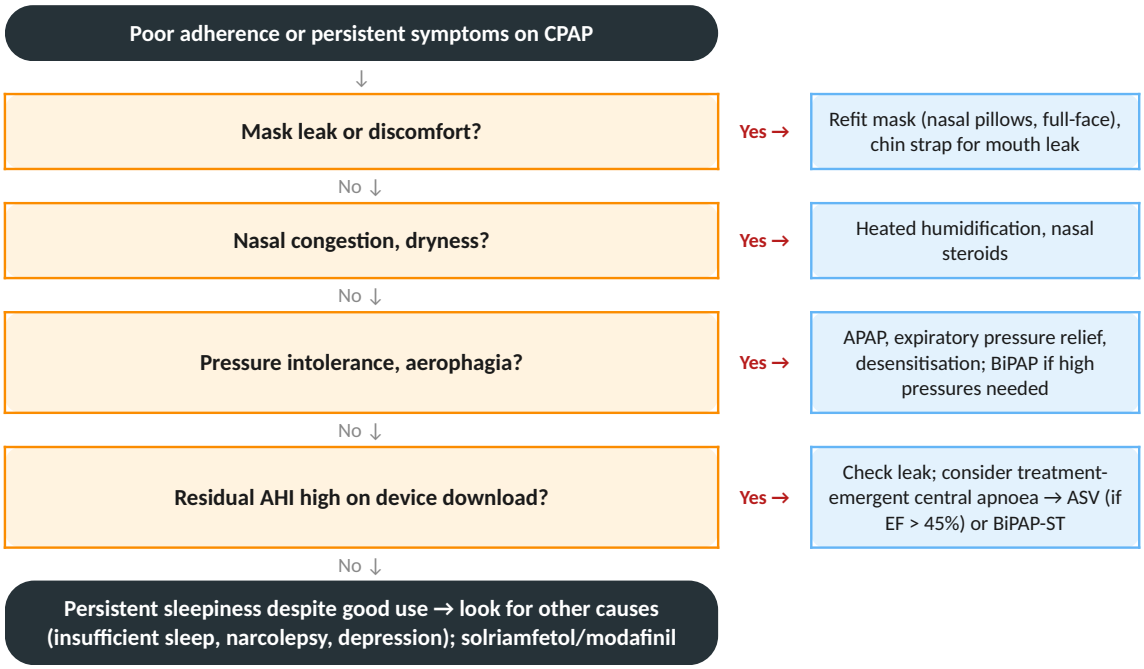
IN BRIEF CPAP is first-line for moderate–severe OSA and symptomatic mild OSA. Adherence (≥ 4 h/night on 70% of nights) is the main challenge — troubleshoot early.

Treatment options in OSA

Option	Best for
CPAP / APAP	All severities with symptoms; cardiovascular risk
Weight loss (≥ 10%), tirzepatide (SURMOUNT-OSA 2024)	Obese patients — tirzepatide FDA-approved for OSA with obesity
Positional therapy	Positional OSA (supine AHI ≥ 2× non-supine)
Mandibular advancement device	Mild–moderate OSA, CPAP intolerance, adequate teeth
Upper-airway surgery (UPPP, MMA, tonsillectomy)	Anatomic obstruction (large tonsils), selected adults; adenotonsillectomy in children
Hypoglossal nerve stimulation	Moderate–severe, BMI < 32–35, CPAP failure, no concentric collapse on DISE
Avoid alcohol, sedatives; treat nasal obstruction	All

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Algorithm E13.2.1 · CPAP troubleshooting



LANDMARK TRIALS & GUIDELINES

- **SAVE (2016):** CPAP did not reduce cardiovascular events in established CVD (adherence 3.3 h/night) but improved sleepiness and quality of life.
- **SERVE-HF:** ASV increased mortality in heart failure with EF ≤ 45% and central sleep apnoea.

★ CLINICAL PEARLS & EXAM FOCUS

- CPAP reduces blood pressure modestly (2–3 mmHg) — more in resistant hypertension.
- Children's OSA presents with hyperactivity, enuresis, poor school performance — adenotonsillectomy is first-line.

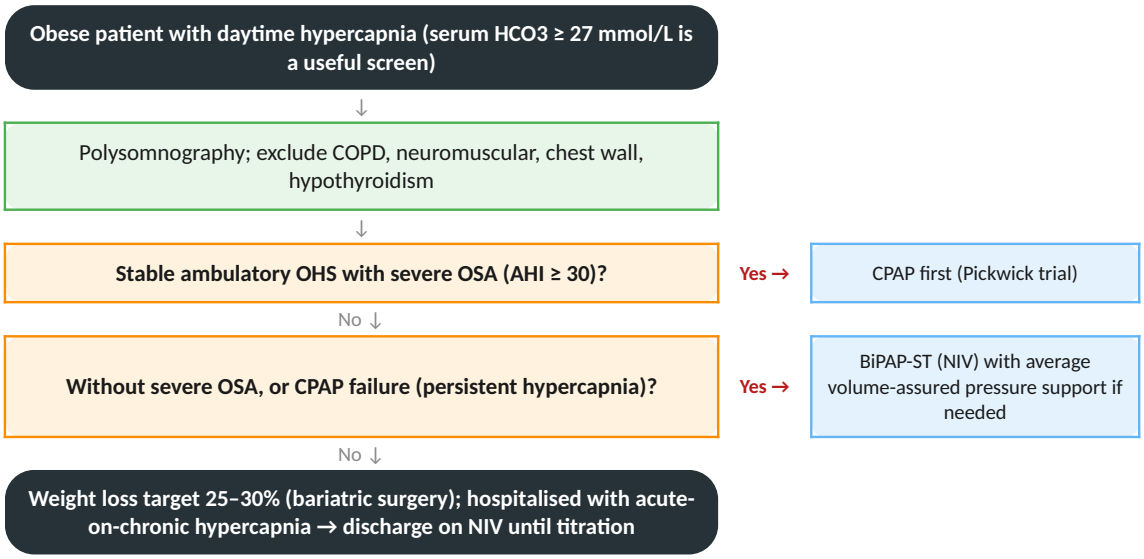
MERCK MANUAL PROFESSIONAL — Obstructive sleep apnea

E13.3 Obesity Hypoventilation, Central Sleep Apnoea & Hypersomnolence

‹ Core topic 13.4 · p.236
Previous-year Qs ›

IN BRIEF Obesity hypoventilation syndrome (OHS) is BMI ≥ 30 + daytime PaCO2 ≥ 45 mmHg without another cause. It is often first recognised during an acute hypercapnic admission misdiagnosed as COPD.

Algorithm E13.3.1 · OHS management (ATS 2019)



Central sleep apnoea — types

Type	Cause	Treatment
Cheyne–Stokes breathing	Heart failure, stroke (high loop gain)	Optimise heart failure; CPAP; ASV only if EF > 45%
High-altitude periodic breathing	Hypoxic ventilatory response	Descent, acetazolamide
Drug-induced	Opioids	Reduce opioid; ASV/BiPAP-ST
Primary / idiopathic	Unknown	CPAP, acetazolamide
Treatment-emergent (complex)	Appears on CPAP	Often resolves; ASV if persistent
Hypoventilation-related	Neuromuscular, OHS	NIV with backup rate

Central disorders of hypersomnolence

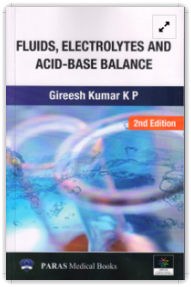
Disorder	Key features	Diagnosis	Treatment
Narcolepsy type 1	Sleepiness + cataplexy, hypnagogic hallucinations, sleep paralysis	MSLT mean latency ≤ 8 min + ≥ 2 SOREMPs; CSF hypocretin ≤ 110 pg/mL	Modafinil, pitolisant, sodium oxybate, solriamfetol
Narcolepsy type 2	No cataplexy	MSLT	Same
Idiopathic hypersomnia	Long unrefreshing sleep, sleep drunkenness	MSLT ≤ 8 min, < 2 SOREMPs or ≥ 11 h sleep	Modafinil, low-sodium oxybate
Kleine–Levin syndrome	Recurrent episodes of hypersomnia with behavioural change in adolescents	Clinical	Supportive, lithium

★ CLINICAL PEARLS & EXAM FOCUS

- OHS has higher mortality than OSA alone — and is under-recognised in COPD clinics.
- Opioid-induced CSA: ataxic (Biot) breathing pattern.

MERCK MANUAL PROFESSIONAL Central sleep apnea | Narcolepsy | Obesity hypoventilation syndrome

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Fluids, Electrolytes & Acid-Base Balance

Clinical reference · 2nd Edition

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E13.4 Drug Chart — Drugs in Sleep-Disordered Breathing

Previous-year Qs ›

Pharmacotherapy in sleep medicine

Class	Drug	Dose	Indication	Caution
Wakefulness-promoting	Modafinil / Armodafinil	100–400 mg morning / 150–250 mg	Residual sleepiness on CPAP, narcolepsy, shift-work	Headache, insomnia, reduces OCP efficacy, SJS (rare)
Dopamine–noradrenaline reuptake inhibitor	Solriamfetol	37.5–150 mg morning	Residual EDS in OSA, narcolepsy	Hypertension, anxiety
H3 receptor antagonist/ inverse agonist	Pitolisant	4.5 → 18–36 mg morning	Narcolepsy ± cataplexy; EDS in OSA	QT prolongation, insomnia
CNS depressant (GHB)	Sodium oxybate	4.5–9 g/night in 2 divided doses	Narcolepsy with cataplexy	Respiratory depression — untreated OSA, alcohol; abuse
Carbonic anhydrase inhibitor	Acetazolamide	250 mg at night	Central sleep apnoea, high-altitude periodic breathing	Paraesthesia, hypokalaemia
GIP/GLP-1 agonist	Tirzepatide	2.5 mg weekly → 10–15 mg weekly	OSA with obesity	GI effects, pancreatitis
GLP-1 agonist	Semaglutide	0.25 → 2.4 mg weekly	Weight loss in OSA/OHS	GI effects
Antidepressant (SNRI)	Venlafaxine	37.5–150 mg	Cataplexy	Hypertension, withdrawal
Antidepressant (TCA)	Clomipramine	10–75 mg	Cataplexy	Anticholinergic
Melatonin agonist	Melatonin	2–5 mg 1–2 h before bedtime	Circadian rhythm disorders, insomnia in elderly	Mild
Dual orexin receptor antagonist	Suvorexant / Lemborexant	10–20 mg / 5–10 mg at night	Insomnia (relatively safe in mild OSA)	Contraindicated in narcolepsy
Drugs to avoid / reduce	Benzodiazepines, opioids, alcohol	—	Worsen OSA and hypoventilation	Respiratory depression

E13.5 Sleep Physiology & the Polysomnogram — Reading the Report

‹ Core topic 13.1 · p.230
Previous-year Qs ›

IN BRIEF A sleep study report is read in a fixed order: architecture, respiratory events, oxygenation, limb movements and arousals. Know the normal values so that the abnormal stands out.

Normal sleep architecture in adults

Stage	Proportion of total sleep time	EEG features
N1	2–5%	Low-amplitude mixed frequency, vertex waves; slow rolling eye movements
N2	45–55%	Sleep spindles, K complexes
N3 (slow-wave)	15–25%	Delta waves (> 20% of the epoch); most in the first third of the night
REM	20–25%	Low-voltage mixed frequency, sawtooth waves, muscle atonia, rapid eye movements; most in the last third

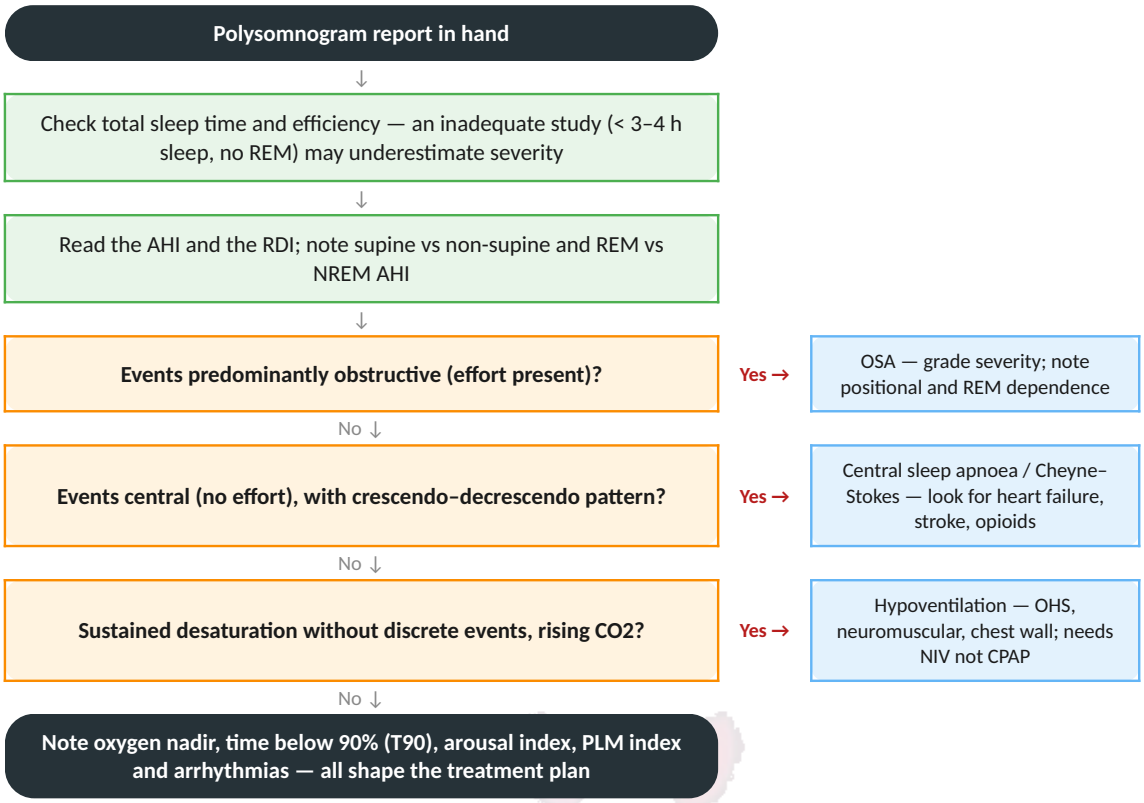
- **Sleep latency** < 30 min; **REM latency** 70–120 min; **sleep efficiency** > 85%; **arousal index** < 10/h; **WASO** (wake after sleep onset) < 30 min.

Channels recorded in a type 1 (attended) polysomnogram

Channel	Purpose
EEG (F4-M1, C4-M1, O2-M1), EOG, chin EMG	Staging sleep
Nasal pressure transducer + oronasal thermistor	Hypopnoea and apnoea detection
Thoracoabdominal belts (RIP)	Respiratory effort — obstructive vs central
Pulse oximetry	Desaturation; oxygen desaturation index (ODI)
ECG	Arrhythmia
Leg EMG (tibialis anterior)	Periodic limb movements
Body position, snore microphone, video	Positional OSA, parasomnia
Transcutaneous or end-tidal CO2	Hypoventilation (essential in neuromuscular disease and OHS)

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Algorithm E13.5.1 · Reading a sleep study report



Severity and additional indices

Index	Meaning
AHI 5–14.9 / 15–29.9 / ≥ 30	Mild / moderate / severe OSA
ODI (≥ 3% or ≥ 4% desaturations per hour)	Used in home studies and oximetry screening
T90	Percentage of sleep time with SpO2 < 90% — correlates with cardiovascular risk
Positional OSA	Supine AHI ≥ 2× non-supine AHI
REM-related OSA	REM AHI ≥ 2× NREM AHI, NREM AHI < 15
PLM index > 15/h with arousals	Periodic limb movement disorder

★ CLINICAL PEARLS & EXAM FOCUS

- A split-night study (diagnostic first half, CPAP titration second) is acceptable when the AHI exceeds 20–40 in the first 2 hours.
- In neuromuscular disease, overnight oximetry alone is inadequate — CO2 monitoring is essential because hypoventilation precedes desaturation.

MERCK MANUAL PROFESSIONAL Polysomnography Obstructive sleep apnea

PART II · SECTION 14

Thoracic Imaging & Pulmonary Procedures

Advanced theory & algorithms

E14.1 Systematic Chest Radiograph Interpretation — with Radiopaedia Image Links 476
E14.2 HRCT Patterns — Nodules, Lines, Cysts & Ground Glass 478
E14.3 Thoracic Ultrasound — BLUE Protocol, Effusion, Pneumothorax & eFAST 479
E14.4 Bronchoscopy, EBUS-TBNA & Cryobiopsy — Indications, Technique & Complications 481
E14.5 Medical Thoracoscopy & Intercostal Drains — Procedure Guide 482
E14.6 Imaging Atlas — Key Radiological Signs for Viva (Radiopaedia Links) 483
E14.7 Drug Chart — Contrast Agents, Radiopharmaceuticals & Procedure Premedication 486
E14.8 Interventional Pulmonology — Stents, Valves, Thermoplasty & Whole-Lung Lavage 486

E14.1 Systematic Chest Radiograph Interpretation — with Radiopaedia Image Links

‹ Core topic 14.1 · p.238
Previous-year Qs ›

IN BRIEF Read every film the same way: patient details and technical quality, then the ABCDE of anatomy, then the review areas where lesions hide. Tap the pink chips to see real examples on Radiopaedia.

Technical assessment — RIPE

Element	Check
Rotation	Medial ends of clavicles equidistant from spinous processes
Inspiration	6 anterior ribs or 10 posterior ribs above the diaphragm (mid-clavicular line)
Projection	PA vs AP (AP magnifies the heart; scapulae over lungs)
Exposure	Vertebral bodies just visible through the heart

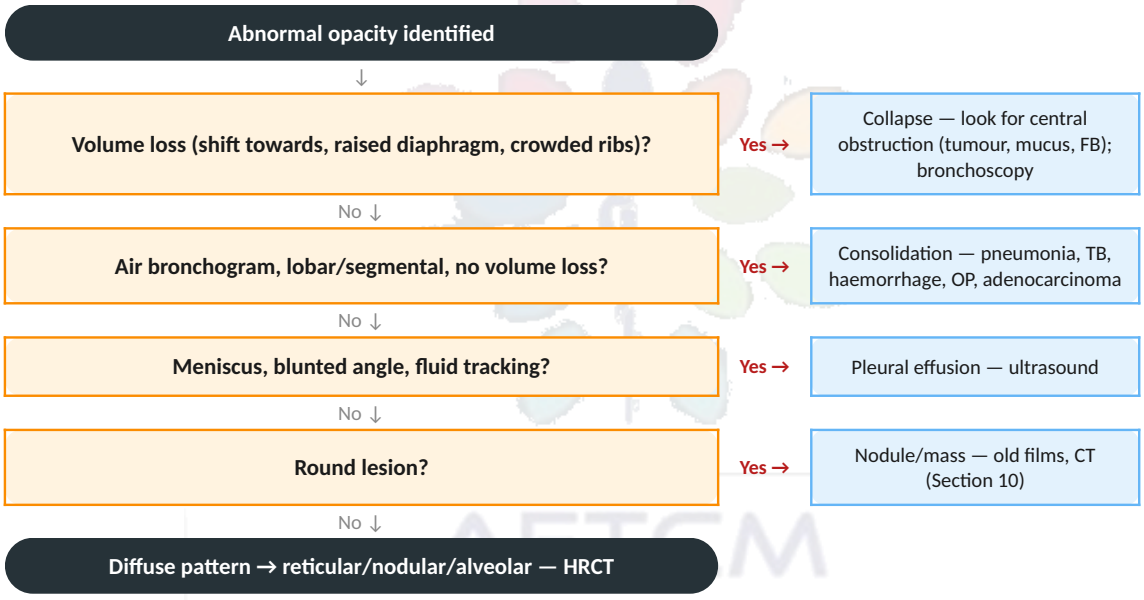
The ABCDE review

Area	Look for
Airway	Trachea central, carina angle, bronchi
Breathing (lungs, pleura)	Zones compared side by side, pleural edges, costophrenic angles, apices
Cardiac / mediastinum	CTR < 0.5 (PA), borders, hila (left higher than right), mediastinal width
Diaphragm	Right higher by 1.5–2 cm, free gas under diaphragm
Everything else	Bones (ribs, clavicles, spine), soft tissues, lines and tubes
Review areas	Apices, behind heart, below diaphragm, hila, retrocardiac space

Classic CXR signs and their meaning

Sign	Meaning
Silhouette sign	Loss of border = lesion in contact (RML → right heart border; lingula → left heart border; lower lobes → diaphragm)
Air bronchogram	Alveolar filling with patent airways — consolidation
Golden S sign	RUL collapse with central mass — think carcinoma
Luftsichel sign	LUL collapse — crescent of aerated superior segment around the aortic knob
Sail sign	LLL collapse behind the heart
Deep sulcus sign	Pneumothorax on supine film
Hampton hump, Westermark sign	Pulmonary infarction, oligaemia in PE
Batwing / butterfly	Perihilar alveolar oedema
Kerley B lines	Interstitial oedema
Cervicothoracic sign	Mass above clavicle with sharp border = posterior (in lung)
Continuous diaphragm sign	Pneumomediastinum

Algorithm E14.1.1 · Opacity on CXR — systematic approach



RADIOPAEDIA · IMAGE LINKS Silhouette sign cases · Golden S sign cases · Luftsichel sign cases · Sail sign cases · Deep sulcus sign cases · Westermark sign cases · Kerley lines cases · Batwing pulmonary opacities cases · Cervicothoracic sign cases · Continuous diaphragm sign cases

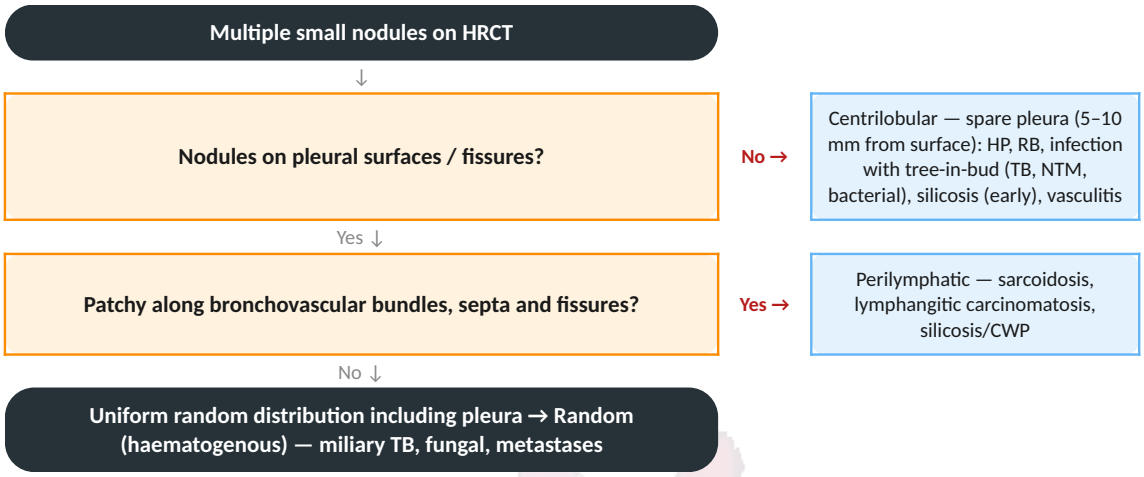
MERCK MANUAL PROFESSIONAL Chest imaging

E14.2 HRCT Patterns — Nodules, Lines, Cysts & Ground Glass

‹ Core topic 14.2 · p.240
Previous-year Qs ›

IN BRIEF HRCT diagnosis rests on the relationship of abnormalities to the secondary pulmonary lobule. Decide the dominant pattern and its distribution.

Algorithm E14.2.1 · Distribution of small nodules on HRCT



Ground-glass, mosaic and crazy-paving

Pattern	Definition	Differential
Ground-glass opacity	Hazy ↑ attenuation with preserved vessel margins	Early infection (viral, PJP), NSIP, HP, DAH, oedema, adenocarcinoma, COVID-19
Mosaic attenuation	Patchwork of different densities	Small-airway disease (air trapping on expiration), vascular (CTEPH), infiltrative
Crazy paving	GGO + interlobular septal thickening	PAP (classic), oedema, ARDS, PJP, DAH, lipoid pneumonia
Reverse halo (atoll)	Central GGO, peripheral ring of consolidation	Organising pneumonia, mucormycosis, sarcoid
Halo sign	Nodule with GGO rim (haemorrhage)	Invasive aspergillosis, haemorrhagic metastases
Head-cheese / three-density	GGO + normal + air trapping lobules	Fibrotic HP

Cysts and cavities

Finding	Think of
Thin-walled cysts, diffuse, round	LAM
Bizarre cysts + nodules, upper zone	PLCH
Basal paramediastinal lentiform cysts	Birt–Hogg–Dubé
Cysts + GGO, perivascular	LIP (Sjögren's)
Thick-walled cavity > 15 mm	Squamous carcinoma, abscess, TB, GPA
Pneumatoceles	PJP, staphylococcal pneumonia

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RADIOPAEDIA · IMAGE LINKS Centrilobular nodules cases · Perilymphatic distribution cases · Random distribution of pulmonary nodules cases · Crazy paving cases · Reverse halo sign cases · Halo sign cases · Mosaic attenuation cases · Secondary pulmonary lobule cases

MERCK MANUAL PROFESSIONAL Interstitial lung disease

E14.3 Thoracic Ultrasound — BLUE Protocol, Effusion, Pneumothorax & eFAST

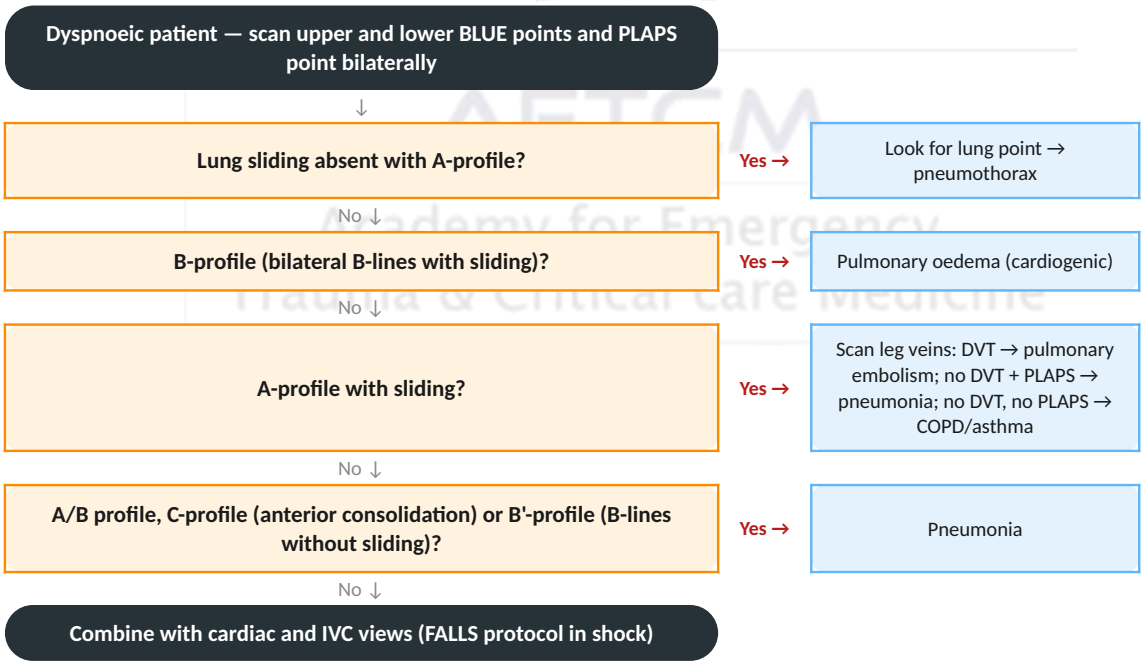
‹ Core topic 14.3 · p.241
Previous-year Qs ›

IN BRIEF Point-of-care lung ultrasound diagnoses the cause of acute respiratory failure in > 90% of cases in minutes (BLUE protocol, Lichtenstein). Tap the orange chips to watch the technique videos.

Basic lung ultrasound signs

Sign	Description	Significance
Bat sign	Ribs with pleural line between	Landmark
Lung sliding	Shimmering pleural line	Excludes pneumothorax at that point
A-lines	Horizontal reverberations	Normal aeration (or air)
B-lines	Vertical laser-like comet tails from pleura to screen bottom, move with sliding	Interstitial syndrome when ≥ 3 per space
Seashore sign (M-mode)	Granular below pleura	Normal sliding
Barcode (stratosphere) sign	Lines throughout	Absent sliding — pneumothorax possible
Lung point	Alternating sliding and no-sliding	Pneumothorax — specific
Quad sign, sinusoid sign	Fluid between pleural lines; inspiratory movement of lung line	Effusion
Shred / tissue-like sign	Hepatised lung with irregular deep border	Consolidation

Algorithm E14.3.1 · BLUE protocol — acute respiratory failure



Pleural effusion by ultrasound

- Anechoic → transudate or exudate; complex septated or homogeneously echogenic → exudate/empyema/haemothorax.
- Volume estimate: V (mL) ≈ 20 × maximal interpleural distance (mm) at the base (Balik formula, supine).
- Mark the site at the end of the scan, in the same position as the procedure.

eFAST in trauma

- Views: right upper quadrant (Morison's pouch), left upper quadrant, pelvis, subxiphoid cardiac, plus bilateral anterior chest for pneumothorax and bases for haemothorax.
- Sensitivity for pneumothorax (≈ 90%) far exceeds supine CXR.

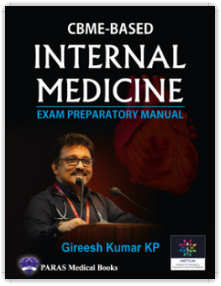
ULTRASOUND VIDEO LINKS ▶ BLUE protocol (JAMA Network video) · ▶ Ultrasound of the chest · ▶ Ultrasound in pleural effusion · ▶ Pneumothorax ultrasound · ▶ eFAST

RADIOPAEDIA · IMAGE LINKS Lung ultrasound cases · BLUE protocol cases · Lung point sign cases · Barcode sign cases · Pleural effusion ultrasound cases · eFAST cases

AETCM YOUTUBE · VIDEO LECTURES ▶ lung ultrasound · ▶ eFAST · ▶ POCUS

MERCK MANUAL PROFESSIONAL Point-of-care ultrasonography

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E14.4 Bronchoscopy, EBUS-TBNA & Cryobiopsy — Indications, Technique & Complications

‹ Core topic 14.5 · p.244
Previous-year Qs ›

IN BRIEF Flexible bronchoscopy is the pulmonologist's core procedure; EBUS-TBNA has replaced mediastinoscopy as the first staging test; transbronchial cryobiopsy is now an accepted alternative to surgical biopsy for ILD in experienced centres.

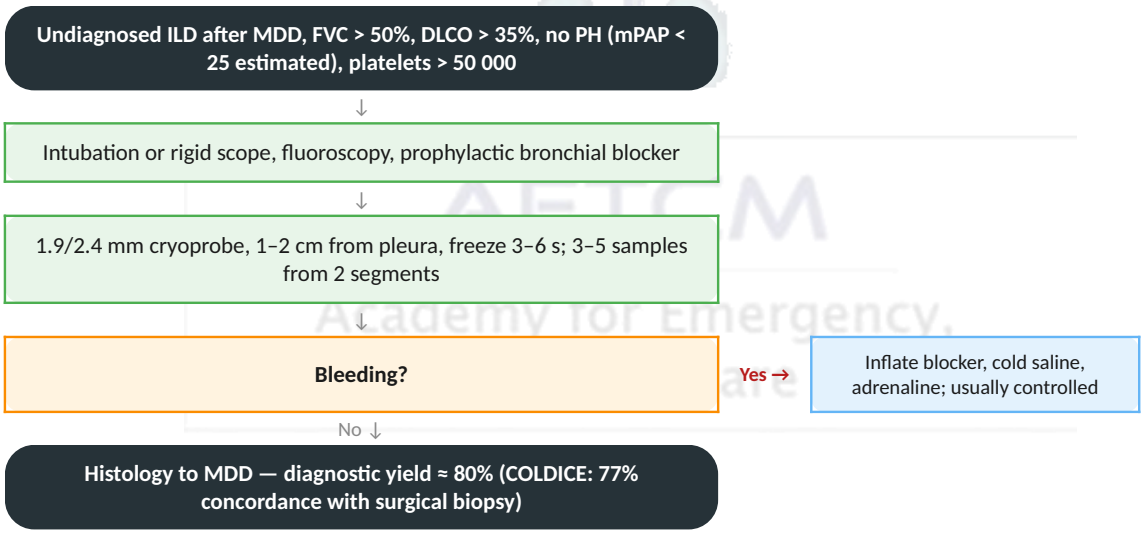
Indications and contraindications of flexible bronchoscopy

Diagnostic	Therapeutic	Contraindications
Unexplained haemoptysis, persistent cough, localised wheeze, non-resolving pneumonia, collapse, suspected cancer, ILD (BAL/TBLB), infection in immunocompromised	Foreign body removal, secretion clearance, haemoptysis control, stent, tumour debulking, whole-lung lavage (PAP), valve placement	Absolute: uncooperative patient, inadequate oxygenation; relative: recent MI, unstable arrhythmia, severe PH (biopsy), uncorrected coagulopathy (biopsy), raised ICP

EBUS-TBNA essentials

Item	Detail
Accessible stations	2R/2L, 4R/4L, 7, 10, 11 (3p and 8, 9 via EUS-B)
Needle	21/22 G; 3–4 passes per node; ROSE improves yield
Sensitivity for mediastinal staging	≈ 90%; NPV ≈ 90% — confirm negative N2 by mediastinoscopy if high suspicion
Other uses	Sarcoidosis (granulomas ≈ 80–90%), TB lymphadenitis (NAAT + culture), lymphoma (with core/cryo-node biopsy)
Complications	< 1% — bleeding, mediastinitis, pneumothorax rare

Algorithm E14.4.1 · Transbronchial lung cryobiopsy for ILD



Complications of bronchoscopy

- Hypoxaemia, bronchospasm, laryngospasm, bleeding (TBLB ≈ 1–4%), pneumothorax (TBLB 1–4%, cryobiopsy up to 10%), fever after BAL, lidocaine toxicity, arrhythmias.
- Post-TBLB CXR only if symptoms suggest pneumothorax (BTS).

★ CLINICAL PEARLS & EXAM FOCUS

- ROSE (rapid on-site evaluation) reduces number of passes and need for repeat procedures.
- For peripheral nodules: radial EBUS + guide sheath, electromagnetic navigation, robotic bronchoscopy (cone-beam CT confirmation) raise yields to 70–85%.

RADIOPAEDIA · IMAGE LINKS Endobronchial ultrasound cases Transbronchial lung cryobiopsy cases

AETCM YOUTUBE · VIDEO LECTURES ▶ bronchoscopy ▶ EBUS ▶ pleural procedures

MERCK MANUAL PROFESSIONAL Bronchoscopy

E14.5 Medical Thoracoscopy & Intercostal Drains — Procedure Guide

‹ Core topic 14.7 · p.246
Previous-year Qs ›

IN BRIEF Medical (local anaesthetic) thoracoscopy combines diagnosis (biopsy under vision) and therapy (talc poudrage) in one procedure. Diagnostic yield for malignancy and TB exceeds 90%.

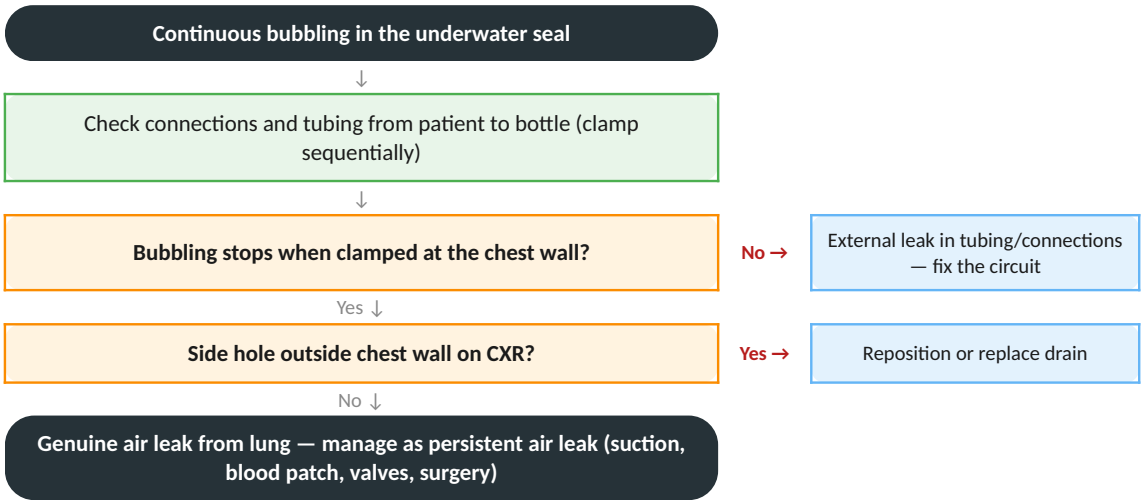
Medical thoracoscopy at a glance

Aspect	Detail
Indications	Undiagnosed exudative effusion, staging of mesothelioma/lung cancer, talc poudrage, early empyema (adhesiolysis), IPC placement
Requirements	Effusion with adequate pleural space (ultrasound), free lung movement; lateral decubitus
Instruments	Rigid thoracoscope (Boutin) or semi-rigid (pleuroscope); single port in 4th–6th ICS mid-axillary line
Contraindications	Obliterated pleural space, uncorrected coagulopathy, severe hypoxaemia, unstable cardiac status
Complications	Subcutaneous emphysema, air leak, fever after talc, infection, bleeding; mortality < 0.1%
Thoracoscopic appearance	TB: "sago grains" studded parietal pleura, adhesions; malignancy: nodules, masses, "candle-wax" drops

Chest drain management

Problem	Action
Drain not swinging	Kinked/blocked tube, lung fully expanded, drain outside pleura — flush (small-bore), check CXR
Persistent bubbling > 5 days	Persistent air leak — suction (−10 to −20 cmH2O), autologous blood patch, endobronchial valves, surgery
Subcutaneous emphysema	Check drain position/side holes, increase suction, larger drain if severe
Pain	Regular analgesia; check position
When to remove	Pneumothorax: no bubbling, lung expanded (clamping not routinely needed); effusion: < 200 mL/24 h (non-pleurodesis)

Algorithm E14.5.1 · Chest drain bubbling — assessment



★ **CLINICAL PEARLS & EXAM FOCUS**

- Never clamp a bubbling drain — risk of tension pneumothorax.
- Pleural fluid in the drain tube that swings with respiration confirms the drain is in the pleural space.

RADIOPAEDIA · IMAGE LINKS Medical thoracoscopy (cases) · Intercostal chest drain (cases)

MERCK MANUAL PROFESSIONAL Thoracoscopy

E14.6 Imaging Atlas — Key Radiological Signs for Viva (Radiopaedia Links)

‹ Core topic 14.2 · p.240
Previous-year Qs ›

IN BRIEF The 40 signs that come up most often in the practical and viva examination, grouped by system. Each chip opens the Radiopaedia article; "cases" opens real image collections.

Airways and parenchyma

Sign	Modality	Meaning
Signet ring sign	CT	Bronchiectasis
Tree-in-bud	CT	Endobronchial spread — TB, infection
Finger-in-glove	CXR/CT	Mucoid impaction — ABPA, bronchial atresia
Air crescent sign	CT	Aspergilloma; recovery phase of invasive aspergillosis
Monod sign	CT	Fungus ball in cavity
Galaxy sign	CT	Sarcoid conglomerate nodules
Cheerio sign	CT	Cystic nodules — LCH, adenocarcinoma
Feeding vessel sign	CT	Septic emboli
Comet-tail sign	CT	Rounded atelectasis
Bulging fissure sign	CXR	Klebsiella / expansile pneumonia

RADIOPAEDIA · IMAGE LINKS Signet ring sign cases · Tree-in-bud sign cases · Finger in glove sign cases · Air crescent sign cases · Monod sign cases · Galaxy sign cases · Feeding vessel sign cases · Comet tail sign cases · Bulging fissure sign cases

Pleura, mediastinum and vessels

Sign	Modality	Meaning
Split pleura sign	CT	Empyema
Meniscus sign	CXR	Effusion
Incomplete border sign	CXR	Extrapulmonary (pleural/chest wall) lesion
Hilum overlay sign	CXR	Mass not arising from hilum
Hilum convergence sign	CXR	Enlarged pulmonary arteries
Doughnut sign	Lateral CXR	Subcarinal lymphadenopathy
1-2-3 sign (Garland triad)	CXR	Sarcoid nodes
Polo mint sign	CTPA	Acute PE in cross-section
Mosaic perfusion	CT	CTEPH, small-airway disease
Pruning of pulmonary arteries	CXR	Pulmonary hypertension

RADIOPAEDIA · IMAGE LINKS Split pleura sign cases · Incomplete border sign cases · Hilum convergence sign cases · Doughnut sign cases · Garland triad cases · Polo mint sign cases · Pulmonary artery pruning cases

Occupational, ILD and miscellaneous

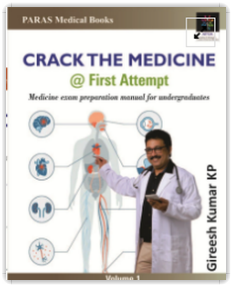
Sign	Modality	Meaning
Eggshell calcification	CXR/CT	Silicosis, sarcoidosis
Holly-leaf plaques	CXR	Calcified pleural plaques (asbestos)
Honeycombing	HRCT	Established fibrosis — UIP
Traction bronchiectasis	HRCT	Fibrosis
Subpleural sparing	HRCT	NSIP
Crazy paving	HRCT	PAP and others
Fleischner sign	CXR	Enlarged pulmonary artery in PE
Tram-track sign	CT	Bronchiectasis
Westermark sign	CXR	Oligaemia in PE
Scimitar sign	CXR	Anomalous venous return

RADIOPAEDIA · IMAGE LINKS Holly leaf sign cases · Subpleural sparing cases · Fleischner sign cases · Scimitar sign cases · Pulmonary alveolar proteinosis cases

MERCK MANUAL PROFESSIONAL Chest imaging

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E14.7 Drug Chart — Contrast Agents, Radiopharmaceuticals & Procedure Premedication

Previous-year Qs ›

Imaging and procedural drugs

Class	Drug	Dose	Indication	Caution
Iodinated contrast (low-osmolar / iso-osmolar)	Iohexol / Iodixanol	1–1.5 mL/kg IV (CTPA ≈ 60–80 mL)	CT chest, CTPA	Contrast nephropathy risk if eGFR < 30 — hydrate; allergy; metformin hold if eGFR < 30
Gadolinium agent	Gadobutrol	0.1 mmol/kg	MRI (cardiac sarcoid, chest wall)	Nephrogenic systemic fibrosis (rare with group II agents)
Radiotracer (PET)	18F-FDG	3–5 MBq/kg IV after 6 h fast	Lung cancer staging, sarcoid activity	Blood glucose < 200 mg/dL; false positives TB, inflammation
Radiotracer (V/Q)	99mTc-MAA (perfusion) + 99mTc-DTPA / Technegas (ventilation)	40–150 MBq	PE in pregnancy/renal failure, CTEPH screening, pre-op split function	Reduce particles in severe PH, right-to-left shunt
Contrast premedication	Prednisolone + antihistamine	Prednisolone 50 mg at 13, 7, 1 h + cetirizine/ chlorphenamine	Prior contrast reaction	Breakthrough reactions possible
Ultrasound contrast	Sulphur hexafluoride microbubbles	2.4 mL IV	Pleural/subpleural lesion characterisation	Rare cardiopulmonary reactions
Bubble study	Agitated saline	9 mL saline + 1 mL air (+ 1 mL blood)	Intracardiac/ intrapulmonary shunt (HPS, PAVM, PFO)	—
Topical airway anaesthesia	Lidocaine	≤ 8.2 mg/kg total	Bronchoscopy, EBUS	Toxicity
Sedation	Midazolam + fentanyl	See Section 1 drug chart	Bronchoscopy, thoracoscopy	Respiratory depression
Sclerosant	Talc	4–5 g poudrage	Pleurodesis at thoracoscopy	Fever, ARDS (ungraded talc)
Haemostatic	Cold saline; adrenaline 1:20 000; tranexamic acid 500 mg topical	Aliquots via scope	Post-biopsy bleeding	Arrhythmia
Antibiotic prophylaxis	Cefazolin	2 g IV	Chest drain in penetrating trauma, IPC insertion (some centres)	Allergy

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E14.8 Interventional Pulmonology — Stents, Valves, Thermoplasty & Whole-Lung Lavage

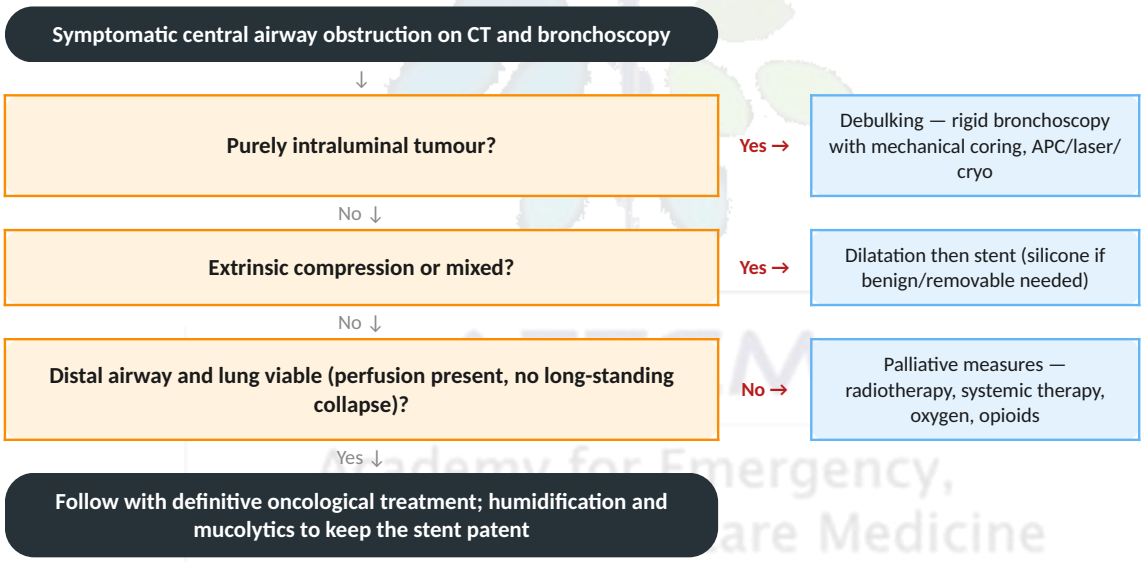
‹ Core topic 14.4 · p.242
Previous-year Qs ›

IN BRIEF Interventional pulmonology now offers definitive treatment, not only diagnosis. Know the indication, the technique in outline, and the main complication of each procedure.

Therapeutic bronchoscopic procedures

Procedure	Indication	Key points / complications
Airway stenting (silicone Dumon, self-expanding metallic, hybrid)	Malignant or benign central airway obstruction, tracheo-oesophageal fistula, tracheomalacia	Metallic stents difficult to remove — avoid in benign disease; granulation tissue, migration, mucus plugging
Laser (Nd:YAG), electrocautery, argon plasma coagulation	Endobronchial tumour debulking, bleeding	Airway fire — keep FiO2 < 40%; perforation
Cryotherapy / cryorecanalisation	Tumour debulking, foreign body, clot	Delayed effect with cryotherapy; immediate with cryo-extraction
Endobronchial valves (Zephyr, Spiration)	Severe emphysema with hyperinflation and no collateral ventilation (Chartis / fissure integrity on CT); persistent air leak	Pneumothorax in 20–30% after valve placement — keep inpatient 3 days (LIBERATE, IMPACT)
Bronchial thermoplasty	Severe asthma, poorly controlled despite maximal therapy, FEV1 > 50%, non-smoker	Three sessions; early exacerbations; benefit debated (AIR2)
Whole-lung lavage	Pulmonary alveolar proteinosis	Double-lumen tube, 10–20 L warm saline per lung under general anaesthesia
Bronchial artery embolisation	Massive haemoptysis	Interventional radiology; spinal artery ischaemia risk
Transbronchial microwave/ radiofrequency ablation	Peripheral tumours in non-surgical candidates	Emerging; cone-beam CT guidance
Foreign body removal	Aspiration	Rigid bronchoscopy preferred for large or sharp objects

Algorithm E14.8.1 · Choosing therapy for central airway obstruction



★ CLINICAL PEARLS & EXAM FOCUS

- Before relieving an obstruction, confirm that the lung beyond is still perfused and not destroyed — otherwise there is no symptomatic gain.
- For endobronchial valves, the absence of collateral ventilation (intact fissure) is the single best predictor of success.

RADIOPAEDIA · IMAGE LINKS Airway stent cases | Endobronchial valve cases | Pulmonary alveolar proteinosis cases

MERCK MANUAL PROFESSIONAL Bronchoscopy | Emphysema

PART II · SECTION 15

Special Environments, Systemic Disease, Pregnancy & Preventive Pulmonology

Advanced theory & algorithms

E15.1 High-Altitude Illness & Flight Fitness — Algorithms 488
E15.2 Respiratory Disease in Pregnancy — Management Algorithm 489
E15.3 Pulmonary Manifestations of Systemic Disease 491
E15.4 Chronic Cough — Diagnostic Algorithm & Treatment of Refractory Cough 493
E15.5 Vaccination in Respiratory Disease 494
E15.6 Drug Chart — Altitude, Cough, Vaccines & Systemic Disease 495
E15.7 Obesity, Metabolic Disease & the Lung 496

E15.1 High-Altitude Illness & Flight Fitness — Algorithms

‹ Core topic 15.1 · p.250
Previous-year Qs ›

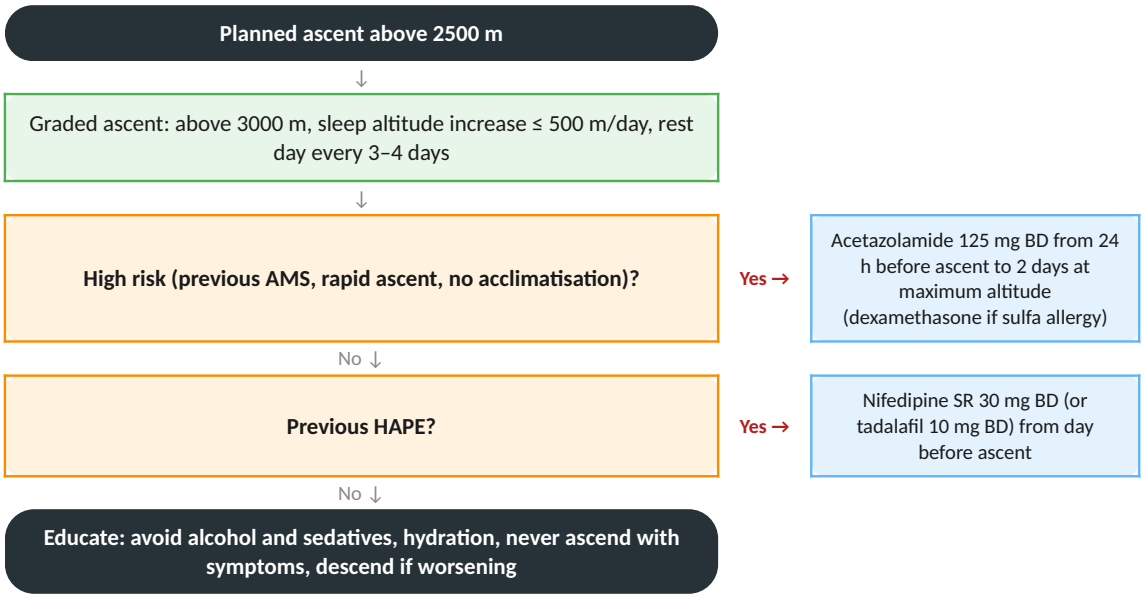
IN BRIEF India sends thousands of pilgrims, trekkers and soldiers above 3000 m every year. Prevention is graded ascent; treatment is descent, oxygen and drugs. Flight fitness is the mirror image — a cabin altitude of 2438 m.

High-altitude syndromes

Syndrome	Onset	Features	Treatment
Acute mountain sickness (AMS)	6–12 h after ascent > 2500 m	Headache + nausea, fatigue, dizziness, poor sleep (Lake Louise score ≥ 3)	Stop ascent, acetazolamide 250 mg BD, analgesia; descend if worsening
High-altitude cerebral oedema (HACE)	1–3 days	Ataxia, altered consciousness, severe AMS	Immediate descent, dexamethasone 8 mg then 4 mg 6-hourly, oxygen, portable hyperbaric bag
High-altitude pulmonary oedema (HAPE)	2–4 days	Dyspnoea at rest, cough, pink frothy sputum, crackles, low SpO2	Descent, oxygen, nifedipine SR 30 mg BD; PDE5 inhibitor alternative; avoid exertion
Chronic mountain sickness (Monge's)	Years of residence	Polycythaemia (Hb ≥ 21 men), hypoxaemia, headache, cyanosis	Descent; phlebotomy, acetazolamide

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Algorithm E15.1.1 · Prevention of altitude illness



Fitness to fly (BTS)

Assessment	Action
SpO2 ≥ 95% at sea level	No in-flight oxygen needed
SpO2 92–95% with risk factor (lung disease, hypercapnia, recent exacerbation)	Hypoxic challenge test (15% FiO2 for 20 min)
SpO2 < 92%	In-flight oxygen 2 L/min
Hypoxic challenge PaO2 < 50 mmHg (6.6 kPa) or SpO2 < 85%	In-flight oxygen
Pneumothorax	Fly ≥ 7 days after full resolution (1 day after surgery in some guidance)
Recent thoracic surgery or PE	Defer 1–2 weeks; anticoagulated PE patients can fly

★ CLINICAL PEARLS & EXAM FOCUS

- Acetazolamide speeds acclimatisation by inducing a metabolic acidosis that stimulates ventilation.
- Boyle's law: a closed gas space expands ≈ 30% at cabin altitude — hence the risk with pneumothorax or large bullae.

MERCK MANUAL PROFESSIONAL High altitude illness | Air travel and medical problems

E15.2 Respiratory Disease in Pregnancy — Management Algorithm

‹ Core topic 15.4 · p.254
Previous-year Qs ›

IN BRIEF Physiology changes the baseline: minute ventilation rises 30–50%, PaCO2 falls to 28–32 mmHg with a compensated respiratory alkalosis, FRC falls 20%, and oxygen consumption rises. A "normal" PaCO2 of 40 in a breathless pregnant woman signals impending failure.

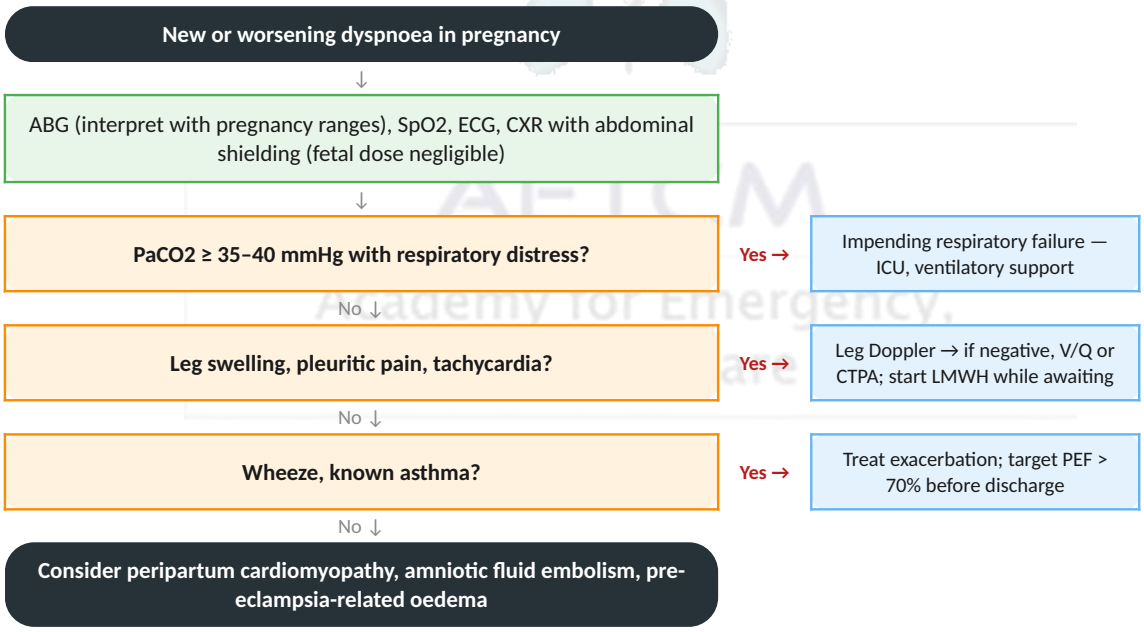
Physiological changes in pregnancy

Parameter	Change
Minute ventilation	↑ 30–50% (tidal volume, progesterone-driven)
FRC, ERV, RV	↓ 20% (diaphragm elevated 4 cm)
TLC	↓ slightly; VC and FEV1 unchanged
PaO2 / PaCO2	↑ 100–105 / ↓ 28–32 mmHg; HCO3 18–21
Oxygen consumption	↑ 20–30%
Airway	Mucosal oedema — difficult intubation, epistaxis

Managing common conditions

Condition	Key points
Asthma	One-third worsen; continue ICS/LABA — uncontrolled asthma is far more dangerous than medication; treat exacerbation as usual (steroids, salbutamol, magnesium); target SpO2 ≥ 95%
Pneumonia	Lower threshold for admission; avoid tetracyclines, fluoroquinolones (relative); macrolides (azithromycin) and β-lactams safe
Tuberculosis	HRE + Z (WHO allows pyrazinamide); streptomycin contraindicated (ototoxic); pyridoxine; TPT with 6H can be given
VTE/PE	Leg Doppler first; V/Q or CTPA (both acceptable — low fetal dose); LMWH treatment through pregnancy + 6 weeks postpartum; warfarin and DOACs contraindicated
Pulmonary hypertension	Pregnancy contraindicated (mortality 10–25%) — contraception advice; ERAs and riociguat teratogenic
Cystic fibrosis / bronchiectasis	Multidisciplinary; most antibiotics usable; watch nutrition
ARDS / COVID	Prone positioning possible with support; deliver if maternal condition demands

Algorithm E15.2.1 · Breathless pregnant woman



★ CLINICAL PEARLS & EXAM FOCUS

- Physiological dyspnoea of pregnancy begins in the first or second trimester and does not interfere with activity — new, severe or exertional dyspnoea needs investigation.
- Radiation from CXR (0.0005–0.01 mGy to fetus) is negligible — never withhold a needed chest radiograph.

MERCK MANUAL PROFESSIONAL Pulmonary disorders in pregnancy

E15.3 Pulmonary Manifestations of Systemic Disease

‹ Core topic 15.3 · p.253
Previous-year Qs ›

IN BRIEF The lung is involved in almost every systemic disease. These tables answer the common short-note questions.

Lung in systemic disease

System	Manifestations
Rheumatoid arthritis	ILD (UIP), pleural effusion (low glucose), nodules, Caplan syndrome, bronchiolitis obliterans, drug toxicity (methotrexate)
SLE	Pleuritis (commonest), acute lupus pneumonitis, DAH, shrinking lung syndrome, PH, infection
Systemic sclerosis	NSIP, PAH, aspiration from oesophageal dysmotility
Inflammatory bowel disease	Bronchiectasis, OP, drug toxicity (mesalazine), VTE
Chronic kidney disease	Uraemic pleuritis, pulmonary oedema ("bat-wing"), calcification, dialysis-related hypoxaemia
Liver disease	Hepatic hydrothorax, hepatopulmonary syndrome (orthodeoxia), portopulmonary hypertension
Diabetes	Increased TB, melioidosis, mucormycosis, OSA; mild restrictive defect
Sickle cell disease	Acute chest syndrome, PH
HIV	TB, PJP, bacterial pneumonia, Kaposi sarcoma, lymphoma, COPD, PH
Thyroid	Goitre with tracheal compression; myxoedema effusion; hyperthyroid dyspnoea and muscle weakness
Neuromuscular	Hypoventilation, aspiration, atelectasis
Obesity	OSA, OHS, asthma-like symptoms, reduced ERV

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Algorithm E15.3.1 · Hepatopulmonary syndrome

Chronic liver disease with dyspnoea and hypoxaemia

↓

ABG — A–a gradient ≥ 15 mmHg (≥ 20 if > 64 years); check orthodeoxia (PaO2 fall ≥ 5% or 4 mmHg from supine to upright)

↓

Contrast-enhanced (agitated saline) echocardiography

↓

Microbubbles in left atrium after ≥ 3 cardiac cycles?

Yes → Intrapulmonary vascular dilatation — hepatopulmonary syndrome

No ↓

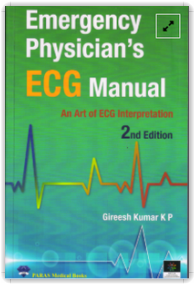
Oxygen; liver transplantation is the only effective treatment (PaO2 < 60 gives MELD exception points)

★ CLINICAL PEARLS & EXAM FOCUS

- Platypnoea–orthodeoxia: dyspnoea and desaturation upright — HPS, PFO, post-pneumonectomy.
- Shrinking lung syndrome in SLE: progressive restriction with elevated diaphragms and clear lung fields.

MERCK MANUAL PROFESSIONAL — Hepatopulmonary syndrome | Pulmonary manifestations of systemic disease

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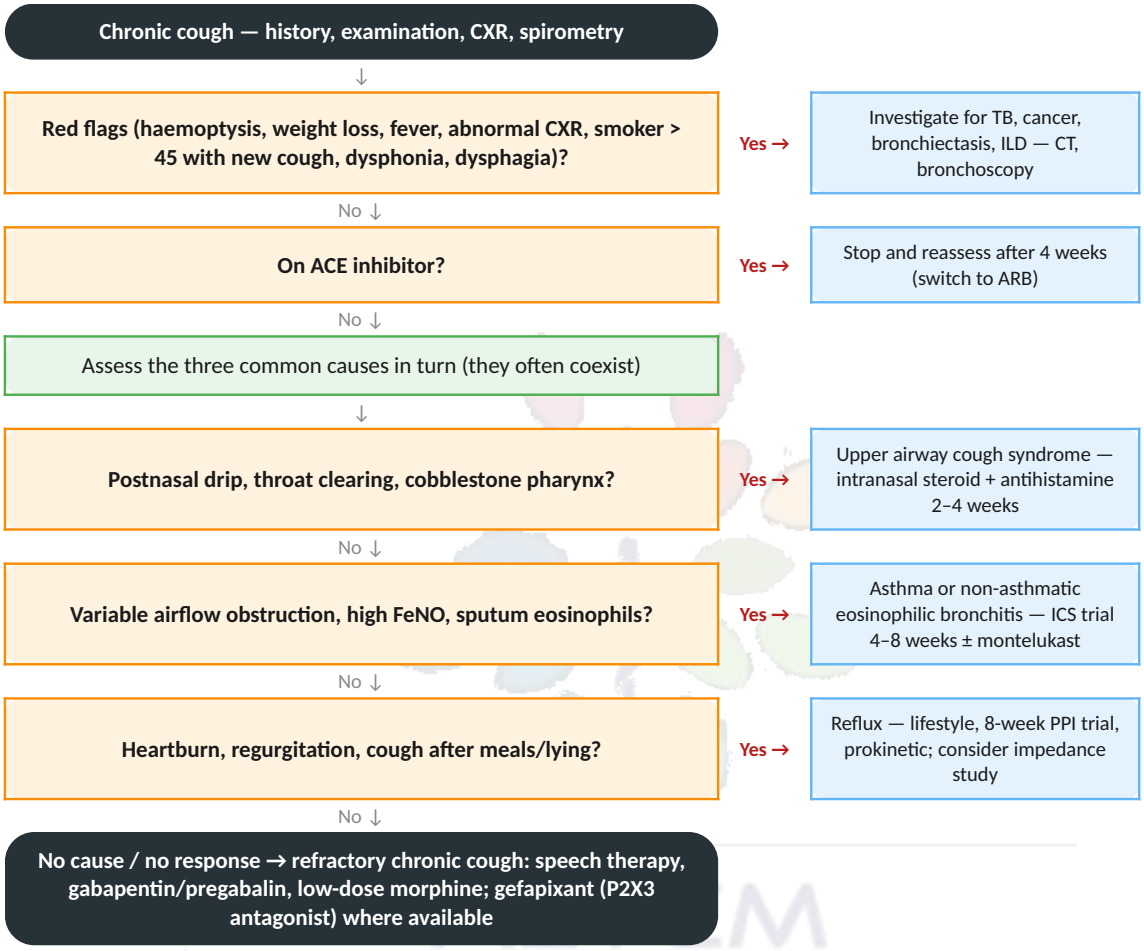
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E15.4 Chronic Cough — Diagnostic Algorithm & Treatment of Refractory Cough

‹ Core topic 15.6 · p.257
Previous-year Qs ›

IN BRIEF Cough lasting > 8 weeks with a normal chest radiograph in a non-smoker not on ACE inhibitors is usually caused by one or more of upper airway cough syndrome, asthma/eosinophilic bronchitis, or reflux — or it is refractory (cough hypersensitivity).

Algorithm E15.4.1 · Chronic cough (> 8 weeks)



Assessment tools

Tool	Use
Leicester Cough Questionnaire (LCQ)	Health status; MCID 1.3
Cough VAS (0–100 mm)	Severity
Cough Severity Diary	Trials
Sputum eosinophils / FeNO	Steroid-responsive cough

★ **CLINICAL PEARLS & EXAM FOCUS**

- ACE inhibitor cough occurs in 10–20% (more in women and in Asians); it may begin months after starting the drug.
- Always exclude TB in India — chronic cough is a presumptive TB symptom under the NTEP.

MERCK MANUAL PROFESSIONAL Cough

E15.5 Vaccination in Respiratory Disease

‹ Core topic 15.5 · p.255
Previous-year Qs ›

IN BRIEF Vaccines prevent exacerbations, hospitalisation and death in chronic lung disease, and every pulmonary consultation is an opportunity to check status.

Vaccines for adults with chronic lung disease (adapted from ACIP/ICS guidance)

Vaccine	Schedule	Indication
Influenza (inactivated, quadrivalent)	Annual	All patients with chronic lung disease; pregnancy; healthcare workers
Pneumococcal conjugate (PCV20/PCV15)	PCV20 single dose; or PCV15 followed by PPSV23 after ≥ 1 year	COPD, asthma, bronchiectasis, immunocompromised, age ≥ 50–65
Pneumococcal polysaccharide (PPSV23)	After PCV15	As above
COVID-19	Per national schedule, updated strain	Chronic lung disease, elderly, immunocompromised
RSV (adjuvanted or mRNA)	Single dose	Age ≥ 60–75; chronic lung disease; pregnancy (maternal, to protect infant)
Tdap	Once, then Td every 10 years	All adults; pregnancy each time (27–36 weeks)
Varicella / Zoster (recombinant)	2 doses	Age ≥ 50, immunocompromised (steroids, biologics)
BCG	At birth (India UIP)	Prevents severe childhood TB (miliary, meningitis)
Hepatitis B	3 doses	Healthcare workers, dialysis, before immunosuppression

★ **CLINICAL PEARLS & EXAM FOCUS**

- Give inactivated vaccines 2 weeks before starting immunosuppression; live vaccines are contraindicated on biologics and high-dose steroids (prednisolone ≥ 20 mg/day for ≥ 2 weeks).
- Influenza vaccination reduces exacerbations and cardiovascular events in COPD.

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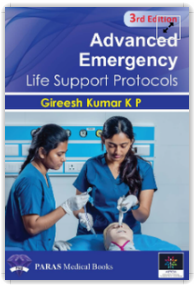
E15.6 Drug Chart — Altitude, Cough, Vaccines & Systemic Disease

Previous-year Qs ›

Drugs in this section

Class	Drug	Dose	Indication	Caution
Carbonic anhydrase inhibitor	Acetazolamide	125 mg BD prophylaxis; 250 mg BD treatment	AMS prevention and treatment; CSA; metabolic alkalosis	Paraesthesia, polyuria, sulfa allergy
Corticosteroid	Dexamethasone	8 mg then 4 mg 6-hourly	HACE; AMS when acetazolamide not suitable	Masks symptoms — descent still essential
Calcium channel blocker	Nifedipine SR	30 mg BD	HAPE prevention and treatment	Hypotension
PDE5 inhibitor	Tadalafil / Sildenafil	10 mg BD / 50 mg TDS	HAPE prophylaxis (alternative)	Headache, hypotension
Antitussive (central, opioid)	Morphine (slow release)	5–10 mg/day	Refractory chronic cough, palliative dyspnoea	Constipation, sedation, dependence
Neuromodulator	Gabapentin / Pregabalin	300–1800 mg/day / 75–300 mg/day	Refractory chronic cough	Dizziness, sedation; taper on stopping
P2X3 antagonist	Gefapixant	45 mg BD	Refractory chronic cough (where approved)	Taste disturbance (dysgeusia)
Antitussive (peripheral)	Levodropropizine / Dextromethorphan	60 mg TDS / 10–20 mg 4-hourly	Symptomatic dry cough	Limited evidence; avoid with SSRIs (DXM)
Mucolytic / expectorant	Guaifenesin, ambroxol	200–400 mg TDS	Productive cough	Weak evidence
Proton pump inhibitor	Pantoprazole / Omeprazole	40 mg OD × 8 weeks	Reflux-related cough (with typical symptoms)	Long-term: fractures, B12, C. difficile
Intranasal corticosteroid	Fluticasone / Mometasone nasal spray	2 sprays each nostril OD	Upper airway cough syndrome, rhinitis	Epistaxis
Antihistamine (first generation)	Chlorpheniramine	4 mg TDS	Postnasal drip (anticholinergic effect helps)	Sedation, urinary retention in elderly
Antihistamine (second generation)	Levocetirizine / Fexofenadine	5 mg OD / 120–180 mg OD	Allergic rhinitis with asthma	Minimal sedation
Vaccines	Influenza, PCV20, RSV, COVID-19, Tdap	See vaccination table	Chronic lung disease	Live vaccines contraindicated on immunosuppression

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E15.7 Obesity, Metabolic Disease & the Lung

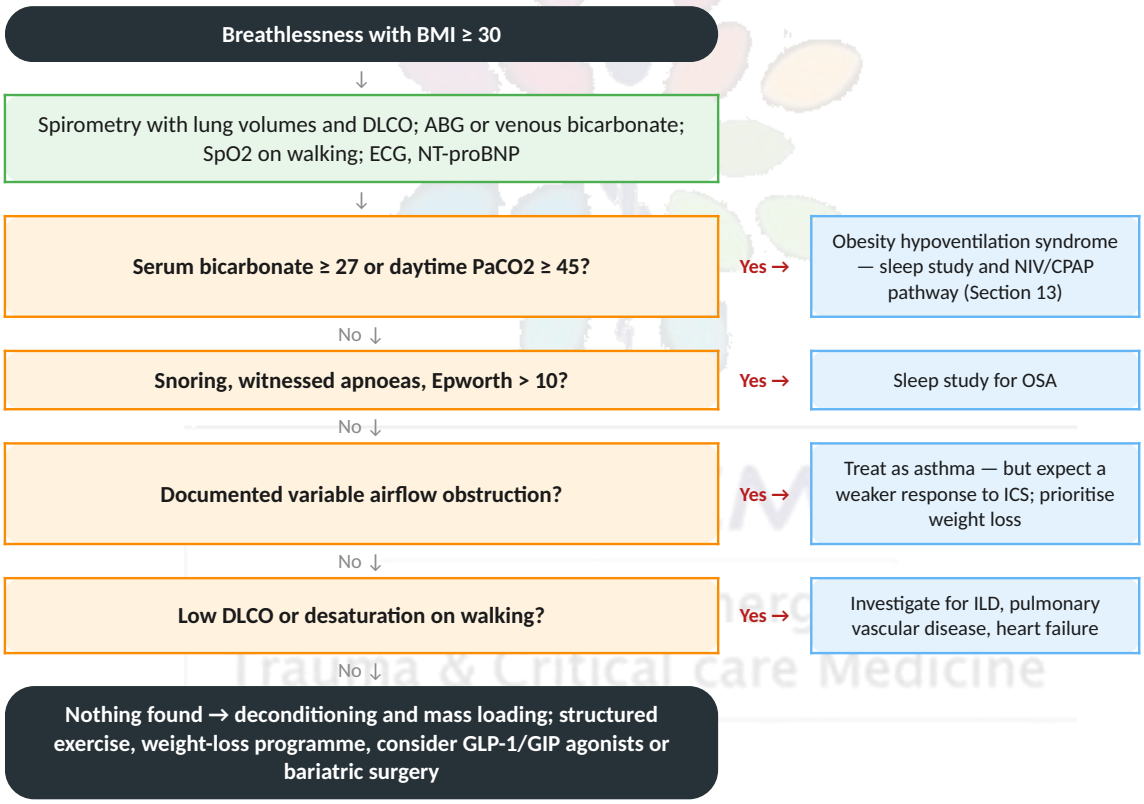
‹ Core topic 15.7 · p.259
Previous-year Qs ›

IN BRIEF Obesity is now one of the commonest reasons for breathlessness in Indian respiratory clinics. It changes physiology, mimics asthma, and drives OSA and hypoventilation.

Respiratory effects of obesity

Effect	Mechanism / consequence
↓ ERV and FRC (the earliest change)	Mass loading of the chest wall and abdomen; small-airway closure at tidal volumes
↓ Compliance, ↑ work of breathing	Chest wall and lung compliance both reduced
V/Q mismatch, basal atelectasis	Dependent airway closure — mild hypoxaemia, worse supine
↑ Oxygen consumption and CO2 production	Higher ventilatory demand
Asthma phenotype	Obesity-associated, non-eosinophilic, poorly steroid-responsive asthma
OSA and OHS	Upper-airway loading; blunted ventilatory response
GERD, deconditioning	Add to cough and dyspnoea

Algorithm E15.7.1 · The breathless obese patient



Benefits of weight loss

Loss	Effect
5–10%	Better asthma control, less GERD, improved spirometry
≥ 10%	Meaningful fall in AHI; improved daytime sleepiness
25–30% (usually bariatric surgery)	Resolution of OHS in many; large AHI reduction

★ CLINICAL PEARLS & EXAM FOCUS

- A "restrictive" spirometry in obesity usually shows low ERV and FVC with a normal or high KCO — this is extraparenchymal restriction, not ILD.
- Never attribute breathlessness to obesity alone before excluding PE, heart failure, anaemia and PH.

MERCK MANUAL PROFESSIONAL Obesity Obesity hypoventilation syndrome

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PART II · SECTION 16

Surgical Aspects of Pulmonary Medicine

Advanced theory & algorithms

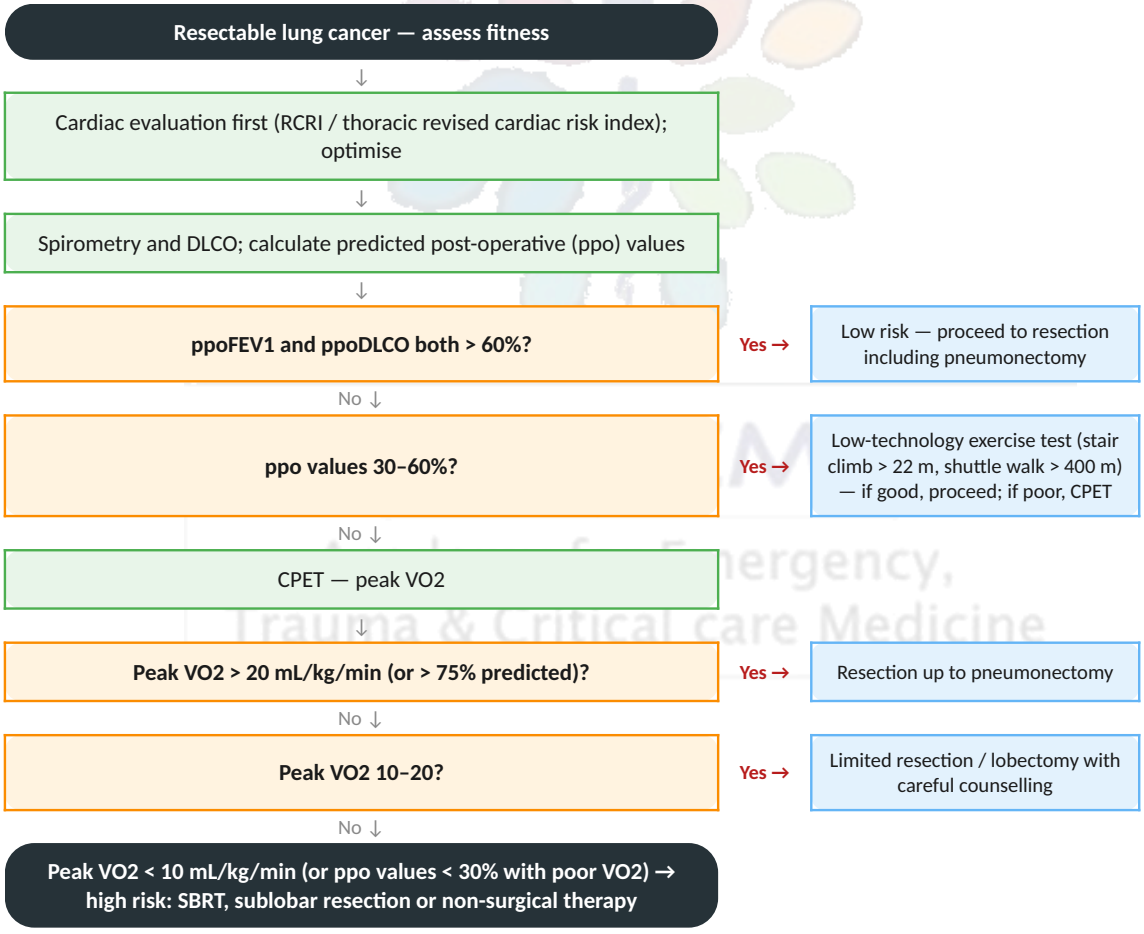
E16.1 Pre-operative Pulmonary Evaluation for Lung Resection — Three-Legged Stool 498
E16.2 Chest Trauma — Primary Survey, Thoracostomy & Operative Triggers 499
E16.3 Lung Transplantation — Referral, Listing & Complications 501
E16.4 Upper Airway Obstruction & Stridor — Emergency Algorithm 503
E16.5 Drug Chart — Peri-operative, Trauma & Transplant Drugs 504

E16.1 Pre-operative Pulmonary Evaluation for Lung Resection — Three-Legged Stool

‹ Core topic 16.1 · p.261
Previous-year Qs ›

IN BRIEF Fitness for lung resection rests on three assessments: respiratory mechanics (FEV1), gas exchange (DLCO) and cardiopulmonary reserve (exercise testing). Cardiac risk is assessed in parallel.

Algorithm E16.1.1 · Functional assessment before lung resection (ERS/ESTS, BTS)



Calculating predicted post-operative function

- **Anatomical (segment) method:** ppoFEV1 = preoperative FEV1 × (1 − segments removed / 19 functional segments).
- Lobectomy segments: RUL 3, RML 2, RLL 5, LUL 5 (incl. lingula), LLL 4.
- **Perfusion scan method** (preferred for pneumonectomy): ppoFEV1 = preoperative FEV1 × (1 − fraction of perfusion to resected lung).

Reducing peri-operative pulmonary complications

Intervention	Evidence
Smoking cessation ≥ 4 weeks before	Fewer wound and pulmonary complications
Pre-habilitation / inspiratory muscle training	Reduced atelectasis and length of stay
Lung-protective intra-operative ventilation	Fewer complications (IMPROVE)
Thoracic epidural / paravertebral analgesia	Better cough, fewer complications
Early mobilisation, chest physiotherapy, incentive spirometry	Standard ERAS element
VATS over thoracotomy	Less pain, fewer complications

★ CLINICAL PEARLS & EXAM FOCUS

- Elderly age alone is not a contraindication — physiological reserve matters.
- A DLCO < 40% predicts complications even when FEV1 is adequate — always measure DLCO.

MERCK MANUAL PROFESSIONAL Preoperative evaluation

E16.2 Chest Trauma — Primary Survey, Thoracostomy & Operative Triggers

‹ Core topic 16.2 · p.263
Previous-year Qs ›

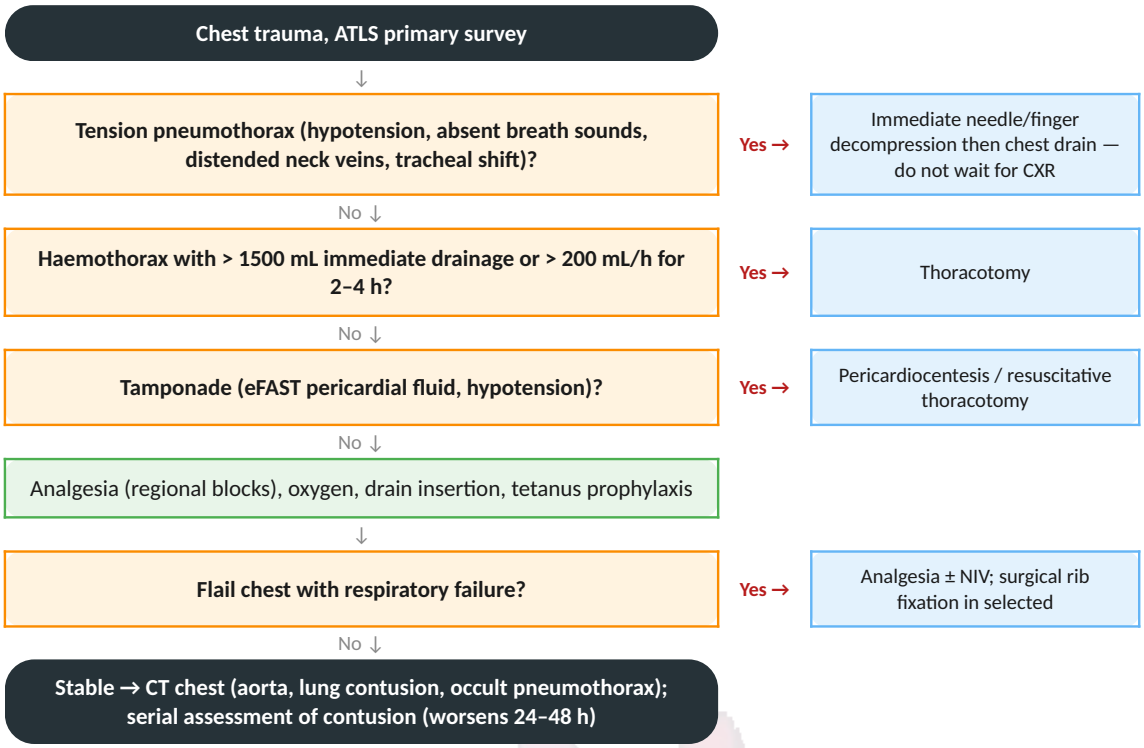
IN BRIEF Twelve immediately life-threatening injuries are found in the primary and secondary surveys. Most are treated with a chest drain and good analgesia; fewer than 10% need thoracotomy.

Life-threatening chest injuries

Primary survey ("lethal six")	Secondary survey ("hidden six")
Airway obstruction	Pulmonary contusion
Tension pneumothorax	Myocardial contusion
Open pneumothorax	Aortic injury
Massive haemothorax	Tracheobronchial injury
Flail chest with pulmonary contusion	Oesophageal rupture
Cardiac tamponade	Diaphragmatic rupture

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Algorithm E16.2.1 · Thoracic trauma — initial management



Specific injuries

Injury	Clue	Management
Tracheobronchial injury	Massive air leak, persistent pneumothorax despite drain, pneumomediastinum, "fallen lung sign"	Bronchoscopy; surgical repair
Traumatic aortic injury	Widened mediastinum, deceleration mechanism	CT angiography; endovascular repair; blood pressure control
Diaphragmatic rupture	NG tube in chest, bowel in hemithorax (left)	Surgical repair
Oesophageal rupture	Pneumomediastinum, left effusion with high amylase	Contrast study/CT; surgery
Pulmonary contusion	Non-segmental opacity within 6 h	Lung-protective support, restrictive fluids, analgesia

★ **CLINICAL PEARLS & EXAM FOCUS**

- Occult pneumothorax on CT (not visible on CXR) in a stable patient can usually be observed, even with positive-pressure ventilation, with close monitoring.
- The most effective measure in blunt chest trauma is adequate analgesia — pain causes hypoventilation and atelectasis.

RADIOPAEDIA · IMAGE LINKS Flail chest cases · Pulmonary contusion cases · Fallen lung sign cases · Traumatic aortic injury cases

MERCK MANUAL PROFESSIONAL Thoracic trauma

E16.3 Lung Transplantation — Referral, Listing & Complications

‹ Core topic 16.3 · p.264
Previous-year Qs ›

IN BRIEF Transplantation is the final option in end-stage lung disease. Early referral matters — many patients are referred too late. India performs an increasing number of lung transplants, with ILD and post-COVID fibrosis prominent indications.

When to refer (ISHLT 2021 consensus)

Disease	Refer when
IPF / fibrotic ILD	At diagnosis of fibrotic ILD (refer all), especially FVC < 80% or DLCO < 40%, any oxygen need or progression
COPD	BODE index 5–6, FEV1 < 25%, hypercapnia, PH, frequent exacerbations
Cystic fibrosis / bronchiectasis	FEV1 < 30% or rapid decline, exacerbations needing ICU, massive haemoptysis, PH
PAH	ESC/ERS intermediate-high or high risk on optimal therapy, including parenteral prostacyclin

Contraindications

Absolute	Relative
Active malignancy (most within 2 years), uncontrolled sepsis, active TB	Age > 70, BMI > 35 or < 16
Significant other organ dysfunction not correctable	Severe deconditioning, osteoporosis
Documented non-adherence, active substance use (including smoking)	Colonisation with highly resistant organisms
Severe chest wall deformity precluding surgery	Previous thoracic surgery with pleurodesis

Complications by timing

Period	Complications
Immediate (< 72 h)	Primary graft dysfunction (ischaemia–reperfusion), bleeding, hyperacute rejection
Early (weeks–months)	Acute cellular rejection, anastomotic dehiscence/stenosis, bacterial and CMV infection
Late (> 1 year)	Chronic lung allograft dysfunction (BOS — obstructive; RAS — restrictive), PTLD, skin cancer, renal failure from calcineurin inhibitors, recurrent disease (sarcoid, LAM)

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Algorithm E16.3.1 · Falling spirometry in a lung transplant recipient

FEV1 falls ≥ 10% from baseline

↓

Rule out infection (bronchoscopy with BAL, CMV PCR), anastomotic stenosis (bronchoscopy), acute rejection (transbronchial biopsy), reflux/aspiration

↓

Acute cellular rejection (perivascular infiltrates, grade ≥ A2)?

Yes → Pulse methylprednisolone 10–15 mg/kg × 3 days

No ↓

Persistent obstructive decline (FEV1 < 80% baseline > 3 weeks) after exclusions?

Yes → Bronchiolitis obliterans syndrome — azithromycin, optimise immunosuppression, consider extracorporeal photopheresis; re-transplant in selected

No ↓

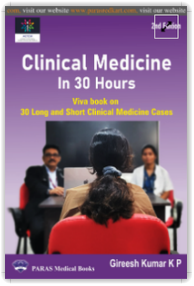
Restrictive pattern with upper-lobe fibrosis → RAS — poorer prognosis

★ CLINICAL PEARLS & EXAM FOCUS

- Median survival after lung transplantation is about 6.5 years; bilateral transplant outperforms single in most indications.
- Chronic lung allograft dysfunction remains the principal barrier to long-term survival.

MERCK MANUAL PROFESSIONAL — Lung transplantation

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E16.4 Upper Airway Obstruction & Stridor — Emergency Algorithm

‹ Core topic 16.4 · p.265
Previous-year Qs ›

IN BRIEF Stridor means the airway is critically narrowed — usually to less than 8 mm in adults. Identify the level (supraglottic, glottic, subglottic, tracheal) from the phase of stridor and the history.

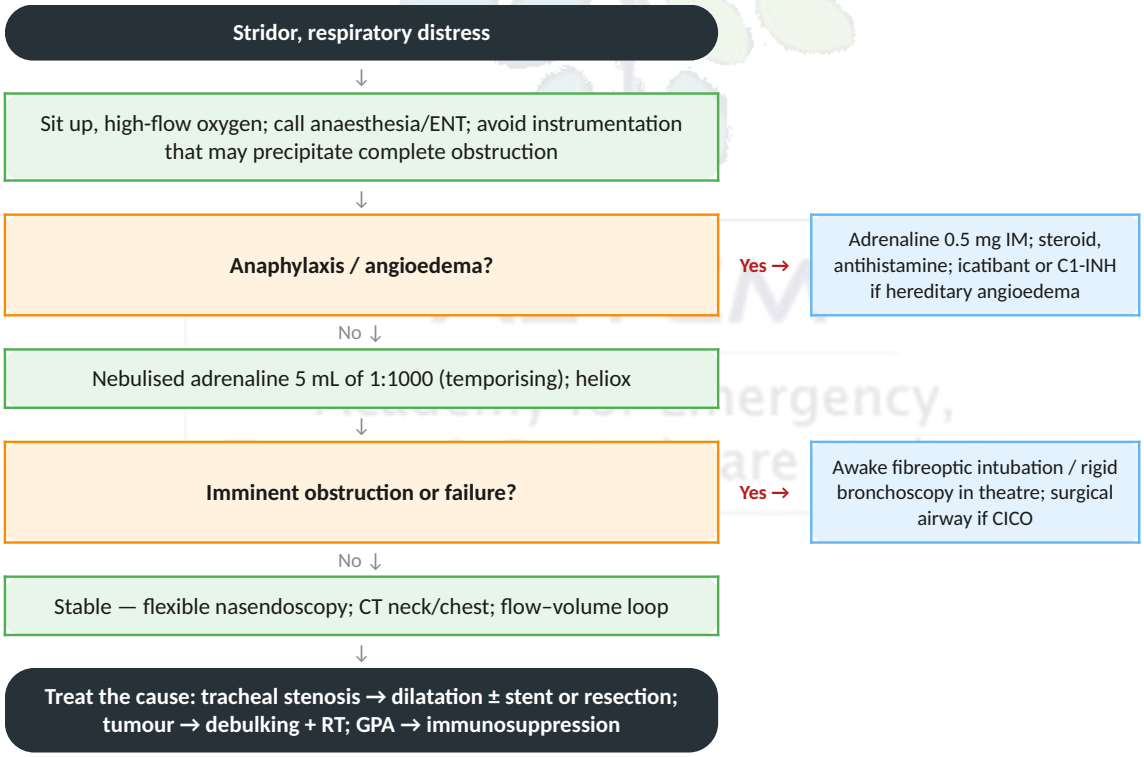
Localising the obstruction

Phase of stridor	Level
Inspiratory	Supraglottic / extrathoracic
Biphasic	Glottic / subglottic (fixed)
Expiratory (wheeze/stridor)	Intrathoracic trachea or large bronchi

Causes by onset

Acute	Chronic
Anaphylaxis, angioedema (ACE inhibitor, hereditary)	Post-intubation / post-tracheostomy stenosis
Epiglottitis, croup, retropharyngeal abscess	Laryngeal or tracheal tumour, thyroid goitre
Foreign body, trauma, burns	Vocal cord palsy, inducible laryngeal obstruction
Bilateral vocal cord palsy (post-thyroidectomy)	Granulomatosis with polyangiitis (subglottic stenosis), amyloid, relapsing polychondritis, tracheomalacia

Algorithm E16.4.1 · Acute stridor in an adult



★ CLINICAL PEARLS & EXAM FOCUS

- A flow–volume loop becomes abnormal when the airway narrows to about 8 mm; stridor appears at about 5 mm.
- Angioedema from ACE inhibitors may occur years after starting; it does not respond to antihistamines or steroids — secure the airway.

RADIOPAEDIA · IMAGE LINKS Tracheal stenosis cases Subglottic stenosis cases

MERCK MANUAL PROFESSIONAL Upper airway obstruction

E16.5 Drug Chart — Peri-operative, Trauma & Transplant Drugs

Previous-year Qs ›

Drugs in surgical pulmonary practice

Class	Drug	Dose	Indication	Caution
Calcineurin inhibitor	Tacrolimus	0.05–0.1 mg/kg/day (trough 8–12 early)	Lung transplant maintenance	Nephrotoxicity, tremor, diabetes, azole interactions
Antiproliferative	Mycophenolate mofetil	1 g BD	Transplant maintenance	Leucopenia, diarrhoea
Corticosteroid	Prednisolone	0.5 mg/kg tapering to 5–10 mg	Transplant maintenance; rejection	Diabetes, osteoporosis
Corticosteroid (pulse)	Methylprednisolone	10–15 mg/kg IV × 3 days	Acute cellular rejection	Hyperglycaemia, infection
Induction agent	Basiliximab	20 mg IV days 0 and 4	Transplant induction	Hypersensitivity
Antiviral	Valganciclovir	900 mg OD (prophylaxis)	CMV prophylaxis after transplant	Marrow suppression
Antifungal	Inhaled amphotericin / voriconazole	Per protocol	Aspergillus prophylaxis after transplant	Voriconazole raises tacrolimus levels (reduce dose)
Macrolide	Azithromycin	250 mg thrice weekly	BOS prevention and treatment	QT
Analgesic (regional)	Bupivacaine 0.25–0.5%	Paravertebral/epidural infusion or serratus block	Rib fractures, thoracotomy	Local anaesthetic toxicity
Analgesic	Paracetamol + NSAID + opioid (multimodal)	Paracetamol 1 g QDS	Chest trauma, post-thoracotomy	NSAID in renal impairment, elderly
Antifibrinolytic	Tranexamic acid	1 g IV over 10 min then 1 g over 8 h	Major trauma within 3 h (CRASH-2)	Thrombosis
Adrenergic	Adrenaline	0.5 mg IM (anaphylaxis); 5 mL 1:1000 nebulised	Anaphylaxis, stridor (temporising)	Arrhythmia, hypertension
Bradykinin B2 antagonist	Icatibant	30 mg SC	Hereditary angioedema	Injection-site reaction
C1-esterase inhibitor	C1-INH concentrate	20 U/kg IV	Hereditary angioedema	Cost
Antibiotic prophylaxis	Cefazolin ± vancomycin	2 g IV at induction	Thoracic surgery	Allergy, MRSA colonisation

Class	Drug	Dose	Indication	Caution
Anticoagulant	Enoxaparin	40 mg SC OD	VTE prophylaxis after thoracic surgery	Bleeding, epidural catheter timing

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PART II · SECTION 17

Research Methodology, Epidemiology & Biostatistics

Advanced theory & algorithms

E17.1 Study Designs, Bias & Confounding — Applied to Respiratory Research 506
E17.2 Biostatistics for the Pulmonologist — Tests, Sample Size & Diagnostic Accuracy 507
E17.3 Critical Appraisal, Trial Ethics & Writing the Thesis Protocol 509
E17.4 Drug Chart — Pharmacovigilance, Drug Trials & Reporting in India 512

E17.1 Study Designs, Bias & Confounding — Applied to Respiratory Research

‹ Core topic 17.1 · p.267
Previous-year Qs ›

IN BRIEF Thesis protocols and paper-one questions both test the same skill: match the question to the right design, and name the biases that threaten it.

Study designs — strengths and weaknesses

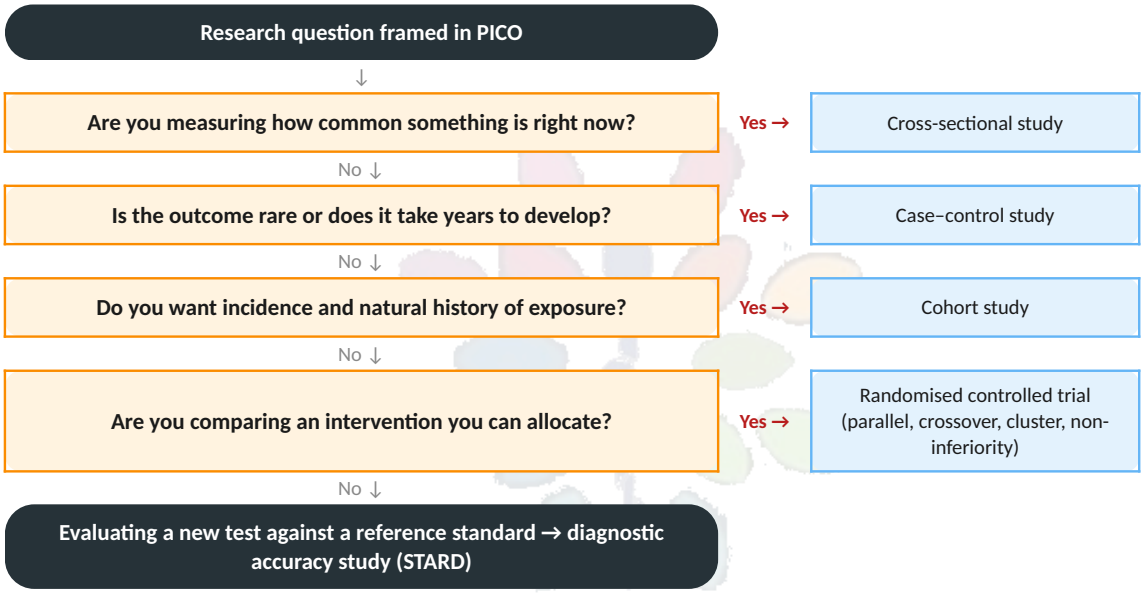
Design	Measures	Strength	Weakness
Cross-sectional	Prevalence; odds ratio	Quick, cheap; good for burden of COPD/ asthma surveys	No temporality; not for rare disease
Case–control	Odds ratio	Efficient for rare diseases (mesothelioma) and long latency	Recall and selection bias; cannot give incidence
Cohort (prospective)	Incidence; relative risk	Temporality clear; multiple outcomes	Expensive, long, loss to follow-up; poor for rare outcomes
Retrospective cohort	Relative risk	Faster, uses records	Data quality, missing confounders
Randomised controlled trial	Efficacy; RR, ARR, NNT	Controls known and unknown confounders	Cost, ethics, external validity
Diagnostic accuracy (cross-sectional)	Sensitivity, specificity, LR	Validates new tests (CBNAAT, LF-LAM)	Spectrum and verification bias
Systematic review / meta-analysis	Pooled effect	Highest level of evidence	Publication bias, heterogeneity

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Biases to name in a viva

Bias	Example in respiratory research
Selection bias	Hospital-based controls in a TB case–control study
Recall bias	Asking mesothelioma patients about asbestos exposure
Information / misclassification	Self-reported smoking; non-differential misclassification dilutes effect
Lead-time and length-time bias	Lung cancer screening studies
Immortal time bias	Cohort studies of inhaled therapy in COPD
Confounding	Smoking confounds the relationship between occupation and lung cancer
Berkson's bias	Hospital admission rates distort associations
Healthy worker effect	Occupational cohorts appear healthier than the general population
Publication bias	Funnel plot asymmetry; Egger's test

Algorithm E17.1.1 · Choosing a study design for a thesis question



★ CLINICAL PEARLS & EXAM FOCUS

- Confounding can be controlled at design (randomisation, restriction, matching) or at analysis (stratification, multivariable regression, propensity scores).
- Reporting guidelines to quote: CONSORT (RCT), STROBE (observational), PRISMA (reviews), STARD (diagnostic), SPIRIT (protocols).

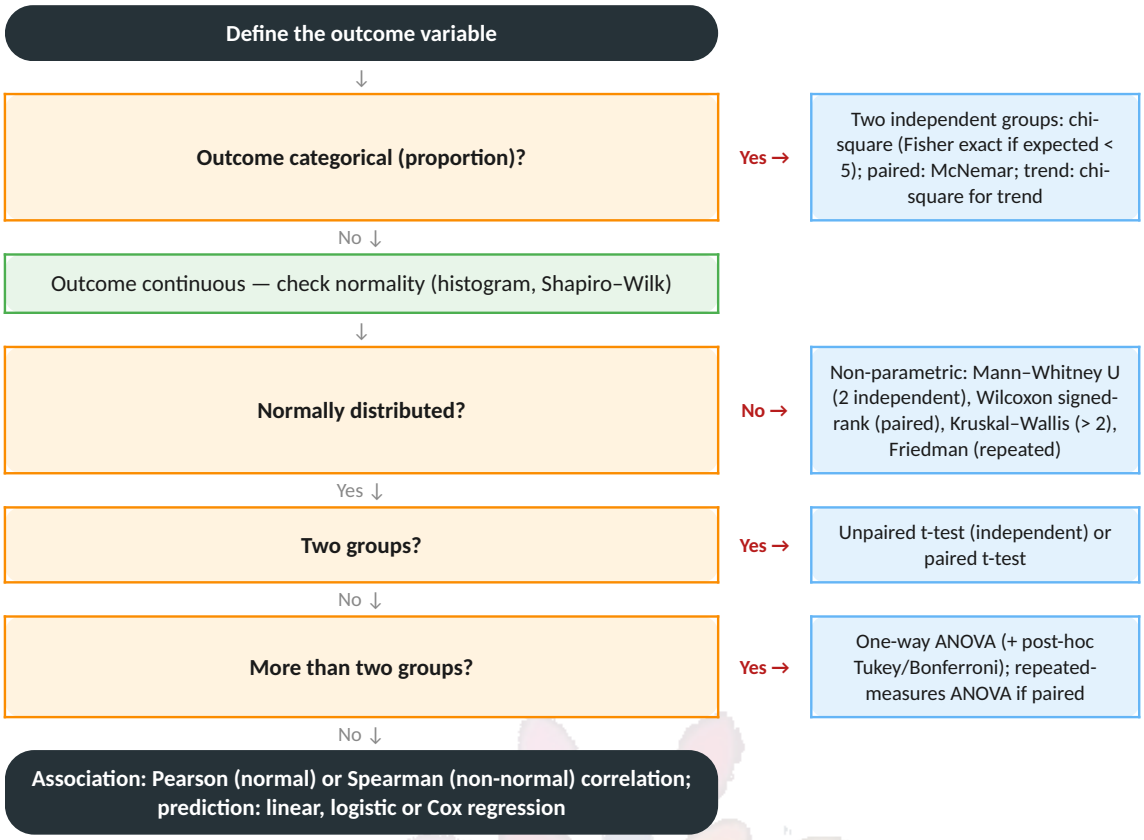
MERCK MANUAL PROFESSIONAL Clinical research

E17.2 Biostatistics for the Pulmonologist — Tests, Sample Size & Diagnostic Accuracy

‹ Core topic 17.2 · p.268
Previous-year Qs ›

IN BRIEF Three questions decide the test: what type of data, how many groups, and are the groups paired?

Algorithm E17.2.1 · Choosing a statistical test



Diagnostic test characteristics — worked example

Measure	Formula	Note
Sensitivity	TP / (TP + FN)	A sensitive test when negative rules OUT (SnNOut)
Specificity	TN / (TN + FP)	A specific test when positive rules IN (SpPIn)
PPV / NPV	TP/(TP+FP) ; TN/(TN+FN)	Depend on prevalence
Positive likelihood ratio	Sens / (1 − Spec)	> 10 strong rule-in
Negative likelihood ratio	(1 − Sens) / Spec	< 0.1 strong rule-out
Accuracy	(TP + TN) / total	Misleading when prevalence is low
Youden index	Sens + Spec − 1	Chooses the optimal cut-off on ROC
AUC (c-statistic)	Area under ROC	0.5 useless; > 0.8 good; 1.0 perfect

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Sample size — what you must state in a protocol

Element	Example
Primary outcome and its measure	Proportion culture-converted at 2 months
Expected values	70% control vs 85% intervention
Alpha (type I error)	0.05, two-sided
Power (1 – beta)	80% or 90%
Effect size / clinically important difference	15% absolute difference
Allowance for loss to follow-up	+10–15%
Software / formula quoted	OpenEpi, G*Power, nMaster, or standard formula

Measures of effect and association

Measure	Interpretation
Relative risk (RR)	Risk ratio; cohort/RCT
Odds ratio (OR)	Case–control; approximates RR when disease is rare
Absolute risk reduction (ARR)	Control event rate – experimental event rate
Number needed to treat (NNT)	1 / ARR
Hazard ratio	Time-to-event (Cox model)
Kappa	Agreement beyond chance (< 0.2 poor; > 0.8 very good)
Bland–Altman	Agreement between two measurement methods (e.g. SpO2 vs SaO2)

★ CLINICAL PEARLS & EXAM FOCUS

- A p value tells you about chance, not clinical importance — always report the confidence interval and the effect size.
- Intention-to-treat analysis preserves randomisation and is the primary analysis for superiority trials; per-protocol is used alongside for non-inferiority.

MERCK MANUAL PROFESSIONAL Biostatistics

E17.3 Critical Appraisal, Trial Ethics & Writing the Thesis Protocol

‹ Core topic 17.3 · p.270
Previous-year Qs ›

IN BRIEF Examiners ask you to appraise a paper in minutes and to defend your own protocol. The same structure serves both.

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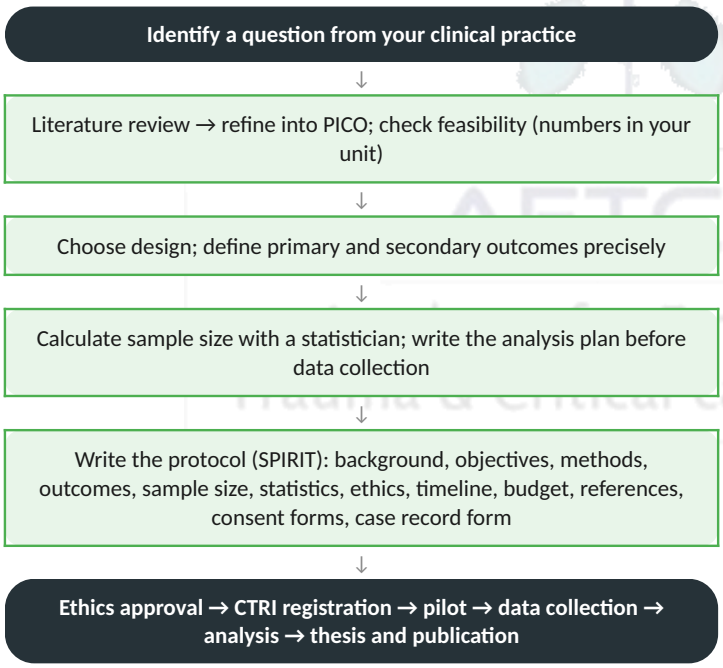
Appraising a randomised trial

Question	What to look for
Was randomisation truly random and concealed?	Computer sequence; sealed opaque envelopes or central allocation
Were groups similar at baseline?	Table 1
Was blinding adequate?	Participants, clinicians, assessors, analysts
Were all patients accounted for?	CONSORT flow diagram; intention-to-treat
Was follow-up complete and long enough?	< 20% loss; outcome timing
Was the primary outcome patient-important?	Mortality or exacerbations rather than surrogate
Are the results precise and applicable?	Confidence intervals; population similar to yours
Was the trial stopped early?	Early stopping exaggerates effect

Ethics essentials for a thesis

Element	Requirement
Approval	Institutional Ethics Committee registered with CDSCO/NECRBHR; approval before recruitment
Regulation	New Drugs and Clinical Trials Rules 2019; ICMR National Ethical Guidelines 2017; Declaration of Helsinki
Registration	CTRI (Clinical Trials Registry – India) before the first patient is enrolled
Consent	Written informed consent in the participant's language; audio-visual consent for vulnerable groups and new drug trials
Core principles	Autonomy, beneficence, non-maleficence, justice; respect for persons
Special groups	Children (assent ≥ 7 years + parental consent), pregnant women, prisoners, the unconscious (legally acceptable representative)
Data	Confidentiality, anonymisation, data-sharing statement, compensation for trial-related injury

Algorithm E17.3.1 · Writing a thesis protocol



MODEL ANSWER FRAMEWORK — *"Critically evaluate the use of a new diagnostic test in your department." (10 marks)*

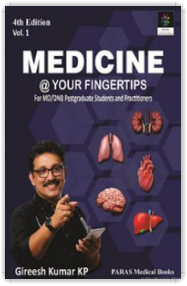
1. Define the clinical problem and the current reference standard.
2. Study design: cross-sectional diagnostic accuracy, consecutive recruitment, blinded comparison.
3. Define index test, reference standard, and timing; sample size for expected sensitivity.
4. Present a 2 × 2 table; calculate sensitivity, specificity, PPV, NPV, likelihood ratios with 95% CI.
5. Discuss spectrum bias, verification bias, prevalence effect on predictive values.
6. Decide on implementation: cost, turnaround time, infrastructure, programme fit (e.g. CBNAAT under NTEP).

★ CLINICAL PEARLS & EXAM FOCUS

- Levels of evidence: systematic review of RCTs > RCT > cohort > case–control > case series > expert opinion.
- GRADE grades the certainty of evidence (high, moderate, low, very low) and the strength of recommendations (strong, conditional).

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E17.4 Drug Chart — Pharmacovigilance, Drug Trials & Reporting in India

Previous-year Qs ›

Regulatory and pharmacovigilance framework

Element	Detail	Practical point	Caution
Regulator	CDSCO (Central Drugs Standard Control Organisation)	Approves new drugs and clinical trials	Phase-wise approval
Programme	PvPI (Pharmacovigilance Programme of India), IPC Ghaziabad	ADR Monitoring Centres (AMC) in medical colleges	Report even suspected reactions
Reporting form	Suspected ADR Reporting Form (red form); ADR PvPI mobile app; helpline 1800-180-3024	Patient details, suspected drug, reaction, outcome, de-challenge/re-challenge	Causality by WHO-UMC scale / Naranjo score
TB-specific	aDSM (active drug safety monitoring and management) in Ni-kshay	Mandatory for all DR-TB patients on newer drugs	Monthly reporting
Device-related	Materiovigilance Programme of India (MvPI)	Report device failures (ventilators, oxygen concentrators, stents)	Under-reported
Serious adverse event	Death, life-threatening, hospitalisation, disability, congenital anomaly	Report within 24 h to sponsor/IEC/CDSCO; analysis within 14 days	Compensation per NDCT Rules 2019
Causality assessment	WHO-UMC: certain, probable, possible, unlikely, conditional, unassessable	Naranjo score ≥ 9 definite, 5–8 probable, 1–4 possible	Document in case notes
Trial phases	Phase I safety; II efficacy/dose; III comparative; IV post-marketing	Bioequivalence for generics	Phase 0 microdosing

★ **CLINICAL PEARLS & EXAM FOCUS**

- Every respiratory unit should report ADRs to anti-TB drugs, biologics and oxygen devices — India's reporting rate remains low.
- Know the three Indian registries for your exam: CTRI (trials), PvPI (drugs), MvPI (devices).

MERCK MANUAL PROFESSIONAL — Adverse drug reactions

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PART II · SECTION 18

Clinical Examination in Respiratory Medicine — Long Case, Short Case & Viva

Advanced theory & algorithms

E18.1 Respiratory History Taking for the Long Case 513
E18.2 General Physical Examination — Respiratory Focus 514
E18.3 Examination of the Respiratory System — Inspection & Palpation 516
E18.4 Examination — Percussion, Auscultation & Putting It Together 518
E18.5 Presenting the Long Case & Common Viva Questions 520
E18.6 Short Cases, Spotters & Procedure Stations 522
E18.7 Rapid Revision — One-Page Answers to the Twenty Commonest Questions 525
E18.8 Bedside Interpretation Stations — ABG, CXR, Spirometry & Ventilator Graphics 526

E18.1 Respiratory History Taking for the Long Case

IN BRIEF The examiner listens for a structured, chronological story with the differential already implied by the questions you asked. Finish the history knowing your diagnosis, its severity, its cause and its effect on the patient's life.

Structure of the respiratory history

Section	Content
Identification	Name, age, sex, occupation (ask in detail — dust, fumes, birds), residence (urban/rural, biomass fuel)
Presenting complaints	In the patient's own words with duration, in chronological order
History of present illness	Each symptom analysed: onset, duration, progression, diurnal and seasonal variation, aggravating and relieving factors, associated symptoms, treatment taken and response
Negative history	Deliberately record relevant negatives — no haemoptysis, no orthopnoea, no joint pain
Past history	TB and its treatment (regimen, duration, completion), measles/pertussis in childhood, asthma, diabetes, hypertension, surgery, blood transfusion
Treatment history	Inhalers (name, device, technique, adherence), steroids, ATT, oxygen, nebulisation, alternative medicine
Personal history	Smoking (pack-years, bidi, hookah, chewing), alcohol, diet, sleep, bowel/bladder, appetite, weight change
Family history	TB contact, asthma/atopy, COPD in non-smokers (AATD), cystic fibrosis, malignancy
Occupational and environmental	Every job held, dust/fume, ventilation, masks, duration, co-workers affected; pets and birds, air-conditioners, coolers, damp housing
Socioeconomic	Income, crowding, ventilation at home, cooking fuel, education, insurance, distance to hospital
Menstrual/obstetric	Where relevant (LAM, catamenial pneumothorax, pregnancy)

Analysing the six cardinal symptoms

Symptom	Points to elicit
Cough	Duration, dry or productive, diurnal variation, postural, bovine (vocal cord palsy), paroxysmal, relation to food (aspiration, fistula)
Sputum	Quantity (cupfuls — bronchiectasis), colour, odour (anaerobes), layering on standing (three layers — bronchiectasis), plugs (ABPA), blood
Haemoptysis	Volume, frequency, pure blood or streaking, precipitants; differentiate from haematemesis and epistaxis
Breathlessness	Onset (sudden — pneumothorax, PE), progression, mMRC grade, orthopnoea, PND, platypnoea, diurnal/seasonal variation, exercise tolerance in metres and stairs
Chest pain	Pleuritic vs constant, site, radiation, relation to respiration and posture
Wheeze	Episodic, nocturnal, triggers (dust, cold, exercise, smoke), monophonic vs polyphonic

Grading breathlessness — mMRC scale

Grade	Description
0	Breathless only on strenuous exercise
1	Short of breath hurrying on level ground or walking up a slight hill
2	Walks slower than people of the same age, or stops for breath walking at own pace
3	Stops for breath after about 100 metres or after a few minutes on level ground
4	Too breathless to leave the house, or breathless on dressing/undressing

★ CLINICAL PEARLS & EXAM FOCUS

- Always quantify smoking in pack-years and ask separately about bidis, hookah and smokeless tobacco — patients often deny "smoking" while using bidis.
- Ask one question about the effect of illness on income and schooling — examiners reward this in the social history.
- End the history with a one-line summary: "A 48-year-old farmer, non-smoker, with 6 months of progressive breathlessness and dry cough, with bird exposure at home…"

E18.2 General Physical Examination — Respiratory Focus

IN BRIEF Before the chest, the hands, face, neck and legs give the diagnosis away. Present findings in a fixed order and never omit the lymph nodes.

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General examination sequence

Step	What to look for
Build, nourishment, posture	Cachexia (malignancy, TB, COPD), obesity (OHS, OSA), tripod position, pursed-lip breathing
Consciousness and orientation	CO2 narcosis, hypoxic confusion
Vitals	Pulse (rate, rhythm, pulsus paradoxus, bounding in CO2 retention), BP, respiratory rate and pattern, temperature, SpO2 (room air and on exertion)
Pallor, icterus, cyanosis	Central cyanosis (tongue, SpO2 < 85%) vs peripheral
Clubbing	Grade I–IV; causes below
Lymph nodes	Cervical, supraclavicular (Virchow), axillary — size, consistency, matting, sinus
Pedal oedema, raised JVP	Cor pulmonale; SVC obstruction (non-pulsatile, fixed)
Skin and joints	Erythema nodosum, lupus pernio (sarcoid), sclerodactyly, rash, arthritis, scars

Respiratory causes of clubbing — and the important exceptions

Clubbing present	Clubbing absent (important negatives)
Bronchogenic carcinoma, mesothelioma	**COPD / emphysema — clubbing is NOT a feature; look for another cause**
Bronchiectasis, lung abscess, empyema	**Uncomplicated asthma**
Idiopathic pulmonary fibrosis, asbestosis	Pneumonia (acute)
Cystic fibrosis	Simple pneumothorax
Chronic cavitary TB (not uncomplicated TB)	Chronic bronchitis without bronchiectasis

Patterns of respiration

Pattern	Association
Tachypnoea	Fever, hypoxaemia, acidosis, anxiety, restrictive disease
Kussmaul	Deep sighing — metabolic acidosis
Cheyne–Stokes	Crescendo–decrescendo with apnoea — heart failure, stroke, high altitude
Biot (ataxic)	Irregular with apnoea — brainstem lesion, opioids
Prolonged expiration with pursed lips	Airflow obstruction
Paradoxical abdominal movement	Diaphragm fatigue or paralysis
Accessory muscle use, intercostal indrawing, tracheal tug	Severe airflow obstruction

Signs of CO2 retention and respiratory failure

Sign	Comment
Flapping tremor (asterixis)	Hypercapnia (also uraemia, hepatic failure)
Bounding pulse, warm peripheries	CO2 vasodilatation
Drowsiness, headache, papilloedema	Severe hypercapnia
Central cyanosis	Hypoxaemia; absent in anaemia, exaggerated in polycythaemia

E18.3 Examination of the Respiratory System — Inspection & Palpation

IN BRIEF Expose the chest fully from the neck to the waist, with the patient at 45°, in good light, with privacy and consent. Compare the two sides at every step.

Inspection

Element	Findings and meaning
Shape of chest	Barrel (COPD — AP:transverse > 0.9), pectus excavatum/carinatum, kyphoscoliosis, flattening (fibrosis, collapse), bulging (effusion, pneumothorax)
Symmetry and movement	Diminished on the affected side; count respiratory rate for a full minute
Trachea and apex beat	Visible deviation
Intercostal spaces	Indrawing (obstruction), fullness (effusion, mass)
Skin	Scars (thoracotomy, drain, BCG), sinuses, dilated veins (direction of flow — SVC obstruction), radiation marks, herpes zoster
Spine and shoulders	Scoliosis, drooping shoulder (fibrosis)
Apical impulse, epigastric pulsation	Cor pulmonale (left parasternal heave)

Palpation — sequence

Step	Technique and significance
Trachea	Three-finger method and Trail's sign (prominence of clavicular head of sternomastoid on the side of shift); shifts **towards** collapse/fibrosis, **away from** effusion/pneumothorax
Apex beat	Confirms mediastinal shift; may be impalpable in emphysema, effusion
Chest expansion	Upper (clavicular), lower (hands around the lower chest, thumbs in the midline); normal ≥ 5 cm; measure with tape at nipple level
Tactile vocal fremitus	Increased in consolidation; decreased in effusion, pneumothorax, collapse with blocked bronchus, thick chest wall
Tenderness, crepitus	Rib fracture, surgical emphysema, costochondritis
Lymph nodes and liver	Hepatomegaly may be pushed down in emphysema

Mediastinal shift at a glance

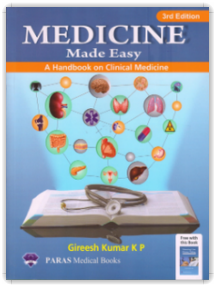
Shift towards the lesion	Shift away from the lesion	No shift
Collapse (obstructive), fibrosis, pneumonectomy, agenesis	Massive pleural effusion, tension pneumothorax, large mass	Consolidation, small effusion, bronchiectasis without collapse

★ **CLINICAL PEARLS & EXAM FOCUS**

- Always confirm tracheal position by two methods (palpation and Trail's sign) — an examiner's favourite.
- Measure chest expansion with a tape and quote the number; "reduced expansion on the right" is stronger with "3 cm versus 5 cm".

ULTRASOUND VIDEO LINKS ▶ Respiratory clinical examination — AETCM

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E18.4 Examination — Percussion, Auscultation & Putting It Together

IN BRIEF Percuss symmetrically from apex to base including the axillae, then auscultate the same areas and finish with vocal resonance. The combination of findings, not any single sign, makes the diagnosis.

Percussion

Note	Causes
Resonant	Normal
Hyper-resonant	Pneumothorax, emphysema, large bulla
Impaired / dull	Consolidation, collapse, fibrosis, thickened pleura
Stony dull	Pleural effusion
Tympanitic	Large cavity, diaphragmatic hernia

- **Areas to percuss:** supraclavicular (Kronig's isthmus), clavicle (direct), infraclavicular, mammary, inframammary, axillary, infra-axillary, suprascapular, interscapular, infrascapular.
- **Liver dullness:** normally upper border at 5th intercostal space in the right mid-clavicular line — pushed down in emphysema and obliterated in pneumoperitoneum.
- **Traube's space:** obliterated in left pleural effusion, splenomegaly, gastric tumour.

Auscultation — breath sounds

Sound	Characteristics	Causes
Normal vesicular	Inspiration longer, no gap, expiration soft	Normal lung
Diminished vesicular	Reduced intensity	Effusion, pneumothorax, collapse, emphysema, thick chest wall
Bronchial	Expiration = or > inspiration, with a gap; high-pitched	Consolidation (tubular), above effusion (tubular/aegophonic), cavity (cavernous), fibrosis, pneumonectomy
Prolonged expiration	—	Airflow obstruction

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Added sounds

Sound	Nature	Causes
Coarse crackles (early inspiratory)	Discontinuous, low-pitched, change with cough	Bronchiectasis, chronic bronchitis, resolving pneumonia
Fine crackles (late inspiratory, velcro)	High-pitched, do not clear with cough	ILD/IPF, pulmonary oedema, early pneumonia
Wheeze (rhonchi)	Continuous, musical, expiratory; polyphonic	Asthma, COPD
Monophonic fixed wheeze	Single-pitch, localised	Tumour, foreign body, bronchial stenosis
Stridor	Inspiratory, loudest over the neck	Upper airway obstruction
Pleural rub	Creaking, both phases, unaltered by cough, painful	Pleurisy, infarction, early effusion
Crunching (Hamman's)	Synchronous with heartbeat	Pneumomediastinum
Succussion splash	Splashing on shaking	Hydropneumothorax
Post-tussive suction / crackles	After cough	Cavity, bronchiectasis

Vocal resonance and special tests

Test	Technique	Positive in
Vocal resonance	"Ninety-nine" / "one-one-one"	Increased: consolidation, cavity, fibrosis. Decreased: effusion, pneumothorax, collapse
Bronchophony	Clear, loud voice	Consolidation
Whispering pectoriloquy	Whispered words heard clearly	Consolidation, cavity — most specific
Aegophony	Nasal "bleating" quality; "ee" sounds like "ay"	Top of a pleural effusion, consolidation
Coin test	Tapping a coin on the chest, auscultating the back — bell-like ring	Pneumothorax
Shifting dullness in chest	Changing position	Hydropneumothorax

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Classic combinations — the examiner's table

Condition	Trachea/ mediastinum	Expansion	Percussion	Breath sounds	Vocal fremitus/ resonance	Added sounds
Consolidation	Central	↓ same side	Dull	Bronchial	Increased	Crackles, rub
Pleural effusion	Away	↓	Stony dull	Absent (bronchial above)	Decreased	Rub above; aegophony
Pneumothorax	Away (tension)	↓	Hyper-resonant	Absent	Decreased	Coin test positive
Collapse (obstructive)	Towards	↓	Dull	Absent/ diminished	Decreased	None
Collapse (non-obstructive, compressive)	Towards	↓	Dull	Bronchial	Increased	—
Fibrosis	Towards	↓	Dull	Bronchial or vesicular	Increased	Fine crackles
Bronchiectasis	Central (or towards)	↓	Normal/dull	Normal/bronchial	Normal/ increased	Coarse crackles, clubbing
COPD / emphysema	Central	↓ bilateral	Hyper-resonant	Diminished, prolonged expiration	Decreased	Wheeze; liver pushed down
Hydropneumothorax	Away	↓	Dull below, hyper-resonant above	Absent/amphoric	Decreased	Succussion splash

★ **CLINICAL PEARLS & EXAM FOCUS**

- Whispering pectoriloquy is the single most specific sign of consolidation — demonstrate it in the viva.
- In a suspected effusion, always look for the aegophony band at the upper border; it earns marks.

ULTRASOUND VIDEO LINKS ▶ Respiratory clinical examination — AETCM

E18.5 Presenting the Long Case & Common Viva Questions

IN BRIEF Marks are given for a confident, structured presentation and for a diagnosis stated with its severity, aetiology, complications and functional impact — not merely a disease label.

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How to present (5–7 minutes)

Step	Content
Opening line	Age, sex, occupation, residence, presenting complaint with duration
History	Chronological narrative; relevant positives and negatives; treatment and response
Risk factors	Smoking, exposure, contact, atopy, comorbidity
General examination	Vitals, clubbing, cyanosis, nodes, oedema
Respiratory system	Inspection, palpation, percussion, auscultation — as positive findings with their side and level
Other systems	Cardiovascular (cor pulmonale), abdomen, CNS
Summary	Two or three sentences restating the key features
Diagnosis	"A clinical diagnosis of … with … (severity), likely due to …, with complications of …, functionally mMRC grade …"
Differential diagnosis	Two or three, with the reason for and against each
Investigations	Confirm diagnosis → assess severity → find aetiology → look for complications → baseline before therapy
Management	Non-pharmacological, pharmacological, treat the cause, prevent complications, rehabilitation, follow-up, counselling

Investigation framework to quote in any case

Purpose	Examples
To confirm the diagnosis	CXR, HRCT, spirometry, pleural fluid analysis, sputum NAAT
To assess severity	ABG, PFT, 6MWT, SpO2, echocardiography (PH), BODE index
To find the cause	Sputum culture and AFB/NAAT, serology (ANA, RF, myositis panel), IgE and Aspergillus IgG, occupational assessment
To detect complications	Cor pulmonale (ECG, echo), polycythaemia, secondary infection, pneumothorax
Baseline before treatment	LFT and uric acid before ATT, vision before ethambutol, TB screening before biologics, ECG before QT-prolonging drugs

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Frequently asked viva questions with one-line answers

Question	Answer
Why is the trachea shifted towards the lesion here?	Loss of volume — collapse or fibrosis pulls the mediastinum towards the affected side
How do you clinically differentiate effusion from consolidation?	Both are dull; effusion has absent breath sounds and reduced fremitus with mediastinal shift away, consolidation has bronchial breathing with increased fremitus and no shift
What is Trail's sign?	Prominence of the clavicular head of sternocleidomastoid on the side to which the trachea is deviated
What is Kronig's isthmus?	The band of resonance over the lung apex — narrowed in apical fibrosis or consolidation
What is amphoric breathing?	Hollow, blowing bronchial breath sound heard over a large superficial cavity or bronchopleural fistula
Causes of bilateral fine end-inspiratory crackles?	Interstitial lung disease, pulmonary oedema, bronchiectasis (coarse), early pneumonia
How do you grade clubbing?	I fluctuation, II loss of angle, III parrot-beak/drumstick, IV hypertrophic osteoarthropathy
Define massive haemoptysis	More than 100–600 mL in 24 h, or any amount causing airway obstruction or haemodynamic compromise
What is Horner's syndrome in a chest case?	Ptosis, miosis, anhidrosis, enophthalmos — sympathetic chain involvement by an apical (Pancoast) tumour
Causes of a dull percussion note with absent breath sounds?	Pleural effusion, thickened pleura, collapse with obstructed bronchus, massive consolidation with blocked bronchus

MODEL ANSWER FRAMEWORK — *"A patient with chronic cough, sputum and clubbing" — how to build the answer*

1. Summarise: chronic productive cough with copious sputum, clubbing, coarse crackles → bronchiectasis.
2. Give the differential: post-TB bronchiectasis, ABPA, cystic fibrosis, primary ciliary dyskinesia, immunodeficiency, foreign body.
3. Investigations: HRCT (bronchoarterial ratio > 1), sputum culture including AFB and fungal, immunoglobulins, Aspergillus IgE/IgG, sweat chloride, nasal NO.
4. Management: airway clearance, exacerbation treatment, eradication of Pseudomonas, macrolide for frequent exacerbations, vaccination, rehabilitation, surgery for localised disease.
5. Complications and counselling: haemoptysis, cor pulmonale, amyloidosis; adherence to physiotherapy.

★ CLINICAL PEARLS & EXAM FOCUS

- Never present a negative you did not look for — examiners test this.
- If you miss a sign, say so honestly and offer to re-examine; fabricated findings lose more marks than missed ones.

E18.6 Short Cases, Spotters & Procedure Stations

IN BRIEF The short case tests speed and accuracy: examine the system in 4–5 minutes, state the findings and give a diagnosis with one or two differentials.

Common short cases and the lead signs

Short case	Lead signs
Pleural effusion	Stony dullness, absent breath sounds, shift away, aegophony at the top
Pneumothorax	Hyper-resonance, absent breath sounds, shift away if tension
Consolidation	Bronchial breathing, increased fremitus, whispering pectoriloquy
Fibrothorax / post-TB fibrosis	Flattened hemithorax, shift towards, crowded ribs, bronchial breathing
COPD	Barrel chest, hyper-resonance, diminished sounds, pursed lips, prolonged expiration
Bronchiectasis	Clubbing, coarse crackles, copious sputum
ILD	Clubbing, fine velcro crackles at bases, tachypnoea, exertional desaturation
Pneumonectomy / lobectomy	Thoracotomy scar, shift towards, absent breath sounds
Superior vena cava obstruction	Facial swelling, fixed raised JVP, dilated chest veins with downward flow
Pancoast tumour	Horner's syndrome, wasting of small muscles of the hand, T1 sensory loss
Kyphoscoliosis	Spinal deformity, restrictive pattern, cor pulmonale

Spotters and instruments to recognise

Group	Items
Devices	pMDI, spacer, DPI (Rotahaler, Turbuhaler, Accuhaler), soft-mist inhaler, nebuliser, peak flow meter, spirometer
Oxygen	Nasal cannula, simple mask, Venturi mask with colour-coded valves, non-rebreathing mask with reservoir, HFNC, oxygen concentrator, CPAP and BiPAP masks
Airway	Endotracheal tube, tracheostomy tube (cuffed, fenestrated), laryngeal mask, Ambu bag, Guedel airway, cricothyroidotomy set
Pleural	Intercostal drain, underwater seal, Heimlich valve, indwelling pleural catheter, Abrams needle, thoracoscope, pleural manometer
Diagnostic	Flexible bronchoscope, rigid bronchoscope, EBUS scope, cryoprobe, TBNA needle, Ryle's tube, sputum container, Mantoux syringe
Drugs/vials	Streptomycin, ATT FDC strip, bedaquiline, salbutamol respule, ipratropium, budesonide, adrenaline, magnesium sulphate
X-rays and CT	Miliary TB, cavity, effusion, pneumothorax, collapse, mass, honeycombing, bronchiectasis, pneumomediastinum, Scimitar

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Procedure and counselling stations

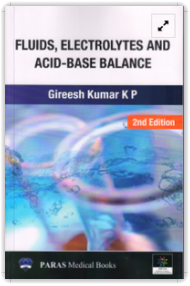
Station	Key steps to demonstrate
Inhaler technique teaching	Check device, shake, exhale, seal lips, slow deep inhalation (pMDI) or forceful (DPI), 10-second breath-hold, rinse mouth after ICS; teach-back
Peak flow / spirometry	Explain, demonstrate, three acceptable attempts, record best, interpret against predicted
Pleural aspiration	Consent, ultrasound marking, above the rib, aseptic technique, send fluid for the right tests, post-procedure CXR if symptomatic
Oxygen prescription	Target range (88–92% or 94–98%), device and flow, monitoring, signature
ABG sampling	Allen's test, 45° angle at radial artery, heparinised syringe, expel air, ice if delay, apply pressure 5 minutes
Breaking bad news	SPIKES — Setting, Perception, Invitation, Knowledge, Emotions, Strategy and summary
Smoking cessation counselling	5 As, set a quit date, pharmacotherapy, follow-up
TB counselling	Adherence, duration, side effects, infection control at home, contact screening, nutrition support, Ni-kshay benefits

★ **CLINICAL PEARLS & EXAM FOCUS**

- In a spotter, say what the device is, one indication and one complication — that is usually all the time allows.
- Practise examining in exactly the same order every time; in the exam, habit rescues nerves.

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E18.7 Rapid Revision — One-Page Answers to the Twenty Commonest Questions

IN BRIEF The final page before the examination. Each answer is the skeleton of a full response: expand each line into a paragraph or table under pressure.

Twenty rapid answers

Question	Skeleton answer
Causes of massive haemoptysis in India	Post-TB bronchiectasis and cavity, Rasmussen aneurysm, aspergilloma, active TB, bronchiectasis, carcinoma, mitral stenosis, vasculitis, AVM
Causes of upper-zone fibrosis	TB, silicosis, sarcoidosis, ankylosing spondylitis, radiation, chronic hypersensitivity pneumonitis, PLCH (mnemonic: "SCHART")
Causes of lower-zone fibrosis	IPF, asbestosis, connective tissue disease, drugs (bleomycin, amiodarone, nitrofurantoin), aspiration
Causes of a cavitating lesion	TB, lung abscess, squamous carcinoma, GPA, septic emboli, fungal (aspergilloma, mucor), rheumatoid nodule, melioidosis
Causes of bilateral hilar lymphadenopathy	Sarcoidosis, TB, lymphoma, metastases, silicosis, berylliosis, infections (histoplasmosis)
Causes of a unilateral white-out with shift away	Massive pleural effusion, large mass with effusion, diaphragmatic hernia
Causes of a white-out with shift towards	Collapse, pneumonectomy, lung agenesis, severe fibrosis
Causes of clubbing with cyanosis	Bronchiectasis, lung abscess, ILD, cyanotic heart disease, hepatopulmonary syndrome
Causes of eosinophilia with lung shadows	ABPA, tropical pulmonary eosinophilia, parasitic (Löffler), drugs, EGPA, AEP/CEP
Indications for NIV	AECOPD with pH < 7.35, cardiogenic pulmonary oedema, immunocompromised hypoxaemic failure, OHS, neuromuscular and chest-wall disease, weaning and post-extubation prophylaxis
Criteria for LTOT	PaO2 ≤ 55 mmHg, or 56–59 with cor pulmonale/polycythaemia/PH, on two stable readings ≥ 3 weeks apart; ≥ 15 h/day
Indications for pleural drainage in infection	Frank pus, positive Gram stain or culture, pH < 7.2, loculation with sepsis
Light's criteria	Protein ratio > 0.5; LDH ratio > 0.6; fluid LDH > two-thirds upper limit of normal serum LDH
Criteria for severe CAP	IDSA/ATS: one major (shock on vasopressors, invasive ventilation) or three minor criteria
ARDS definition (2023 global)	Acute onset within 1 week of a risk factor; bilateral opacities on CXR/CT/ultrasound; not fully explained by cardiac failure; P/F ≤ 300 or S/F ≤ 315 with HFNC ≥ 30 L/min, NIV or CPAP ≥ 5
Treatment of MDR-TB	BPaLM for 6 months if FQ-susceptible and eligible; otherwise BPaL, 9-month all-oral regimen, or longer individualised regimen from Groups A–C
Definition of PH and PAH	mPAP > 20 mmHg; pre-capillary PAH adds PAWP ≤ 15 and PVR > 2 WU
Features of a massive PE	Shock or systolic BP < 90 mmHg (or drop ≥ 40 mmHg for > 15 min) — thrombolysis indicated
Risk factors for lung cancer	Smoking, biomass and air pollution, asbestos, silica, radon, arsenic, previous TB or fibrosis, family history, HIV
Indications for lung transplantation	End-stage ILD, COPD, cystic fibrosis/bronchiectasis and PAH after maximal therapy, with no absolute contraindication

Numbers worth memorising

Value	Number
Normal A–a gradient	< 10–15 mmHg (age/4 + 4)
Normal DLCO	75–140% predicted; corrected for haemoglobin
Pleural fluid ADA cut-off for TB	> 40 IU/L
Transudate–exudate albumin gradient	> 1.2 g/dL = transudate
NIV threshold in AECOPD	pH < 7.35 with PaCO2 > 45 mmHg
Tidal volume in ARDS	6 mL/kg predicted body weight
Plateau pressure limit	≤ 30 cmH2O; driving pressure ≤ 15
Prone positioning threshold	P/F < 150, for ≥ 16 h/day
Severe asthma PEF	< 33% of best or predicted (life-threatening)
6MWD minimal important difference	≈ 30 metres
Smoking pack-years for LDCT screening	≥ 20 pack-years, age 50–80
ICS threshold by eosinophils in COPD	≥ 300/µL favours; < 100/µL against

★ CLINICAL PEARLS & EXAM FOCUS

- Walk into the examination able to draw four things from memory: the flow–volume loops, the NTEP diagnostic algorithm, the ARDS ventilation plan and the pleural effusion pathway.

E18.8 Bedside Interpretation Stations — ABG, CXR, Spirometry & Ventilator Graphics

IN BRIEF The data-interpretation station gives you a printout and four minutes. Use the same opening sentence every time: identify the test, comment on its quality, describe the abnormality, then give the diagnosis and the next step.

How to open each station

Station	Opening sentence
ABG	"This is an arterial blood gas on room air / on FiO2 … The patient has a [acidaemia/alkalaemia] with a primary [respiratory/metabolic] …"
Chest radiograph	"This is a PA erect chest radiograph of an adult, adequately inspired, not rotated, adequately exposed. The abnormality is …"
Spirometry	"This is a flow–volume loop and volume–time curve. The test is acceptable and repeatable. The pattern is …"
Ventilator screen	"These are pressure, flow and volume–time waveforms in [volume/pressure] control. The abnormality is …"
Sleep study	"This is a polysomnogram showing an AHI of … with the events predominantly [obstructive/central] and oxygen nadir of …"
Pleural fluid report	"The fluid is an exudate by Light's criteria, lymphocyte-predominant, with an ADA of … suggesting …"

Worked ABG examples for practice

ABG	Interpretation	Likely clinical setting
pH 7.21, PaCO2 78, HCO3 30, PaO2 48 (room air)	Acute-on-chronic respiratory acidosis with hypoxaemia	AECOPD — start NIV
pH 7.48, PaCO2 26, PaO2 58, A–a gradient wide	Acute respiratory alkalosis with hypoxaemia	Pulmonary embolism
pH 7.30, PaCO2 44, HCO3 21, PaO2 62, lactate 4	Mixed metabolic acidosis with inadequate respiratory compensation	Septic shock with pneumonia
pH 7.52, PaCO2 50, HCO3 40, K 2.8	Metabolic alkalosis with compensatory hypoventilation	Diuretic therapy in COPD — acetazolamide
pH 7.33, PaCO2 38, HCO3 19, AG 24, osmolar gap high	High-anion-gap acidosis	Toxic alcohol ingestion
pH 7.40, PaCO2 40, PaO2 95, SpO2 86% on pulse oximeter	Saturation gap	Methaemoglobinaemia or CO poisoning — co-oximetry

Reading a ventilator screen in four steps

Step	Question	What it tells you
1	Which mode and who is triggering?	Controlled vs assisted breaths; negative deflection before each breath = patient trigger
2	Is the flow waveform square or decelerating?	Volume control vs pressure control
3	Does expiratory flow return to zero before the next breath?	If not — air trapping and auto-PEEP
4	What is the gap between peak and plateau pressure?	Wide gap = resistance problem; both high = compliance problem

Spirometry station — the five questions

Question	Answer to give
Is the test acceptable?	Good start, no cough in the first second, ≥ 6 s or plateau, two values within 150 mL
Is there obstruction?	FEV1/FVC below the lower limit of normal (or < 0.70 by GOLD)
Is there restriction?	Low FVC with normal/high ratio — confirm with TLC
Is it reversible?	Change in FEV1 or FVC > 10% of predicted (ATS/ERS 2022)
What is the severity?	By z-score: mild −1.65 to −2.5; moderate to −4.0; severe below −4.0

★ **CLINICAL PEARLS & EXAM FOCUS**

- Never say "the X-ray is normal" until you have checked the apices, behind the heart, below the diaphragms and the bones.
- In the ABG station, always calculate the A–a gradient and the anion gap aloud — examiners award method, not just the final answer.

PART II · SECTION 19

Oxygen Delivery, Non-Invasive Support & Mechanical Ventilation — A Practical Handbook

Advanced theory & algorithms

E19.1 Oxygen Delivery Devices — Principles & How to Choose 528
E19.2 Device 1 · Nasal Cannula (Nasal Prongs) 531
E19.3 Device 2 · Simple Face Mask 532
E19.4 Device 3 · Partial Rebreathing Mask 534
E19.5 Device 4 · Non-Rebreather Mask (NRB) 535
E19.6 Device 5 · Reservoir Cannula (Oxymizer) & Pulse-Dose Devices 536
E19.7 Device 6 · Venturi Mask (Air-Entrainment Mask) 538
E19.8 Device 7 · High-Flow Nasal Cannula (HFNC) 539
E19.9 Device 8 · Tracheostomy Mask, T-Piece & Face Tent 541
E19.10 Device 9 · Bag-Valve-Mask & Manual Ventilation Devices 543
E19.11 Device 10 · Oxygen Sources — Cylinders, Concentrators & Liquid Oxygen 544
E19.12 Oxygen Prescription, Weaning & Hazards 545
E19.13 Non-Invasive Ventilation 1 · Principles, CPAP vs BiPAP 548
E19.14 Non-Invasive Ventilation 2 · Indications, Contraindications & Interfaces 549
E19.15 Non-Invasive Ventilation 3 · Setting Up CPAP & BiPAP at the Bedside 551
E19.16 Non-Invasive Ventilation 4 · Troubleshooting & Weaning 553
E19.17 Non-Invasive Ventilation 5 · Domiciliary NIV, CPAP for OSA & Cough Augmentation 554
E19.18 Invasive Mechanical Ventilation 1 · Indications & The Four Basic Questions 555
E19.19 Invasive Mechanical Ventilation 2 · Modes Explained 557
E19.20 Invasive Mechanical Ventilation 3 · Disease-Specific Ventilator Settings 559
E19.21 Invasive Mechanical Ventilation 4 · Alarms, Complications & Daily Care 560
E19.22 Rapid Sequence Intubation 1 · Preparation & The Seven Ps 562
E19.23 Rapid Sequence Intubation 2 · Drug Table with Doses & Cautions 563
E19.24 Rapid Sequence Intubation 3 · Delayed & Awake Techniques, Failed Airway 564
E19.25 Quick Reference — Oxygen, NIV & Ventilation on One Spread 567

E19.1 Oxygen Delivery Devices — Principles & How to Choose

IN BRIEF Oxygen delivery devices divide into **low-flow (variable-performance)** systems, in which room air is entrained around and through the device so that the delivered FiO_2 changes with the patient's own inspiratory flow, and **high-flow (fixed-performance)** systems, which deliver a flow equal to or greater than the patient's peak inspiratory demand and therefore give a predictable FiO_2.

Why the distinction matters

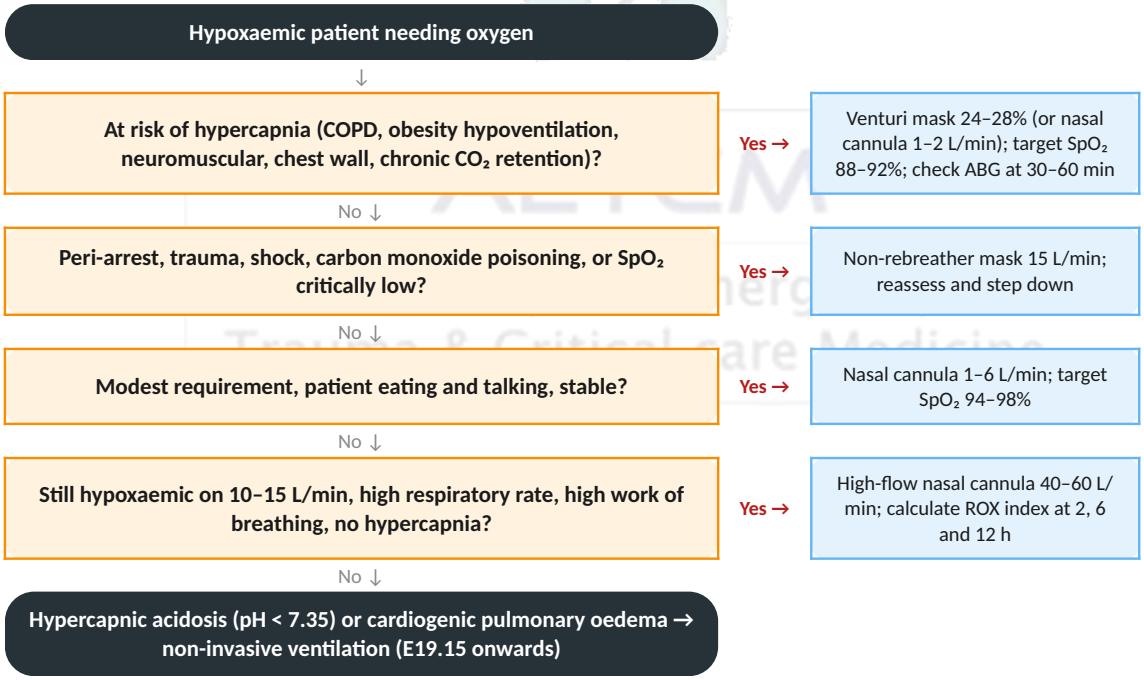
- An adult in respiratory distress has a peak inspiratory flow of **30–60 L/min** or more. Any device supplying less than this must make up the difference with room air (21% oxygen), so the FiO_2 actually breathed is lower and more variable than the flowmeter suggests.

- **Low-flow devices:** the sicker and more breathless the patient, the lower the FiO_2 actually delivered — exactly the opposite of what is needed.
- **High-flow devices:** FiO_2 stays constant whatever the respiratory pattern, which is what makes the Venturi mask the device of choice when hypercapnia is a risk and HFNC the device of choice in severe hypoxaemia.
- The anatomical reservoir (nose, nasopharynx, oropharynx) holds about **50 mL** and is filled during the expiratory pause; this is why nasal cannula works at all, and why mouth breathing matters less than students expect.

The complete range of devices at a glance

Device	Type	Flow rate	Approximate FiO_2
Nasal cannula	Low flow	1–6 L/min	24–44% (≈ 4% per L/min)
Simple face mask	Low flow	5–10 L/min	40–60%
Partial rebreathing mask	Low flow (reservoir)	6–10 L/min	60–80%
Non-rebreather mask	Low flow (reservoir)	10–15 L/min	90–100%
Reservoir cannula (Oxymizer)	Low flow (reservoir)	0.5–4 L/min	Equivalent to 2–4× the same flow by plain cannula
Venturi mask	High flow (fixed performance)	2–15 L/min by adapter	24–60%
High-flow nasal cannula	High flow	up to 40–60+ L/min	21–100%
Tracheostomy mask / T-piece	Variable or fixed	8–15 L/min	28–100%
Face tent	Low flow	8–15 L/min	≈ 28–50% (variable)
Bag-valve-mask with reservoir	Manual ventilation	15 L/min	≈ 100%
Oxygen concentrator	Source	0.5–10 L/min (5 L common)	87–96% at the outlet

Algorithm E19.1.1 · Choosing an oxygen device at the bedside



Oxygen targets (BTS / national guidance)

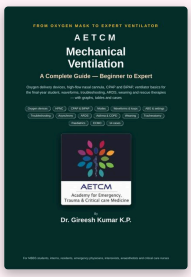
Group	Target SpO_2
Most acutely ill adults	94–98%
At risk of hypercapnic respiratory failure	88–92%
Carbon monoxide poisoning, cluster headache, pneumothorax, sickle crisis	High-concentration oxygen regardless of saturation
Stable chronic lung disease on long-term oxygen	Per prescription, usually 88–92%

★ CLINICAL PEARLS & EXAM FOCUS

- Oxygen is a drug: write the device, the flow or FiO_2, the target saturation range and who may adjust it.
- Hyperoxia harms — liberal oxygen increases mortality (IOTA meta-analysis). Titrate down as soon as the patient allows.
- Humidify when flow exceeds 4 L/min by cannula, in any patient with a tracheostomy, and always with HFNC.

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E19.2 Device 1 · Nasal Cannula (Nasal Prongs)

Nasal cannula at a glance

Item	Detail
Description	Lightweight two-pronged tube sitting in the anterior nares, looped over the ears and secured under the chin
Category	Low flow — variable performance
Flow rate	1–6 L/min (above 6 L/min is poorly tolerated and does not raise FiO_2 much further)
Approximate FiO_2	24–44%; a rough rule is 20% + (4 × flow in L/min)
Humidification	Not needed up to 4 L/min; bubble humidifier above that, or if the patient complains of dryness
Indications	Mild hypoxaemia, stable patients, long-term oxygen therapy, during meals and speech, post-operative, weaning from a mask
Advantages	Comfortable, cheap, allows eating, drinking and talking; patient can wear it continuously; very widely available
Limitations	FiO_2 unpredictable and falls with high inspiratory flow; nasal dryness and epistaxis; pressure sores behind the ears and on the nasal septum; displaced easily
Monitoring	SpO_2, respiratory rate, nasal mucosa, skin behind the ears

FiO_2 by flow rate (approximate, adult, normal minute ventilation)

Flow (L/min)	Approximate FiO_2
1	24%
2	28%
3	32%
4	36%
5	40%
6	44%

★ CLINICAL PEARLS & EXAM FOCUS

- The figures above assume a normal respiratory pattern. In a tachypnoeic patient with a high inspiratory flow, 4 L/min may deliver barely 28%.
- Mouth breathing does not abolish the effect: oxygen still fills the nasopharyngeal reservoir and is drawn in with each breath.
- Place a hydrocolloid dressing behind the ears in any patient expected to need cannula for more than 24 hours.

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E19.3 Device 2 · Simple Face Mask

Simple face mask at a glance

Item	Detail
Description	Soft plastic mask covering the nose and mouth with open side ports for exhalation and air entrainment; no reservoir bag and no valves
Category	Low flow — variable performance
Flow rate	**5–10 L/min** — never run below 5 L/min
Approximate FiO_2	40–60%
Indications	Moderate hypoxaemia in a patient not at risk of hypercapnia; short-term use in the emergency department, recovery room or ward
Advantages	Higher FiO_2 than cannula; simple; no special fittings
Limitations	FiO_2 unpredictable; must be removed to eat, drink or take oral drugs; claustrophobic; muffles speech; risk of CO_2 rebreathing if the flow is too low; pressure on the bridge of the nose
Monitoring	SpO_2, seal and fit, skin over the nasal bridge, ability to communicate

The 5 L/min rule

- The mask body itself is a **100–250 mL reservoir of exhaled gas**. A flow of at least 5 L/min is needed to flush carbon dioxide from that volume between breaths.
- Running a simple mask at 2–4 L/min causes rebreathing and rising $PaCO_2$ while delivering less oxygen than a nasal cannula at the same flow — a common and dangerous ward error.

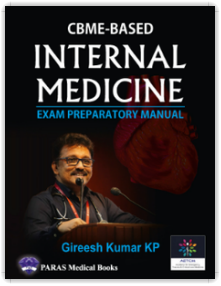
Approximate FiO_2 by flow

Flow (L/min)	Approximate FiO_2
5–6	40%
6–7	50%
7–8	55%
8–10	60%

★ CLINICAL PEARLS & EXAM FOCUS

- If a patient needs a simple mask but is at risk of CO_2 retention, use a Venturi mask instead — never a simple mask with the flow turned down.
- Fit the mask so that the metal nose clip is moulded to the nasal bridge; most "mask intolerance" is a fitting problem.

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E19.4 Device 3 · Partial Rebreathing Mask

Partial rebreathing mask at a glance

Item	Detail
Description	Simple face mask with a 600–1000 mL reservoir bag attached, **no valve between the mask and the bag** and no (or only one) side-port valve
Category	Low flow with reservoir — variable performance
Flow rate	**6–10 L/min**, set high enough that the bag never collapses by more than about one-third on inspiration
Approximate FiO_2	60–80%
Indications	Moderate-to-severe hypoxaemia when a non-rebreather is unavailable; patients in whom some CO_2 rebreathing is acceptable or desired
Advantages	Higher FiO_2 than a simple mask; some conservation of oxygen
Limitations	The first third of exhaled gas (anatomical dead-space gas, low in CO_2 and still rich in oxygen) re-enters the bag and is rebreathed; FiO_2 still variable; same comfort problems as any mask
Monitoring	SpO_2, bag movement with each breath, respiratory rate, $PaCO_2$ where relevant

How it works

- On expiration, the first portion of exhaled gas — which comes from the **anatomical dead space** and has therefore taken no part in gas exchange — returns to the reservoir bag and mixes with fresh oxygen.
- The remainder of the exhaled breath, which carries the carbon dioxide, escapes through the side ports.
- This design conserves oxygen and was historically valued where supply was limited; in modern practice the **non-rebreather is preferred** whenever high FiO_2 is the aim.

★ CLINICAL PEARLS & EXAM FOCUS

- The single most important check is the bag: if it collapses completely on inspiration, the flow is too low — increase it.
- Distinguish the two masks at a glance: the partial rebreather has **no one-way valve** between bag and mask; the non-rebreather has one.

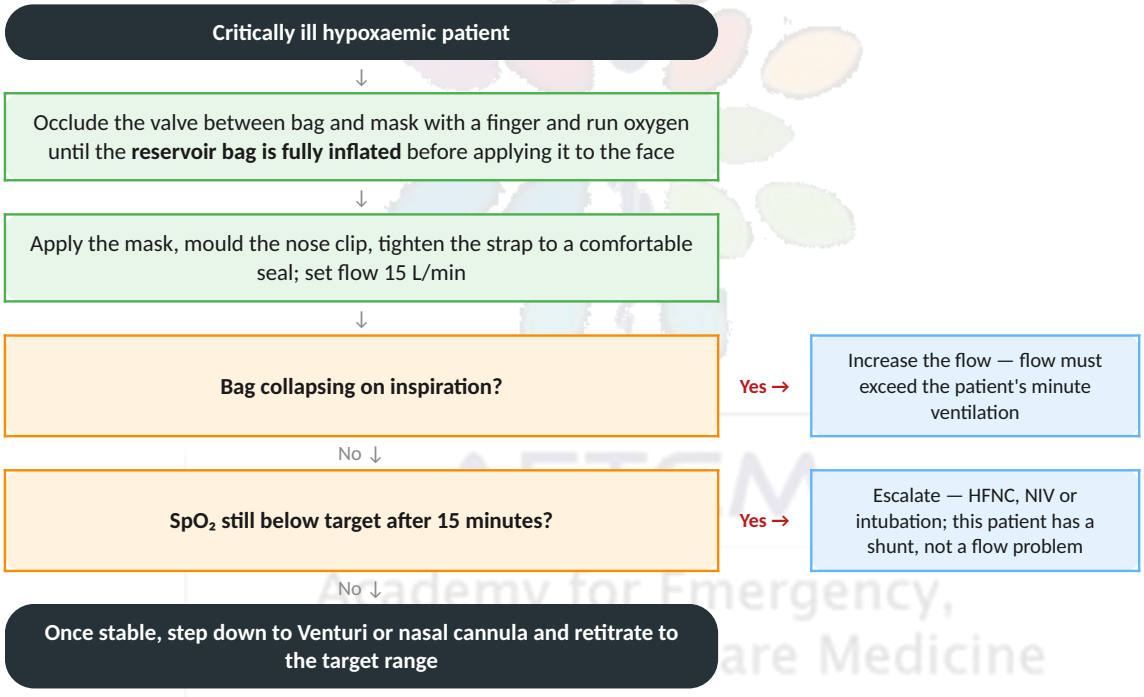
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E19.5 Device 4 · Non-Rebreather Mask (NRB)

Non-rebreather mask at a glance

Item	Detail
Description	Face mask with a 1000–1500 mL reservoir bag, a **one-way valve between bag and mask** and one-way flap valves over one or both side ports
Category	Low flow with reservoir — the highest FiO_2 achievable without positive pressure
Flow rate	**10–15 L/min** (15 L/min in practice)
Approximate FiO_2	90–100% in theory; **60–80% in practice** because of leak around the mask and entrainment through the unvalved port
Indications	Critical illness — major trauma, shock, sepsis, cardiac arrest and the peri-arrest period, carbon monoxide poisoning, pneumothorax, cluster headache, sickle-cell crisis, pre-oxygenation before intubation
Advantages	Fastest route to a high FiO_2 with no special equipment
Limitations	Not a fixed-performance device; dangerous in CO_2 retainers unless clearly indicated; drying; must be removed to eat or speak; cannot be used long term
Monitoring	SpO_2 continuously, ABG, bag inflation, level of consciousness

Algorithm E19.5.1 · Using a non-rebreather correctly



★ CLINICAL PEARLS & EXAM FOCUS

- Always prime the reservoir bag before putting the mask on the patient — an empty bag delivers less oxygen than a simple mask.
- A non-rebreather gives **no positive pressure**; if a patient is failing on 15 L/min, the answer is HFNC, NIV or intubation, not more flow.
- Removing both side-port valves (as some manufacturers supply) converts it into a partial rebreather.

E19.6 Device 5 · Reservoir Cannula (Oxymizer) & Pulse-Dose Devices

Oxygen-conserving devices at a glance

Item	Detail
Description	Nasal cannula incorporating a 20 mL reservoir — either a moustache-shaped chamber under the nose (pendant type hangs on the chest) — that fills during expiration and delivers a bolus at the start of inspiration
Category	Low flow with reservoir — oxygen conserving
Flow rate	0.5–4 L/min
Equivalence	Delivers an FiO_2 roughly equal to **2–4 times** the same flow through a plain cannula; a 2 L/min reservoir cannula may equal 4–6 L/min conventional
Indications	Long-term oxygen therapy, ambulatory oxygen, patients with very high requirements, cylinder conservation during transport and air travel
Advantages	Prolongs cylinder life two- to four-fold; lighter portable cylinders; lower cost
Limitations	Visible and cosmetically unpopular; must be changed weekly; the reservoir membrane can stick if humidified; not suitable for mouth breathers

Demand (pulse-dose) oxygen delivery systems

- An electronic or pneumatic valve senses the **negative pressure at the start of inspiration** and releases a pulse of oxygen only during the first third of the breath, when gas actually reaches the alveoli.
- Oxygen delivered during late inspiration and expiration is wasted, so pulse delivery saves up to 75% of the supply.
- Settings are expressed as a **pulse setting (1–6)**, not as L/min — the two are not interchangeable, and a patient must always be titrated on the actual device they will use, walking, before discharge.
- Not suitable for patients with a weak inspiratory effort, during sleep (some devices fail to trigger), or when a continuous flow has been specifically prescribed.

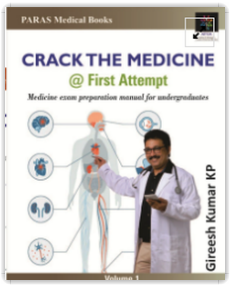
★ CLINICAL PEARLS & EXAM FOCUS

- Always check the saturation on exertion with the exact portable device and setting the patient is going home with — bench equivalence figures are unreliable.
- Airlines usually permit approved portable oxygen concentrators in pulse mode only; confirm the model and battery requirement well before travel.

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E19.7 Device 6 · Venturi Mask (Air-Entrainment Mask)

Venturi mask at a glance

Item	Detail
Description	Face mask with colour-coded, interchangeable jet adapters that entrain a fixed ratio of room air to oxygen by the Bernoulli principle
Category	**High flow — fixed performance**: total gas flow exceeds the patient's peak inspiratory flow, so FiO_2 is constant
Flow rate	2–15 L/min, specified for each adapter (printed on the barrel)
Approximate FiO_2	24, 28, 31, 35, 40, 60%
Indications	**The device of choice in COPD and anyone at risk of hypercapnic respiratory failure**; any situation requiring an accurate, titratable FiO_2
Advantages	Predictable FiO_2 independent of the breathing pattern; easy stepwise titration; no rebreathing
Limitations	Noisy; must be removed to eat; the correct flow must be set for each adapter; high ambient temperature and humidity alter entrainment slightly
Monitoring	ABG 30–60 min after any change, SpO_2, respiratory rate, level of consciousness

Standard adapters, flows and entrainment ratios

Colour	FiO_2	Minimum oxygen flow	Air : oxygen ratio	Approximate total flow
Blue	24%	2–4 L/min	25 : 1	≈ 52–104 L/min
White	28%	4–6 L/min	10 : 1	≈ 44–66 L/min
Orange	31%	6–8 L/min	8 : 1	≈ 54–72 L/min
Yellow	35%	8–10 L/min	5 : 1	≈ 48–60 L/min
Red	40%	10–12 L/min	3 : 1	≈ 40–48 L/min
Green	60%	12–15 L/min	1 : 1	≈ 24–30 L/min

- Colour coding varies between manufacturers — **always read the value printed on the adapter**, never rely on colour alone. Note the paradox: the **higher** the FiO_2, the **lower** the total gas flow, because less air is entrained, so at 60% the device is no longer truly fixed performance in a breathless patient.

★ CLINICAL PEARLS & EXAM FOCUS

- Bernoulli effect: oxygen through a narrow jet accelerates and draws room air through fixed side windows — occluding them with bedding raises FiO_2 unpredictably.
- Start an acutely breathless COPD patient on 24% or 28%, check an ABG at 30–60 min, and step up only if the $PaCO_2$ and pH allow.

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E19.8 Device 7 · High-Flow Nasal Cannula (HFNC)

HFNC at a glance

Item	Detail
Description	Wide-bore soft nasal prongs delivering heated (31–37 °C) and fully humidified gas from an air–oxygen blender through a heated circuit
Category	High flow — fixed performance
Flow rate	Start 40–50 L/min; up to **60–70 L/min** in adults (1–2 L/kg/min in children; 2–8 L/min in neonates)
FiO_2	21–100%, set independently of flow
Indications	Acute hypoxaemic respiratory failure (pneumonia, COVID-19, early ARDS), post-extubation support in high-risk patients, pre-oxygenation and apnoeic oxygenation at intubation, bronchoscopy, palliative relief of breathlessness, immunocompromised hypoxaemia
Advantages	Comfortable; allows speech, eating and physiotherapy; generates low-level PEEP; washes out nasopharyngeal dead space; preserves mucociliary clearance
Limitations	No reliable control of tidal volume; risk of **delaying necessary intubation**; nasal discomfort at very high flow; power and water dependent; costly
Monitoring	ROX index at 2, 6 and 12 h; respiratory rate; SpO_2; work of breathing; ABG

The five physiological effects

- **Precise FiO_2** — flow exceeds the patient's peak inspiratory flow, so no room air is entrained.
- **Dead-space washout** — continuous flushing of the nasopharynx removes exhaled CO_2, improving alveolar ventilation.
- **Positive end-expiratory pressure** — roughly **1 cmH_2O for every 10 L/min** with the mouth closed (about half that with the mouth open); typically 3–5 cmH_2O at 50 L/min.
- **Reduced work of breathing** — lower inspiratory resistance and less need to generate high flows.
- **Conditioned gas** — warm, 100% humidified gas preserves ciliary function, loosens secretions and is far better tolerated than dry oxygen.

Setting up and weaning HFNC

Step	Practice
Initial settings	Flow 40–50 L/min, FiO_2 1.0, temperature 37 °C (reduce to 34 °C if not tolerated)
Prong size	Should occupy no more than **half** the nostril diameter to allow leak
Titration	Correct hypoxaemia first with FiO_2; reduce work of breathing with flow
Weaning	Reduce **FiO_2 to ≤ 0.4 first**, then reduce flow in steps of 5–10 L/min; switch to conventional oxygen at flow ≤ 20 L/min and FiO_2 ≤ 0.4
Failure	ROX index < 3.85 at 12 h, rising respiratory rate, worsening hypoxaemia, altered consciousness → intubate without delay

The ROX index

- **ROX = (SpO_2 / FiO_2) ÷ respiratory rate.**
- ≥ 4.88 at 2, 6 or 12 h predicts success; < 3.85 predicts failure and should prompt intubation.
- Worked example: SpO_2 92%, FiO_2 0.6, respiratory rate 28 → (92/0.6)/28 = **5.5** — reassuring.

LANDMARK TRIALS & GUIDELINES

- **FLORALI (2015):** HFNC reduced 90-day mortality compared with standard oxygen and NIV in hypoxaemic failure with $PaO_2/FiO_2 \leq 200$.
- **HIGH (2018):** no benefit over standard oxygen in immunocompromised patients.
- **RECOVERY-RS, HENIVOT:** CPAP/helmet NIV reduced intubation more than HFNC in COVID-19.

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E19.9 Device 8 · Tracheostomy Mask, T-Piece & Face Tent

Devices for the patient with a tracheostomy or facial injury

Device	Description	Flow	FiO_2	Notes
Tracheostomy mask (collar)	Soft mask held over the stoma by a neck strap, with an exhalation port	8–15 L/min through a heated humidifier or nebuliser	≈ 28–60% (variable)	Humidification is mandatory — the upper airway is bypassed
T-piece (Briggs adapter)	T-shaped connector attaching directly to the tracheostomy or endotracheal tube, with reservoir tubing on the expiratory limb	10–15 L/min, flow at least 2–3 × minute ventilation	Up to 100% with an adequate reservoir	Used for spontaneous breathing trials; watch for mist disappearing at the far end on inspiration — a sign of inadequate flow
Face tent (face shield)	Soft open bucket-shaped shield under the chin and around the face	8–15 L/min	≈ 28–50%, very variable	For facial trauma, burns, claustrophobia, after facial surgery; useful for humidified aerosol
Aerosol (nebuliser) mask	Mask with wide-bore tubing for continuous nebulised saline or bronchodilator	8–12 L/min	Variable	Large-volume nebuliser can be set to a chosen FiO_2

Humidification in the tracheostomised patient

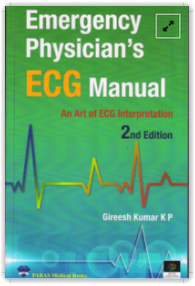
- The nose and pharynx normally warm gas to 37 °C and saturate it with water. A tracheostomy bypasses them entirely.
- Dry gas causes crusting, mucus plugging, tube blockage, bleeding and loss of ciliary function — the commonest avoidable cause of a tracheostomy emergency.
- Options: **heated humidifier** (best for the acute or ventilated patient), **heat and moisture exchanger ("Swedish nose")** for the stable patient, and regular saline nebulisation.

★ CLINICAL PEARLS & EXAM FOCUS

- A blocked tracheostomy tube presents as sudden respiratory distress with an inability to pass a suction catheter — remove the inner cannula first, then suction, then consider tube change.
- Keep the emergency box at the bedside: spare tube of the same size, one size smaller, tracheal dilators, suction, and the patient's tracheostomy details.

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E19.10 Device 9 · Bag-Valve-Mask & Manual Ventilation Devices

Manual ventilation devices

Device	Description	Oxygen	Delivered FiO_2	Key point
Self-inflating bag (Ambu / BVM)	Self-refilling bag with a non-rebreathing valve; works with or without an oxygen supply	15 L/min with reservoir	≈ 100% with reservoir; 40–60% without	The only device that works with no gas supply — essential for resuscitation and transport
Flow-inflating (anaesthetic) bag	Refills only from fresh gas flow; needs a tight seal	Required	Up to 100%	Allows the operator to feel compliance; used in anaesthesia and neonatal care
T-piece resuscitator (Neopuff)	Delivers set peak pressure and PEEP	Required	Set	Standard for neonatal resuscitation
Mapleson C circuit	Bag, valve and reservoir tubing	Required	Up to 100%	Common in UK recovery and intensive care

Using a bag-valve-mask well

- **Two-person technique is superior:** one operator holds the mask with both hands using a two-handed jaw thrust and "C-E" grip, the other squeezes the bag.
- Use an **oropharyngeal or nasopharyngeal airway** to relieve soft-tissue obstruction; the commonest cause of failed bag-mask ventilation is a poor airway, not a poor seal.
- Squeeze only enough to produce visible chest rise: roughly **500–600 mL over 1 second, 10–12 breaths per minute**. Over-vigorous ventilation causes gastric insufflation, regurgitation and reduced venous return.
- Attach a **PEEP valve** (5–10 cmH_2O) for pre-oxygenation of the hypoxaemic patient and waveform **capnography** whenever available.
- **Predictors of difficult bag-mask ventilation (MOANS):** Mask seal (beard), Obesity or Obstruction, Age > 55, No teeth, Stiff lungs or Snoring.

★ CLINICAL PEARLS & EXAM FOCUS

- During cardiac arrest with an advanced airway, ventilate at 10 breaths per minute without pausing compressions — hyperventilation reduces coronary perfusion pressure and worsens survival.
- Remember that a self-inflating bag held to the face of a spontaneously breathing patient delivers very little oxygen unless the patient can open the valve; use a non-rebreather instead.

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E19.11 Device 10 · Oxygen Sources — Cylinders, Concentrators & Liquid Oxygen

Oxygen sources compared

Source	Output / purity	Advantages	Limitations
Pipeline (wall) oxygen	100%, 4 bar (400 kPa)	Unlimited supply, no handling	Hospital only; manifold failure affects the whole building
Compressed gas cylinder	100% at 137 bar (size E ≈ 680 L)	Portable, no power needed, high flows	Finite; heavy; fire hazard; needs regulator
Oxygen concentrator	87–96% by zeolite molecular sieve, 0.5–10 L/min	Continuous supply from room air; cheap to run; ideal for home LTOT	Needs mains power; purity falls at high flow; noisy; needs servicing and filter cleaning
Portable concentrator	Pulse dose mostly; some continuous to 2–3 L/min	Battery powered, airline-approvable	Low output; may not trigger reliably in sleep
Liquid oxygen	100%, stored at −183 °C	Large volume in small weight; good for high-flow home users	Evaporative loss; specialist refilling; limited availability in India

Calculating how long a cylinder will last

- **Remaining minutes = (gauge pressure in bar × cylinder factor) ÷ flow in L/min.**
- Cylinder factor (litres per bar): size D ≈ 2.5, size E ≈ 5, size F ≈ 10, size J ≈ 50 (check the label — values differ by manufacturer).
- *Worked example:* an E cylinder reading 100 bar, running at 10 L/min → (100 × 5) ÷ 10 = **50 minutes**. Always plan transfers with at least twice the calculated requirement.

Safety rules that are examined

Rule	Reason
No oil, grease or alcohol-based hand rub near valves	Spontaneous ignition in high-pressure oxygen
Never smoke; keep 2 m from open flames and heaters	Oxygen vigorously supports combustion
Secure cylinders upright, chained or in a trolley	Falling cylinders can shear the valve and become projectiles
Open valves slowly	Adiabatic compression can ignite contaminants
Store in a cool, ventilated, locked area	Pressure rises with temperature
Check the expiry and test date on each cylinder	Legal requirement

★ CLINICAL PEARLS & EXAM FOCUS

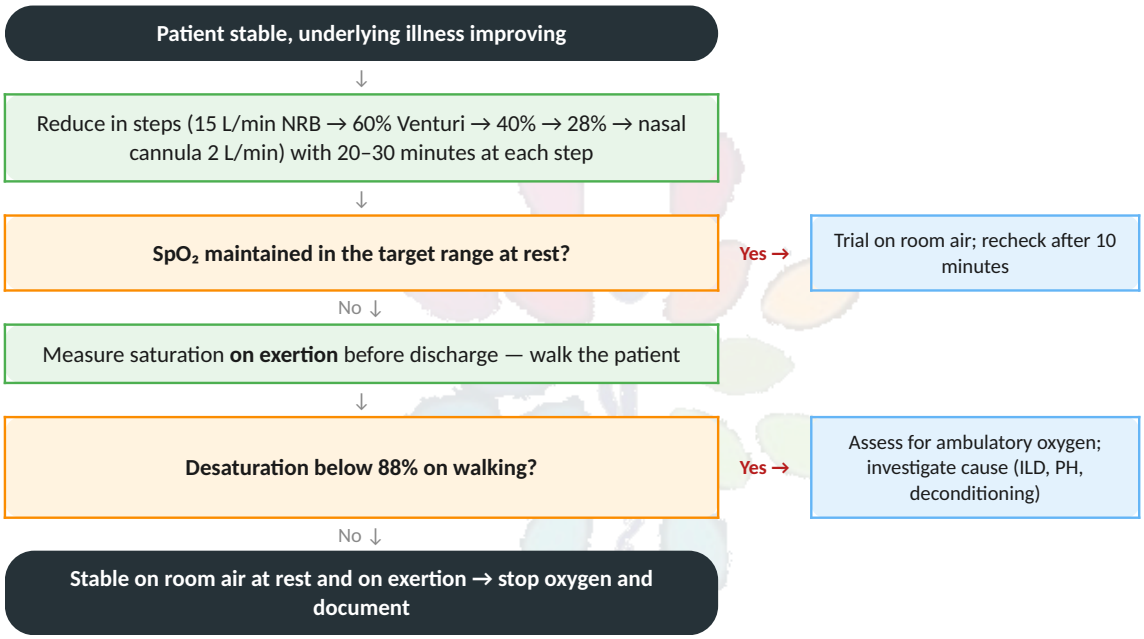
- During the COVID-19 surge India learnt the importance of **PSA (pressure swing adsorption) oxygen plants** — on-site generation from ambient air, producing 93 ± 3% oxygen, now mandated in most district hospitals.
- An oxygen concentrator at 5 L/min delivers roughly the same FiO_2 as a cylinder at 5 L/min for practical purposes; above 5 L/min the purity of most domestic concentrators falls.

E19.12 Oxygen Prescription, Weaning & Hazards

Writing a safe oxygen prescription

Element	Example
Target saturation range	94–98% (or 88–92% if at risk of hypercapnia)
Starting device and dose	Venturi 28%, or nasal cannula 2 L/min
Who may adjust	Nursing staff may titrate within the target range
Monitoring	Continuous SpO_2; ABG at 30–60 min after any significant change in a CO_2 retainer
Escalation plan	Named doctor to review if FiO_2 must be increased or target not met
Review and stop date	Reassess daily; stop when SpO_2 stays in range on room air

Algorithm E19.12.1 · Weaning supplemental oxygen



Hazards of oxygen therapy

Hazard	Mechanism / prevention
Hypercapnic respiratory failure	V/Q mismatch from release of hypoxic pulmonary vasoconstriction, Haldane effect and reduced drive — use controlled oxygen with target 88–92%
Absorption atelectasis	Nitrogen washout at high FiO_2 allows alveolar collapse — use the lowest effective FiO_2, add PEEP
Oxygen toxicity	Free-radical lung injury after prolonged FiO_2 > 0.6; tracheobronchitis then diffuse alveolar damage
Retinopathy of prematurity	Target SpO_2 90–95% in preterm neonates
Fire and burns	Smoking on oxygen; dry dressings and emollients; strict no-smoking policy
Drying, epistaxis, pressure injury	Humidify above 4 L/min; protect the ears and nasal bridge
Masking deterioration	A rising oxygen requirement is a warning sign — do not simply turn the flow up

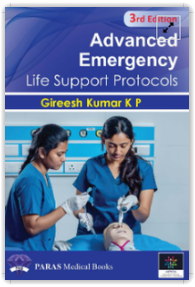
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- Oxygen relieves hypoxaemia; it does not relieve breathlessness in a patient who is not hypoxaemic. For palliative breathlessness with normal saturations, a fan is as effective and safer.

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E19.13 Non-Invasive Ventilation 1 · Principles, CPAP vs BiPAP

IN BRIEF Non-invasive ventilation delivers positive airway pressure through a mask or helmet without an artificial airway. **CPAP** supplies one constant pressure throughout the respiratory cycle; **BiPAP** supplies two — a higher pressure during inspiration and a lower one during expiration — and therefore actively augments the tidal volume.

CPAP and BiPAP compared

Feature	CPAP	BiPAP (bilevel)
Pressures	One pressure (e.g. 10 cmH_2O) throughout	IPAP (e.g. 16) + EPAP (e.g. 5)
Pressure support	**None** — the patient does all the work of breathing	**IPAP − EPAP** = pressure support (e.g. 11 cmH_2O)
Main effect	Recruits alveoli, raises FRC, reduces preload and afterload — improves **oxygenation**	Augments tidal volume and unloads the respiratory muscles — improves **ventilation** (clears CO_2)
Typical indications	Cardiogenic pulmonary oedema, obstructive sleep apnoea, selected hypoxaemic failure, post-operative atelectasis	AECOPD with acidosis, obesity hypoventilation, neuromuscular and chest-wall disease, acute-on-chronic hypercapnia, palliative breathlessness
Effect on $PaCO_2$	Little or none	Falls — the principal reason for choosing it
Backup rate	None	Available (S/T mode) — essential in central apnoea and neuromuscular disease

The physiological actions of positive airway pressure

- **Alveolar recruitment** — reopens collapsed alveoli and raises functional residual capacity, improving V/Q matching and oxygenation.
- **Counterbalancing intrinsic PEEP** — in COPD, EPAP of 4–5 cmH_2O offsets auto-PEEP so the patient no longer has to generate large negative pressures to trigger a breath.
- **Reduced work of breathing** — pressure support takes over part of the inspiratory muscle load; the diaphragm rests.
- **Haemodynamic effects** — raised intrathoracic pressure reduces venous return (preload) and left ventricular transmural pressure (afterload): beneficial in pulmonary oedema, harmful in hypovolaemia and right ventricular failure.
- **Splinting of the upper airway** — the basis of CPAP in obstructive sleep apnoea.

Terminology you must be able to define

Term	Meaning
IPAP	Inspiratory positive airway pressure
EPAP	Expiratory positive airway pressure (equivalent to PEEP)
Pressure support	IPAP − EPAP; the driver of tidal volume
Rise time	Time taken to reach IPAP (0.05–0.4 s); shorter in air hunger, longer for comfort
Trigger	Threshold of flow or pressure at which the machine starts a breath
Cycling	Point at which inspiration ends (usually when flow falls to 25% of peak)
S / S-T / T mode	Spontaneous / spontaneous-timed with backup rate / timed (fully controlled)
AVAPS	Average volume-assured pressure support — machine varies IPAP to reach a target tidal volume

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E19.14 Non-Invasive Ventilation 2 · Indications, Contraindications & Interfaces

Evidence for NIV by indication

Indication	Modality	Strength of evidence
AECOPD with pH < 7.35 and $PaCO_2$ > 45 mmHg	BiPAP	**Strong** — reduces intubation, mortality and length of stay
Cardiogenic pulmonary oedema	CPAP or BiPAP	**Strong** — reduces intubation (3CPO: no mortality difference between modes)
Obesity hypoventilation syndrome	BiPAP (or CPAP if severe OSA)	Strong
Neuromuscular disease and chest-wall deformity	BiPAP S/T with backup rate	Strong
Immunocompromised hypoxaemic failure	NIV or HFNC	Moderate
Weaning and post-extubation prophylaxis in high-risk patients	BiPAP	Moderate
Chest trauma with rib fractures	CPAP	Moderate — with good analgesia
De novo hypoxaemic failure / pneumonia / ARDS	Helmet NIV or HFNC	Weak — high failure rate; monitor closely
Asthma	BiPAP	Weak — only in a monitored setting
Do-not-intubate patients	CPAP/BiPAP	Appropriate as a ceiling of care or for palliation

Contraindications

Absolute	Relative
Respiratory or cardiac arrest	Confusion or agitation (pH < 7.25 with encephalopathy)
Inability to protect the airway; coma unrelated to CO_2 narcosis	Copious secretions
Severe vomiting or upper gastrointestinal bleeding	Haemodynamic instability requiring vasopressors
Fixed upper-airway obstruction	Recent upper gastrointestinal or facial surgery
Facial trauma, burns or surgery preventing a seal	Untreated pneumothorax (drain first)
Patient refusal	Severe hypoxaemia with multi-organ failure

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Choosing an interface

Interface	Advantages	Problems
Oronasal (full-face) mask	**First choice in acute failure** — avoids mouth leak	Claustrophobia, aspiration risk, nasal bridge pressure sores
Nasal mask	Comfortable; speech and expectoration possible; first choice for domiciliary use	Mouth leak reduces efficacy
Nasal pillows	Minimal contact; good for claustrophobia and long-term CPAP	Nasal irritation; limited pressures
Total face mask	Even pressure distribution, fewer sores, rapid application	Bulky; large dead space
Helmet	Tolerates high pressures; no facial sores; good in hypoxaemic failure	CO_2 rebreathing at low flow, noise, poor triggering, cost
Mouthpiece	Daytime support in neuromuscular disease	Needs bulbar function and cooperation

★ CLINICAL PEARLS & EXAM FOCUS

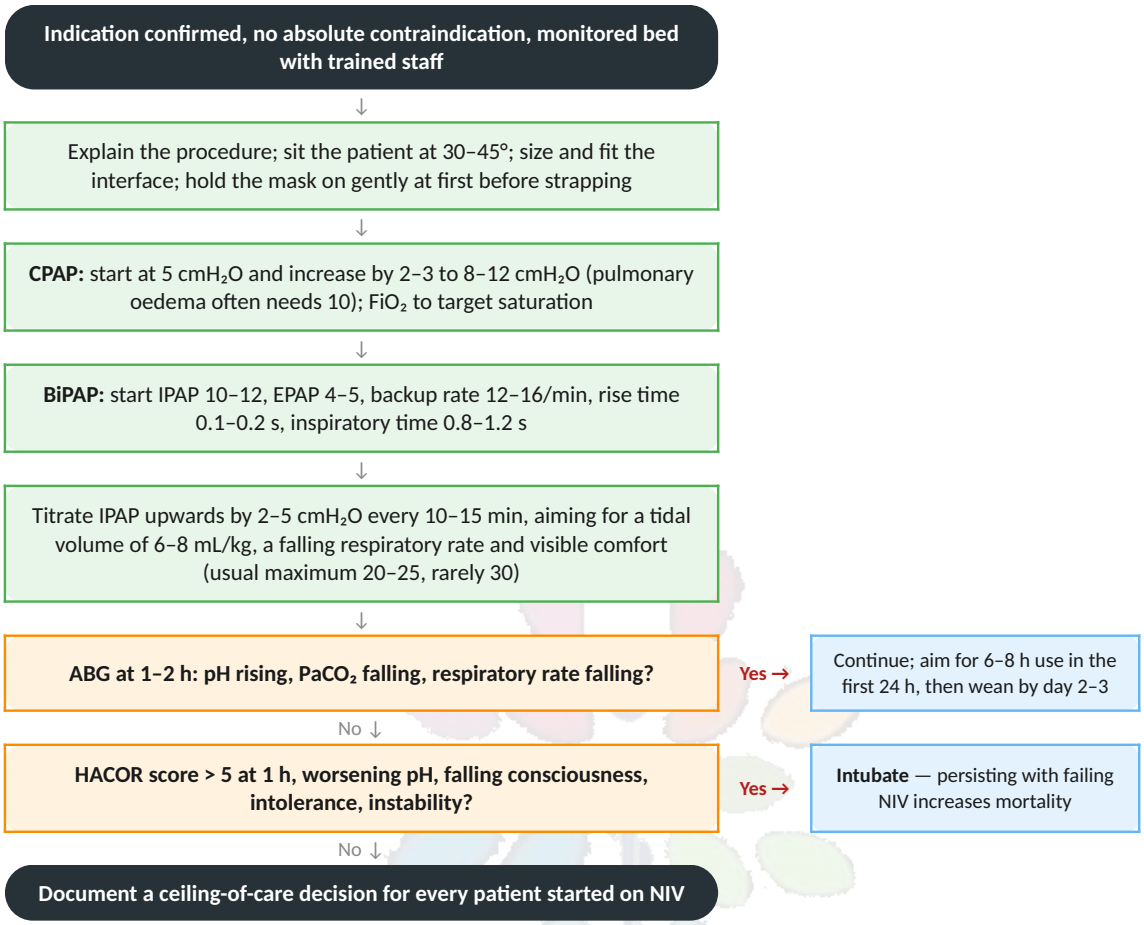
- Size the mask with the manufacturer's gauge — the commonest fitting error is choosing a mask that is too large.
- Two fingers should slide under the headgear strap. Overtightening increases leak by distorting the cushion and guarantees a pressure sore.

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E19.15 Non-Invasive Ventilation 3 · Setting Up CPAP & BiPAP at the Bedside

Algorithm E19.15.1 · Starting acute non-invasive ventilation



Typical starting settings by condition

Condition	Mode	Settings	Oxygen target
AECOPD with acidosis	BiPAP S/T	IPAP 14–20, EPAP 4–5, rate 14	SpO_2 88–92%
Cardiogenic pulmonary oedema	CPAP	10 cmH_2O (range 5–12.5)	SpO_2 94–98%
Obesity hypoventilation	BiPAP S/T or AVAPS	IPAP 18–24, EPAP 8–10, rate 14	SpO_2 88–92%
Neuromuscular / chest wall	BiPAP S/T	IPAP 12–18, EPAP 4, rate 14–16, long Ti	SpO_2 ≥ 92%
Chest trauma	CPAP	5–10 cmH_2O with analgesia	SpO_2 94–98%
Palliative breathlessness	CPAP or BiPAP, low pressures	Comfort-led	Comfort-led

Monitoring in the first two hours

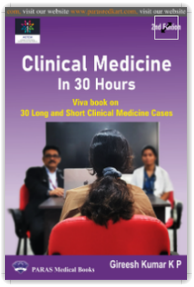
Parameter	Frequency
Clinical — respiratory rate, accessory muscle use, consciousness, comfort	Continuously for 30 min, then every 15–30 min
SpO_2	Continuous
Arterial blood gas	Baseline, 1 h, 4 h, and after any significant change
Tidal volume, leak, respiratory rate on the machine display	Hourly
Haemodynamics	Continuous ECG and hourly blood pressure

★ CLINICAL PEARLS & EXAM FOCUS

- **Pressure support = IPAP − EPAP.** Raising both by the same amount improves oxygenation but does nothing for the $PaCO_2$.
- Nasogastric tubes are not routine; insert one only for vomiting or marked gastric distension, and use a fine-bore tube with a sealing pad.

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E19.16 Non-Invasive Ventilation 4 · Troubleshooting & Weaning

Troubleshooting NIV

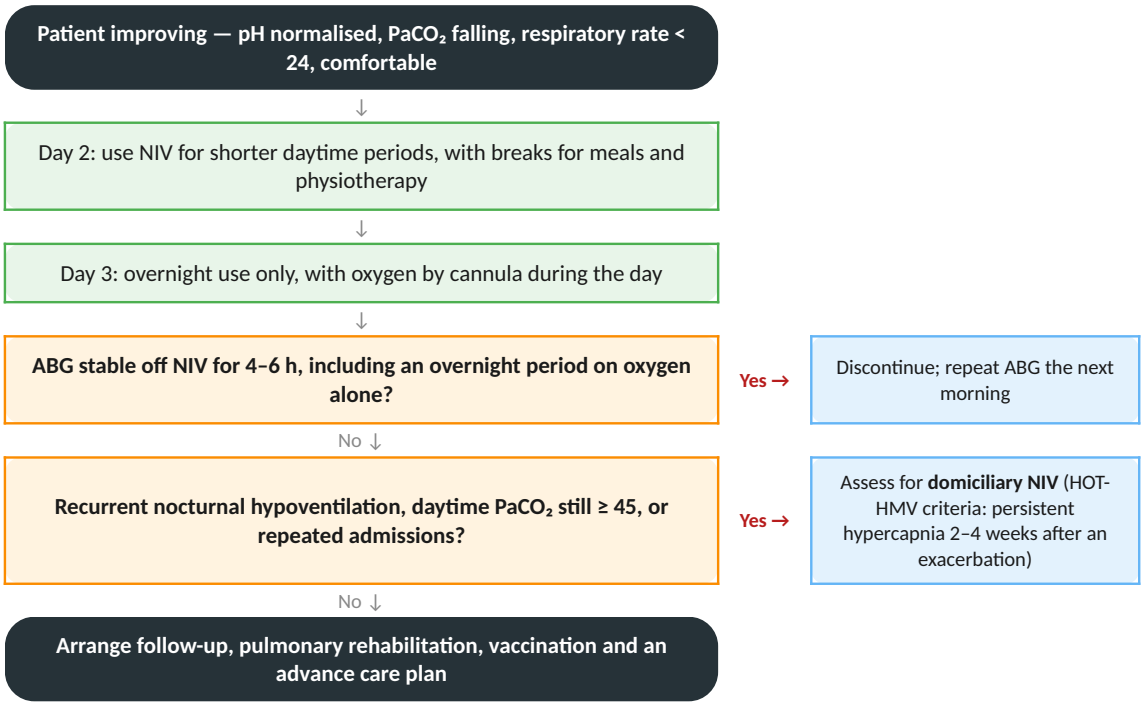
Problem	Cause	Solution
Excess leak	Wrong mask size, overtightened or loose headgear, beard, edentulous face	Refit and resize; chin strap for mouth leak; loosen rather than tighten; consider total-face mask
Nasal bridge pressure sore	Prolonged tight contact	Hydrocolloid dressing from the start, alternate interfaces, release pressure periodically
Dry mouth and nose	Mouth leak, inadequate humidification	Heated humidifier, chin strap
Eye irritation	Leak directed upwards	Refit mask, reduce pressure slightly
Gastric distension	High IPAP exceeding oesophageal opening pressure (> 20–25)	Reduce IPAP, sit upright, nasogastric tube if severe
Asynchrony / auto-triggering	Leak, wrong trigger or cycling settings	Fix the leak first; adjust trigger sensitivity, cycling threshold and rise time
Persistent hypercapnia	Inadequate pressure support, leak, metabolic alkalosis, exhausted patient	Increase IPAP – EPAP gap; correct alkalosis (acetazolamide); reassess for intubation
Intolerance and agitation	Fear, discomfort, delirium	Coaching and reassurance first; low-dose dexmedetomidine or morphine in a monitored setting
Claustrophobia	Mask type	Nasal mask, nasal pillows, helmet

Predicting NIV failure — the HACOR score

Variable	Points
Heart rate ≥ 121/min	1
Acidosis pH < 7.35 (increasing points as pH falls below 7.30, 7.25)	2–4
Consciousness — GCS 13–14 / 11–12 / ≤ 10	2 / 5 / 10
Oxygenation PaO_2/FiO_2 (lower = more points, up to ≤ 100)	1–6
Respiratory rate 31–35 / 36–40 / 41–45 / ≥ 46	1 / 2 / 3 / 4
HACOR > 5 at 1 hour predicts NIV failure — early intubation improves survival	—

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Algorithm E19.16.1 · Weaning from acute NIV



LANDMARK TRIALS & GUIDELINES

- **Plant et al. (2000):** ward-based NIV in acidotic AECOPD reduced intubation and mortality.
- **3CPO (2008):** CPAP and BiPAP both reduce distress in pulmonary oedema; neither improves mortality over standard therapy.
- **HOT-HMV (2017):** home NIV with oxygen after a hypercapnic exacerbation prolonged time to readmission or death.

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E19.17 Non-Invasive Ventilation 5 · Domiciliary NIV, CPAP for OSA & Cough Augmentation

Indications for long-term home NIV

Disease	Criteria to start
COPD	Persistent daytime $PaCO_2$ ≥ 52 mmHg measured 2–4 weeks after an exacerbation, or recurrent acidotic admissions
Obesity hypoventilation	Daytime $PaCO_2$ ≥ 45 with nocturnal hypoventilation; CPAP first if severe OSA coexists
Neuromuscular disease (including motor neurone disease)	Symptoms plus any of: $PaCO_2$ ≥ 45, nocturnal SpO_2 < 88% for ≥ 5 min, FVC < 50% predicted, MIP weaker than −60 cmH_2O, SNIP < 40
Chest-wall disease (kyphoscoliosis, post-thoracoplasty)	As for neuromuscular disease
Bronchiectasis / cystic fibrosis	Hypercapnia, bridge to transplantation

CPAP for obstructive sleep apnoea

Element	Practice
Indication	AHI ≥ 15, or 5–14.9 with sleepiness, cardiovascular disease, or occupational risk
Titration	Auto-titrating CPAP at home, or attended laboratory titration; usual therapeutic pressure 6–12 cmH_2O
Adherence target	≥ 4 h per night on ≥ 70% of nights
Download review	Usage hours, residual AHI, leak — review at 1 month and then yearly
Alternatives	Mandibular advancement device, positional therapy, weight loss (including tirzepatide), surgery, hypoglossal nerve stimulation
Driving advice	Do not drive while sleepy; notify the licensing authority as locally required

Cough augmentation — essential alongside NIV in neuromuscular disease

Technique	Detail
Peak cough flow	Measure at every visit; < 270 L/min = at risk, < 160 L/min = ineffective cough
Breath stacking	Lung volume recruitment with a bag and one-way valve, 3–5 breaths stacked, 2–3 times daily
Manually assisted cough	Abdominal thrust timed with the patient's cough effort
Mechanical insufflation–exsufflation (cough assist)	+40 to −40 cmH_2O, 4–6 cycles per set; the single most effective measure for secretion clearance
Suction and nebulised saline	Adjuncts

★ CLINICAL PEARLS & EXAM FOCUS

- Domiciliary NIV fails most often for interface and adherence reasons — arrange a nurse-led review within two weeks of setting it up.
- In motor neurone disease, NIV improves survival and quality of life, but only if bulbar function permits a seal; discuss it before the FVC falls below 50%.

MERCK MANUAL PROFESSIONAL Noninvasive ventilation Obstructive sleep apnea

E19.18 Invasive Mechanical Ventilation 1 · Indications & The Four Basic Questions

Indications for intubation and mechanical ventilation

Category	Examples
Failure of oxygenation	ARDS, severe pneumonia, pulmonary oedema not responding to NIV
Failure of ventilation	Severe asthma or AECOPD with exhaustion, neuromuscular weakness, drug overdose
Failure to protect the airway	GCS ≤ 8, bulbar palsy, massive haemoptysis, severe vomiting
Anticipated clinical course	Airway burns, expanding neck haematoma, transfer of an unstable patient
Reduction of work of breathing	Shock with high respiratory workload, severe metabolic acidosis
Therapeutic	Permissive hypercapnia, controlled ventilation for raised intracranial pressure, surgery

Every ventilator order answers four questions

- **1. What triggers the breath?** The patient (flow or pressure trigger) or the machine (time, i.e. the set rate).
- **2. What is the target — volume or pressure?** Volume control guarantees a tidal volume and lets pressure vary; pressure control guarantees a pressure and lets volume vary.
- **3. What ends the breath (cycling)?** Volume delivered, inspiratory time elapsed, or a fall in flow to a set percentage of peak (as in pressure support).
- **4. What is the baseline pressure?** PEEP.

Core settings and what each one does

Setting	Typical starting value	Chiefly affects
Tidal volume	6 mL/kg predicted body weight	$PaCO_2$; lung stress
Respiratory rate	12–20/min	$PaCO_2$
FiO_2	1.0 initially, then lowest to meet target	PaO_2
PEEP	5 cmH_2O (higher in ARDS)	PaO_2; recruitment; preload
Inspiratory flow / time	60 L/min; Ti 0.8–1.0 s	I:E ratio; comfort
Trigger sensitivity	Flow 1–3 L/min	Work of breathing; asynchrony

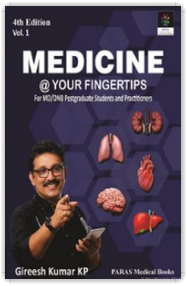
- **Predicted body weight:** men 50 + 0.91 × (height in cm − 152.4); women 45.5 + 0.91 × (height in cm − 152.4). Tidal volume is **always** set on predicted, never actual, body weight.

Safety limits to set on every ventilator

Limit	Value
Plateau pressure	≤ 30 cmH_2O
Driving pressure (Pplat – PEEP)	≤ 15 cmH_2O
High-pressure alarm	10 cmH_2O above usual peak
Low minute-ventilation alarm	2 L/min below current
Apnoea backup	Always enabled in spontaneous modes

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E19.19 Invasive Mechanical Ventilation 2 · Modes Explained

Conventional modes

Mode	How it works	Advantages	Disadvantages
Volume control (VC-AC / CMV)	Set tidal volume and rate delivered with a fixed flow; every patient effort triggers a full set breath	Guaranteed minute ventilation; simple; ARDSNet protocols are built on it	Pressure varies with compliance; flow starvation and asynchrony in the awake patient
Pressure control (PC-AC)	Set inspiratory pressure and time; decelerating flow; tidal volume varies with compliance and effort	Limits peak alveolar pressure; better distribution of gas; more comfortable	**No guaranteed tidal volume** — volume falls as compliance worsens
SIMV (synchronised intermittent mandatory ventilation)	Set number of mandatory breaths synchronised with effort; spontaneous breaths between them, usually with pressure support	Allows spontaneous effort	Slower weaning than pressure support alone; asynchrony; largely historical
Pressure support ventilation (PSV)	Every breath is patient-triggered, pressure-targeted and flow-cycled (usually at 25% of peak flow)	Comfortable; used for weaning and spontaneous breathing trials	No backup unless apnoea alarm set; apnoea risk
CPAP	Continuous pressure, no support	Spontaneous breathing trial, oxygenation	No ventilatory assistance

Dual-control and advanced modes

Mode	Principle	Use
PRVC / VC+ / AutoFlow	Pressure-controlled breath with the pressure automatically adjusted breath-to-breath to deliver a target tidal volume	Combines volume guarantee with decelerating flow; popular default in modern ICUs
APRV (airway pressure release ventilation)	Prolonged high pressure (P-high) with brief timed releases (P-low), allowing spontaneous breathing throughout	Refractory hypoxaemia; open-lung strategy; needs experience
NAVA (neurally adjusted ventilatory assist)	Support proportional to diaphragmatic electrical activity (oesophageal catheter)	Excellent synchrony; weaning, paediatrics
PAV+ (proportional assist ventilation)	Support proportional to the patient's instantaneous effort	Weaning; preserves respiratory drive
ASV / adaptive support	Closed-loop selection of rate and tidal volume to minimise work	Automated weaning
HFOV	Very small tidal volumes at 3–15 Hz around a constant mean pressure	Abandoned in adult ARDS (OSCILLATE harm); neonatal rescue

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Algorithm E19.19.1 · Choosing an initial mode

Patient newly intubated

↓

Deeply sedated or paralysed, ARDS, or strict volume control required? — Yes → Volume control (or PRVC) with 6 mL/kg PBW

No ↓

Severe obstructive disease needing long expiratory time? — Yes → Volume control with high flow and low rate — gives direct control of inspiratory time

No ↓

High airway pressures with poor compliance and a sedated patient? — Yes → Pressure control or PRVC, limiting driving pressure

No ↓

Awake, cooperative, ready to begin weaning? — Yes → Pressure support 8–12 cmH_2O with PEEP 5

No ↓

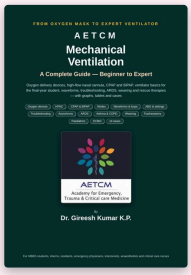
Reassess the mode at every ward round — modes are tools for the current problem, not permanent prescriptions

★ CLINICAL PEARLS & EXAM FOCUS

- No mode has ever been shown to reduce mortality; **how** you set it (tidal volume, plateau and driving pressure) is what matters.
- In pressure control, a falling tidal volume at unchanged settings means worsening compliance — look for pneumothorax, collapse, oedema or abdominal distension.

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E19.20 Invasive Mechanical Ventilation 3 · Disease-Specific Ventilator Settings

Initial ventilator settings by disease

Condition	Mode	Tidal volume	Rate	PEEP	I:E / flow	Targets and cautions
ARDS	VC or PRVC	6 mL/kg PBW (4–8)	20–30 (up to 35)	High, by ARDSNet FiO_2/PEEP table (often 10–18)	I:E 1:1–1:2	Pplat ≤ 30, driving pressure ≤ 15, pH ≥ 7.20; prone if P/F < 150
Severe asthma	VC	6–8 mL/kg PBW	**8–12**	0–5	Flow 80–100 L/min, **I:E 1:4–1:5**	Permissive hypercapnia (pH ≥ 7.15); watch auto-PEEP; disconnect if hypotensive
AECOPD	VC or PSV	6–8 mL/kg PBW	10–14	3–5 (≈ 80% of intrinsic PEEP)	Long expiration	Do not over-ventilate — target the patient's **baseline** $PaCO_2$, not 40
Neuromuscular / chest wall	VC or PC	6–8 mL/kg PBW	12–16	5	Standard	Normal lungs — avoid atelectasis; sigh breaths; aggressive secretion clearance
Traumatic brain injury	VC	6–8 mL/kg PBW	14–18	5–8	Standard	$PaCO_2$ 35–38 mmHg; avoid hypoxia and hypotension; hyperventilate only briefly for herniation
Cardiogenic pulmonary oedema	VC or PC	6–8 mL/kg PBW	14–18	8–12	Standard	PEEP reduces preload and afterload — beneficial; watch for hypotension
Bronchopleural fistula / air leak	PC or VC, lowest pressures	4–6 mL/kg PBW	Higher	Lowest tolerable	Short Ti	Minimise mean airway pressure; permissive hypercapnia; consider independent lung ventilation
Massive haemoptysis	VC	Standard	Standard	Low	—	Bleeding side **down**; large tube; consider selective intubation or blocker
Post-operative / normal lungs	VC or PRVC	6–8 mL/kg PBW	12–16	5	Standard	Protective ventilation benefits normal lungs too (IMPROVE trial)
Right ventricular failure / PAH	VC	6 mL/kg PBW	Moderate	Lowest effective	—	Avoid hypercapnia, acidosis, hypoxia and high PEEP — all raise PVR
Obesity	VC or PRVC	6–8 mL/kg **predicted** body weight	14–18	8–12	Reverse Trendelenburg	Higher PEEP and recruitment; head-up position
Pregnancy	VC	6–8 mL/kg PBW	Higher	5–8	Left lateral tilt	Target $PaCO_2$ 28–32 mmHg (pregnancy baseline); SpO_2 ≥ 95%

ARDSNet lower-PEEP FiO_2/PEEP table (abridged)

FiO_2	0.3	0.4	0.5	0.6	0.7	0.8	0.9	1.0
PEEP (cmH_2O)	5	5–8	8–10	10	10–14	14	14–18	18–24

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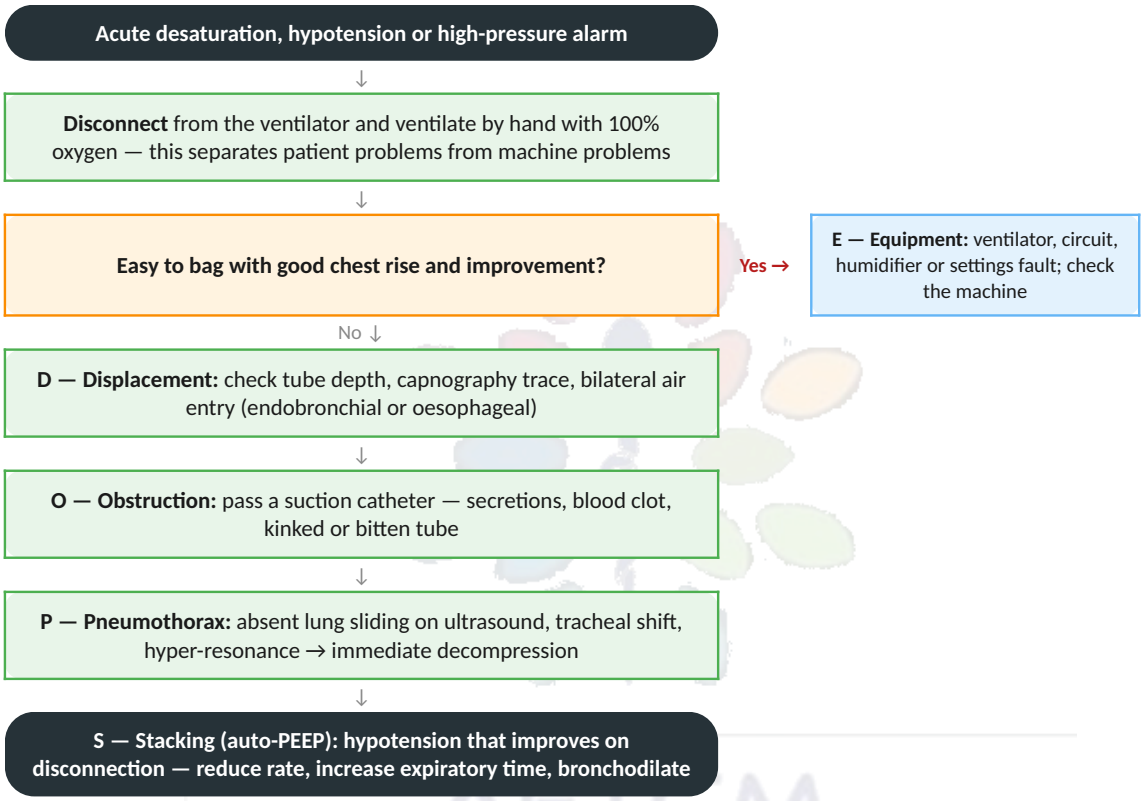
★ CLINICAL PEARLS & EXAM FOCUS

- One rule covers nearly every disease: **6 mL/kg predicted body weight, plateau ≤ 30, driving pressure ≤ 15**. Then adjust rate and PEEP for the specific disease.
- In obstructive disease, the key variable is **expiratory time**, which you control through rate and inspiratory flow — not through PEEP.

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E19.21 Invasive Mechanical Ventilation 4 · Alarms, Complications & Daily Care

Algorithm E19.21.1 · Sudden deterioration in a ventilated patient — the DOPES approach



High-pressure alarm — peak versus plateau

Finding	Meaning	Causes
Peak high, plateau normal (gap > 10 cmH_2O)	Resistance problem	Secretions, bronchospasm, kinked or bitten tube, small tube, water in the circuit
Peak and plateau both high	Compliance problem	Pneumothorax, endobronchial intubation, atelectasis, pulmonary oedema, ARDS progression, abdominal distension, chest-wall rigidity, auto-PEEP

The daily ventilator bundle (FAST HUG)

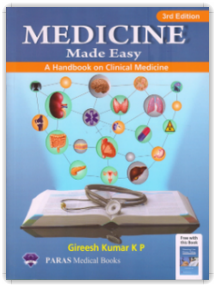
Letter	Element
F	Feeding — enteral within 24–48 h
A	Analgesia first, then sedation; daily sedation interruption
S	Sedation — target RASS −2 to 0; assess delirium (CAM-ICU)
T	Thromboprophylaxis
H	Head of bed elevated 30–45°
U	Ulcer prophylaxis — only if coagulopathy, shock or steroids
G	Glucose control 140–180 mg/dL
Plus	Oral care, cuff pressure 20–30 cmH_2O, subglottic suction, spontaneous breathing trial, early mobilisation

Complications of mechanical ventilation

Complication	Prevention
Ventilator-associated pneumonia	Bundle above; subglottic drainage; avoid unnecessary circuit changes
Ventilator-induced lung injury (volutrauma, barotrauma, atelectrauma, biotrauma)	6 mL/kg, Pplat ≤ 30, driving pressure ≤ 15, adequate PEEP
Pneumothorax, pneumomediastinum, surgical emphysema	Limit pressures; daily ultrasound in high-risk patients
Haemodynamic compromise	Lowest effective mean airway pressure; volume status
Oxygen toxicity, absorption atelectasis	Lowest FiO_2 that meets the target
ICU-acquired weakness, diaphragm atrophy	Minimise paralysis, early mobilisation, allow spontaneous effort when safe
Tracheal stenosis, tracheomalacia, tracheo-oesophageal fistula	Cuff pressure 20–30 cmH_2O; appropriate tube size
Delirium, PTSD	Light sedation, orientation, sleep hygiene, family presence

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E19.22 Rapid Sequence Intubation 1 · Preparation & The Seven Ps

IN BRIEF Rapid sequence intubation is the near-simultaneous administration of a potent induction agent and a rapidly acting neuromuscular blocking drug to produce optimal intubating conditions quickly, minimising the time the airway is unprotected.

The seven Ps of RSI

Step	Timing	Content
Preparation	−10 min	SOAP-ME: Suction, Oxygen, Airway devices (two laryngoscopes, bougie, supraglottic airway, surgical kit), Pharmacy (drugs drawn and labelled), Monitors (ECG, SpO_2, blood pressure, **waveform capnography**), End-tidal CO_2; assign roles; verbalise the airway plan A–B–C–D; check IV access ×2
Pre-oxygenation	−5 min	3 minutes of tidal breathing on a non-rebreather at 15 L/min, or 8 vital-capacity breaths; HFNC 60 L/min or NIV in the hypoxaemic patient (PREOXI trial favours NIV); head up 20–30°
Pre-intubation optimisation	−5 min	Correct hypotension (fluid, push-dose vasopressor), treat acidosis, prepare the "resuscitate before you intubate" plan; apnoeic oxygenation via nasal cannula at 15 L/min
Paralysis with induction	0	Induction agent, immediately followed by the neuromuscular blocker, both as a rapid push
Positioning	0 to +45 s	Ear-to-sternal-notch alignment; ramped position in obesity; manual in-line stabilisation in trauma
Placement with proof	+45–60 s	Laryngoscopy (videolaryngoscope first — DEVICE trial), tube, cuff inflation; confirm with **waveform capnography** — "no trace = wrong place"
Post-intubation care	+2 min	Secure tube, note depth (21 cm women, 23 cm men at the teeth), sedation and analgesia infusion, ventilator settings, chest radiograph, nasogastric tube, analgesia before more sedation

Predicting the difficult airway

Tool	Elements
LEMON	Look externally; Evaluate 3-3-2 (3 fingers mouth opening, 3 fingers hyomental, 2 fingers thyrohyoid); Mallampati; Obstruction/Obesity; Neck mobility
MACOCHA (ICU-validated)	Mallampati III–IV, obstructive sleep apnoea, reduced cervical mobility, mouth opening < 3 cm, coma, severe hypoxaemia, non-anaesthetist operator
MOANS (difficult BVM)	Mask seal/beard, Obesity/Obstruction, Age > 55, No teeth, Stiff lungs
SHORT (difficult surgical airway)	Surgery/scar, Haematoma, Obesity, Radiation, Tumour
HOp killers (physiologically difficult)	**H**ypotension, **O**xygenation failure, **p**H (metabolic acidosis)

★ **CLINICAL PEARLS & EXAM FOCUS**

- The physiologically difficult airway kills more often than the anatomically difficult airway. Give fluid and have a vasopressor running **before** induction in a shocked patient.
- Apnoeic oxygenation — nasal cannula at 15 L/min left in place throughout laryngoscopy — prolongs safe apnoea time and costs nothing.

MERCK MANUAL PROFESSIONAL Airway establishment and control

E19.23 Rapid Sequence Intubation 2 · Drug Table with Doses & Cautions

Induction (hypnotic) agents

Class	Drug	Dose	Onset / duration	Indication	Caution
Phenol derivative	**Propofol**	1.5–2.5 mg/kg IV (0.5–1 mg/kg if shocked or elderly)	15–45 s / 5–10 min	Haemodynamically stable patient; status epilepticus; bronchodilator properties	Dose-dependent **hypotension** and myocardial depression; apnoea; pain on injection; avoid in shock
Imidazole	**Etomidate**	0.3 mg/kg IV (0.15 mg/kg if shocked)	15–45 s / 5–15 min	Haemodynamic instability, head injury (lowers ICP, preserves MAP)	Transient **adrenal suppression** (single dose); myoclonus; no analgesia; nausea
NMDA antagonist	**Ketamine**	1–2 mg/kg IV (0.5–1 mg/kg if shocked); 4 mg/kg IM	45–60 s / 10–20 min	**Shock, sepsis, severe asthma** (bronchodilator), trauma, delayed sequence intubation	Hypersalivation; emergence phenomena; direct myocardial depression in catecholamine-depleted patients; raises blood pressure
Benzodiazepine	**Midazolam**	0.1–0.3 mg/kg IV	60–90 s / 15–30 min	Only if nothing else available	Slow, unreliable induction; hypotension; prolonged sedation
Barbiturate	**Thiopentone**	3–5 mg/kg IV	15–30 s / 5–10 min	Status epilepticus, raised ICP	Severe hypotension; histamine release; **contraindicated in porphyria**; avoid in asthma (histamine release); tissue necrosis if extravasated

Neuromuscular blocking agents

Class	Drug	Dose	Onset / duration	Indication	Caution
Depolarising	**Succinylcholine**	1.5 mg/kg IV (2 mg/kg in children; 4 mg/kg IM)	45–60 s / 6–10 min	Fastest onset and offset; classic RSI agent; anticipated difficult airway where quick return of breathing is wanted	**Hyperkalaemia** — contraindicated in burns > 24 h, crush injury, denervation, prolonged immobility > 72 h, neuromuscular disease, pre-existing hyperkalaemia, rhabdomyolysis; malignant hyperthermia; bradycardia (children — atropine); raised intraocular and intragastric pressure
Non-depolarising	**Rocuronium**	1.2 mg/kg IV for RSI (0.6 mg/kg for maintenance)	45–60 s / 45–70 min	When succinylcholine is contraindicated; now first choice in many units	Long duration — **must be able to oxygenate**; reverse with sugammadex 16 mg/kg
Non-depolarising	**Vecuronium**	0.15–0.3 mg/kg IV	90–120 s / 60–90 min	Alternative where rocuronium unavailable	Slower onset; prolonged in hepatic and renal failure
Non-depolarising	**Cisatracurium**	0.15–0.2 mg/kg IV, then 1–3 µg/kg/min	2–3 min / 45–60 min	Infusion in severe ARDS; organ-independent (Hofmann) elimination	Too slow for RSI

Adjuncts, pressors and reversal agents

Class	Drug	Dose	Indication	Caution
Opioid	Fentanyl	1–3 µg/kg IV 2–3 min before induction	Blunts the sympathetic response (aortic dissection, raised ICP, ischaemia)	Chest-wall rigidity with rapid high doses; apnoea; hypotension — omit in shock
Anticholinergic	Atropine	0.02 mg/kg IV (minimum 0.1 mg)	Bradycardia with succinylcholine in children	Tachycardia
Lidocaine	Lignocaine	1.5 mg/kg IV	Formerly for raised ICP and reactive airways — evidence weak	Not routine
Push-dose vasopressor	Phenylephrine	50–200 µg IV boluses	Peri-intubation hypotension	Reflex bradycardia
Push-dose vasopressor	Adrenaline	10–20 µg IV boluses (1 mL of 10 µg/mL)	Peri-intubation hypotension with low cardiac output	Tachyarrhythmia; dilution errors — label clearly
Reversal	**Sugammadex**	16 mg/kg IV for immediate reversal of rocuronium	"Can't intubate, can't oxygenate" after rocuronium	Does not reverse succinylcholine; cost; hypersensitivity
Reversal	Neostigmine + glycopyrrolate	50 µg/kg + 10 µg/kg	Reversal of partial non-depolarising block	Ineffective for deep block; bradycardia
Post-intubation sedation	Propofol / midazolam / dexmedetomidine + fentanyl	Infusion	Must be started **immediately** after tube confirmation	Awareness with paralysis is a serious harm — never leave a paralysed patient unsedated

★ CLINICAL PEARLS & EXAM FOCUS

- Reduce the induction dose and increase the paralytic dose in shock: the hypnotic distributes to a contracted circulation, while the relaxant needs circulation time to work.
- Rocuronium 1.2 mg/kg and succinylcholine 1.5 mg/kg give equivalent intubating conditions at 60 seconds.

AETCM YOUTUBE · VIDEO LECTURES ▶ rapid sequence intubation ▶ RSI drugs ▶ intubation

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E19.24 Rapid Sequence Intubation 3 · Delayed & Awake Techniques, Failed Airway

IN BRIEF **Delayed sequence intubation (DSI)** is procedural sedation for the procedure of pre-oxygenation. A dissociative dose of ketamine is given to an agitated, hypoxaemic patient who will not tolerate a mask, pre-oxygenation is then completed properly, and only afterwards is the paralytic given.

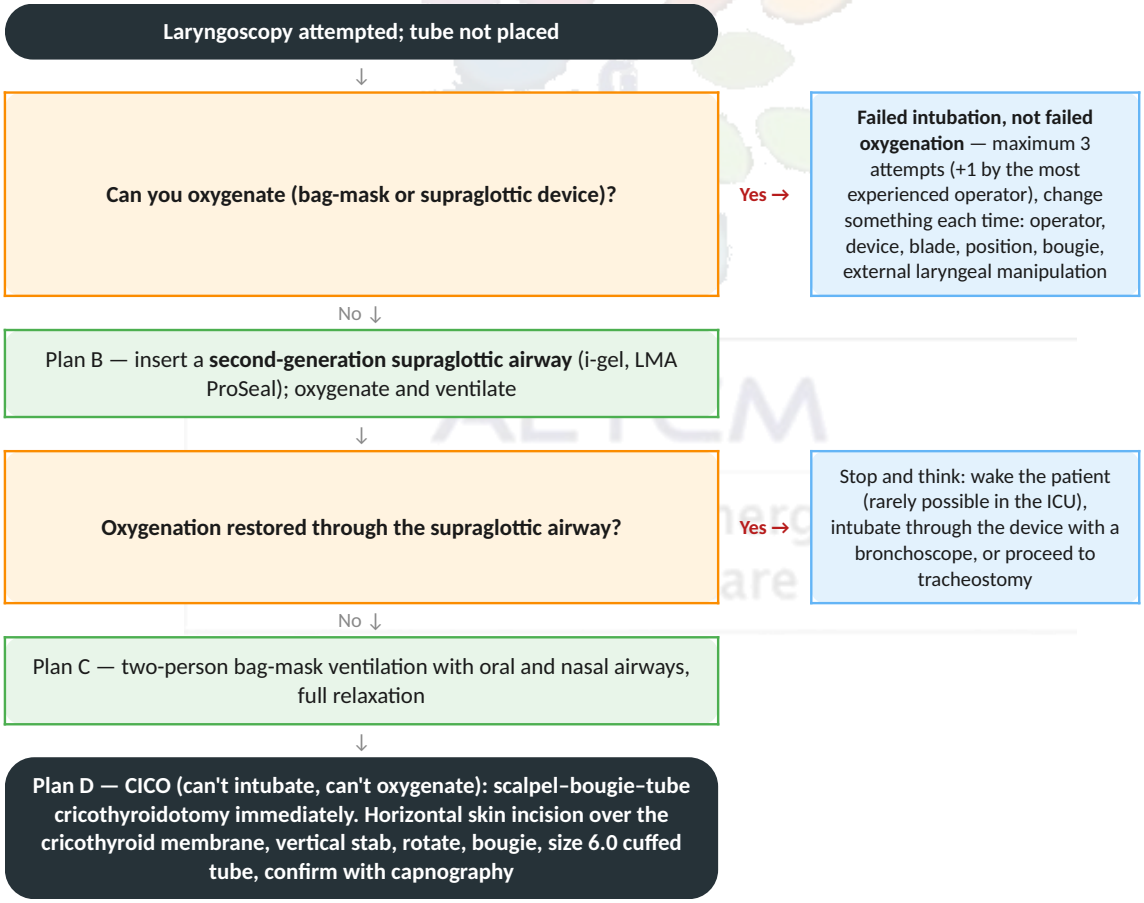
Delayed sequence intubation

Element	Detail
Indication	Agitated, delirious or uncooperative hypoxaemic patient who pulls off the oxygen mask; saturations too low for safe apnoea
Agent	**Ketamine 1–1.5 mg/kg IV** given slowly over 15–30 s — preserves airway reflexes, respiratory drive and haemodynamics
Then	Apply non-rebreather at 15 L/min, HFNC 60 L/min, or NIV for **3 minutes** of true pre-oxygenation; add nasal cannula at 15 L/min for apnoeic oxygenation
Then	Once SpO_2 has plateaued (ideally ≥ 95%), give the paralytic (rocuronium 1.2 mg/kg or succinylcholine 1.5 mg/kg) and intubate
Evidence	Weingart's observational series showed a mean rise in SpO_2 of about 9%; widely adopted in emergency medicine
Cautions	Not a substitute for resuscitation; laryngospasm is rare but possible; avoid in patients who will not tolerate any apnoea

Awake tracheal intubation

Element	Detail
Indication	Predicted difficult airway with a patient who can cooperate — upper-airway tumour, severe ankylosis, previous radiotherapy, large goitre, unstable cervical spine
Technique	Topical anaesthesia (lidocaine ≤ 9 mg/kg total; nebulised 4%, spray-as-you-go, nerve blocks), minimal sedation (dexmedetomidine or low-dose remifentanil), flexible bronchoscope or videolaryngoscope
Confirmation	Visualise the carina, then railroad the tube; confirm with capnography **before** giving any induction agent
Caution	Never abolish spontaneous ventilation until the tube is confirmed in the trachea

Algorithm E19.24.1 · The failed airway — a declared emergency



Post-intubation checklist

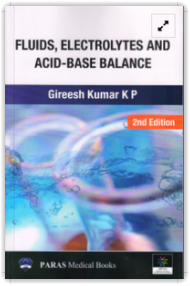
Item	Detail
Confirm	Waveform capnography; bilateral chest rise and auscultation; chest radiograph (tip 3–5 cm above the carina)
Secure	Tape or holder; record depth at the teeth
Sedate	Analgesia first, then sedation to RASS −2 to 0
Ventilate	Lung-protective settings by disease (E19.20)
Decompress	Nasogastric tube; cuff pressure 20–30 cmH_2O
Monitor	Continuous SpO_2, capnography, blood pressure; ABG at 30 min
Document	Grade of view, number of attempts, device used, difficulties — and alert the patient's record for future anaesthetics

★ CLINICAL PEARLS & EXAM FOCUS

- Declare the emergency out loud: "This is a can't-intubate, can't-oxygenate situation — I am performing a front-of-neck airway." Verbalising it breaks the fixation that causes delay.
- A failed airway should always end with a written alert in the notes and a letter to the patient so that the next team is warned.

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E19.25 Quick Reference — Oxygen, NIV & Ventilation on One Spread

Oxygen devices — flow and FiO_2 summary

Device	Flow	FiO_2
Nasal cannula	1–6 L/min	24–44%
Simple mask	5–10 L/min	40–60%
Partial rebreather	6–10 L/min	60–80%
Non-rebreather	10–15 L/min	90–100% (60–80% in practice)
Venturi	2–15 L/min by adapter	24, 28, 31, 35, 40, 60%
HFNC	40–60+ L/min	21–100%

NIV at a glance

Setting	CPAP	BiPAP
Start	5 cmH_2O	IPAP 10–12 / EPAP 4–5
Usual therapeutic	8–12 cmH_2O	IPAP 14–20 / EPAP 4–5
Maximum	15 cmH_2O	IPAP 25–30
Backup rate	—	12–16/min
Review	ABG at 1 h	ABG at 1 h; HACOR > 5 → intubate

Ventilator starting points

Variable	Value
Tidal volume	6 mL/kg predicted body weight
Rate	12–20/min (8–12 in asthma; up to 35 in ARDS)
PEEP	5 cmH_2O (ARDSNet table in ARDS; 0–5 in asthma)
FiO_2	1.0, then lowest to meet target
Plateau pressure	≤ 30 cmH_2O
Driving pressure	≤ 15 cmH_2O

RSI drug doses — the four numbers to remember

Drug	Standard dose	Shock dose
Ketamine	1.5 mg/kg	0.5–1 mg/kg
Etomidate	0.3 mg/kg	0.15 mg/kg
Propofol	2 mg/kg	0.5–1 mg/kg (avoid)
Succinylcholine	1.5 mg/kg	1.5 mg/kg
Rocuronium	1.2 mg/kg	1.2 mg/kg
Sugammadex (rescue)	16 mg/kg	16 mg/kg

★ **CLINICAL PEARLS & EXAM FOCUS**

- Three numbers carry most of this chapter: **4% per litre** of nasal cannula flow, **6 mL/kg** predicted body weight, and **IPAP minus EPAP** is the pressure support.

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