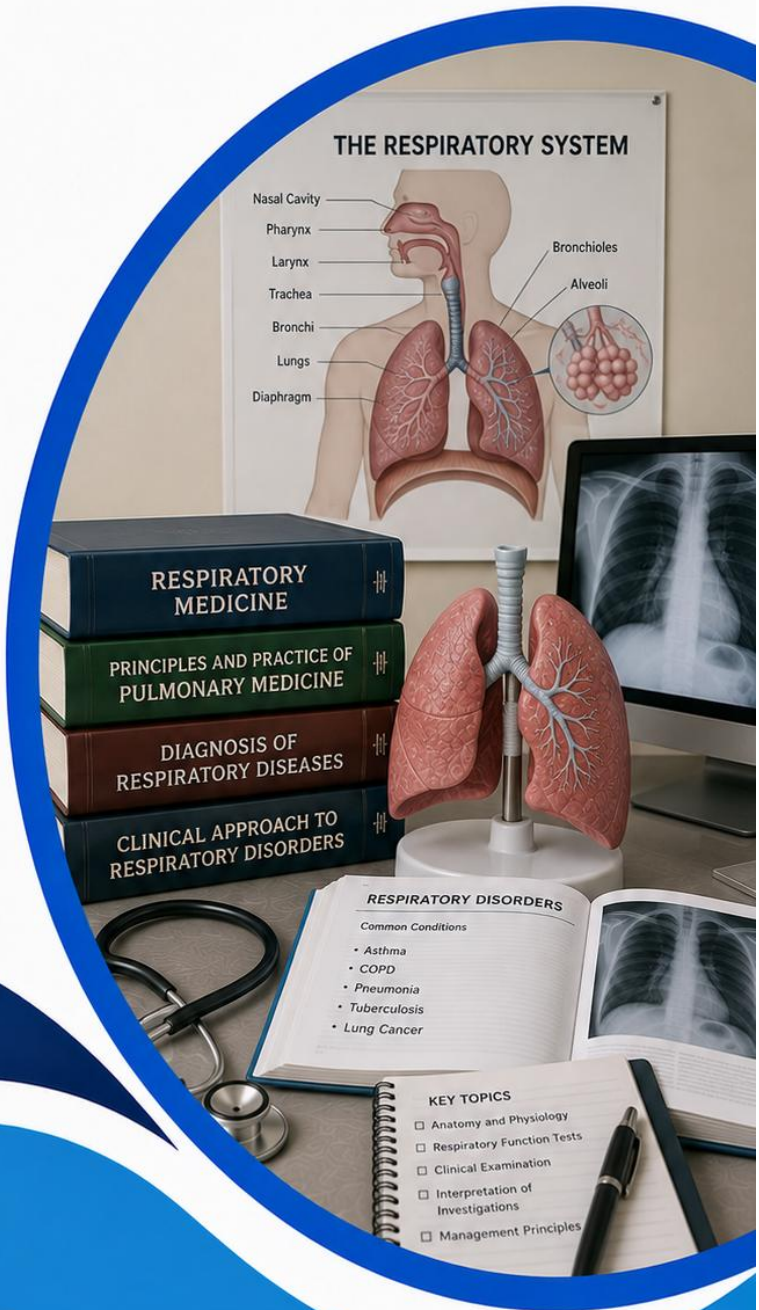
La-Net

MED 402

RESPIRATORY



Download on the App Store

GET IT ON Google Play

Course video is available on our app

lanetonline.com

Course code: MED 402

Course title: Respiratory

Course credit load: 2 Units

Series 1: Fundamentals of Respiratory Evaluation and Clinical Skills

- **Part 1.1: Anatomy and Physiology of the Respiratory System**
  - Sub Part 1.1.1: Gross anatomy of the respiratory tract
  - Sub Part 1.1.2: Mechanics of breathing and lung volumes
  - Sub Part 1.1.3: Gas exchange and control of ventilation
- **Part 1.2: Respiratory History Taking**
  - Sub Part 1.2.1: Cardinal respiratory symptoms
  - Sub Part 1.2.2: Past medical, drug, occupational, and social history
  - Sub Part 1.2.3: Systems review and functional assessment
- **Part 1.3: Physical Examination of the Respiratory System**
  - Sub Part 1.3.1: General inspection and peripheral signs
  - Sub Part 1.3.2: Chest inspection and palpation
  - Sub Part 1.3.3: Percussion and auscultation
- **Part 1.4: Common Respiratory Investigations**
  - Sub Part 1.4.1: Chest X-ray interpretation
  - Sub Part 1.4.2: Pulmonary function tests

- Sub Part 1.4.3: Arterial blood gas analysis

Introduction

The respiratory system is responsible for delivering oxygen to the body and eliminating carbon dioxide. Diseases of the respiratory system are among the most common causes of morbidity and mortality in Nigeria and globally, including pneumonia, tuberculosis, asthma, and chronic obstructive pulmonary disease. Competent evaluation of the respiratory patient requires a foundation in respiratory anatomy and physiology, systematic history taking and physical examination, and the ability to select and interpret the key investigations.

This Series provides that foundation. It covers the anatomy of the respiratory tract, the mechanics and physiology of breathing, and the clinical approach to the respiratory patient from history through to physical examination. It then introduces the most important investigations in respiratory medicine: the chest X-ray, pulmonary function tests, and arterial blood gas analysis. These skills underpin all the clinical respiratory knowledge covered in subsequent Series of this course.

A systematic approach to respiratory assessment, applied consistently to every patient, ensures that important findings are not missed and that the clinical reasoning leading to diagnosis and management is grounded in objective data.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 1.1** describe the gross anatomy of the respiratory tract and the mechanics of breathing, lung volumes, and gas exchange (Linked to Part 1.1),
- **SLO 1.2** explain the control of ventilation and the physiological basis of common respiratory symptoms (Linked to Part 1.1),

- **SLO 1.3** conduct a systematic respiratory history covering the cardinal symptoms, past history, occupational and social history, and functional assessment (Linked to Part 1.2),
- **SLO 1.4** perform and describe the findings of a systematic respiratory examination including inspection, palpation, percussion, and auscultation (Linked to Part 1.3), and
- **SLO 1.5** interpret a chest X-ray, pulmonary function tests, and arterial blood gas analysis in the context of common respiratory conditions (Linked to Part 1.4).

1.1 Anatomy and Physiology of the Respiratory System

1.1.1 Gross anatomy of the respiratory tract

The respiratory tract is divided into the **upper respiratory tract** (nose, nasopharynx, oropharynx, larynx) and the **lower respiratory tract** (trachea, bronchi, bronchioles, and alveoli). The trachea bifurcates at the **carina** (at the level of the angle of Louis, T4/5) into the right and left main bronchi; the right main bronchus is shorter, wider, and more vertical, making it the more common site of inhaled foreign bodies.

The lungs are paired organs occupying the pleural cavities. The right lung has three lobes (upper, middle, and lower); the left lung has two lobes (upper and lower, with the lingula corresponding to the right middle lobe). The functional unit of gas exchange is the **acinus**, comprising the respiratory bronchioles, alveolar ducts, and alveoli. The **pleura** consists of visceral (covering lung surface) and parietal (lining chest wall) layers; the potential space between them normally contains a small amount of lubricating fluid.

Fill in the blank: The right main bronchus is shorter, wider, and more vertical than the left, making it the more common site of inhaled foreign _____; the right lung has three lobes; the left lung has two lobes.

1.1.2 Mechanics of breathing and lung volumes

Inspiration is an active process driven by contraction of the **diaphragm** (the primary muscle of inspiration) and the external intercostal muscles, which increase chest dimensions and reduce

intrathoracic pressure, drawing air into the lungs. Expiration is passive at rest but active during exercise and in obstructive lung disease (using internal intercostals and abdominal muscles).

Key lung volumes include: **tidal volume (TV)** (volume breathed in and out in one normal breath; approximately 500 mL at rest), **vital capacity (VC)** (maximum volume exhaled after maximum inhalation; normally 4 to 5 litres), **total lung capacity (TLC)** (all air in the lungs at full inspiration), **residual volume (RV)** (air remaining after maximum exhalation), and **functional residual capacity (FRC)** (air remaining after normal exhalation). FEV1 (forced expiratory volume in 1 second) and FVC (forced vital capacity) are the key spirometric measurements.

Fill in the blank: Inspiration is active (diaphragm and external intercostals); expiration is passive at rest; key lung volumes include tidal volume (500 mL), vital capacity, TLC, residual volume, and FRC; FEV1 and FVC are key _____ measurements.

1.1.3 Gas exchange and control of ventilation

Gas exchange occurs across the alveolar-capillary membrane: oxygen diffuses from alveolar air (PO2 approximately 100 mmHg) into pulmonary capillary blood, and carbon dioxide diffuses in the opposite direction. Efficient gas exchange requires **ventilation-perfusion (V/Q) matching**; V/Q mismatch is the most common cause of hypoxaemia in respiratory disease. The normal arterial PO2 is 10 to 13 kPa (75 to 100 mmHg) and PaCO2 is 4.7 to 6.0 kPa (35 to 45 mmHg).

Ventilation is controlled by the **respiratory centre in the brainstem** (medulla and pons), which responds to chemical stimuli: central chemoreceptors in the medulla respond to PaCO2 (the primary drive to breathe), and peripheral chemoreceptors in the carotid and aortic bodies respond to PaO2, PaCO2, and pH. In patients with chronic hypercapnia (as in severe COPD), the **hypoxic drive** (low PaO2 stimulating peripheral chemoreceptors) may become the primary respiratory stimulus, making high-flow oxygen dangerous.

Fill in the blank: Gas exchange requires V/Q matching; hypoxaemia is most commonly caused by V/Q _____; the primary drive to breathe is PaCO2 via central chemoreceptors; in chronic hypercapnia, the hypoxic drive may become primary.

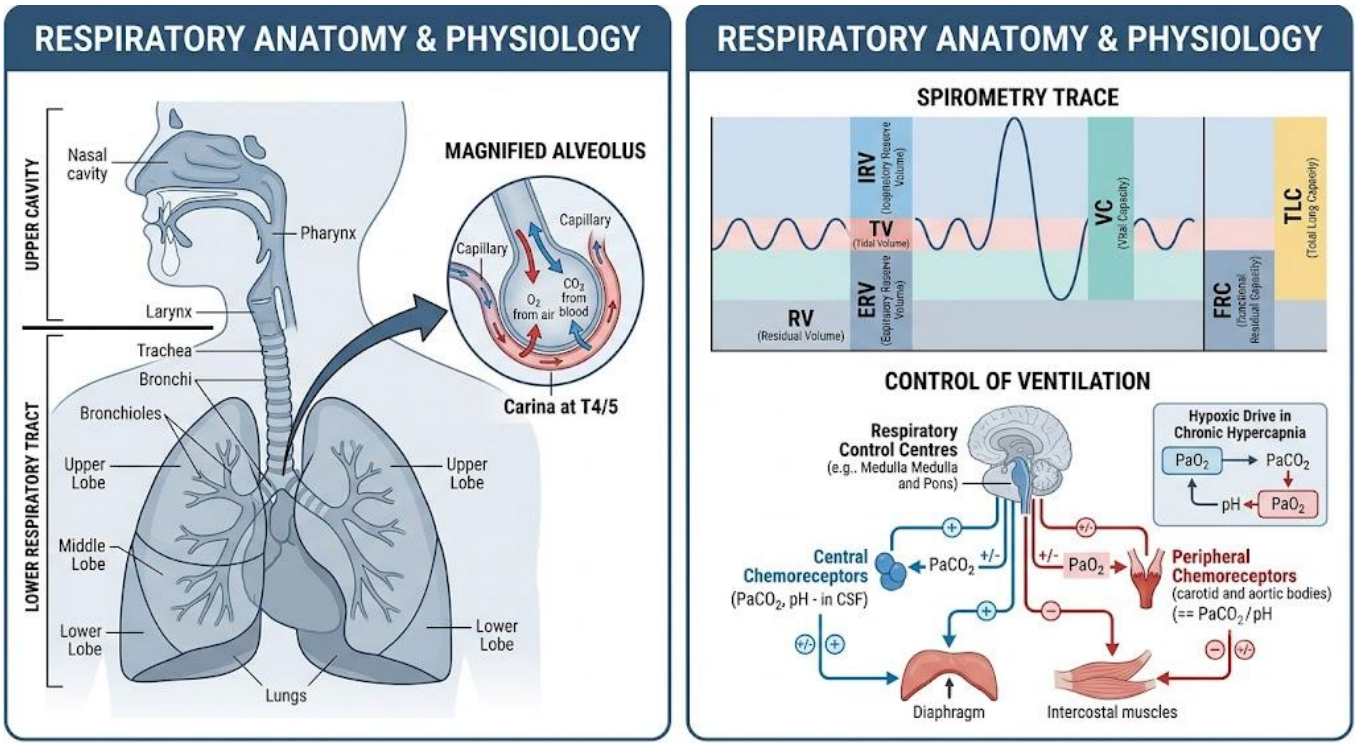


Figure 1.1: Respiratory anatomy, lung volumes, and the control of ventilation

Pilot questions 1.1

1. Describe the gross anatomy of the respiratory tract including the upper and lower respiratory tracts. (Linked to SLO 1.1)
2. Explain why the right main bronchus is more commonly affected by inhaled foreign bodies. (Linked to SLO 1.1)
3. Define tidal volume, vital capacity, total lung capacity, and residual volume. (Linked to SLO 1.1)
4. Explain the mechanics of inspiration and expiration. (Linked to SLO 1.2)
5. Explain why high-flow oxygen is dangerous in patients with chronic hypercapnia. (Linked to SLO 1.2)

1.2 Respiratory History Taking

1.2.1 Cardinal respiratory symptoms

The cardinal respiratory symptoms are: **cough** (productive or dry; duration; character of sputum: mucoid, purulent, blood-stained), **dyspnoea** (onset: sudden or gradual; severity graded by MRC scale; associated wheeze or orthopnoea), **haemoptysis** (coughing up blood; must be distinguished from haematemesis and epistaxis; important causes: TB, lung cancer, bronchiectasis, pulmonary embolism), **wheeze** (musical expiratory sound; suggests airflow obstruction), **chest pain** (pleuritic: sharp, worse on inspiration; suggests pleurisy or pneumonia), and **stridor** (inspiratory high-pitched noise from upper airway obstruction).

Each symptom is explored using SOCRATES and in the context of the patient's overall functional capacity. The **MRC dyspnoea scale** grades breathlessness from Grade 1 (breathless on strenuous exercise) to Grade 5 (too breathless to leave the house or breathless when dressing). A thorough review of the pattern, severity, duration, and associated symptoms guides the differential diagnosis of the presenting respiratory problem.

Fill in the blank: Cardinal respiratory symptoms include cough, dyspnoea (graded by _____ scale), haemoptysis (TB, lung cancer, bronchiectasis), wheeze (airflow obstruction), pleuritic chest pain, and stridor (upper airway obstruction).

1.2.2 Past medical, drug, occupational, and social history

The past medical history in respiratory disease must include: previous respiratory conditions (asthma, TB, pneumonia, bronchiectasis), childhood respiratory illness, atopy (eczema, allergic rhinitis, food allergies), cardiovascular disease (left heart failure causes dyspnoea and pulmonary oedema), and malignancy (lung metastases or primary lung cancer).

The drug history is critical: several drugs cause pulmonary toxicity, including **amiodarone** (pulmonary fibrosis), **methotrexate** (pneumonitis), **NSAIDs and aspirin** (exacerbate asthma), and **ACE inhibitors** (dry persistent cough in approximately 15 percent of patients). The **occupational history** is essential: ask about dust, fume, chemical, asbestos, and animal exposure; occupational lung diseases include pneumoconiosis (coal dust), asbestosis, silicosis,

and occupational asthma. The **social history** covers smoking (pack years: number of packs per day multiplied by years smoked), alcohol use, and housing conditions.

Fill in the blank: Drug-induced respiratory conditions include ACE inhibitor cough, amiodarone pulmonary fibrosis, and aspirin-exacerbated asthma; the occupational history identifies dust, asbestos, and fume exposure causing pneumoconiosis, silicosis, or _____ asthma.

1.2.3 Systems review and functional assessment

The systems review in a respiratory patient should cover cardiovascular symptoms (orthopnoea, PND, ankle oedema: cardiac causes of dyspnoea), upper respiratory symptoms (nasal polyps, post-nasal drip, sinusitis: contributing to asthma), constitutional symptoms (fever, weight loss, night sweats: TB, malignancy), and musculoskeletal symptoms (joint pains and skin rashes in connective tissue disease causing interstitial lung disease).

Functional assessment documents the impact of respiratory disease on daily activities: exercise tolerance (what distances or activities are possible), activities of daily living (dressing, bathing, climbing stairs), work capacity, sleep quality (nocturnal cough, wheeze, or breathlessness), and quality of life. The MRC dyspnoea scale and validated questionnaires such as the St George's Respiratory Questionnaire provide structured functional assessment. In Nigeria, specific inquiry about indoor air pollution exposure (cooking fires, kerosene stoves, biomass fuel) is essential as a major and underrecognised respiratory risk factor.

Fill in the blank: Systems review covers cardiovascular (orthopnoea, PND), upper respiratory (nasal polyps), constitutional (fever, weight loss: TB), and musculoskeletal symptoms; functional assessment documents exercise tolerance, _____ of daily living, sleep quality, and quality of life.

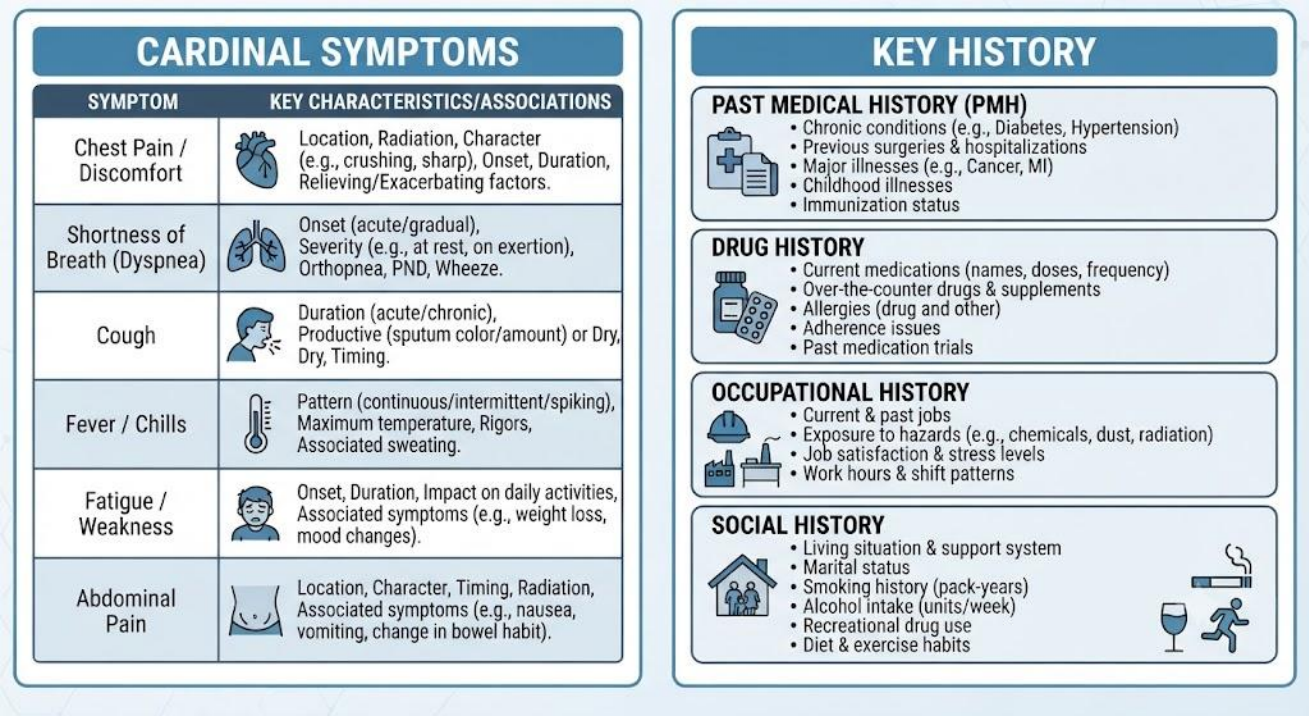


Figure 1.2: Cardinal respiratory symptoms and key history components

Pilot questions 1.2

1. List and define the cardinal respiratory symptoms. (Linked to SLO 1.3)
2. Describe the MRC dyspnoea scale and explain how it is used clinically. (Linked to SLO 1.3)
3. List four drugs that cause respiratory complications and describe the effect of each. (Linked to SLO 1.3)
4. Explain why the occupational history is important in the respiratory patient. (Linked to SLO 1.3)
5. Describe what functional assessment covers in a respiratory patient. (Linked to SLO 1.3)

1.3 Physical Examination of the Respiratory System

1.3.1 General inspection and peripheral signs

General inspection of the respiratory patient begins with: **respiratory rate** (normal 12 to 20 per minute; tachypnoea: above 20), **use of accessory muscles** (sternocleidomastoid and scalene muscles active in respiratory distress), **breathing pattern** (Kussmaul: deep sighing in metabolic acidosis; Cheyne-Stokes: periodic breathing with apnoea; pursed-lip breathing in COPD), and **cyanosis** (central: lips and tongue; peripheral: fingertips).

Peripheral signs of respiratory disease include: **clubbing** (loss of nail-bed angle; causes: lung cancer, bronchiectasis, lung abscess, mesothelioma, idiopathic pulmonary fibrosis, not COPD), **tar staining of fingers** (smoking), **fine tremor and bounding pulse** (CO_2 retention: carbon dioxide narcosis), and **asterixis (flapping tremor)** (hypercarbia). The face shows **Horner's syndrome** (miosis, ptosis, anhidrosis: from apical lung tumour compressing the sympathetic chain Pancoast tumour).

Fill in the blank: Clubbing causes in respiratory disease include lung cancer, bronchiectasis, lung abscess, mesothelioma, and IPF; clubbing is notably _____ in COPD; CO_2 retention causes a bounding pulse, tremor, and asterixis.

1.3.2 Chest inspection and palpation

Chest inspection assesses: **shape** (barrel chest: increased AP diameter in hyperinflation of COPD; pectus excavatum; kyphoscoliosis reducing lung capacity), **symmetry of movement** (unilateral reduced movement: pneumonia, pleural effusion, pneumothorax on the affected side), **tracheal position** (central normally; deviation towards: collapse, fibrosis; deviation away: tension pneumothorax, large pleural effusion), and **intercostal retractions** in respiratory distress.

Palpation of the chest confirms tracheal position, assesses **expansion** (hands placed on lower chest; thumbs at midline; observe degree and symmetry of thumb separation), **tactile vocal fremitus** (TVF: vibration felt when patient says '99'; increased over consolidation; reduced or absent over effusion or pneumothorax), and detects **rib tenderness** (fracture) or **subcutaneous**

emphysema (crackling sensation from air in subcutaneous tissues: pneumothorax or oesophageal rupture).

Fill in the blank: Tracheal deviation towards the lesion occurs in collapse and fibrosis; deviation away occurs in tension pneumothorax and large pleural effusion; TVF is increased over _____ and reduced or absent over effusion or pneumothorax.

1.3.3 Percussion and auscultation

Percussion of the chest generates resonant (normal air-filled lung), dull (consolidation, collapse, or haemothorax), **stony dull** (pleural effusion), or **hyper-resonant** (pneumothorax or emphysema) notes. Percussion starts at the apices and moves down each side systematically; cardiac and hepatic dullness are normal landmarks.

Auscultation with the stethoscope assesses: **breath sounds** (vesicular: normal; bronchial: heard over consolidation or above an effusion, with equal inspiration and expiration and a gap between; reduced or absent: effusion, pneumothorax, collapse), and **added sounds crackles** (fine: pulmonary oedema, interstitial lung disease; coarse: bronchiectasis, pneumonia), **wheeze** (expiratory: asthma, COPD; monophonic: single airway obstruction), and **pleural rub** (leathery scratching sound: pleurisy). **Vocal resonance** (patient says '99'; increased in consolidation; decreased in effusion) and whispered pectoriloquy (words heard clearly over consolidation) complete the assessment.

Fill in the blank: Percussion: consolidation = dull; pleural effusion = stony dull; pneumothorax = hyper-resonant; bronchial breath sounds indicate _____; fine crackles suggest pulmonary oedema or ILD; coarse crackles suggest bronchiectasis or pneumonia.

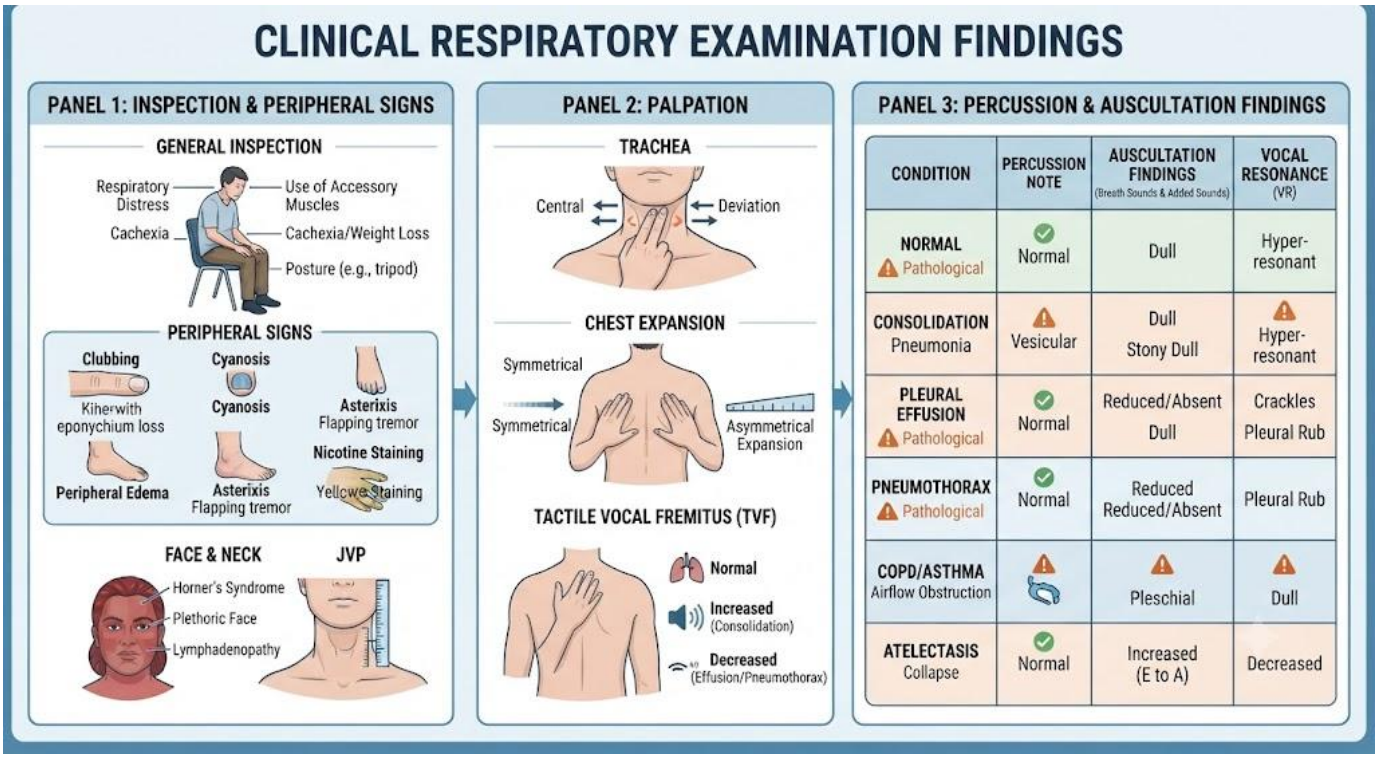


Figure 1.3: Systematic respiratory examination findings by condition

Pilot questions 1.3

1. List four causes of clubbing in respiratory disease and state one condition in which clubbing does not occur despite chronic lung disease. (Linked to SLO 1.4)
2. Describe the causes and clinical significance of tracheal deviation. (Linked to SLO 1.4)
3. Describe the percussion notes heard over consolidation, pleural effusion, and pneumothorax. (Linked to SLO 1.4)
4. Distinguish between vesicular and bronchial breath sounds and state the conditions in which each is heard. (Linked to SLO 1.4)
5. Describe the added breath sounds and state the conditions associated with each. (Linked to SLO 1.4)

1.4 Common Respiratory Investigations

1.4.1 Chest X-ray interpretation

The chest X-ray (CXR) is the most widely used and immediately available imaging investigation in respiratory medicine. A systematic approach to CXR interpretation: **adequacy** (rotation: clavicular heads equidistant from the spinous process; penetration: vertebrae visible through the heart), **airway** (trachea central; carina angle below 70 degrees), **bones** (ribs, clavicles, vertebrae), **cardiac silhouette** (cardiothoracic ratio normally below 0.5), **diaphragm** (right normally higher than left; loss of costophrenic angle sharpness: effusion), and **lung fields** (consolidation, collapse, mass, pleural effusion, pneumothorax). A mnemonic: **ABCDE** (Airway, Bones, Cardiac, Diaphragm, Everything else).

Key CXR patterns in common conditions: consolidation (homogeneous opacification with air bronchogram: pneumonia), collapse (volume loss, mediastinal shift towards affected side), pleural effusion (blunting of costophrenic angle, meniscus sign), pneumothorax (visible lung edge with absent lung markings laterally), pulmonary oedema (bilateral perihilar bat-wing infiltrates, cardiomegaly, upper lobe diversion, Kerley B lines), and **cavitation** (air-fluid level within an opacity: TB, lung abscess, cavitating malignancy).

Fill in the blank: CXR interpretation uses ABCDE: Airway, Bones, Cardiac (CTR below 0.5), Diaphragm, Everything else; consolidation shows homogeneous opacification with _____ bronchogram; pleural effusion blunts the costophrenic angle.

1.4.2 Pulmonary function tests

Spirometry measures the FEV1 (forced expiratory volume in 1 second), FVC (forced vital capacity), and the **FEV1/FVC ratio**. In **obstructive disease** (asthma, COPD): FEV1 is reduced, FVC is normal or mildly reduced, and FEV1/FVC ratio is below 0.70 (below 70 percent). In **restrictive disease** (pulmonary fibrosis, pleural effusion, chest wall deformity, neuromuscular disease): both FEV1 and FVC are proportionally reduced, and the FEV1/FVC ratio is normal or elevated.

Peak expiratory flow rate (PEFR) measures the maximal flow rate during a forced expiratory effort and is used to monitor asthma and assess bronchodilator reversibility. **Full lung volume measurements** (TLC, RV, FRC by body plethysmography or helium dilution) are needed to confirm restrictive disease (low TLC) and to detect air trapping (elevated RV in COPD). **Transfer factor (DLCO)** measures the capacity of the alveolar-capillary membrane to transfer CO; it is reduced in emphysema, pulmonary fibrosis, and pulmonary vascular disease.

Fill in the blank: Obstructive spirometry: FEV1 reduced, FVC normal or mildly reduced, FEV1/FVC ratio below 0.70; restrictive spirometry: FEV1 and FVC both reduced with _____ FEV1/FVC ratio; transfer factor (DLCO) is reduced in emphysema, pulmonary fibrosis, and pulmonary vascular disease.

1.4.3 Arterial blood gas analysis

Arterial blood gas (ABG) analysis measures pH (normal 7.35 to 7.45), PaO2 (normal 10 to 13 kPa on air), PaCO2 (normal 4.7 to 6.0 kPa), and bicarbonate (normal 22 to 26 mmol/L). A systematic ABG interpretation approach: (1) assess oxygenation (PaO2); (2) assess acid-base status (pH); (3) determine the primary disturbance (respiratory: CO2 change; metabolic: HCO3 change); (4) assess compensation.

Type 1 respiratory failure (hypoxaemia without hypercapnia: PaO2 below 8 kPa with normal or low PaCO2) results from V/Q mismatch (pneumonia, pulmonary embolism, pulmonary oedema). **Type 2 respiratory failure** (hypoxaemia with hypercapnia: PaO2 below 8 kPa with PaCO2 above 6 kPa) results from hypoventilation (COPD exacerbation, neuromuscular disease, opioid overdose). The A-a gradient (alveolar-arterial oxygen gradient) helps distinguish causes of hypoxaemia: normal A-a gradient suggests hypoventilation; elevated A-a gradient suggests V/Q mismatch or diffusion impairment.

Fill in the blank: Type 1 respiratory failure: hypoxaemia without hypercapnia (V/Q mismatch); Type 2: hypoxaemia with hypercapnia (hypoventilation: COPD exacerbation, neuromuscular disease); normal A-a gradient suggests _____ as the cause of hypoxaemia.

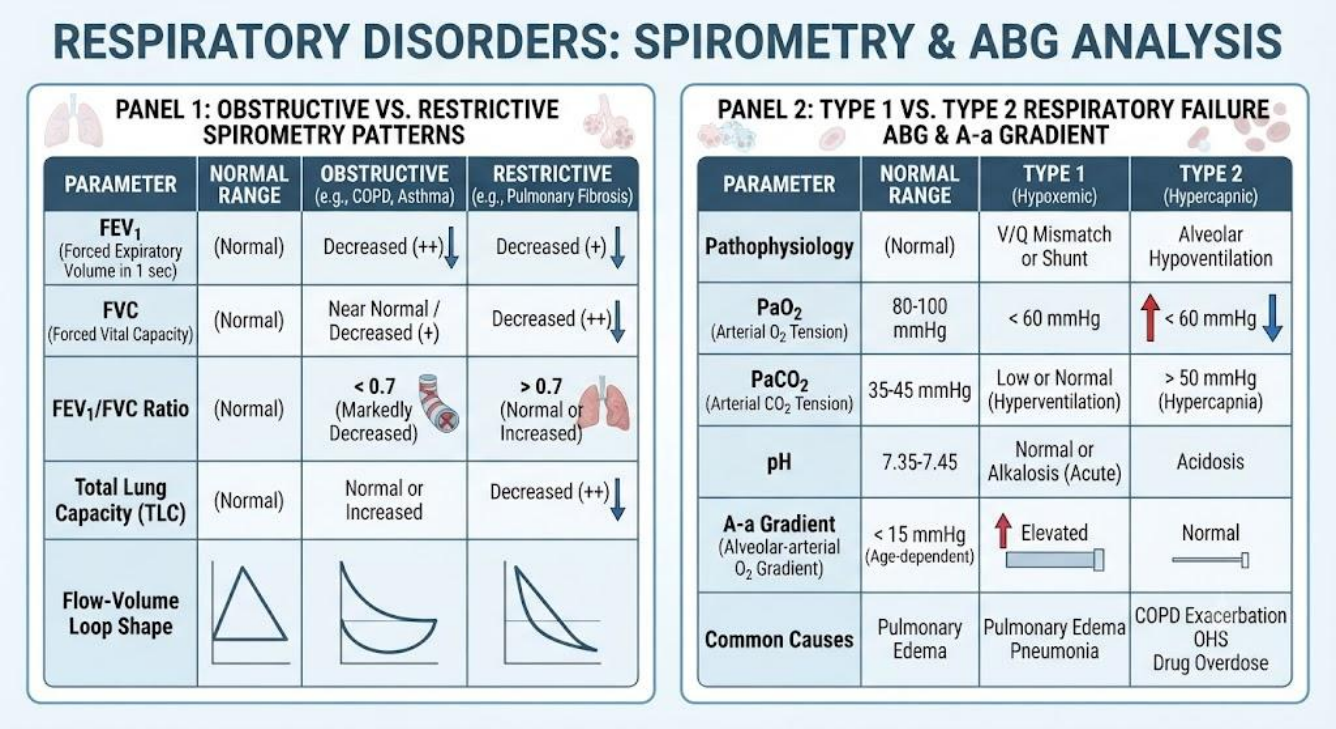


Figure 1.4: Spirometry patterns and arterial blood gas analysis in common respiratory conditions

Pilot questions 1.4

1. Describe the ABCDE systematic approach to chest X-ray interpretation. (Linked to SLO 1.5)
2. Describe the CXR appearances of consolidation, pleural effusion, and pneumothorax. (Linked to SLO 1.5)
3. Distinguish between obstructive and restrictive spirometry patterns. (Linked to SLO 1.5)
4. Explain what the transfer factor (DLCO) measures and name three conditions in which it is reduced. (Linked to SLO 1.5)
5. Distinguish between Type 1 and Type 2 respiratory failure using arterial blood gas criteria. (Linked to SLO 1.5)

Series Summary

This Series established the foundations of respiratory medicine. The respiratory tract divides into upper (nose, pharynx, larynx) and lower (trachea, bronchi, bronchioles, alveoli); the right main bronchus is shorter and more vertical, making it the more common site of foreign body inhalation; the right lung has three lobes, the left two; the acinus is the functional unit of gas exchange. Inspiration is active (diaphragm and external intercostals); key volumes include tidal volume (500 mL), vital capacity, TLC, RV, and FRC; FEV1 and FVC are key spirometric measurements. Gas exchange requires V/Q matching; V/Q mismatch is the most common cause of hypoxaemia; the primary drive to breathe is PaCO2; in chronic hypercapnia the hypoxic drive becomes primary, making high-flow oxygen dangerous. The respiratory history covers cough, dyspnoea (MRC graded), haemoptysis, wheeze, pleuritic chest pain, and stridor; past history must include atopy and previous lung disease; the drug history must identify ACE inhibitor cough, amiodarone fibrosis, and aspirin-exacerbated asthma; the occupational history identifies pneumoconiosis, silicosis, and occupational asthma; indoor air pollution is a critical risk factor in Nigeria.

Respiratory examination: general inspection (respiratory rate, accessory muscle use, cyanosis, clubbing not in COPD, tar staining, asterixis in CO2 retention, Horner's syndrome in Pancoast tumour); chest inspection (barrel chest, tracheal position, asymmetric movement); palpation (expansion, TVF increased in consolidation and reduced in effusion); percussion (dull: consolidation; stony dull: effusion; hyper-resonant: pneumothorax); auscultation (bronchial breath sounds: consolidation; reduced: effusion or pneumothorax; fine crackles: pulmonary oedema or ILD; coarse crackles: bronchiectasis or pneumonia; wheeze: asthma or COPD; pleural rub: pleurisy). CXR interpretation: ABCDE; consolidation = opacification with air bronchogram; effusion = costophrenic blunting; pneumothorax = lung edge with absent markings. Spirometry: obstruction (FEV1/FVC below 0.70); restriction (normal ratio, reduced FVC and FEV1); DLCO reduced in emphysema, fibrosis, pulmonary vascular disease. ABG: Type 1 RF (low PaO2, normal PaCO2: V/Q mismatch); Type 2 (low PaO2, high PaCO2: hypoventilation).

Glossary of Terms

- **Tidal volume:** the volume of air breathed in and out in one normal breath; approximately 500 mL at rest.
- **Vital capacity (VC):** the maximum volume of air that can be exhaled after a maximal inhalation.
- **FEV1:** forced expiratory volume in 1 second; the volume of air exhaled in the first second of a forced expiratory manoeuvre.
- **FVC:** forced vital capacity; the total volume exhaled during a maximal forced expiratory effort from full inspiration.
- **FEV1/FVC ratio:** the ratio of FEV1 to FVC; below 0.70 indicates obstructive ventilatory defect; normal in restriction.
- **Transfer factor (DLCO):** a measure of the capacity of the alveolar-capillary membrane to transfer CO; reduced in emphysema, pulmonary fibrosis, and pulmonary vascular disease.
- **Tactile vocal fremitus (TVF):** vibration felt over the chest wall when a patient speaks; increased over consolidation; reduced over effusion or pneumothorax.
- **Clubbing:** loss of the normal nail-bed angle with increased nail curvature; causes in respiratory disease include lung cancer, bronchiectasis, lung abscess, mesothelioma, and IPF.
- **Type 1 respiratory failure:** hypoxaemia (PaO_2 below 8 kPa) without hypercapnia; caused by V/Q mismatch.
- **Type 2 respiratory failure:** hypoxaemia with hypercapnia ($PaCO_2$ above 6 kPa); caused by hypoventilation.
- **MRC dyspnoea scale:** a five-grade scale rating breathlessness from Grade 1 (breathless only on strenuous exercise) to Grade 5 (too breathless to leave the house or breathless when dressing).
- **Pancoast tumour:** an apical lung tumour compressing the sympathetic chain causing Horner's syndrome (miosis, ptosis, anhidrosis) and the brachial plexus causing arm pain.

Concept Check Questions [Series 1]

Objective Questions

1. The right main bronchus is shorter, wider, and more _____ than the left, making it more prone to foreign body inhalation. (Linked to SLO 1.1)
2. The primary drive to breathe is PaCO2 via _____ chemoreceptors; in chronic hypercapnia the hypoxic drive becomes primary. (Linked to SLO 1.2)
3. Haemoptysis has important causes including TB, lung cancer, bronchiectasis, and _____ embolism. (Linked to SLO 1.3)
4. Clubbing occurs in lung cancer and bronchiectasis but is notably _____ in COPD despite chronic lung disease. (Linked to SLO 1.4)
5. Obstructive spirometry shows FEV1 reduced, FVC normal, and FEV1/FVC ratio below _____. (Linked to SLO 1.5)
6. Type 2 respiratory failure is defined as PaO2 below 8 kPa with PaCO2 above _____ kPa; caused by hypoventilation. (Linked to SLO 1.5)

Short-Answer Questions

7. List the cardinal respiratory symptoms and briefly define each. (Linked to SLO 1.3)
8. Describe the findings on chest examination in a patient with a left-sided pleural effusion. (Linked to SLO 1.4)
9. Distinguish between obstructive and restrictive spirometry patterns. (Linked to SLO 1.5)

Long-Answer Questions

10. Describe the systematic physical examination of the respiratory system from inspection to auscultation. (Linked to SLO 1.4)
11. Describe the ABCDE approach to chest X-ray interpretation and the CXR appearances of consolidation, pleural effusion, and pneumothorax. (Linked to SLO 1.5)

Answers to Concept Check Questions [Series 1]

Objective Questions

1. Vertical.
2. Central.
3. Pulmonary.
4. Absent.
5. 0.70.
6. 6.

Short-Answer Questions

7. Cough: productive or dry; sputum character. Dyspnoea: difficulty breathing; MRC graded 1 to 5. Haemoptysis: coughing blood; causes include TB, lung cancer, bronchiectasis, PE. Wheeze: musical expiratory sound; airflow obstruction. Pleuritic chest pain: sharp, worse on inspiration; pleurisy or pneumonia. Stridor: inspiratory high-pitched noise; upper airway obstruction.
8. Inspection: reduced movement on the left; trachea deviated to the right (away from a large effusion). Palpation: reduced expansion on left; TVF reduced or absent on left. Percussion: stony dull at the left base. Auscultation: breath sounds reduced or absent at the left base; bronchial breathing may be heard above the effusion; vocal resonance reduced.
9. Obstructive: FEV1 reduced, FVC normal or mildly reduced, FEV1/FVC ratio below 0.70; causes: asthma, COPD. Restrictive: FEV1 and FVC both reduced proportionally, FEV1/FVC ratio normal or elevated (above 0.70); causes: pulmonary fibrosis, pleural effusion, chest wall deformity, neuromuscular disease.

Long-Answer Questions

10. **Basic response:** Inspection: respiratory rate (normal 12 to 20), accessory muscle use, breathing pattern (Kussmaul, Cheyne-Stokes, pursed-lip), cyanosis (central: lips and tongue; peripheral: fingertips). Peripheral signs: clubbing (lung cancer, bronchiectasis, lung abscess, mesothelioma, IPF; not COPD), tar staining, CO2 retention (bounding pulse, tremor, asterixis), Horner's syndrome (Pancoast tumour). Chest inspection: shape

(barrel chest in COPD hyperinflation), tracheal position (towards: collapse or fibrosis; away: tension pneumothorax or large effusion), symmetry of movement, intercostal retractions. Palpation: expansion (symmetry and degree), TVF (increased in consolidation; reduced in effusion or pneumothorax), subcutaneous emphysema. Percussion: resonant (normal), dull (consolidation or collapse), stony dull (effusion), hyper-resonant (pneumothorax or emphysema). Auscultation: breath sounds (vesicular: normal; bronchial: consolidation or above effusion; reduced or absent: effusion, pneumothorax, collapse); added sounds (fine crackles: pulmonary oedema, ILD; coarse crackles: bronchiectasis, pneumonia; wheeze: asthma, COPD; pleural rub: pleurisy); vocal resonance (increased in consolidation; decreased in effusion).

Value-added response: The power of the systematic respiratory examination lies in the pattern of findings it produces: in consolidation, all signs point to the same area (dull percussion, bronchial breath sounds, increased TVF and vocal resonance, crackles); in pleural effusion, all signs also converge (stony dull, absent breath sounds, reduced TVF and vocal resonance); recognising these patterns as coherent clinical pictures rather than isolated findings is the skill the examination is designed to develop.

11. **Basic response:** ABCDE: Adequacy (rotation: clavicular heads equidistant from spinous process; penetration: vertebrae visible through heart), Airway (trachea central; carina below 70 degrees), Bones (ribs, clavicles, vertebrae: fractures, erosion), Cardiac (CTR below 0.5; enlarged: heart failure, pericardial effusion), Diaphragm (right normally higher; blunted costophrenic angle: effusion; subphrenic air), Everything else (lung fields, hila, soft tissues, tubes, lines). Consolidation: homogeneous opacification of a lobe or segment with air bronchogram (air-filled bronchi visible through opacified lung); no volume loss; causes: pneumonia, aspiration. Pleural effusion: blunting of costophrenic angle; meniscus sign (concave upper border); mediastinum deviated away in large effusions; no air bronchogram. Pneumothorax: visible lung edge with absent lung markings lateral to it; trachea deviated towards the unaffected side in tension pneumothorax; may show collapsed lung near hilum in large pneumothorax.

    Value-added response: The air bronchogram is a key distinguishing feature: it is seen in consolidation (because the surrounding lung is opacified but the airways remain air-filled) but not in pleural effusion (the fluid obscures everything including the airways) or

collapse (the airways are also collapsed); its presence therefore reliably indicates alveolar filling whether by fluid (pneumonia, pulmonary oedema) or cellular infiltration (lymphoma) rather than atelectasis or effusion.

Answers to Pilot Questions [Series 1]

Part 1.1

1. Upper respiratory tract: nose, nasopharynx, oropharynx, larynx; functions include warming, humidifying, and filtering inspired air. Lower respiratory tract: trachea (cartilaginous; bifurcates at carina at level of T4/5 angle of Louis), right and left main bronchi, lobar bronchi, segmental bronchi, bronchioles (no cartilage), terminal bronchioles, respiratory bronchioles, alveolar ducts, and alveoli (the site of gas exchange).
2. The right main bronchus is shorter (approximately 2.5 cm vs 5 cm for the left), wider in diameter, and departs from the trachea at a less acute angle (more vertical) than the left; these anatomical features mean that an inhaled foreign body is directed by gravity and airflow preferentially into the right bronchus, most commonly lodging in the right lower lobe bronchus.
3. Tidal volume (TV): volume breathed in one normal resting breath (approximately 500 mL). Vital capacity (VC): maximum volume exhaled after maximum inhalation (normally 4 to 5 litres). Total lung capacity (TLC): all air in the lungs at full inspiration (TV + IRV + ERV + RV; normally 5 to 6 litres). Residual volume (RV): air remaining in lungs after maximum exhalation (cannot be expelled; normally 1 to 1.5 litres).
4. Inspiration is an active process: the diaphragm contracts and descends (increasing thoracic vertical dimension); the external intercostal muscles contract (increasing AP and transverse diameter); intrathoracic pressure falls below atmospheric pressure; air flows into the lungs. Expiration at rest is passive: the diaphragm and intercostal muscles relax; elastic recoil of the lungs drives air out. During exercise or in obstructive disease, expiration is active using the internal intercostal muscles and abdominal muscles to force air out.

5. Patients with severe chronic COPD develop chronic CO2 retention; over time their central chemoreceptors become relatively insensitive to elevated PaCO2 (they reset their threshold); their primary respiratory drive shifts to the peripheral chemoreceptors responding to low PaO2 (the hypoxic drive); administering high-flow oxygen in this setting abolishes the hypoxic drive, leading to hypoventilation, further CO2 retention, and worsening respiratory acidosis; controlled low-flow oxygen (target saturation 88 to 92 percent) is therefore used.

Part 1.2

1. Cough: productive (mucoid, purulent, blood-stained) or dry; duration. Dyspnoea: difficulty breathing; MRC Grades 1 to 5; sudden onset or gradual. Haemoptysis: coughing blood; distinguish from haematemesis (GI source) and epistaxis; causes: TB, lung cancer, bronchiectasis, PE. Wheeze: musical expiratory sound; airflow obstruction in asthma or COPD. Chest pain: pleuritic (sharp, worse on inspiration): pleurisy, pneumonia. Stridor: inspiratory high-pitched noise: upper airway obstruction from tumour, foreign body, or croup.
2. MRC dyspnoea scale: Grade 1 (not troubled except by strenuous exercise), Grade 2 (short of breath when hurrying on level ground or walking up a slight hill), Grade 3 (walks slower than contemporaries on level ground due to breathlessness or has to stop after about 15 minutes), Grade 4 (stops for breath after walking about 100 metres or after a few minutes on level ground), Grade 5 (too breathless to leave the house or breathless when dressing). Used to grade severity, monitor progression, and assess response to treatment.
3. ACE inhibitors (approximately 15 percent of users: dry persistent cough from bradykinin accumulation; switch to ARB). Amiodarone (pulmonary fibrosis or pneumonitis; dose-dependent; check CXR and DLCO on treatment). Methotrexate (pneumonitis: non-productive cough, dyspnoea, fever; may require cessation). NSAIDs and aspirin (exacerbate asthma in aspirin-exacerbated respiratory disease; prostaglandin E2 inhibition shifts arachidonic acid to leukotriene production, causing bronchoconstriction).
4. Many respiratory diseases have occupational causes that will not be diagnosed if the occupational history is omitted: coal dust exposure causes pneumoconiosis (coal workers'

pneumoconiosis); silica exposure causes silicosis; asbestos exposure causes asbestosis, mesothelioma, and lung cancer; isocyanates, grain dust, animal allergens, and many other workplace exposures cause occupational asthma; identifying the occupation and specific exposures may reveal a treatable and legally compensable cause of the patient's respiratory disease.

5. Exercise tolerance: what distances can the patient walk before stopping, what activities are limited by breathlessness. Activities of daily living: dressing, bathing, climbing stairs, household tasks. Work capacity: impact on employment and duties. Sleep quality: nocturnal cough, wheeze, breathlessness, Cheyne-Stokes breathing. Quality of life: social activities, hobbies, emotional impact. Functional assessment is documented using the MRC dyspnoea scale and validated questionnaires such as the St George's Respiratory Questionnaire.

Part 1.3

1. Lung cancer (primary or secondary), bronchiectasis, lung abscess, mesothelioma, idiopathic pulmonary fibrosis (IPF), congenital heart disease with cyanosis. COPD: clubbing is notably absent in COPD despite being a chronic lung disease; its absence in COPD and its presence in the other conditions listed is a clinically useful discriminating feature.
2. Tracheal deviation occurs because of differential pressure or volume changes between the two hemithoraces. Deviation towards the lesion: collapse or fibrosis (volume loss on the affected side pulls the trachea towards it). Deviation away from the lesion: tension pneumothorax (air under pressure pushes the mediastinum away; a medical emergency requiring immediate needle decompression) and large pleural effusion (fluid under pressure pushes the mediastinum away). Central trachea: a normal finding; also seen in small pneumothorax or effusion.
3. Consolidation: dull percussion note (the alveolar air is replaced by fluid or cells, reducing resonance; the percussion note is the same quality as percussing over a solid muscle). Pleural effusion: stony dull percussion note (characteristic quality; denser and flatter than the dullness of consolidation because of the fluid mass). Pneumothorax: hyper-resonant percussion note (the air-filled pleural space is more resonant than normal air-filled lung).

4. Vesicular breath sounds: normal breath sounds; heard in inspiration and early expiration; rustling quality; inspiration is longer than expiration; no pause between phases. Bronchial breath sounds: harsh, tubular quality; inspiration and expiration are equal in length with a pause between them; heard over consolidation (where sound is transmitted through the solid lung) and just above a pleural effusion; they are pathological in any other location.
5. Fine crackles: high-pitched, brief discontinuous sounds; heard in late inspiration; caused by explosive re-opening of small airways or alveoli that have collapsed; conditions: pulmonary oedema, idiopathic pulmonary fibrosis, early pneumonia. Coarse crackles: low-pitched, longer crackling sounds throughout inspiration; from secretions in large airways; conditions: bronchiectasis, COPD, resolving pneumonia. Wheeze: continuous musical sounds from oscillating airway walls in narrowed airways; expiratory predominance: asthma, COPD; monophonic: single airway obstruction (tumour or foreign body). Pleural rub: leathery scratching sound synchronous with breathing from inflamed pleural surfaces; pleurisy.

Part 1.4

1. Adequacy: is the film rotated (clavicular heads equidistant from spinous process) and adequately penetrated (vertebrae just visible through cardiac shadow)? Airway: trachea central; carina angle below 70 degrees; check for tracheal deviation. Bones: ribs (fractures, erosions), clavicles, vertebrae (metastases, collapse), scapulae. Cardiac: cardiothoracic ratio (below 0.5); left and right heart borders; aortic knuckle; prominent hila. Diaphragm: right normally higher than left; costophrenic angle sharpness (blunted: effusion); subphrenic air (perforated viscus). Everything else: lung fields (zones, density, masses), pleura, soft tissues, foreign bodies, tubes, lines.
2. Consolidation: homogeneous opacification (white) of part or all of a lobe; air bronchogram present (air-filled airways visible within the opacification); no volume loss; lobar or segmental distribution; causes: pneumonia, aspiration, pulmonary infarction. Pleural effusion: blunting of costophrenic angle (earliest sign); meniscus sign (concave upper border slanting up laterally); completely white hemithorax in massive effusion; mediastinum deviated away in large effusion; no air bronchogram. Pneumothorax: visible lung edge (pleural line) with absent lung markings lateral to it; trachea and mediastinum pushed away in tension pneumothorax; collapsed lung near hilum in large pneumothorax.

3. Obstructive pattern (asthma, COPD): FEV1 is disproportionately reduced relative to FVC; FEV1/FVC ratio is below 0.70 (post-bronchodilator for COPD diagnosis); FVC may be normal or mildly reduced; the obstruction means that not all the air can be expelled in 1 second even though the total capacity may be relatively preserved; air trapping (elevated RV) is also present in COPD. Restrictive pattern (pulmonary fibrosis, pleural effusion, chest wall deformity): FEV1 and FVC are both reduced proportionally; the FEV1/FVC ratio is normal (above 0.70) or elevated; the lungs are small and stiff or restricted by external factors.
4. Transfer factor (DLCO) measures the capacity of the alveolar-capillary membrane to transfer carbon monoxide from inspired gas to capillary blood, reflecting the functional surface area available for gas diffusion. Three conditions where it is reduced: emphysema (destruction of alveolar-capillary membrane, reducing surface area), idiopathic pulmonary fibrosis (thickening of alveolar-capillary membrane, impairing diffusion), pulmonary vascular disease (reduced pulmonary capillary blood volume, reducing the amount of haemoglobin available to bind CO).
5. Type 1 respiratory failure: PaO2 below 8 kPa (60 mmHg) with PaCO2 normal or low; caused by V/Q mismatch where ventilated alveoli are not matched by perfusion or vice versa; examples: pneumonia, pulmonary embolism, pulmonary oedema, ARDS. Type 2 respiratory failure: PaO2 below 8 kPa with PaCO2 above 6 kPa (45 mmHg); caused by alveolar hypoventilation where insufficient fresh gas reaches the alveoli to excrete CO2; examples: COPD exacerbation, neuromuscular disease (Guillain-Barre syndrome, myasthenia gravis), severe asthma, opioid or sedative overdose, obesity hypoventilation syndrome.

References and Attributions [Series 1]

Textual References

- Kumar, P. and Clark, M. (2017). Kumar and Clark's Clinical Medicine (9th ed.). Edinburgh: Elsevier.
- Talley, N. J. and O'Connor, S. (2018). Clinical Examination: A Systematic Guide to Physical Diagnosis (8th ed.). Sydney: Elsevier.
- West, J. B. and Luks, A. M. (2020). West's Respiratory Physiology: The Essentials (11th ed.). Philadelphia: Wolters Kluwer.
- British Thoracic Society. (2019). BTS/SIGN British Guideline on the Management of Asthma. London: BTS.

Figures, Plates, Tables, and Boxes

- Figure 1.1: Respiratory anatomy, lung volumes, and control of ventilation. AI prompt: "Two-panel diagram: left panel labelled respiratory tract with upper and lower components, right and left lung lobes, carina at T4/5, magnified alveolus showing gas exchange; right panel top spirometry trace with labelled lung volumes (TV, IRV, ERV, RV, VC, TLC, FRC) and bottom control of ventilation diagram (brainstem respiratory centre, central and peripheral chemoreceptors, hypoxic drive); clean professional infographic." OER search: "respiratory anatomy lung volumes spirometry control of ventilation diagram".
- Figure 1.2: Cardinal respiratory symptoms and key history components. AI prompt: "Two-panel diagram: six-row cardinal symptoms table and four-section key history; clean professional infographic." OER search: "respiratory history cardinal symptoms drug occupational history diagram".
- Figure 1.3: Systematic respiratory examination findings. AI prompt: "Three-panel diagram: inspection, palpation, percussion and auscultation findings by condition; clean professional infographic." OER search: "respiratory examination inspection palpation percussion auscultation findings diagram".
- Figure 1.4: Spirometry patterns and ABG analysis. AI prompt: "Two-panel diagram: obstructive vs restrictive spirometry and Type 1 vs Type 2 ABG table; clean professional

infographic." OER search: "spirometry obstructive restrictive ABG Type 1 Type 2 respiratory failure diagram".

Series 2: Approach to Respiratory Symptoms and Investigations

- **Part 2.1: Approach to Dyspnoea**
  - Sub Part 2.1.1: Acute versus chronic dyspnoea
  - Sub Part 2.1.2: Differential diagnosis of dyspnoea
  - Sub Part 2.1.3: Investigation of dyspnoea
- **Part 2.2: Approach to Cough and Haemoptysis**
  - Sub Part 2.2.1: Acute and chronic cough
  - Sub Part 2.2.2: Haemoptysis
  - Sub Part 2.2.3: Investigation of cough and haemoptysis
- **Part 2.3: Approach to Chest Pain and Wheeze**
  - Sub Part 2.3.1: Pleuritic and non-pleuritic chest pain
  - Sub Part 2.3.2: Wheeze and stridor
  - Sub Part 2.3.3: Investigation of chest pain and wheeze
- **Part 2.4: Advanced Respiratory Investigations**
  - Sub Part 2.4.1: CT imaging and bronchoscopy
  - Sub Part 2.4.2: Pleural investigations
  - Sub Part 2.4.3: Microbiological and haematological investigations

Introduction

A symptom-based approach to respiratory medicine is an essential clinical skill. Patients present with symptoms not diagnoses and the ability to systematically construct a differential diagnosis from the presenting complaint, history, examination, and investigations determines the quality and speed of clinical decision-making. The major respiratory symptoms of dyspnoea, cough, haemoptysis, chest pain, and wheeze each have characteristic causes and an evidence-based investigation pathway.

This Series builds on the examination skills of Series 1 to develop a structured diagnostic approach to each major respiratory symptom. For each symptom, the important differential diagnoses are reviewed, and the appropriate investigation pathway is described. The Series then covers advanced respiratory investigations that go beyond the basic CXR and spirometry introduced in Series 1, including CT imaging, bronchoscopy, pleural investigation, and microbiological testing.

These skills are directly applicable to clinical practice in Nigeria, where the differential diagnosis of respiratory symptoms includes a distinctive mix of infectious, occupational, and non-communicable respiratory diseases.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 2.1** construct a differential diagnosis for dyspnoea based on the mode of onset, associated features, and examination findings (Linked to Part 2.1),
- **SLO 2.2** describe the approach to investigation of dyspnoea in different clinical contexts (Linked to Part 2.1),
- **SLO 2.3** describe the major causes of cough and haemoptysis and their investigation (Linked to Part 2.2),

- **SLO 2.4** describe the causes of pleuritic chest pain, wheeze, and stridor, and their clinical differentiation (Linked to Part 2.3), and
- **SLO 2.5** describe the indications and findings of CT imaging, bronchoscopy, pleural investigations, and microbiological tests in respiratory disease (Linked to Part 2.4).

2.1 Approach to Dyspnoea

2.1.1 Acute versus chronic dyspnoea

Dyspnoea is the subjective sensation of difficult or laboured breathing. The mode of onset is the first critical diagnostic discriminator. **Acute dyspnoea (onset over minutes to hours)** suggests a life-threatening emergency: **pulmonary embolism** (sudden onset, pleuritic chest pain, risk factors for DVT), **pneumothorax** (sudden pleuritic pain, unilateral absent breath sounds), **acute severe asthma** (wheeze, diurnal variation, atopy), **pulmonary oedema** (orthopnoea, PND, cardiac history), and **anaphylaxis** (urticaria, stridor, exposure history).

Chronic dyspnoea (onset over weeks to months) suggests: **COPD** (smoking history, progressive, with wheeze and reduced exercise tolerance), **pulmonary fibrosis** (progressive, dry cough, fine crackles, occupational or connective tissue disease history), **heart failure** (orthopnoea, bilateral ankle oedema, cardiac history), **anaemia** (pallor, fatigue, dietary or menstrual history), and **pleural effusion** (dullness on percussion, reduced breath sounds).

Fill in the blank: Acute dyspnoea causes include pulmonary embolism, pneumothorax, severe asthma, and pulmonary oedema; chronic dyspnoea causes include COPD (smoking history), pulmonary fibrosis, heart failure (orthopnoea), anaemia, and _____ effusion.

2.1.2 Differential diagnosis of dyspnoea

A systematic approach to the differential diagnosis of dyspnoea uses associated features. Dyspnoea with **orthopnoea and PND** suggests left heart failure or severe asthma. Dyspnoea with **pleuritic chest pain** suggests pulmonary embolism, pneumothorax, pleurisy, or pneumonia. Dyspnoea with **wheeze** suggests asthma, COPD, or acute pulmonary oedema (cardiac asthma).

Dyspnoea with **haemoptysis** suggests pulmonary embolism, lung cancer, or TB. Dyspnoea with **weight loss and night sweats** suggests TB or malignancy.

In Nigeria, the differential diagnosis of dyspnoea must prominently include **pulmonary tuberculosis** (the most common cause of chronic respiratory symptoms in adults, with weight loss, night sweats, and haemoptysis), **HIV-related respiratory disease** (Pneumocystis jirovecii pneumonia, bacterial pneumonia, pulmonary Kaposi sarcoma), **indoor air pollution-related COPD** (from biomass fuel use, predominantly in women), and **schistosomiasis-related pulmonary hypertension**.

Fill in the blank: In Nigeria, the differential diagnosis of chronic dyspnoea must prominently include pulmonary TB (weight loss, night sweats, haemoptysis), HIV-related respiratory disease, biomass fuel-related COPD, and _____-related pulmonary hypertension.

2.1.3 Investigation of dyspnoea

First-line investigations for dyspnoea include: **CXR** (consolidation: pneumonia; effusion; cardiomegaly: heart failure; hyperinflation: COPD; pneumothorax), **FBC** (anaemia, eosinophilia in allergic conditions), **ECG** (AF: cardiac cause; right heart strain: PE or pulmonary hypertension; LVH: heart failure), **echocardiography** (LV function: heart failure; pulmonary hypertension), and **spirometry** (obstructive: COPD or asthma; restrictive: pulmonary fibrosis).

Second-line investigations for selected presentations: **D-dimer and CT pulmonary angiography (CTPA)** (for suspected PE), **high-resolution CT (HRCT) chest** (interstitial lung disease, bronchiectasis), **V/Q scan** (PE in patients unable to have CTPA), **BNP or NT-proBNP** (elevated in heart failure), and **ABG analysis** (to classify respiratory failure and guide oxygen therapy).

Fill in the blank: First-line dyspnoea investigations include CXR, FBC (anaemia, eosinophilia), ECG (right heart strain: PE), spirometry, and echocardiography; second-line investigations include CTPA (suspected PE), HRCT (ILD, bronchiectasis), and _____ (elevated in heart failure).

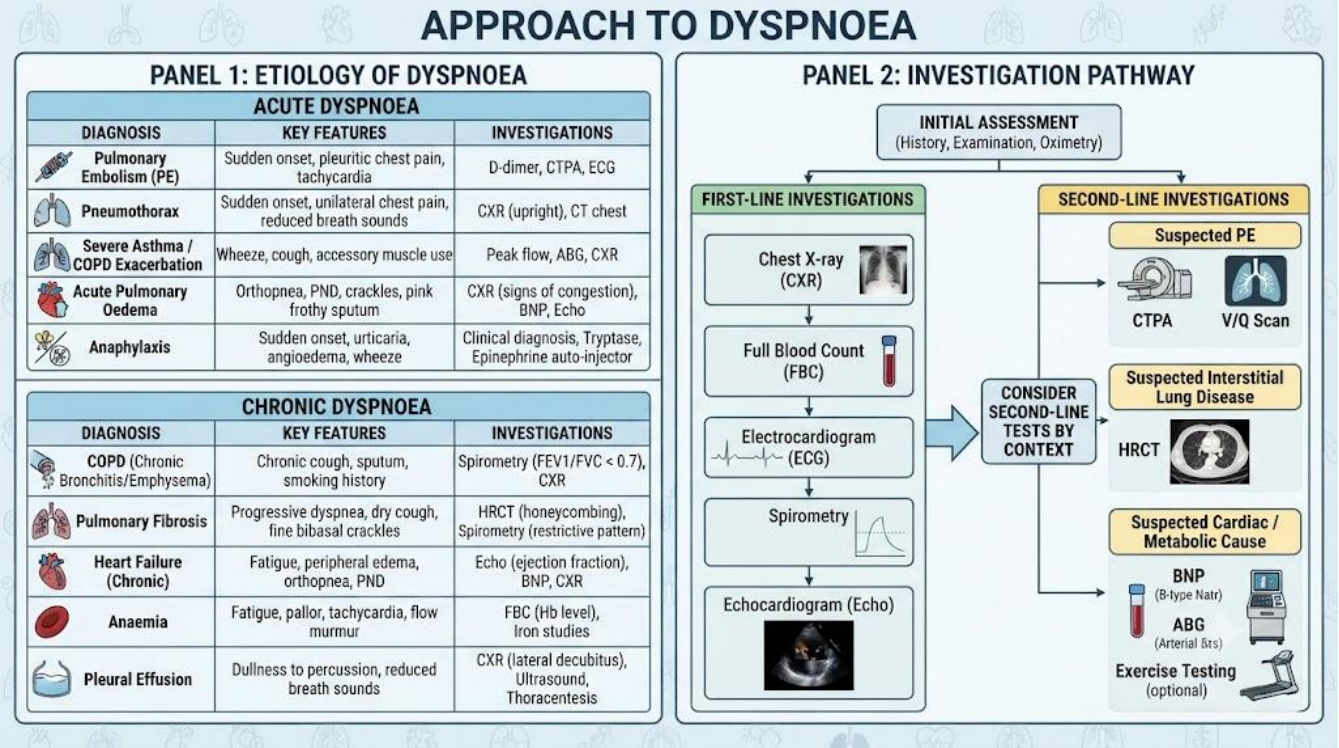


Figure 2.1: Approach to dyspnoea differential diagnosis by onset and associated features, and investigation pathway

Pilot questions 2.1

1. Distinguish between acute and chronic dyspnoea and list three causes of each. (Linked to SLO 2.1)
2. Describe the clinical features that suggest pulmonary embolism in a patient with acute dyspnoea. (Linked to SLO 2.1)
3. Describe the differential diagnosis of dyspnoea in the Nigerian clinical context. (Linked to SLO 2.1)
4. Describe the first-line investigations for a patient presenting with dyspnoea. (Linked to SLO 2.2)
5. Explain when CTPA and HRCT chest are indicated in the investigation of dyspnoea. (Linked to SLO 2.2)

2.2 Approach to Cough and Haemoptysis

2.2.1 Acute and chronic cough

Cough is the most common respiratory symptom. **Acute cough (below 3 weeks)** is most commonly caused by **upper respiratory tract infection (URTI)** (viral: rhinovirus, influenza), **acute bronchitis** (usually viral; productive cough, wheeze; self-limiting), and **pneumonia** (productive cough, fever, pleuritic pain, consolidation on CXR).

Chronic cough (above 8 weeks) is most commonly caused by the **post-nasal drip syndrome (UACS)** (upper airway cough syndrome: post-nasal drip from rhinitis or sinusitis; throat clearing, nasal symptoms), **asthma** (cough variant asthma: dry nocturnal cough; wheeze; bronchodilator responsive), **gastro-oesophageal reflux disease (GORD)** (acid reflux triggering cough reflex; worse after meals and when supine), and **ACE inhibitor-induced cough** (dry, persistent, in approximately 15 percent of patients on ACEi; resolves on stopping the drug). In Nigeria, **pulmonary TB** must always be considered in any patient with cough lasting above 2 weeks.

Fill in the blank: The most common causes of chronic cough are post-nasal drip (UACS), asthma (cough variant), GORD, and ACE inhibitor cough; in Nigeria, _____ TB must always be excluded in cough lasting above 2 weeks.

2.2.2 Haemoptysis

Haemoptysis (coughing up blood from the respiratory tract) must be distinguished from **haematemesis** (vomiting blood from the GI tract: darker, mixed with food, preceded by nausea) and **epistaxis** (nasal bleeding: blood drips down the back of the throat). Important causes of haemoptysis include: **pulmonary tuberculosis** (the most common cause in Nigeria: blood-streaked sputum or frank haemoptysis, with weight loss, night sweats, cavitation on CXR), **lung cancer** (particularly squamous cell carcinoma; associated with smoking, weight loss, clubbing), **bronchiectasis** (chronic productive cough, copious sputum, coarse crackles; haemoptysis may be massive), and **pulmonary embolism** (haemoptysis with pleuritic chest pain and dyspnoea).

Massive haemoptysis (above 200 to 600 mL in 24 hours) is a life-threatening emergency from airways flooding with blood, predominantly caused by bronchiectasis, TB, and aspergilloma; immediate management includes positioning the patient with the bleeding lung dependent, calling for senior help, and securing IV access for resuscitation and bronchial artery embolisation.

Fill in the blank: The most common cause of haemoptysis in Nigeria is pulmonary _____; other causes include lung cancer, bronchiectasis, and PE; massive haemoptysis is life-threatening and managed with positioning and bronchial artery embolisation.

2.2.3 Investigation of cough and haemoptysis

Investigation of chronic cough begins with a **CXR** (to exclude TB, malignancy, and other significant pathology); if normal, a therapeutic trial approach is used: **trial of nasal steroid spray** (for post-nasal drip), **spirometry and bronchodilator reversibility test** (for cough variant asthma), and **stopping the ACE inhibitor** (to confirm ACE inhibitor-induced cough). Sputum for AFB smear and culture is mandatory when TB is suspected.

Investigation of haemoptysis always requires a **CXR** (cavity: TB; mass: malignancy; diffuse shadowing: vasculitis or bleeding), **sputum for AFB** (TB), **CT chest** (better characterisation of mass, cavity, bronchiectasis; nodal staging in malignancy), and **bronchoscopy** (to localise the bleeding source, obtain biopsies, and in some cases apply haemostatic treatment). Fibre-optic bronchoscopy is the investigation of choice for haemoptysis of unknown cause after CXR and CT.

Fill in the blank: Investigation of haemoptysis requires CXR, sputum for AFB (TB), CT chest (characterisation), and _____ (to localise the bleeding source, obtain biopsies, and apply haemostatic treatment).

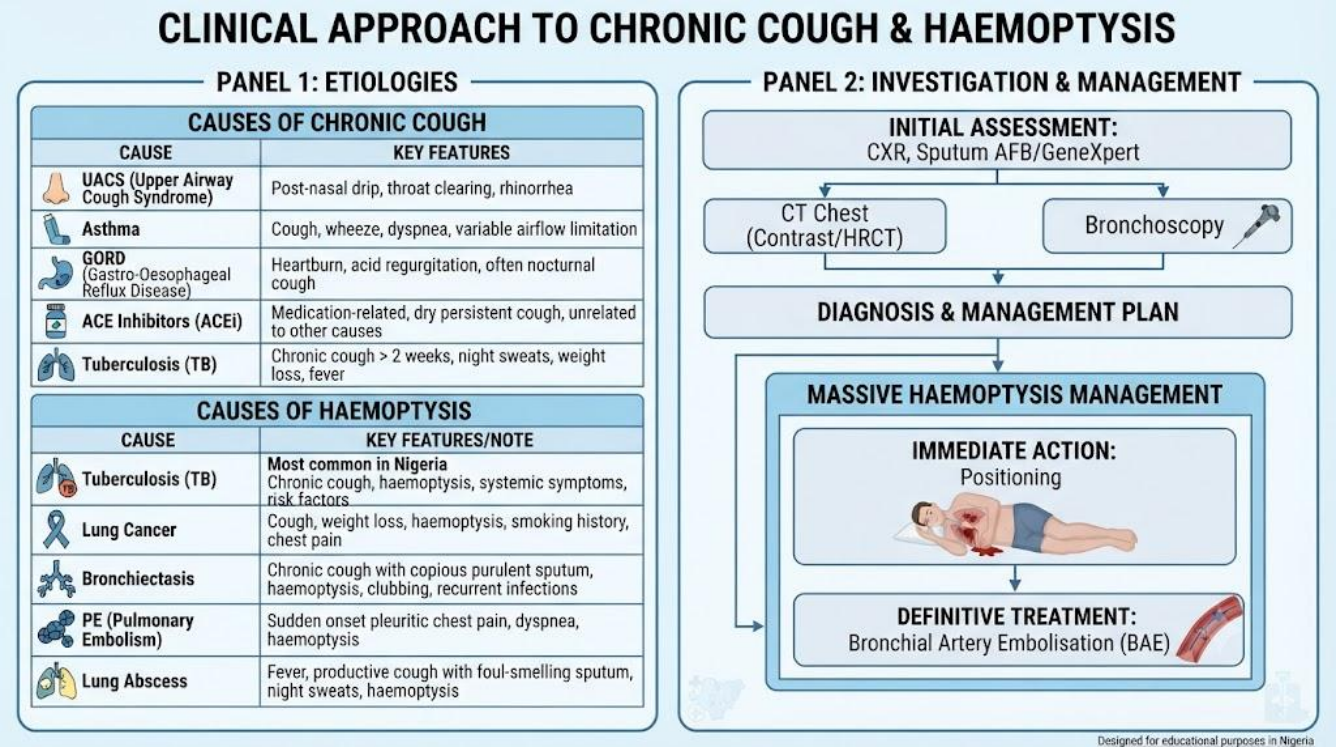


Figure 2.2: Causes of chronic cough and haemoptysis with investigation pathway

Pilot questions 2.2

1. Distinguish between acute and chronic cough and list the most common causes of each. (Linked to SLO 2.3)
2. Explain why pulmonary TB must always be considered in the Nigerian patient with chronic cough. (Linked to SLO 2.3)
3. List the important causes of haemoptysis and describe the features of each. (Linked to SLO 2.3)
4. Distinguish haemoptysis from haematemesis and epistaxis. (Linked to SLO 2.3)
5. Describe the investigation pathway for a patient with haemoptysis. (Linked to SLO 2.3)

2.3 Approach to Chest Pain and Wheeze

2.3.1 Pleuritic and non-pleuritic chest pain

Pleuritic chest pain is sharp, stabbing pain that is worse on inspiration or coughing, caused by inflammation of the parietal pleura. Causes include: **pleurisy** (primary viral: often with preceding URTI; or secondary to pneumonia, PE, TB, or connective tissue disease), **pneumonia** (with pleuritic pain from adjacent pleural involvement), **pulmonary embolism** (pleuritic pain with dyspnoea, tachycardia, and haemoptysis), and **pneumothorax** (sudden onset pleuritic pain with dyspnoea).

Non-respiratory causes of chest pain that must be distinguished from pleuritic pain include: **musculoskeletal chest pain** (reproduced by chest wall palpation; Tietze syndrome: costochondritis), **pericarditis** (positional: worse lying flat, relieved leaning forward; friction rub; saddle-shaped ST elevation on ECG), and **oesophageal pain** (burning, related to meals, relieved by antacids; dysphagia). Ischaemic cardiac pain is crushing, central, radiating to the arm or jaw, and not affected by inspiration or position.

Fill in the blank: Pleuritic chest pain is sharp, worse on inspiration, and caused by pleural inflammation; causes include pleurisy, pneumonia, PE, and pneumothorax; pericarditis is positional (worse flat, relieved leaning _____).

2.3.2 Wheeze and stridor

Wheeze is a musical, continuous expiratory sound from airflow through narrowed airways. **Polyphonic wheeze** (multiple pitches: widespread airway narrowing) suggests **asthma or COPD**. **Monophonic wheeze** (single pitch: localised obstruction) suggests a **foreign body, endobronchial tumour, or extrinsic compression**. Wheeze must be distinguished from cardiac asthma (heart failure presenting with wheeze from pulmonary oedema and bronchospasm: 'all that wheezes is not asthma').

Stridor is a harsh, high-pitched inspiratory noise from obstruction of the upper airway or large airways. Causes in adults include: **laryngeal carcinoma** (progressive hoarseness and stridor; smoking history), **thyroid mass or goitre** (tracheal deviation and compression), **foreign body**

aspiration (sudden onset, choking episode), and **anaphylaxis** (stridor with urticaria and angioedema). Stridor in children is most commonly from **croup (laryngotracheobronchitis)** (parainfluenza virus; barking cough; inspiratory stridor) or **epiglottitis** (Haemophilus influenzae type b; high fever; drooling; tripod position; life-threatening).

Fill in the blank: Polyphonic wheeze suggests asthma or COPD (widespread narrowing); monophonic wheeze suggests foreign body or endobronchial tumour (localised obstruction); stridor (inspiratory) from upper airway obstruction adult causes include laryngeal carcinoma, thyroid _____, and foreign body.

2.3.3 Investigation of chest pain and wheeze

Investigation of pleuritic chest pain starts with **CXR** (consolidation: pneumonia; pneumothorax: lung edge; pleural effusion: costophrenic blunting; clear: primary pleurisy or PE), **ECG** (saddle ST elevation: pericarditis; right heart strain S1Q3T3: PE), and **D-dimer plus CTPA** (if PE suspected and D-dimer elevated). Pericarditis is confirmed by echocardiography (pericardial effusion) and a clinical picture of positional chest pain with a friction rub.

Investigation of wheeze includes: **spirometry and bronchodilator reversibility** (significant reversibility: FEV1 improvement above 12 percent and above 200 mL confirms asthma), **PEFR diary** (diurnal variation above 20 percent in morning versus evening supports asthma diagnosis), and **CXR** (hyperinflation: COPD; foreign body; cardiac shadow: pulmonary oedema). Investigation of stridor requires **direct or fibre-optic laryngoscopy** and **CT neck and chest** (to assess the level and cause of obstruction).

Fill in the blank: Wheeze investigation: spirometry with bronchodilator reversibility (FEV1 improvement above 12 percent and 200 mL confirms asthma); PEFR diary (diurnal variation above 20 percent supports asthma); stridor investigation requires laryngoscopy and CT _____ and chest.

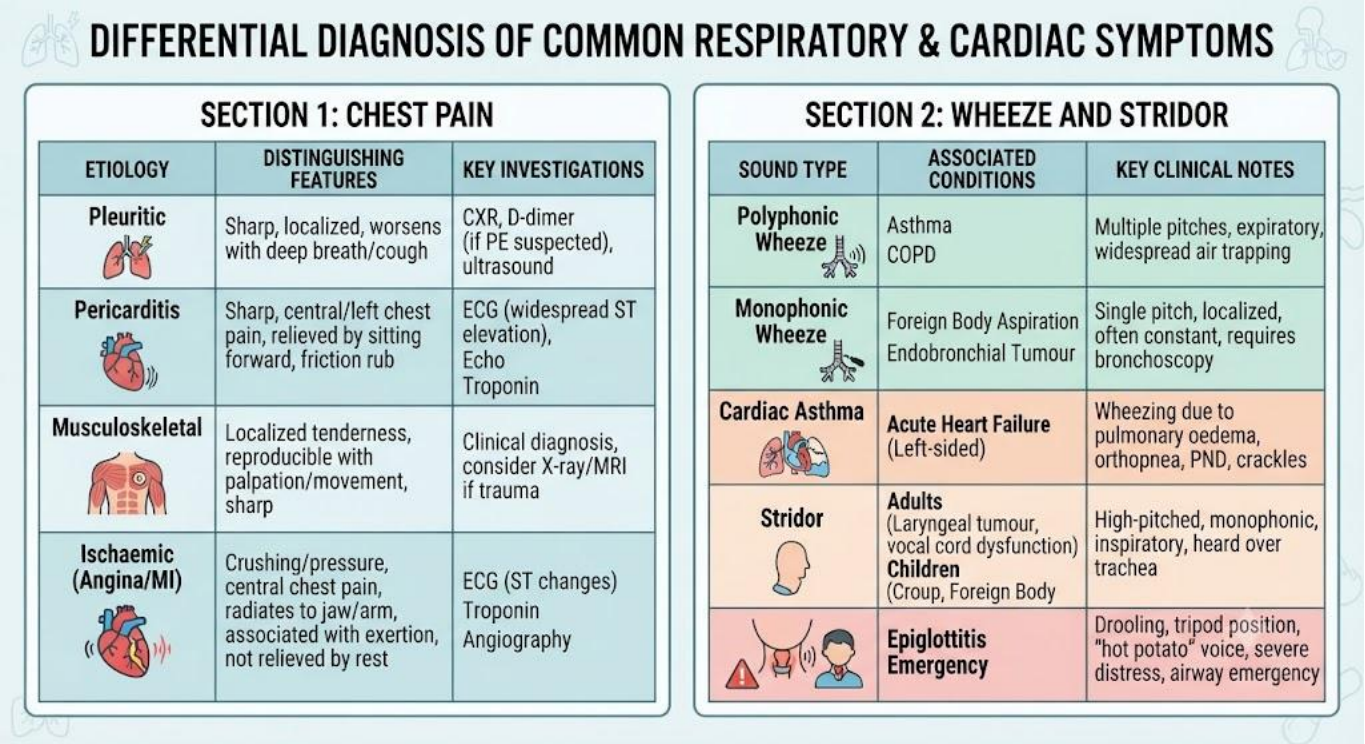


Figure 2.3: Approach to chest pain and wheeze or stridor causes, features, and investigation

Pilot questions 2.3

1. Describe the clinical features of pleuritic chest pain and list four causes. (Linked to SLO 2.4)
2. Explain how to distinguish pleuritic chest pain from ischaemic cardiac pain. (Linked to SLO 2.4)
3. Distinguish between polyphonic and monophonic wheeze and state the conditions associated with each. (Linked to SLO 2.4)
4. Describe the causes of stridor in adults and children. (Linked to SLO 2.4)
5. Describe the investigation of a patient presenting with wheeze. (Linked to SLO 2.4)

2.4 Advanced Respiratory Investigations

2.4.1 CT imaging and bronchoscopy

High-resolution CT (HRCT) chest provides detailed imaging of the lung parenchyma and is the investigation of choice for: **interstitial lung disease** (ILD: shows honeycombing, ground-glass opacity, reticular shadowing; distinguishes UIP pattern of IPF from other ILD patterns), **bronchiectasis** (bronchial dilatation, thickened bronchial walls, signet ring sign), **small nodules** (lung cancer screening and characterisation), and **pulmonary embolism** (as part of CTPA with contrast). CT is more sensitive than CXR for all these conditions.

Fibre-optic bronchoscopy (FOB) allows direct visualisation of the airways down to the segmental bronchi, with the ability to obtain **bronchial biopsies** (endobronchial tumour), **bronchoalveolar lavage (BAL)** (cell count and microbiology in ILD and opportunistic infection), and **transbronchial biopsies** (lung parenchyma sampling). FOB is indicated for: unexplained haemoptysis, central lung mass, suspected endobronchial disease, foreign body removal, and investigation of localised wheeze. **Endobronchial ultrasound (EBUS)** allows sampling of mediastinal and hilar lymph nodes.

Fill in the blank: HRCT chest is the investigation of choice for ILD (honeycombing, ground-glass opacity), bronchiectasis (signet ring sign), and small nodules; bronchoscopy allows biopsies, BAL, and transbronchial sampling; EBUS allows sampling of _____ lymph nodes.

2.4.2 Pleural investigations

Pleural aspiration (thoracocentesis) is performed for diagnostic and therapeutic purposes. Pleural fluid is analysed for: **appearance** (straw-coloured: transudate; turbid or purulent: exudate; haemorrhagic: malignancy or PE; milky: chylothorax), **protein and LDH** (Light's criteria: exudate if pleural fluid protein above 30 g/L, or pleural/serum protein ratio above 0.5, or pleural/serum LDH ratio above 0.6), **glucose** (low in empyema, TB, rheumatoid), **cytology** (malignant cells: mesothelioma, metastatic carcinoma), and **microbiology** (culture and AFB in TB).

Pleural biopsy (Abrams needle biopsy or CT-guided or thoracoscopic biopsy) is indicated when thoracocentesis is non-diagnostic, particularly when TB or malignancy is suspected. **Medical thoracoscopy (pleuroscopy)** allows direct visualisation of the pleural cavity and targeted biopsy of pleural lesions under local anaesthesia, with sensitivity above 90 percent for pleural malignancy. **Pleural pH below 7.2** in an exudative effusion indicates complicated parapneumonic effusion or empyema requiring drainage.

Fill in the blank: Light's criteria classify pleural fluid as exudate (protein above 30 g/L or pleural/serum protein ratio above 0.5); pleural pH below 7.2 indicates complicated parapneumonic effusion or _____; thoracoscopy has sensitivity above 90 percent for pleural malignancy.

2.4.3 Microbiological and haematological investigations

Microbiological investigations in respiratory disease include: **sputum Gram stain and culture** (community-acquired pneumonia: S. pneumoniae is the most common isolate), **sputum for AFB (acid-fast bacilli) smear and culture** (TB: three early morning sputum samples; culture on Lowenstein-Jensen medium takes 4 to 8 weeks; GeneXpert MTB/RIF provides rapid PCR-based TB diagnosis and rifampicin resistance detection within 2 hours), **urinary antigen tests** (Legionella and pneumococcal urinary antigens in severe CAP), and **serology and PCR** (influenza, COVID-19, Mycoplasma, Chlamydia).

Haematological investigations include: **FBC** (leucocytosis: bacterial infection; lymphopenia: viral infection or TB; eosinophilia: allergic disease, Loeffler's syndrome, tropical eosinophilia), **CRP and ESR** (elevated in infection and inflammation; ESR markedly elevated in TB), **serum ACE** (elevated in active sarcoidosis), and **ANCA, ANA, and anti-GBM antibodies** (vasculitis and pulmonary-renal syndromes). Serum **LDH** is elevated in PCP (Pneumocystis jirovecii pneumonia) and correlates with disease severity.

Fill in the blank: GeneXpert MTB/RIF provides rapid PCR-based TB diagnosis and _____ resistance detection within 2 hours; FBC shows eosinophilia in allergic disease; serum ACE is elevated in active sarcoidosis.

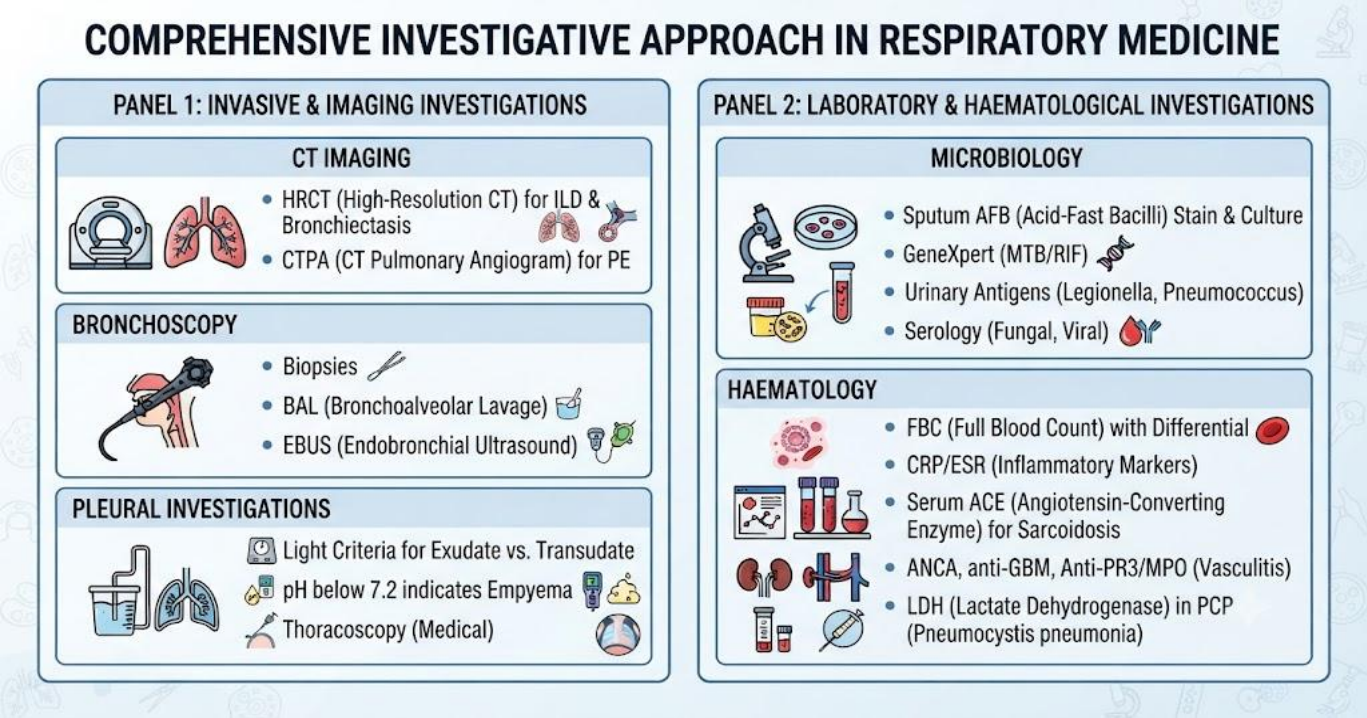


Figure 2.4: Advanced respiratory investigations CT imaging, bronchoscopy, pleural investigations, and microbiology

Pilot questions 2.4

1. Describe the indications for HRCT chest and the findings in ILD and bronchiectasis. (Linked to SLO 2.5)
2. Describe the indications for fibre-optic bronchoscopy and the specimens that can be obtained. (Linked to SLO 2.5)
3. Describe how pleural fluid is analysed and explain Light's criteria. (Linked to SLO 2.5)
4. Describe the microbiological investigation of a patient with suspected pulmonary tuberculosis. (Linked to SLO 2.5)
5. Describe the haematological investigations in respiratory disease and their interpretation. (Linked to SLO 2.5)

Series Summary

This Series developed a symptom-based approach to respiratory medicine. Dyspnoea: acute causes include PE (sudden onset, pleuritic pain, tachycardia), pneumothorax (hyper-resonance, absent sounds), severe asthma, acute pulmonary oedema, and anaphylaxis; chronic causes include COPD (smoking, obstructive spirometry), pulmonary fibrosis (crackles, HRCT), heart failure (orthopnoea, elevated BNP), anaemia, and pleural effusion; in Nigeria TB must be included in chronic dyspnoea. First-line investigation: CXR, FBC, ECG, spirometry, echo; second-line: CTPA (PE), HRCT (ILD or bronchiectasis), BNP (heart failure), ABG. Cough: acute causes (URTI, bronchitis, pneumonia); chronic causes (post-nasal drip, asthma, GORD, ACEi, TB in Nigeria); chronic cough investigation: CXR; therapeutic trials. Haemoptysis: causes include TB (most common in Nigeria), lung cancer, bronchiectasis, PE; investigation: CXR, sputum AFB, CT chest, bronchoscopy; massive haemoptysis: position with bleeding lung dependent and proceed to bronchial artery embolisation.

Chest pain: pleuritic (worse on inspiration; causes: pleurisy, pneumonia, PE, pneumothorax); pericarditis (positional, friction rub, saddle ST elevation); musculoskeletal (reproduced by palpation); distinguish from ischaemic pain (crushing, central, not affected by inspiration). Wheeze: polyphonic (asthma, COPD); monophonic (foreign body, tumour); cardiac asthma (heart failure); wheeze investigation: spirometry, bronchodilator reversibility (FEV1 above 12 percent and 200 mL: asthma), PEFR diary. Stridor: laryngeal carcinoma, thyroid mass, foreign body (adults); croup (children), epiglottitis (life-threatening). Advanced investigations: HRCT (ILD: honeycombing; bronchiectasis: signet ring); bronchoscopy (biopsies, BAL, EBUS); pleural fluid: Light's criteria (exudate: protein above 30 g/L); pH below 7.2: empyema; thoracoscopy: above 90 percent sensitivity for pleural malignancy; sputum AFB and GeneXpert (TB rapid diagnosis and rifampicin resistance); FBC (eosinophilia: allergy), serum ACE (sarcoidosis), ANCA and anti-GBM (vasculitis).

Glossary of Terms

- **CTPA:** CT pulmonary angiography; the investigation of choice for suspected pulmonary embolism; visualises filling defects in the pulmonary arterial tree.
- **HRCT chest:** high-resolution CT of the chest; uses thin slices to provide detailed imaging of lung parenchyma; the investigation of choice for ILD and bronchiectasis.
- **GeneXpert MTB/RIF:** a rapid PCR-based test for Mycobacterium tuberculosis and rifampicin resistance in sputum; provides results within 2 hours; now the first-line TB diagnostic test.
- **Bronchoalveolar lavage (BAL):** a procedure during bronchoscopy where saline is instilled and aspirated from a lung segment to obtain cells and microorganisms from the alveolar space.
- **Light's criteria:** a set of criteria to distinguish exudate from transudate: pleural fluid is an exudate if protein is above 30 g/L, or pleural/serum protein ratio is above 0.5, or pleural/serum LDH ratio is above 0.6.
- **EBUS:** endobronchial ultrasound; a bronchoscopic technique allowing visualisation and needle aspiration of mediastinal and hilar lymph nodes.
- **PEFR:** peak expiratory flow rate; the maximum flow rate during a forced exhalation; used to monitor asthma and assess bronchodilator reversibility.
- **Post-nasal drip (UACS):** upper airway cough syndrome; the most common cause of chronic cough; caused by post-nasal drip from rhinitis or sinusitis triggering the cough reflex.
- **Massive haemoptysis:** expectoration of above 200 to 600 mL of blood in 24 hours; a life-threatening emergency; causes include bronchiectasis, TB, and aspergilloma.
- **Empyema:** pus in the pleural space; indicated by pleural pH below 7.2 or glucose below 2.2 mmol/L; requires drainage by chest tube.
- **Croup (laryngotracheobronchitis):** viral infection of the upper airway caused by parainfluenza virus; presents with a barking cough and inspiratory stridor; managed with oral dexamethasone.
- **D-dimer:** a fibrin degradation product elevated in pulmonary embolism; a normal D-dimer effectively excludes PE in patients with low to intermediate pre-test probability.

Concept Check Questions [Series 2]

Objective Questions

1. Acute onset dyspnoea with pleuritic chest pain and tachycardia suggests pulmonary _____. (Linked to SLO 2.1)
2. In Nigeria, chronic dyspnoea and cough lasting more than 2 weeks must always raise suspicion for pulmonary _____. (Linked to SLO 2.1)
3. The most common cause of haemoptysis in Nigeria is pulmonary _____; other causes include lung cancer and bronchiectasis. (Linked to SLO 2.3)
4. Polyphonic wheeze suggests widespread airway narrowing (asthma, COPD); monophonic wheeze suggests _____ obstruction from foreign body or tumour. (Linked to SLO 2.4)
5. GeneXpert MTB/RIF detects Mycobacterium tuberculosis and _____ resistance within 2 hours. (Linked to SLO 2.5)
6. Pleural pH below 7.2 indicates complicated parapneumonic effusion or _____; requires chest tube drainage. (Linked to SLO 2.5)

Short-Answer Questions

7. Describe the differential diagnosis of chronic cough. (Linked to SLO 2.3)
8. Describe the Light's criteria for distinguishing exudate from transudate in pleural fluid. (Linked to SLO 2.5)
9. Describe the clinical features that distinguish pleuritic chest pain from ischaemic cardiac pain. (Linked to SLO 2.4)

Long-Answer Questions

10. Describe the approach to a patient with haemoptysis including the differential diagnosis, investigation pathway, and management of massive haemoptysis. (Linked to SLO 2.3)
11. Describe the advanced respiratory investigations available for the investigation of respiratory disease. (Linked to SLO 2.5)

Answers to Concept Check Questions [Series 2]

Objective Questions

1. Embolism.
2. TB.
3. TB.
4. Localised.
5. Rifampicin.
6. Empyema.

Short-Answer Questions

7. Post-nasal drip syndrome (UACS: rhinitis or sinusitis; throat clearing, nasal symptoms). Cough variant asthma (dry nocturnal cough; bronchodilator responsive; spirometry with reversibility). GORD (worse after meals and when supine; burning; relieved by antacids). ACE inhibitor-induced cough (dry, persistent; resolves on stopping). Pulmonary TB (Nigeria: cough above 2 weeks, weight loss, night sweats; AFB sputum).
8. Exudate if any one of three Light's criteria are met: pleural fluid protein above 30 g/L (or above 3 g/dL); pleural fluid/serum protein ratio above 0.5; pleural fluid/serum LDH ratio above 0.6. Causes of exudate: pneumonia, TB, malignancy, PE, connective tissue disease. Causes of transudate: heart failure, cirrhosis, hypoalbuminaemia, nephrotic syndrome.
9. Pleuritic: sharp, stabbing; worse on deep inspiration and coughing; relieved by shallow breathing; localised to one area; may have pleural rub on auscultation; associated with fever (pneumonia, pleurisy), dyspnoea (PE), or sudden onset (pneumothorax). Ischaemic: crushing or pressure; central or left-sided; radiates to left arm or jaw; not affected by inspiration or position; associated with diaphoresis and nausea; relieved by nitrates.

Long-Answer Questions

10. **Basic response:** Differential diagnosis: pulmonary TB (most common in Nigeria: blood-streaked or frank haemoptysis; weight loss, night sweats, cavitation on CXR); lung cancer (squamous cell carcinoma: smoking history, weight loss, clubbing, central mass on CXR/CT); bronchiectasis (chronic productive cough, copious sputum, coarse crackles, bronchial dilatation on HRCT); pulmonary embolism (pleuritic pain, dyspnoea, tachycardia, risk factors for DVT); lung abscess (fever, productive cough, air-fluid level

on CXR or CT); aspergilloma (Aspergillus fungus ball in a pre-existing cavity). Investigation: CXR (cavity: TB or abscess; mass: malignancy; diffuse shadowing: vasculitis; clear: PE); sputum for AFB smear and GeneXpert (TB); CT chest (better characterisation of all lesions; nodal staging in malignancy); bronchoscopy (localise bleeding source; biopsies; BAL). Massive haemoptysis management: position patient with the bleeding lung dependent (to protect the non-bleeding lung from flooding); secure IV access and resuscitate; urgent chest CT; bronchoscopy (to localise the source); bronchial artery embolisation (BAE: catheter-based occlusion of the bleeding bronchial artery: the most effective acute treatment); surgical resection in selected cases.

Value-added response: In Nigeria, the first diagnosis to exclude in any patient with haemoptysis is pulmonary TB: the combination of haemoptysis, weight loss, night sweats, and upper lobe cavitation on CXR constitutes a clinical TB diagnosis until proven otherwise, and immediate sputum AFB and GeneXpert testing is mandatory; delaying this investigation in the face of haemoptysis risks both ongoing disease transmission and progression to life-threatening haemoptysis.

11. **Basic response:** CT imaging: HRCT chest (thin-slice, no contrast): investigation of choice for ILD (honeycombing and traction bronchiectasis: UIP pattern of IPF; ground-glass opacity: NSIP or early ILD; mosaic attenuation: HPOA), bronchiectasis (bronchial dilatation, signet ring sign, wall thickening, mucous plugging), small nodules (Lung-RADS scoring for lung cancer risk), and small pneumothorax or effusion missed on CXR. CTPA (CT pulmonary angiography with contrast): investigation of choice for PE (filling defects in pulmonary arteries). Bronchoscopy: direct visualisation of airways to segmental bronchi; bronchial biopsy (endobronchial tumour); BAL (cell count and microbiology in ILD, PCP, opportunistic infections); transbronchial biopsy (lung parenchyma); EBUS (mediastinal and hilar lymph node sampling). Pleural investigations: thoracocentesis (diagnostic or therapeutic: aspirate fluid for protein, LDH, glucose, cytology, microbiology, AFB); pleural biopsy (Abrams needle or CT-guided or thoracoscopic); medical thoracoscopy (sensitivity above 90 percent for pleural malignancy). Microbiology: sputum AFB and GeneXpert MTB/RIF (TB and rifampicin resistance in 2 hours); urinary antigen (Legionella and pneumococcal); serology and PCR (Mycoplasma, Chlamydia, influenza, COVID-19). Haematology: FBC (eosinophilia:

allergy; leucocytosis: bacterial infection); CRP and ESR (infection, inflammation, TB); serum ACE (sarcoidosis); ANCA and anti-GBM (vasculitis); LDH (elevated in PCP).

Value-added response: The choice of investigation is guided by the clinical question: HRCT answers structural questions (what does the parenchyma look like?), bronchoscopy answers luminal and cellular questions (what is in the airway or alveolar space?), pleural investigation answers fluid composition questions (is this an exudate and what is causing it?), and microbiology answers aetiological questions (what organism is responsible?); using each tool for its appropriate clinical question avoids unnecessary invasive procedures and reaches the diagnosis most efficiently.

Answers to Pilot Questions [Series 2]

Part 2.1

1. Acute dyspnoea (onset over minutes to hours): pulmonary embolism (sudden, pleuritic pain, tachycardia), pneumothorax (sudden, unilateral, hyper-resonance), acute severe asthma (wheeze, accessory muscles). Chronic dyspnoea (onset over weeks to months): COPD (progressive, smoking, obstructive spirometry), pulmonary fibrosis (progressive, dry cough, fine crackles), heart failure (orthopnoea, ankle oedema, elevated BNP).
2. Sudden onset dyspnoea, pleuritic chest pain (sharp, worse on inspiration), tachycardia (heart rate above 100 bpm), tachypnoea, low-grade fever; risk factors for deep vein thrombosis (immobility, recent surgery, malignancy, oral contraceptive use, pregnancy); signs of DVT (calf swelling, tenderness); oxygen saturation may be reduced; CXR may be normal or show a wedge-shaped peripheral opacity (pulmonary infarction), elevated hemidiaphragm, or pleural effusion.
3. Pulmonary TB (the most common cause: cough above 2 weeks, weight loss, night sweats, haemoptysis, upper lobe infiltrates or cavitation on CXR; use GeneXpert and AFB sputum). HIV-related respiratory disease (PCP: dry cough, dyspnoea, low-grade fever, bilateral perihilar infiltrates; HIV status essential). Biomass fuel-related COPD (women, indoor cooking fire or kerosene exposure, progressive dyspnoea, obstructive spirometry; not just a smoker's disease). Schistosomiasis-related pulmonary hypertension (right heart

failure in endemic areas; parasites in portal circulation cause immune-mediated pulmonary vascular disease).

4. CXR (consolidation: pneumonia; effusion: malignancy, heart failure, TB; cardiomegaly: heart failure; hyperinflation: COPD; pneumothorax; mass or nodule). FBC (anaemia: cause of dyspnoea; eosinophilia: allergic or parasitic; leucocytosis: infection). ECG (right heart strain in PE: S1Q3T3; AF: cardiac cause of dyspnoea; LVH: hypertensive heart failure; ischaemic changes: cardiac dyspnoea). Echocardiography (LV dysfunction: heart failure; RV enlargement: PE or cor pulmonale; pulmonary artery pressure). Spirometry (obstructive: COPD or asthma; restrictive: fibrosis or neuromuscular).
5. CTPA (CT pulmonary angiography): indicated when PE is suspected with elevated D-dimer or high pre-test probability; demonstrates filling defects in the pulmonary arteries; provides definitive diagnosis; contraindicated in severe renal impairment or contrast allergy. HRCT chest (high-resolution CT): indicated for interstitial lung disease (shows honeycombing, ground-glass opacity, reticular shadowing; distinguishes UIP from other ILD patterns), bronchiectasis (bronchial dilatation, signet ring sign), and detailed characterisation of nodules or masses missed on CXR.

Part 2.2

1. Acute cough (below 3 weeks): most commonly upper respiratory tract infection (viral: rhinovirus, influenza; runny nose, sore throat, mild fever; self-limiting), acute bronchitis (productive cough with clear or purulent sputum, wheeze; usually viral), pneumonia (productive cough, fever, pleuritic pain, consolidation). Chronic cough (above 8 weeks): post-nasal drip (UACS: most common; nasal symptoms, throat clearing), asthma (cough variant: nocturnal, dry, bronchodilator responsive), GORD (worse after meals and supine), ACE inhibitor (dry persistent; 15 percent of users), pulmonary TB (Nigeria: weight loss, night sweats, haemoptysis).
2. Pulmonary TB is the most common infectious disease in Nigeria and the most common cause of chronic respiratory symptoms; it is highly prevalent due to overcrowding, malnutrition, high HIV prevalence, and limited healthcare access; any cough lasting above 2 weeks should be evaluated for TB with three early morning sputum samples for AFB smear and GeneXpert MTB/RIF (providing both TB diagnosis and rifampicin

resistance in 2 hours); missing TB leads to ongoing transmission, progression to severe disease, and risk of drug-resistant TB.

3. TB (most common in Nigeria): blood-streaked or frank haemoptysis; weight loss, night sweats, low-grade fever; upper lobe infiltrates or cavitation on CXR. Lung cancer: progressive haemoptysis; smoker; weight loss; clubbing; central mass, hilar lymphadenopathy, or pleural effusion on CXR or CT. Bronchiectasis: chronic productive cough with copious sputum; coarse crackles; dilated bronchi on HRCT; haemoptysis may be massive. PE: haemoptysis with dyspnoea, pleuritic chest pain, tachycardia; risk factors for DVT. Lung abscess: fever, productive foul-smelling sputum; air-fluid level in cavity on CXR.
4. Haemoptysis: blood coughed up from respiratory tract; bright red or pink frothy; mixed with sputum; preceded by respiratory symptoms; pH alkaline. Haematemesis: blood vomited from GI tract; dark red or coffee-ground colour; mixed with food; preceded by nausea; often clotted; pH acid. Epistaxis: nasal bleeding; blood drips from anterior nares or drips down the back of the throat; associated with nasal congestion, nasal trauma, or anticoagulant use; no respiratory prodrome.
5. CXR (mandatory first step: cavity or infiltrate: TB; mass: malignancy; clear: PE or early bronchiectasis). Sputum for AFB smear and GeneXpert MTB/RIF (three early morning samples; mandatory if TB suspected). CT chest (characterises mass, cavity, or bronchiectasis; CT angiography for PE; nodal staging in malignancy). Bronchoscopy (fibre-optic: localises bleeding source in up to 90 percent of cases; biopsies endobronchial tumour; BAL for microbiology; therapeutic: endobronchial tamponade or adrenaline application). Bronchial artery embolisation for massive haemoptysis.

Part 2.3

1. Sharp, stabbing chest pain; worsened by deep inspiration, coughing, or sneezing; relieved by shallow breathing or lying on the affected side; well-localised. Four causes: pleurisy (primary viral or secondary to pneumonia, PE, TB, connective tissue disease), pneumonia (adjacent pleural inflammation), pulmonary embolism (distal emboli cause pleural infarction), pneumothorax (sudden onset, ipsilateral).

2. Pleuritic: sharp, stabbing; well-localised; worsened by deep inspiration, coughing; not affected by exertion; pleural rub on auscultation; may have associated fever, dyspnoea, or haemoptysis; reproducible with deep breath. Ischaemic (angina or MI): crushing, tight, or pressure quality; central or left-sided; radiates to left arm, jaw, or back; precipitated by exertion and relieved by rest or nitrates; not affected by breathing or position; associated with diaphoresis, nausea, pallor.
3. Polyphonic wheeze (multiple musical pitches simultaneously): caused by widespread airway narrowing affecting many airways at different levels; conditions: asthma (variable, reversible), COPD (fixed, chronic), acute pulmonary oedema (cardiac asthma from bronchospasm and secretions). Monophonic wheeze (single musical pitch; consistent across breathing cycle): caused by localised, fixed obstruction of a single large airway; conditions: endobronchial tumour (primary or secondary lung cancer), inhaled foreign body (sudden onset, choking history), extrinsic airway compression (mediastinal mass or enlarged lymph node).
4. Adults: laryngeal carcinoma (progressive hoarseness preceding stridor; smoking and alcohol history; hoarse voice), thyroid mass or goitre (tracheal deviation and compression; goitre palpable on examination), foreign body aspiration (sudden onset stridor; choking episode; more common in elderly with dysphagia), anaphylaxis (urticaria, angioedema, hypotension, exposure history), bilateral vocal cord palsy (from thyroid surgery, malignancy). Children: croup or laryngotracheobronchitis (parainfluenza virus; barking seal-like cough; inspiratory stridor; managed with oral dexamethasone and cool moist air), epiglottitis (Haemophilus influenzae type b or Group A streptococcus; high fever, drooling, tripod position; life-threatening airway emergency; management: senior anaesthesia and ENT immediately; IV ceftriaxone).
5. Spirometry with bronchodilator reversibility test (FEV1 and FVC before and 20 minutes after salbutamol 400 micrograms via spacer; significant reversibility: FEV1 improvement above 12 percent and above 200 mL from baseline; confirms asthma diagnosis). PEFR diary over 2 to 4 weeks (diurnal variation: morning PEFR minus evening PEFR divided by the higher value, multiplied by 100; above 20 percent variation supports asthma). CXR (hyperinflation and flat diaphragms: COPD; cardiomegaly and pulmonary oedema: cardiac asthma; foreign body). Fractional exhaled nitric oxide (FeNO: elevated in

eosinophilic airway inflammation; supports asthma diagnosis and predicts steroid response).

Part 2.4

1. HRCT indications: ILD (symptoms of dyspnoea and dry cough with fine crackles, particularly with occupational history or connective tissue disease; shows honeycombing, traction bronchiectasis, ground-glass opacity; distinguishes UIP from NSIP and other patterns), bronchiectasis (chronic productive cough, coarse crackles, recurrent chest infections; shows bronchial dilatation greater than adjacent artery, signet ring sign, bronchial wall thickening), lung cancer screening (low-dose CT in high-risk patients), and small pneumothorax or pleural disease not seen on CXR. ILD findings: UIP pattern (honeycombing in subpleural basal distribution, traction bronchiectasis, reticular shadowing: IPF); ground-glass opacity with peripheral consolidation: COP or NSIP. Bronchiectasis findings: cylindrical (mild), varicose (moderate), cystic (severe) dilatation; signet ring sign (bronchus diameter larger than adjacent vessel); mucous plugging.
2. Indications for FOB: unexplained haemoptysis (to localise the bleeding source and obtain biopsies), central lung mass on imaging (to biopsy endobronchial tumour), suspected endobronchial disease (localised wheeze, stridor, collapse), foreign body removal, localised consolidation or collapse of unknown cause, investigation of ILD or opportunistic infection (via BAL), sputum collection in patients unable to expectorate. Specimens: bronchial biopsy (endobronchial or peribronchial tumour), BAL (cell count: lymphocytosis in HP and sarcoidosis; eosinophilia in eosinophilic pneumonia; PCP on silver stain; microbiology for bacteria, fungi, AFB), protected specimen brush (quantitative culture for VAP), transbronchial biopsy (lung parenchyma: sarcoidosis, cryptogenic organising pneumonia), EBUS-TBNA (mediastinal and hilar lymph node fine needle aspiration for staging and diagnosis).
3. Appearance (straw-coloured: transudate; turbid: exudate or empyema; bloody: malignancy, PE, haemothorax; milky white: chylothorax from thoracic duct disruption). Protein and LDH (Light's criteria: exudate if protein above 30 g/L, or pleural/serum protein ratio above 0.5, or pleural/serum LDH ratio above 0.6; transudate criteria: heart failure, cirrhosis, nephrotic syndrome). Glucose (low below 2.2 mmol/L: empyema, TB,

rheumatoid pleurisy). pH (below 7.2: complicated parapneumonic effusion or empyema: requires drainage). Cytology (malignant cells: mesothelioma, metastatic carcinoma; lymphocytosis: TB or lymphoma). Microbiology (Gram stain and culture: bacteria; AFB culture: TB; GeneXpert: rapid TB detection from pleural fluid with lower sensitivity than sputum).

4. Three early morning sputum samples (before antibiotic treatment): collected on three consecutive mornings; early morning sputum is most concentrated in mycobacteria. Sputum AFB smear (Ziehl-Neelsen staining: rapid, cheap, but less sensitive than culture; positive in approximately 50 to 70 percent of smear-positive TB). GeneXpert MTB/RIF (first-line diagnostic test in Nigeria: PCR-based; detects Mycobacterium tuberculosis and rifampicin resistance in under 2 hours; sensitivity approximately 88 percent for smear-negative TB; WHO-recommended). Mycobacterial culture (Lowenstein-Jensen medium: takes 4 to 8 weeks; gold standard for drug susceptibility testing). If sputum cannot be obtained: induced sputum (inhale hypertonic saline to stimulate sputum production), bronchoscopy and BAL (if unable to produce sputum), or lymph node biopsy if lymphadenopathy present.
5. FBC: leucocytosis with neutrophilia (bacterial infection: pneumonia, empyema); lymphopenia (viral infection, TB, HIV); eosinophilia (allergic asthma, Loeffler's syndrome, tropical eosinophilia from filariasis). CRP and ESR: elevated in acute infection and inflammation; ESR markedly elevated (above 100 mm/hr) in active TB, malignancy, connective tissue disease. Serum ACE (angiotensin-converting enzyme): elevated in approximately 75 percent of patients with active pulmonary sarcoidosis; used to monitor disease activity; not diagnostic alone. ANCA (anti-neutrophil cytoplasmic antibody): positive in ANCA-associated vasculitis (granulomatosis with polyangiitis: c-ANCA or PR3-ANCA; microscopic polyangiitis: p-ANCA or MPO-ANCA) causing pulmonary-renal syndrome. Anti-GBM antibodies: positive in Goodpasture syndrome (pulmonary haemorrhage and glomerulonephritis). Serum LDH: markedly elevated in PCP (Pneumocystis jirovecii pneumonia), correlating with disease severity.

References and Attributions [Series 2]

Textual References

- Kumar, P. and Clark, M. (2017). Kumar and Clark's Clinical Medicine (9th ed.). Edinburgh: Elsevier.
- World Health Organization. (2022). WHO Consolidated Guidelines on Tuberculosis: Diagnostic Testing. Geneva: WHO.
- British Thoracic Society. (2010). BTS Pleural Disease Guideline 2010. Thorax, 65(Suppl 2), ii1 to ii76.
- Light, R. W. (2002). Pleural Effusion. New England Journal of Medicine, 346(25), 1971 to 1977.

Figures, Plates, Tables, and Boxes

- Figure 2.1: Approach to dyspnoea differential diagnosis and investigation. AI prompt: "Two-panel diagram: left two-section table Acute Dyspnoea (PE, pneumothorax, severe asthma, acute pulmonary oedema, anaphylaxis) and Chronic Dyspnoea (COPD, pulmonary fibrosis, heart failure, anaemia, pleural effusion); right investigation pathway first-line and second-line; clean professional infographic." OER search: "approach to dyspnoea differential diagnosis investigation pathway acute chronic diagram".
- Figure 2.2: Chronic cough and haemoptysis investigation. AI prompt: "Two-panel diagram: causes of chronic cough and haemoptysis; investigation pathway (CXR, sputum AFB, GeneXpert, CT, bronchoscopy); massive haemoptysis management; clean infographic." OER search: "chronic cough haemoptysis investigation bronchoscopy Nigeria TB diagram".
- Figure 2.3: Chest pain and wheeze or stridor approach. AI prompt: "Two-panel table: chest pain types (pleuritic, pericarditis, musculoskeletal, ischaemic) and wheeze or stridor (polyphonic, monophonic, cardiac asthma, stridor adults and children including epiglottitis); clean infographic." OER search: "pleuritic chest pain wheeze stridor differential diagnosis investigation diagram".

- Figure 2.4: Advanced respiratory investigations. AI prompt: "Two-panel diagram: CT imaging, bronchoscopy, pleural investigations; microbiology (sputum AFB, GeneXpert, urinary antigens); haematology (FBC, CRP, serum ACE, ANCA, LDH in PCP); clean infographic." OER search: "advanced respiratory investigations HRCT bronchoscopy pleural fluid GeneXpert TB diagram".

Series 3: Pneumonias and Tuberculosis Management

- **Part 3.1: Community-Acquired Pneumonia**
  - Sub Part 3.1.1: Aetiology and pathogenesis of CAP
  - Sub Part 3.1.2: Clinical features and diagnosis of CAP
  - Sub Part 3.1.3: Severity assessment and management of CAP
- **Part 3.2: Special Pneumonias**
  - Sub Part 3.2.1: Staphylococcal and Klebsiella pneumonia
  - Sub Part 3.2.2: Atypical pneumonia
  - Sub Part 3.2.3: Aspiration and hospital-acquired pneumonia
- **Part 3.3: Pulmonary Tuberculosis**
  - Sub Part 3.3.1: Epidemiology and pathogenesis of TB
  - Sub Part 3.3.2: Clinical features and diagnosis of TB
  - Sub Part 3.3.3: Treatment of TB
- **Part 3.4: Drug-Resistant TB and TB Complications**
  - Sub Part 3.4.1: Drug-resistant tuberculosis
  - Sub Part 3.4.2: TB and HIV co-infection
  - Sub Part 3.4.3: Complications of pulmonary tuberculosis

Introduction

Pneumonia and tuberculosis together constitute the most important cause of respiratory morbidity and mortality in Nigeria. Community-acquired pneumonia (CAP) is a leading cause of death in children under five and in the elderly, while pulmonary tuberculosis remains the most prevalent infectious disease in Nigeria, with the country ranked among the top 10 high-burden TB nations globally. Understanding the aetiology, clinical features, diagnosis, and management of these conditions is therefore a clinical priority.

This Series covers community-acquired pneumonia systematically, including severity assessment and antibiotic selection. Special pneumonias staphylococcal, Klebsiella, atypical, aspiration, and hospital-acquired are examined separately. The Series then provides a comprehensive account of pulmonary TB from epidemiology through treatment, including drug-resistant TB, TB-HIV co-infection, and the major complications of pulmonary tuberculosis.

Mastery of this content prepares the student to recognise, investigate, and initiate treatment for these common and potentially fatal respiratory infections.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 3.1** describe the aetiology, pathogenesis, clinical features, and diagnosis of community-acquired pneumonia (Linked to Part 3.1),
- **SLO 3.2** apply the CURB-65 severity score to guide site of care and antibiotic selection in CAP (Linked to Part 3.1),
- **SLO 3.3** describe the distinguishing features and management of staphylococcal, Klebsiella, atypical, aspiration, and hospital-acquired pneumonias (Linked to Part 3.2),
- **SLO 3.4** describe the epidemiology, pathogenesis, clinical features, diagnosis, and treatment of pulmonary tuberculosis (Linked to Part 3.3), and

- **SLO 3.5** describe drug-resistant TB, TB-HIV co-infection, and the major complications of pulmonary tuberculosis (Linked to Part 3.4).

3.1 Community-Acquired Pneumonia

3.1.1 Aetiology and pathogenesis of CAP

Community-acquired pneumonia (CAP) is defined as pneumonia acquired outside hospital or within 48 hours of admission, in a patient who has not resided in a long-term care facility within the preceding 14 days. The most common causative organism is **Streptococcus pneumoniae** (the pneumococcus; responsible for 30 to 40 percent of CAP requiring hospitalisation), followed by **Haemophilus influenzae** (particularly in COPD and smokers), **Staphylococcus aureus** (post-influenza; cavitating; severe), and atypical organisms (Mycoplasma pneumoniae, Chlamydophila pneumoniae, Legionella pneumophila).

Pathogenesis involves aspiration of oropharyngeal flora or inhalation of droplet nuclei into the lower respiratory tract, overcoming the defences of mucociliary clearance, cough reflex, and alveolar macrophages. Pneumonia progresses through four classical stages: **congestion** (vascular engorgement, alveolar oedema), **red hepatisation** (RBC and neutrophil exudate filling alveoli: lung solidification), **grey hepatisation** (RBC lysis; fibrin and macrophage predominance), and **resolution** (macrophage clearance of debris; restoration of normal architecture).

Fill in the blank: The most common cause of CAP is S. pneumoniae; pathogenesis involves aspiration of oropharyngeal flora overcoming mucociliary clearance; pneumonia progresses through congestion, red hepatisation, grey hepatisation, and _____.

3.1.2 Clinical features and diagnosis of CAP

Clinical features of CAP: **acute onset of fever** (above 38 degrees C), **productive cough** (purulent or rust-coloured sputum in pneumococcal pneumonia), **pleuritic chest pain** (from adjacent pleural involvement), **dyspnoea** (tachypnoea: respiratory rate above 20), and **systemic**

features (rigors, malaise, myalgia). Elderly patients may present atypically with confusion, falls, or functional decline without fever.

Examination reveals: tachycardia, tachypnoea, fever, reduced chest expansion on the affected side, dull percussion, bronchial breath sounds, and fine crackles over the affected lobe. **CXR** shows lobar or segmental consolidation (homogeneous opacification with air bronchogram). Sputum Gram stain and culture, blood cultures (before antibiotics), FBC (leucocytosis), CRP, and **urinary pneumococcal and Legionella antigens** complete the investigation. A normal CXR in the first 24 hours does not exclude pneumonia.

Fill in the blank: CAP presents with fever, productive cough (rust-coloured in pneumococcal), pleuritic chest pain, and dyspnoea; CXR shows lobar consolidation with _____ bronchogram; urinary pneumococcal and Legionella antigens are key investigations in severe CAP.

3.1.3 Severity assessment and management of CAP

The CURB-65 score assesses CAP severity: **C** (Confusion: new onset), **U** (Urea above 7 mmol/L), **R** (Respiratory rate above 30 per minute), **B** (Blood pressure: systolic below 90 or diastolic below 60 mmHg), **65** (age 65 years or above). Score 0 to 1: low severity treat at home (5-day oral amoxicillin). Score 2: moderate severity consider hospital admission (oral amoxicillin plus clarithromycin). Score 3 to 5: high severity hospital admission required, consider ICU (IV co-amoxiclav plus clarithromycin; or IV ceftriaxone plus clarithromycin).

Supportive treatment includes oxygen (target SpO2 94 to 98 percent; 88 to 92 percent in COPD), IV fluids, analgesia (pleuritic pain), and physiotherapy (to aid sputum clearance). **Duration of antibiotics**: 5 days for low to moderate severity; 7 to 10 days for severe; 14 to 21 days for Staphylococcal or Legionella pneumonia. Review 48 hours after starting antibiotics; if no improvement, review diagnosis and consider broadening cover.

Fill in the blank: CURB-65: Confusion, Urea above 7, Respiratory rate above 30, BP systolic below 90, age above 65; score 0 to 1: treat at home with oral amoxicillin; score 3 to 5: _____ admission and IV antibiotics.

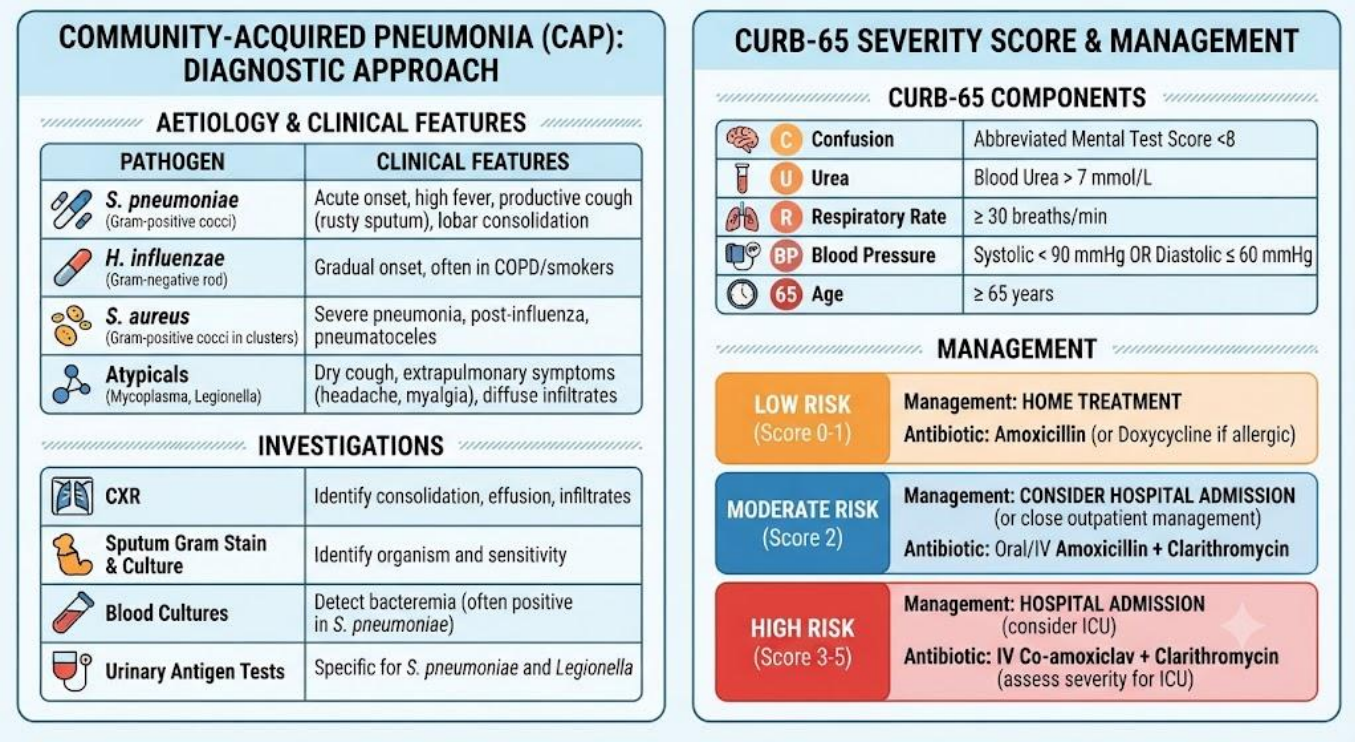


Figure 3.1: CAP aetiology, clinical features, investigations, and CURB-65 severity-guided management

Pilot questions 3.1

1. Describe the most common causative organisms of CAP and the clinical features of pneumococcal pneumonia. (Linked to SLO 3.1)
2. Describe the pathological stages of pneumonia. (Linked to SLO 3.1)
3. Describe the investigations used to diagnose and assess the severity of CAP. (Linked to SLO 3.1)
4. Describe the CURB-65 score and explain how it guides antibiotic choice and site of care. (Linked to SLO 3.2)
5. Describe the supportive treatment of a patient with severe CAP. (Linked to SLO 3.2)

3.2 Special Pneumonias

3.2.1 Staphylococcal and Klebsiella pneumonia

Staphylococcal pneumonia is caused by Staphylococcus aureus and has distinctive features: it occurs after influenza (influenza virus damages the bronchial epithelium, predisposing to secondary bacterial invasion), in IV drug users, and in patients with haematogenous seeding from another infective focus. CXR shows **bilateral patchy consolidation with rapid cavitation and pneumatocele formation**. It is severe and rapidly progressive; treatment requires **IV fluoxacillin (or vancomycin for MRSA)** for 14 to 21 days.

Klebsiella pneumonia (Friedlander's pneumonia) is caused by Klebsiella pneumoniae, a Gram-negative bacillus. It classically affects **alcoholics and debilitated patients** and produces a **thick, gelatinous (currant-jelly) sputum** from haemorrhagic necrosis of the lung. CXR shows **upper lobe consolidation with a bulging fissure** (the hallmark of Klebsiella pneumonia: the lobe expands due to the voluminous inflammatory exudate, pushing the fissure downwards). It is severe with a high mortality; treatment is with **a third-generation cephalosporin or carbapenem**.

Fill in the blank: Staphylococcal pneumonia is post-influenza; CXR shows bilateral cavitation and pneumatoceles; Klebsiella pneumonia affects alcoholics; CXR shows upper lobe consolidation with a _____ fissure; currant-jelly sputum is characteristic.

3.2.2 Atypical pneumonia

Atypical pneumonia is caused by organisms that do not grow on standard culture media and do not respond to beta-lactam antibiotics: **Mycoplasma pneumoniae** (the most common atypical pathogen in young adults; epidemic every 3 to 4 years; presents with gradual onset, headache, dry cough, and bilateral patchy lower lobe infiltrates; extrapulmonary complications include haemolytic anaemia cold agglutinins erythema multiforme, and encephalitis), **Chlamydophila pneumoniae** (similar to Mycoplasma; pharyngitis may precede pneumonia), **Legionella pneumophila** (Legionnaires' disease: from contaminated water sources: cooling towers, air conditioning, hospital water systems; severe CAP with prominent systemic features: confusion,

diarrhoea, hyponatraemia, marked elevation of LDH and CRP; urinary Legionella antigen is the key diagnostic test).

All atypical organisms are treated with macrolides (azithromycin, clarithromycin) or doxycycline; fluoroquinolones (levofloxacin, moxifloxacin) are an alternative for severe disease. Legionella pneumonia requires **azithromycin or a fluoroquinolone**; rifampicin may be added in severe cases. Cold agglutinin test is positive in above 50 percent of Mycoplasma cases.

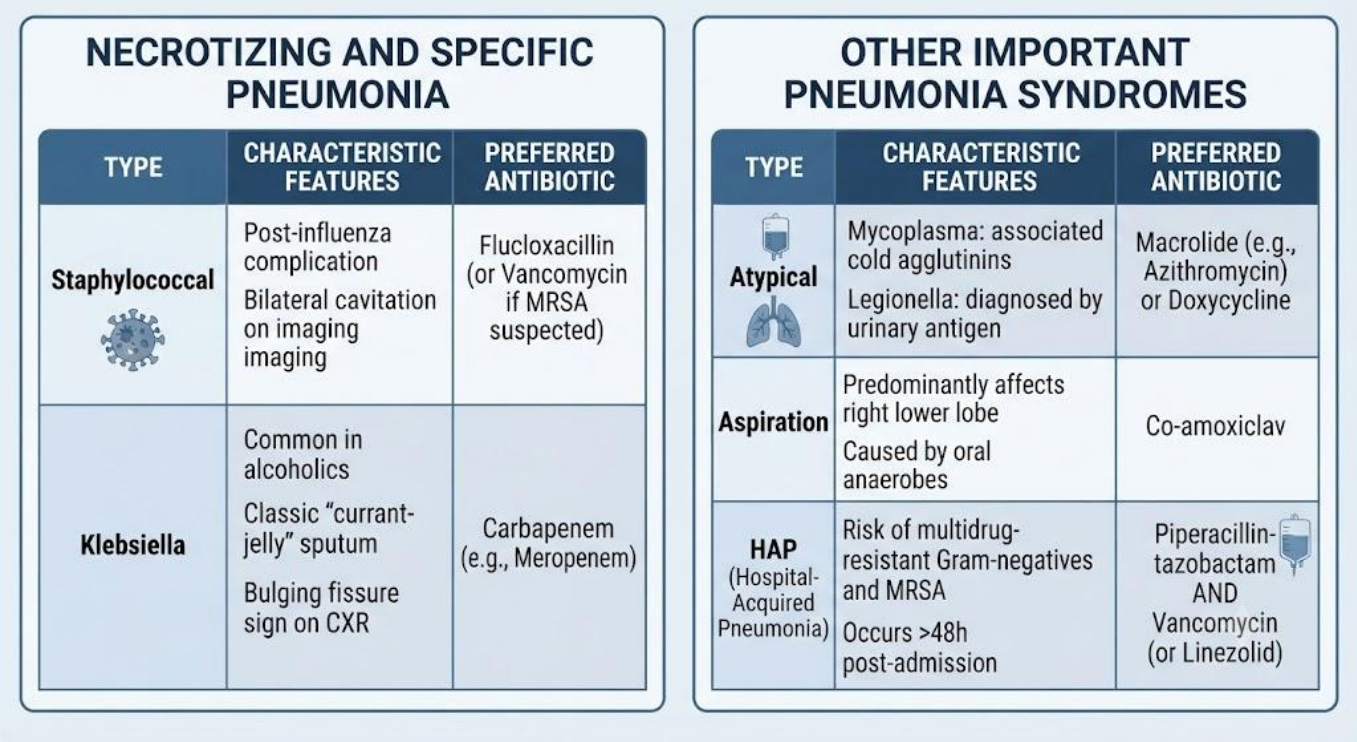
Fill in the blank: Mycoplasma pneumoniae causes atypical pneumonia in young adults with cold agglutinins (haemolytic anaemia); Legionella causes severe CAP with confusion, diarrhoea, and _____; all atypical pneumonias are treated with macrolides or doxycycline.

3.2.3 Aspiration and hospital-acquired pneumonia

Aspiration pneumonia results from aspiration of oropharyngeal contents into the lower respiratory tract. Risk factors include: **reduced level of consciousness** (alcohol, anaesthesia, seizures, stroke), **dysphagia** (neurological disease, oesophageal disease), and **poor dental hygiene**. Aspiration typically affects the **right lower lobe** (dependent position in the supine patient; from the anatomical characteristics of the right main bronchus). Causative organisms are mixed oral anaerobes and Gram-negative bacteria. Treatment includes **co-amoxiclav or clindamycin** to cover anaerobes.

Hospital-acquired pneumonia (HAP) is defined as pneumonia developing above 48 hours after hospital admission. It is predominantly caused by **Gram-negative bacteria** (Pseudomonas aeruginosa, Klebsiella, Escherichia coli, Acinetobacter) and **MRSA**. **Ventilator-associated pneumonia (VAP)** is a specific subtype occurring in mechanically ventilated patients. HAP carries a high mortality (above 30 percent). Treatment requires broad-spectrum antibiotics guided by local resistance patterns: **piperacillin-tazobactam or ceftazidime plus an aminoglycoside** for Gram-negative organisms; vancomycin for MRSA.

Fill in the blank: Aspiration pneumonia affects the right lower lobe; treat with co-amoxiclav or clindamycin (anaerobic cover); HAP is defined as pneumonia above 48 hours after admission; caused by Gram-negative organisms and MRSA; treat with _____ or piperacillin-tazobactam.



NECROTIZING AND SPECIFIC PNEUMONIA

TYPE	CHARACTERISTIC FEATURES	PREFERRED ANTIBIOTIC
Staphylococcal	Post-influenza complication Bilateral cavitation on imaging imaging	Flucloxacillin (or Vancomycin if MRSA suspected)
Klebsiella	Common in alcoholics Classic "currant-jelly" sputum Bulging fissure sign on CXR	Carbapenem (e.g., Meropenem)

OTHER IMPORTANT PNEUMONIA SYNDROMES

TYPE	CHARACTERISTIC FEATURES	PREFERRED ANTIBIOTIC
Atypical	Mycoplasma: associated cold agglutinins Legionella: diagnosed by urinary antigen	Macrolide (e.g., Azithromycin) or Doxycycline
Aspiration	Predominantly affects right lower lobe Caused by oral anaerobes	Co-amoxiclav
HAP (Hospital-Acquired Pneumonia)	Risk of multidrug-resistant Gram-negatives and MRSA Occurs >48h post-admission	Piperacillin-tazobactam AND Vancomycin (or Linezolid)

Figure 3.2: Special pneumonias features, CXR findings, and management

Pilot questions 3.2

1. Describe the distinctive clinical and radiological features of staphylococcal pneumonia. (Linked to SLO 3.3)
2. Describe the clinical features and CXR findings of Klebsiella pneumonia. (Linked to SLO 3.3)
3. Describe the features of Mycoplasma pneumonia including its extrapulmonary complications. (Linked to SLO 3.3)
4. Describe Legionnaires' disease including its source, clinical features, and diagnosis. (Linked to SLO 3.3)
5. Define hospital-acquired pneumonia and describe its management. (Linked to SLO 3.3)

3.3 Pulmonary Tuberculosis

3.3.1 Epidemiology and pathogenesis of TB

Tuberculosis (TB) is caused by Mycobacterium tuberculosis, an aerobic, non-motile, non-spore-forming, acid-fast bacillus. Nigeria has one of the highest TB burdens globally, with an estimated incidence of 219 per 100,000 population. TB is transmitted by airborne droplet nuclei (particles of 1 to 5 microns diameter) released when an infectious person coughs, sneezes, or speaks. A single infectious case may infect 10 to 15 contacts per year.

Following inhalation, M. tuberculosis is phagocytosed by alveolar macrophages; it resists intracellular killing and replicates within macrophages. The macrophage-mycobacteria complex migrates to hilar lymph nodes, forming the **Ghon complex (primary complex)** the primary lung focus plus the draining hilar lymph node. In most immunocompetent individuals, cell-mediated immunity (T-lymphocyte activation and granuloma formation) contains the infection; mycobacteria may persist in a **latent state**. In immunocompromised hosts (HIV, malnutrition, diabetes, immunosuppressive therapy), **reactivation or progressive primary TB** develops.

Fill in the blank: M. tuberculosis is transmitted by airborne droplet nuclei; primary infection forms the Ghon complex (lung focus plus hilar lymph node); most infections become _____ TB; reactivation occurs in the immunocompromised host.

3.3.2 Clinical features and diagnosis of TB

Pulmonary TB presents with a subacute or chronic illness: **persistent cough (above 2 weeks)** (initially dry, then productive; blood-stained sputum in advanced disease), **constitutional symptoms** (fever classically low-grade in the afternoon; drenching night sweats; significant weight loss; fatigue; anorexia), and **haemoptysis** (from cavity erosion into a blood vessel). Examination may show consolidation or cavity signs at the lung apex (upper lobe is the predilection site due to high V/Q ratio and high oxygen tension).

Diagnosis relies on: **GeneXpert MTB/RIF** (WHO-recommended first-line test: PCR-based; detects M. tuberculosis and rifampicin resistance in under 2 hours; sensitivity approximately 88 percent for smear-negative TB), **AFB smear microscopy** (rapid and cheap; positive in 50 to 70

percent of pulmonary TB; defines smear-positive infectious cases), and **mycobacterial culture** (gold standard: Lowenstein-Jensen medium, 4 to 8 weeks; MGIT liquid culture, 10 to 14 days; essential for drug susceptibility testing). CXR: upper lobe infiltrates, cavitation, hilar lymphadenopathy, and calcified Ghon complex.

Fill in the blank: TB diagnosis: GeneXpert MTB/RIF (M. tuberculosis and rifampicin resistance in 2 hours; first-line); AFB smear (rapid, 50 to 70 percent sensitivity); mycobacterial _____ (gold standard; drug susceptibility testing); CXR: upper lobe infiltrates and cavitation.

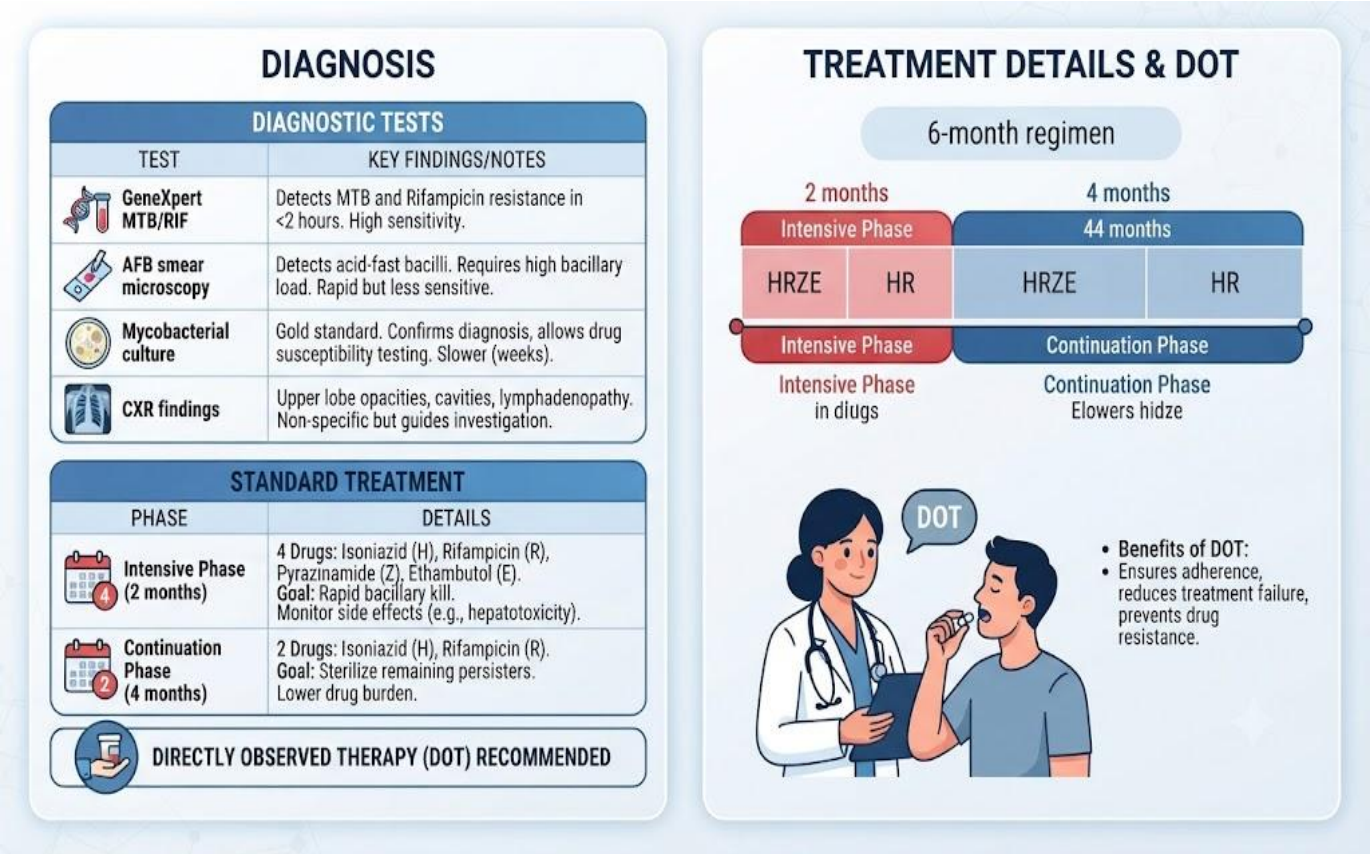


Figure 3.3: Pulmonary TB diagnosis and the standard 2HRZE/4HR treatment regimen with drug side effects

3.3.3 Treatment of TB

Standard TB treatment follows the **2HRZE/4HR regimen**: an **intensive phase of 2 months** with four drugs isoniazid (H), rifampicin (R), pyrazinamide (Z), and ethambutol (E) followed by a **continuation phase of 4 months** with isoniazid and rifampicin. This regimen achieves cure

in above 95 percent of drug-sensitive TB when taken correctly. Directly observed therapy (**DOT**) is recommended to ensure adherence and prevent acquired resistance.

Key side effects of first-line TB drugs: **isoniazid** (peripheral neuropathy: prevented by pyridoxine vitamin B6 supplementation; hepatotoxicity), **rifampicin** (orange discolouration of body fluids; hepatotoxicity; induces liver enzymes reduces efficacy of oral contraceptives and antiretrovirals), **pyrazinamide** (hyperuricaemia and gout; hepatotoxicity), **ethambutol** (optic neuritis: monitor visual acuity and colour vision monthly). All four drugs are hepatotoxic; monitor LFTs and stop all four drugs if transaminases exceed five times normal.

Fill in the blank: Standard TB treatment is 2HRZE/4HR: 2 months of isoniazid, rifampicin, pyrazinamide, and ethambutol, then 4 months of _____ and rifampicin; isoniazid causes peripheral neuropathy (prevented by pyridoxine); ethambutol causes optic neuritis.

Pilot questions 3.3

1. Describe the transmission and pathogenesis of primary tuberculosis. (Linked to SLO 3.4)
2. Describe the clinical features of pulmonary tuberculosis. (Linked to SLO 3.4)
3. Describe the diagnostic tests used in pulmonary tuberculosis and their relative sensitivities. (Linked to SLO 3.4)
4. Describe the standard first-line TB treatment regimen and the side effects of each drug. (Linked to SLO 3.4)
5. Explain why directly observed therapy (DOT) is recommended for TB treatment. (Linked to SLO 3.4)

3.4 Drug-Resistant TB and TB Complications

3.4.1 Drug-resistant tuberculosis

Drug-resistant TB (DR-TB) arises from inadequate or incomplete treatment, poor adherence, or transmission of a resistant strain. **Multidrug-resistant TB (MDR-TB)** is defined as resistance to

at least **isoniazid and rifampicin** the two most potent first-line drugs. **Extensively drug-resistant TB (XDR-TB)** is MDR-TB with additional resistance to any fluoroquinolone and at least one injectable second-line drug. GeneXpert Ultra detects rifampicin resistance as a proxy for MDR-TB within 2 hours.

MDR-TB treatment uses second-line drugs for a minimum of **18 to 24 months**: newer shorter regimens (BPaL: bedaquiline, pretomanid, and linezolid; 6 to 9 months) are increasingly recommended. Key second-line drugs include **bedaquiline** (inhibits mycobacterial ATP synthase; QTc prolongation: monitor ECG), **linezolid** (myelosuppression, peripheral neuropathy), and **clofazimine** (skin discolouration; abdominal pain). Treatment of MDR-TB requires specialist supervision and is associated with high rates of adverse effects.

Fill in the blank: MDR-TB is resistance to isoniazid and rifampicin; XDR-TB adds resistance to fluoroquinolones and injectable agents; MDR-TB treatment uses second-line drugs for 18 to 24 months; the BPaL regimen (bedaquiline, pretomanid, _____) lasts 6 to 9 months.

3.4.2 TB and HIV co-infection

TB is the leading cause of death in people living with HIV (PLHIV) globally and in Nigeria. HIV infection increases the risk of TB reactivation from latent infection by 20- to 30-fold, and increases TB progression from primary infection. TB-HIV co-infection alters the clinical presentation: **smear-negative and extrapulmonary TB are more common** in HIV-positive patients (because inadequate cell-mediated immunity impairs granuloma formation and limits cavitation); CXR may show atypical features including lower lobe involvement, diffuse infiltrates, or be near-normal.

Management of TB-HIV requires starting **anti-TB treatment first** (within 2 weeks of TB diagnosis), then introducing **antiretroviral therapy (ART)** within 2 to 8 weeks (earlier for patients with CD4 below 50 cells/microlitre). Key interactions: **rifampicin is a potent CYP450 inducer** that markedly reduces levels of most antiretrovirals; efavirenz is the preferred NNRTI (less affected by rifampicin) in TB-HIV co-treatment. **Immune reconstitution inflammatory syndrome (IRIS)** (paradoxical worsening of TB symptoms after starting ART from restored immune response) occurs in 15 to 40 percent of TB-HIV patients.

Fill in the blank: In TB-HIV co-infection, start anti-TB treatment first, then ART within 2 to 8 weeks; rifampicin induces CYP450 reducing antiretroviral levels; efavirenz is preferred; _____ (IRIS) occurs in 15 to 40 percent when ART is started.

3.4.3 Complications of pulmonary tuberculosis

Pulmonary complications of TB include: **haemoptysis** (from cavity erosion into a pulmonary artery: Rasmussen's aneurysm can be massive and fatal), **pneumothorax** (from subpleural cavity rupture), **pleural effusion** (primary TB pleurisy: lymphocytic exudate with low glucose; or secondary to pulmonary TB), **empyema necessitans** (TB empyema eroding through the chest wall), **aspergilloma** (Aspergillus colonisation of a pre-existing TB cavity: causes haemoptysis; treated with antifungal or embolisation), and **bronchiectasis** (from airway destruction by TB-related inflammation).

Extrapulmonary complications include: TB lymphadenitis (most common site: cervical lymph nodes scrofula), TB pericarditis (constrictive pericarditis causing heart failure), TB meningitis (the most lethal form: neck stiffness, low CSF glucose, high protein, lymphocytosis), TB spine (Pott's disease: vertebral destruction and paraplegia), adrenal TB (Addison's disease from adrenal destruction), and miliary TB (haematogenous dissemination producing millet-seed-sized granulomas in all organs: characterised by high fever, hepatosplenomegaly, and diffuse bilateral micronodular CXR pattern).

Fill in the blank: Pulmonary TB complications include haemoptysis (Rasmussen's aneurysm), pneumothorax, pleural effusion, aspergilloma in a TB cavity, and bronchiectasis; miliary TB shows _____ dissemination with bilateral micronodular CXR pattern.

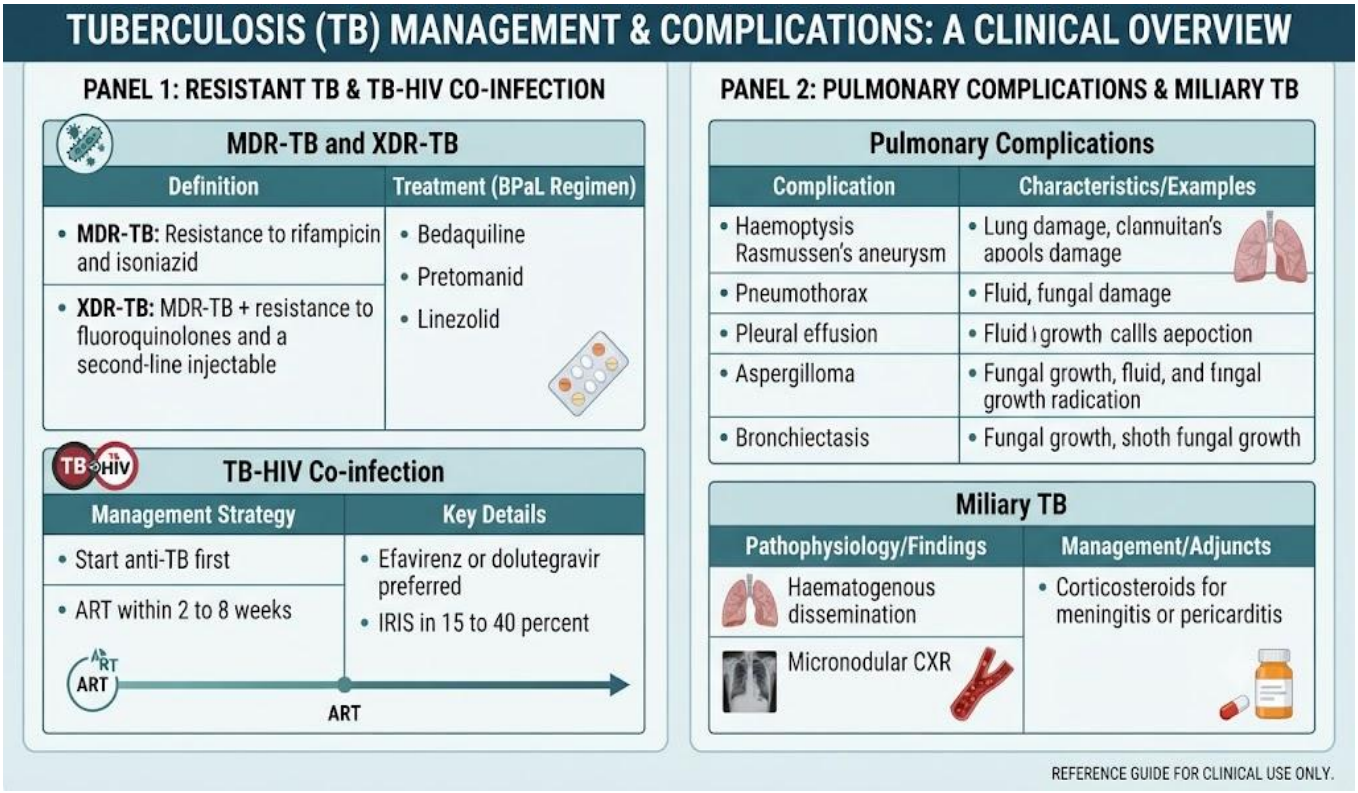


Figure 3.4: Drug-resistant TB, TB-HIV co-infection, IRIS, and complications of pulmonary TB

Pilot questions 3.4

1. Define MDR-TB and XDR-TB and describe the principles of MDR-TB treatment. (Linked to SLO 3.5)
2. Explain how HIV co-infection alters the clinical presentation of TB. (Linked to SLO 3.5)
3. Describe the management of TB-HIV co-infection including the timing of ART. (Linked to SLO 3.5)
4. Describe the pulmonary complications of tuberculosis. (Linked to SLO 3.5)
5. Describe miliary tuberculosis including its clinical features and CXR appearance. (Linked to SLO 3.5)

Series Summary

This Series covered pneumonia and TB. CAP is most commonly caused by S. pneumoniae; clinical features include fever, productive cough (rust-coloured in pneumococcal), pleuritic chest pain, and tachypnoea; CXR shows lobar consolidation with air bronchogram; investigations include sputum culture, blood cultures, FBC, CRP, and urinary antigens in severe CAP. CURB-65 guides management: score 0 to 1 treat at home (amoxicillin); score 2 consider hospital; score 3 to 5 hospital admission and IV antibiotics. Special pneumonias: staphylococcal (post-influenza; bilateral cavitation and pneumatoceles; IV flucloxacillin or vancomycin for MRSA; 14 to 21 days); Klebsiella (alcoholics; currant-jelly sputum; bulging fissure on CXR; cephalosporin or carbapenem); atypical Mycoplasma (young adults; cold agglutinins; haemolytic anaemia; macrolide or doxycycline), Legionella (contaminated water; confusion, diarrhoea, hyponatraemia, elevated LDH; urinary antigen; azithromycin or fluoroquinolone); aspiration (right lower lobe; anaerobes; co-amoxiclav); HAP (Gram-negatives and MRSA; broad-spectrum IV antibiotics).

TB is caused by M. tuberculosis; transmitted by airborne droplet nuclei; Nigeria is a high-burden country. Primary infection forms the Ghon complex; latent TB persists; reactivation occurs in HIV, malnutrition, and diabetes. Clinical features: cough above 2 weeks, night sweats, weight loss, haemoptysis, upper lobe infiltrates and cavitation on CXR. Diagnosis: GeneXpert MTB/RIF (first-line: M. tuberculosis and rifampicin resistance in 2 hours); AFB smear (50 to 70 percent sensitivity); culture (gold standard; drug susceptibility). Treatment: 2HRZE/4HR; isoniazid causes peripheral neuropathy (prevent with pyridoxine); rifampicin induces liver enzymes; ethambutol causes optic neuritis; monitor LFTs. MDR-TB: resistance to isoniazid and rifampicin; treat 18 to 24 months (BPaL regimen 6 to 9 months). TB-HIV: start anti-TB first; ART within 2 to 8 weeks; rifampicin reduces antiretroviral levels; use efavirenz; IRIS occurs in 15 to 40 percent. Complications: haemoptysis (Rasmussen's aneurysm), pneumothorax, empyema, aspergilloma, miliary TB (haematogenous; bilateral micronodular CXR).

Glossary of Terms

- **CURB-65:** a severity scoring system for CAP: Confusion, Urea above 7 mmol/L, Respiratory rate above 30, Blood pressure systolic below 90 or diastolic below 60 mmHg, age 65 or above; guides site of care and antibiotic selection.
- **Ghon complex:** the primary focus of tuberculosis infection (a subpleural granuloma usually in the middle or lower zone) combined with the draining hilar lymph node; calcification of both forms the Ranke complex.
- **Latent TB:** infection with M. tuberculosis where the host immune response contains the organism without eliminating it; the patient is asymptomatic and non-infectious but at risk of reactivation.
- **MDR-TB:** multidrug-resistant TB; defined as M. tuberculosis resistant to at least isoniazid and rifampicin.
- **XDR-TB:** extensively drug-resistant TB; MDR-TB with additional resistance to any fluoroquinolone and at least one injectable second-line drug.
- **BPaL regimen:** a 6 to 9-month regimen for MDR-TB comprising bedaquiline, pretomanid, and linezolid; increasingly recommended as a shorter alternative to 18 to 24-month regimens.
- **IRIS:** immune reconstitution inflammatory syndrome; paradoxical worsening of TB symptoms after starting ART in TB-HIV patients from restoration of immune response.
- **Aspergilloma:** a fungal ball of Aspergillus species colonising a pre-existing pulmonary cavity (typically from previous TB); causes haemoptysis; appears as a solid mass with an air crescent on CXR or CT.
- **Miliary TB:** haematogenous dissemination of M. tuberculosis producing millet-seed-sized granulomas throughout all organs; characterised by high fever and bilateral micronodular shadowing on CXR.
- **Rasmussen's aneurysm:** a pseudoaneurysm of the pulmonary artery adjacent to a TB cavity; erosion causes massive haemoptysis.

- **DOT:** directly observed therapy; a strategy for TB treatment where each dose is taken under observation by a healthcare worker or trained community member to ensure adherence and prevent resistance.
- **Cold agglutinins:** IgM antibodies against red blood cell surface antigens produced in Mycoplasma pneumoniae infection; cause haemolytic anaemia; agglutinate red cells at low temperatures.

Concept Check Questions [Series 3]

Objective Questions

1. The most common causative organism of CAP is S. _____, responsible for 30 to 40 percent of cases requiring hospitalisation. (Linked to SLO 3.1)
2. A CURB-65 score of 3 to 5 indicates high severity CAP requiring _____ admission and IV antibiotics. (Linked to SLO 3.2)
3. Klebsiella pneumonia classically produces thick currant-jelly sputum and upper lobe consolidation with a _____ fissure on CXR. (Linked to SLO 3.3)
4. Legionella pneumonia is diagnosed by the urinary _____ antigen test; features include confusion, diarrhoea, and hyponatraemia. (Linked to SLO 3.3)
5. Standard TB treatment is 2HRZE/4HR; isoniazid causes peripheral neuropathy prevented by _____ (vitamin B6). (Linked to SLO 3.4)
6. MDR-TB is resistance to isoniazid and rifampicin; in TB-HIV co-infection, rifampicin induces CYP450 and reduces antiretroviral levels; _____ is the preferred NNRTI. (Linked to SLO 3.5)

Short-Answer Questions

7. Describe the clinical and radiological features of staphylococcal pneumonia. (Linked to SLO 3.3)
8. Describe the clinical features of pulmonary tuberculosis. (Linked to SLO 3.4)
9. Describe the complications of pulmonary tuberculosis. (Linked to SLO 3.5)

Long-Answer Questions

10. Describe the diagnosis and standard treatment of pulmonary tuberculosis, including drug side effects. (Linked to SLO 3.4)
11. Describe MDR-TB, TB-HIV co-infection, and IRIS. (Linked to SLO 3.5)

Answers to Concept Check Questions [Series 3]

Objective Questions

1. Pneumoniae.
2. Hospital.
3. Bulging.
4. Legionella.
5. Pyridoxine.
6. Efavirenz.

Short-Answer Questions

7. Staphylococcal pneumonia: occurs post-influenza, in IV drug users, and from haematogenous seeding. Features: severe, rapidly progressive; high fever; purulent sputum. CXR: bilateral patchy consolidation with rapid cavitation and pneumatocele formation (thin-walled cavities). Treatment: IV flucloxacillin 14 to 21 days; vancomycin or teicoplanin for MRSA.
8. Cough above 2 weeks (initially dry, then productive; blood-stained in advanced disease), low-grade afternoon fever, drenching night sweats, significant weight loss, fatigue, anorexia, haemoptysis (from cavity erosion). Examination: signs of consolidation or cavitation at the lung apex (upper lobe predilection from high V/Q ratio and high oxygen tension). CXR: upper lobe infiltrates, cavitation, hilar lymphadenopathy.
9. Haemoptysis (Rasmussen's aneurysm: pseudoaneurysm of pulmonary artery adjacent to a cavity; can be massive and fatal). Pneumothorax (subpleural cavity rupture). Pleural effusion (TB pleurisy: lymphocytic exudate). Empyema necessitans (TB empyema eroding through chest wall). Aspergilloma (Aspergillus colonising a TB cavity; haemoptysis; air crescent on CXR). Bronchiectasis (airway destruction). Miliary TB (haematogenous dissemination; bilateral micronodular CXR).

Long-Answer Questions

10. **Basic response:** Diagnosis: three early morning sputum samples before antibiotics. GeneXpert MTB/RIF: first-line WHO-recommended test; PCR-based; detects M. tuberculosis and rifampicin resistance in under 2 hours; sensitivity approximately 88

percent for smear-negative TB. AFB smear microscopy (Ziehl-Neelsen): rapid and cheap; positive in 50 to 70 percent of pulmonary TB; defines smear-positive infectious cases. Mycobacterial culture: gold standard; Lowenstein-Jensen solid medium (4 to 8 weeks) or MGIT liquid culture (10 to 14 days); essential for drug susceptibility testing. CXR: upper lobe infiltrates, cavitation, hilar lymphadenopathy, calcified Ghon complex. Tuberculin skin test and IGRA (interferon-gamma release assay) detect latent TB but do not diagnose active disease. Treatment (2HRZE/4HR): Intensive phase 2 months: isoniazid (H: peripheral neuropathy prevent with pyridoxine; hepatotoxicity), rifampicin (R: orange body fluids; CYP450 inducer; reduces OCP and ART efficacy; hepatotoxicity), pyrazinamide (Z: hyperuricaemia and gout; hepatotoxicity), ethambutol (E: optic neuritis monitor visual acuity monthly). Continuation phase 4 months: isoniazid and rifampicin. DOT (directly observed therapy) recommended. All four drugs are hepatotoxic; stop all if transaminases exceed 5 times normal.

Value-added response: The 2HRZE/4HR regimen represents one of the most elegant examples of combination chemotherapy: each drug targets a different bacterial population (isoniazid kills rapidly dividing bacilli, rifampicin kills both dividing and intermittently dividing bacilli, pyrazinamide kills bacilli in acidic caseous foci, and ethambutol prevents resistance acquisition), so that the combination achieves complete sterilisation that no single drug could achieve alone, while making the emergence of acquired resistance practically impossible when all four drugs are taken correctly.

11. **Basic response:** MDR-TB: M. tuberculosis resistant to at least isoniazid and rifampicin (the two most potent first-line drugs); arises from inadequate treatment or poor adherence; detected by GeneXpert (rifampicin resistance as proxy) or drug susceptibility testing. XDR-TB: MDR-TB plus resistance to any fluoroquinolone and at least one injectable second-line drug. MDR-TB treatment: minimum 18 to 24 months of second-line drugs; newer BPaL regimen (bedaquiline, pretomanid, linezolid) achieves cure in 6 to 9 months with fewer drugs; bedaquiline causes QTc prolongation (monitor ECG); linezolid causes myelosuppression and neuropathy; specialist supervision required. TB-HIV co-infection: HIV increases risk of TB reactivation 20- to 30-fold; smear-negative and extrapulmonary TB more common; start anti-TB treatment first; introduce ART 2 to 8 weeks later (2 weeks for CD4 below 50); rifampicin induces CYP450 reducing most

NNRTI and PI levels; efavirenz-based ART preferred; integrase inhibitor-based regimens (dolutegravir) are an alternative. IRIS: paradoxical worsening of TB symptoms (enlarging lymph nodes, worsening lung infiltrates, fever) after starting ART from restored immune response; occurs in 15 to 40 percent; managed by continuing both anti-TB treatment and ART; corticosteroids for severe IRIS.

Value-added response: IRIS exemplifies a fundamental paradox of medicine: the very treatment that saves the patient's life (ART, restoring the immune system) temporarily makes the disease appear worse; clinicians who do not understand IRIS may incorrectly diagnose treatment failure and change antibiotics when they should instead reassure the patient and continue both treatments, since IRIS is self-limiting in the vast majority of cases.

Answers to Pilot Questions [Series 3]

Part 3.1

1. S. pneumoniae: most common (30 to 40 percent of hospitalised CAP); Gram-positive diplococcus; lobar consolidation; rust-coloured sputum; bacteraemia common; diagnose with urinary pneumococcal antigen and blood culture. H. influenzae: second most common; particularly in COPD and smokers; lower lobe predilection. S. aureus: severe; post-influenza; cavitating; bilateral patchy consolidation. Atypicals: Mycoplasma (young adults), Chlamydophila, Legionella (severe; from water sources).
2. Congestion (days 1 to 2): initial inflammatory response; vascular engorgement; alveolar oedema; macrophage recruitment; lung appears dark red and heavy. Red hepatisation (days 3 to 4): alveoli filled with red blood cells, neutrophils, and fibrin; lung solidifies resembling liver in consistency; air bronchogram visible on CXR. Grey hepatisation (days 5 to 8): red cell lysis and degeneration; neutrophils replaced by macrophages; fibrin network; lung becomes grey. Resolution (days 8 to 10): macrophages phagocytose debris; fibrin dissolved; normal architecture restored; CXR clears over 4 to 6 weeks.
3. CXR (lobar or segmental consolidation with air bronchogram; bilateral infiltrates in atypical or staphylococcal; cavity in S. aureus or Klebsiella). Sputum Gram stain and

culture (before antibiotics; Gram-positive diplococci: pneumococcal; Gram-negative: H. influenzae or Klebsiella). Blood cultures (before antibiotics; positive in 10 to 15 percent of hospitalised CAP; mandatory in severe CAP). FBC (leucocytosis with neutrophilia; leucopaenia in severe sepsis). CRP (elevated; correlates with severity). Urinary antigens (pneumococcal urinary antigen: sensitivity 70 to 80 percent; Legionella urinary antigen: sensitivity 70 to 90 percent; test in moderate to severe CAP). ABG (PaO2: assess oxygenation and respiratory failure).

4. CURB-65: 1 point each for Confusion (new onset), Urea above 7 mmol/L (above 19 mg/dL), Respiratory rate above 30 per minute, Blood pressure systolic below 90 or diastolic below 60 mmHg, age 65 or above. Score 0 to 1: low severity; treat at home; oral amoxicillin 500 mg three times daily for 5 days. Score 2: moderate severity; consider hospital admission; oral amoxicillin plus clarithromycin. Score 3 to 5: high severity; hospital admission; consider ICU referral; IV co-amoxiclav 1.2 g three times daily plus IV or oral clarithromycin 500 mg twice daily; or IV ceftriaxone 2 g once daily plus clarithromycin.
5. Oxygen: target SpO2 94 to 98 percent in most patients (88 to 92 percent in COPD to avoid suppressing hypoxic drive); use high-flow oxygen via non-rebreather mask in severe hypoxaemia; consider NIV in type 2 respiratory failure. IV fluids: resuscitate if haemodynamically compromised; maintain adequate renal perfusion. Analgesia: pleuritic chest pain limits deep breathing and cough; use paracetamol or NSAIDs. Physiotherapy: aided sputum clearance in severe or aspiration pneumonia. VTE prophylaxis: subcutaneous low-molecular-weight heparin during hospital admission. Treat complications: empyema requires chest tube drainage; septic shock requires vasopressors.

Part 3.2

1. Staphylococcal pneumonia: caused by S. aureus; occurs after influenza (influenza damages bronchial epithelium predisposing to secondary infection), in IV drug users (haematogenous seeding from right-sided endocarditis), and in immunocompromised patients. Clinical: severe; high swinging fever; purulent sputum; rapidly progressive; may develop septicaemia. CXR: bilateral patchy consolidation with rapid (within hours to days) cavitation forming thin-walled cavities (pneumatoceles), pyopneumothorax, or lung

abscess. Treatment: IV flucloxacillin 1 to 2 g four times daily for 14 to 21 days; vancomycin or teicoplanin for MRSA.

2. Klebsiella pneumonia: caused by Klebsiella pneumoniae (Gram-negative rod; mucoid capsule). Affects alcoholics, diabetics, and debilitated patients. Clinical: abrupt onset; high fever; rigors; thick gelatinous sputum (currant-jelly colour from haemorrhagic necrosis and mucus); dyspnoea; severe toxicity. CXR: upper lobe (or right-sided) consolidation with a bulging fissure the hallmark finding: the voluminous inflammatory exudate causes lobar expansion, pushing the fissure downward or forward; cavitation may develop. Treatment: third-generation cephalosporin (ceftriaxone or cefotaxime) or carbapenem (imipenem or meropenem) for resistant strains.
3. Mycoplasma pneumoniae: gradual onset over 1 to 2 weeks; prodrome of headache, malaise, sore throat; dry or minimally productive cough; low-grade fever; CXR: bilateral patchy lower lobe infiltrates. Extrapulmonary complications: haemolytic anaemia from cold agglutinins (IgM antibodies causing complement-mediated haemolysis at low temperatures; positive cold agglutinin test in above 50 percent), erythema multiforme (target lesions on skin and mucous membranes), Stevens-Johnson syndrome, meningoencephalitis, pericarditis, and myocarditis.
4. Source: contaminated warm water in cooling towers, air conditioning systems, hospital hot water systems, and whirlpool spas; not transmitted person to person. Clinical: severe CAP; high fever; dry cough; prominent extrapulmonary features: confusion (encephalopathy), diarrhoea, abdominal pain; laboratory: hyponatraemia (SIADH), markedly elevated CRP and LDH, lymphopaenia; CXR: bilateral patchy consolidation; can progress rapidly. Diagnosis: urinary Legionella antigen (sensitivity 70 to 90 percent; detects Legionella pneumophila serogroup 1 only; available within hours). Treatment: azithromycin 500 mg daily or levofloxacin 500 mg twice daily for 10 to 14 days; rifampicin may be added in severe cases.
5. HAP: pneumonia developing more than 48 hours after hospital admission; predominantly caused by Gram-negative organisms (Pseudomonas aeruginosa, Klebsiella, Escherichia coli, Acinetobacter baumannii) and MRSA; associated with mechanical ventilation (VAP), invasive procedures, and immunosuppression; carries mortality above 30 percent. Management: obtain blood cultures and respiratory specimens before antibiotics; choose

antibiotics based on local resistance patterns; Gram-negative cover: piperacillin-tazobactam 4.5 g three times daily IV, or ceftazidime plus an aminoglycoside; MRSA cover: vancomycin 1 g twice daily IV (dose adjusted by trough levels) or linezolid 600 mg twice daily; review and de-escalate after 48 to 72 hours based on culture results.

Part 3.3

1. Transmission: airborne droplet nuclei (1 to 5 microns diameter) produced when an infectious person coughs, sneezes, or speaks; inhaled droplet nuclei reach the alveoli. Primary infection: M. tuberculosis phagocytosed by alveolar macrophages; resists killing using various mechanisms (inhibiting phagosome-lysosome fusion, producing superoxide dismutase); replicates within macrophages; spreads to regional hilar lymph nodes. Ghon complex: subpleural granuloma (usually mid or lower zone) plus reactive hilar lymphadenopathy. Granuloma formation (T-cell mediated): macrophages, T lymphocytes, and Langhans giant cells form the granuloma; central caseous necrosis may develop. In immunocompetent hosts, granuloma contains infection (latent TB); in immunocompromised hosts (HIV, malnutrition, diabetes), progressive primary TB or reactivation occurs.
2. Cough: above 2 weeks duration; initially dry, becoming productive; rust-coloured or blood-stained sputum in advanced disease. Constitutional symptoms: low-grade fever (characteristically in the afternoon), drenching night sweats, significant unintentional weight loss, fatigue, anorexia. Haemoptysis: from direct cavity erosion into a blood vessel or from Rasmussen's aneurysm. Dyspnoea: from extensive parenchymal involvement, pleural effusion, or pneumothorax. Examination: signs of consolidation or cavitation at the lung apex (upper lobe predilection due to high V/Q ratio and high oxygen tension favouring mycobacterial growth): dullness, bronchial breathing, amphoric breathing over a large cavity, post-tussive crepitations.
3. GeneXpert MTB/RIF: WHO-recommended first-line test; PCR-based; detects M. tuberculosis and rifampicin resistance (proxy for MDR-TB) in under 2 hours; sensitivity approximately 88 percent for smear-negative TB; sensitivity approximately 98 percent for smear-positive TB. AFB smear microscopy: rapid and cheap; Ziehl-Neelsen stain; positive in 50 to 70 percent of pulmonary TB; three early morning samples required; defines smear-positive (most infectious) cases. Mycobacterial culture: gold standard;

most sensitive; Lowenstein-Jensen solid medium (4 to 8 weeks) or MGIT liquid culture (10 to 14 days); essential for drug susceptibility testing. CXR: upper lobe infiltrates, cavitation, hilar lymphadenopathy, fibrosis, calcified Ghon complex.

4. Intensive phase (2 months): four drugs given daily. Isoniazid (H): inhibits mycolic acid synthesis; kills rapidly dividing bacilli; side effects: peripheral neuropathy (prevent with pyridoxine 10 mg daily), hepatotoxicity, rash. Rifampicin (R): inhibits RNA polymerase; kills dividing and dormant bacilli; side effects: orange discolouration of urine, sweat, and tears (warn patient); hepatotoxicity; CYP450 induction (reduces OCP, warfarin, ART efficacy). Pyrazinamide (Z): kills bacilli in acidic caseous lesions; side effects: hyperuricaemia and gout, hepatotoxicity. Ethambutol (E): inhibits arabinosyl transferase; prevents resistance to isoniazid and rifampicin; side effects: optic neuritis (monitor visual acuity and colour vision before and monthly). Continuation phase (4 months): isoniazid and rifampicin only.
5. DOT (directly observed therapy): each dose of anti-TB treatment is administered in the presence of a trained healthcare worker or community volunteer who observes the patient swallowing the tablets. Rationale: TB treatment requires 6 months of uninterrupted therapy; poor adherence (taking only some doses or stopping early) selectively kills drug-susceptible bacteria while allowing drug-resistant mutants to survive and multiply; this is the mechanism by which MDR-TB arises; DOT ensures that every dose is taken, preventing acquired resistance, ensuring treatment completion, and monitoring side effects; it is particularly important in the context of Nigeria's high TB burden and the risk of generating drug-resistant strains that are far more difficult and expensive to treat.

Part 3.4

1. MDR-TB: M. tuberculosis resistant to at least isoniazid and rifampicin (defined by WHO); arises from inadequate treatment (incorrect regimen, poor quality drugs, drug shortages, or poor adherence creating selection pressure); detected by GeneXpert (rifampicin resistance as surrogate) or phenotypic drug susceptibility testing. XDR-TB: MDR-TB with additional resistance to any fluoroquinolone and at least one of the group A drugs (bedaquiline, linezolid). MDR-TB treatment principles: use at least four likely effective drugs; include bedaquiline plus linezolid plus clofazimine plus pyrazinamide if susceptible; newer BPaL regimen (bedaquiline, pretomanid, linezolid) for 6 to 9 months

has shown cure rates above 90 percent in XDR-TB; minimum duration 18 to 24 months for conventional regimens; specialist supervision required; high rates of adverse effects; regular monitoring of ECG (bedaquiline: QTc), FBC and nerves (linezolid: myelosuppression and neuropathy).

2. HIV causes progressive loss of CD4 T-lymphocytes (the cells responsible for macrophage activation and granuloma formation); without effective cellular immunity, mycobacteria are not contained within granulomas and can disseminate freely. Consequences for TB presentation: smear-negative TB more common (granulomas do not develop properly so less cavitation and fewer bacilli in sputum); extrapulmonary TB more frequent (lymphadenitis, meningitis, miliary TB); CXR may show atypical features including lower lobe involvement, diffuse bilateral infiltrates, hilar adenopathy without apical changes, or near-normal CXR (particularly at low CD4 counts); skin testing (TST) and IGRA may be falsely negative due to anergy.
3. Start anti-TB treatment first (regardless of CD4 count) to reduce TB-related mortality. Introduce ART 2 to 8 weeks after starting anti-TB treatment (earlier ART within 2 weeks for patients with CD4 below 50 cells/microlitre, who have highest mortality risk). Drug interactions: rifampicin is a potent CYP450 inducer, markedly reducing plasma levels of most NNRTIs and PIs; efavirenz-based ART is the preferred regimen (its CYP450 metabolism is less affected; use standard dose 600 mg daily); dolutegravir-based ART is increasingly used (requires dose adjustment to 50 mg twice daily due to rifampicin induction). Monitor for IRIS: paradoxical worsening of TB symptoms 2 to 8 weeks after ART initiation; manage by continuing both treatments; use corticosteroids (prednisolone 1 mg/kg per day tapered over 4 weeks) for life-threatening IRIS.
4. Haemoptysis: caused by cavity erosion into a pulmonary artery (Rasmussen's aneurysm) or smaller bronchial vessels; ranges from blood-streaked sputum to massive haemoptysis; massive haemoptysis managed with bronchial artery embolisation or surgical resection. Pneumothorax: rupture of a subpleural cavity into the pleural space; TB pneumothorax is often associated with bronchopleural fistula; managed with chest tube drainage. Pleural effusion: TB pleurisy (primary: lymphocytic exudate with high protein, low glucose, elevated ADA; or secondary to direct pleural extension). Empyema necessitans: TB empyema extending through the chest wall to form a subcutaneous abscess.

Aspergilloma: Aspergillus fumigatus colonises a pre-existing TB cavity; causes haemoptysis; visible as a solid mass with an air crescent (Monad sign) on CXR or CT; treated with antifungal (itraconazole or voriconazole) or bronchial artery embolisation. Bronchiectasis: chronic airway destruction and dilatation from repeated TB-related inflammation.

5. Miliary TB: haematogenous dissemination of M. tuberculosis throughout the body; occurs when the immune response is overwhelmed (HIV, malnutrition, extremes of age, immunosuppressive therapy). Clinical: high swinging fever, night sweats, weight loss, hepatosplenomegaly, generalised lymphadenopathy, choroidal tubercles on fundoscopy (pathognomonic: multiple white lesions at the posterior pole); may develop meningitis, pericarditis, or adrenal failure. CXR: bilateral, uniformly distributed, small (1 to 3 mm) nodular shadows throughout both lung fields (millet-seed appearance; hence miliary from the Latin milium, millet seed). CT chest: more sensitive; shows nodules in a random distribution relative to the secondary lobule. Diagnosis confirmed by sputum, urine, blood, or bone marrow culture; liver or bone marrow biopsy may show granulomas. Treatment: standard 2HRZE/4HR; corticosteroids added for TB meningitis and pericarditis.

References and Attributions [Series 3]

Textual References

- World Health Organization. (2022). WHO Consolidated Guidelines on Tuberculosis. Module 4: Treatment. Geneva: WHO.
- British Infection Association. (2009). British Thoracic Society Guidelines for the Management of Community-Acquired Pneumonia in Adults. Thorax, 64(Suppl 3), iii1 to iii55.
- Kumar, P. and Clark, M. (2017). Kumar and Clark's Clinical Medicine (9th ed.). Edinburgh: Elsevier.
- Nigeria Centre for Disease Control. (2021). National TB and Leprosy Control Programme Annual Report 2021. Abuja: NCDC.

Figures, Plates, Tables, and Boxes

- Figure 3.1: CAP aetiology, diagnosis, and CURB-65 management. AI prompt: "Two-panel diagram: left two-section table (aetiology and clinical features of CAP; investigations); right CURB-65 score and three-tier management (score 0 to 1: home amoxicillin; score 2: consider hospital; score 3 to 5: hospital IV antibiotics); clean professional infographic." OER search: "community-acquired pneumonia CURB-65 severity score management antibiotic diagram".
- Figure 3.2: Special pneumonias. AI prompt: "Two-panel table: staphylococcal and Klebsiella pneumonias (left); atypical, aspiration, and HAP (right); clean infographic." OER search: "special pneumonia staphylococcal Klebsiella atypical aspiration hospital-acquired management diagram".
- Figure 3.3: Pulmonary TB diagnosis and treatment. AI prompt: "Two-panel diagram: diagnosis (GeneXpert, AFB smear, culture, CXR) and 2HRZE/4HR treatment with side effects; clean infographic." OER search: "pulmonary TB diagnosis GeneXpert AFB smear treatment 2HRZE 4HR side effects diagram".
- Figure 3.4: Drug-resistant TB, TB-HIV, and complications. AI prompt: "Two-panel diagram: MDR-TB and XDR-TB treatment (BPaL); TB-HIV co-infection and IRIS; pulmonary complications and miliary TB; clean infographic." OER search: "MDR-TB XDR-TB treatment TB HIV co-infection IRIS complications miliary TB diagram".

Series 4: Airway Diseases and Complications

- **Part 4.1: Asthma**
    - Sub Part 4.1.1: Epidemiology, pathogenesis, and classification of asthma
    - Sub Part 4.1.2: Clinical features, diagnosis, and monitoring of asthma
    - Sub Part 4.1.3: Management of stable and acute severe asthma
- **Part 4.2: Chronic Obstructive Pulmonary Disease**
    - Sub Part 4.2.1: Aetiology and pathophysiology of COPD
    - Sub Part 4.2.2: Clinical features and diagnosis of COPD
    - Sub Part 4.2.3: Management of stable COPD and COPD exacerbations
- **Part 4.3: Pleural Effusion and Pneumothorax**
    - Sub Part 4.3.1: Pleural effusion: causes and management
    - Sub Part 4.3.2: Pneumothorax
    - Sub Part 4.3.3: Empyema and pleural malignancy
- **Part 4.4: Pulmonary Fibrosis and Pulmonary Embolism**
    - Sub Part 4.4.1: Idiopathic pulmonary fibrosis
    - Sub Part 4.4.2: Pulmonary embolism
    - Sub Part 4.4.3: Pulmonary hypertension

Introduction

Airway diseases, asthma and chronic obstructive pulmonary disease, are among the most prevalent chronic respiratory conditions in Nigeria and globally. Asthma affects children and

young adults and is largely reversible; COPD affects older adults with smoking or biomass fuel exposure and is progressive. Both cause significant disability and avoidable death when poorly managed. The complications of chest disease, including pleural effusion, pneumothorax, empyema, pulmonary fibrosis, and pulmonary embolism, are encountered across all clinical settings.

This Series covers asthma in systematic detail from pathogenesis through management, including acute severe asthma. COPD is examined with attention to the Nigerian context where biomass fuel exposure is a major aetiological factor in women. The common pleural complications are then covered, followed by idiopathic pulmonary fibrosis, pulmonary embolism, and pulmonary hypertension.

Together, these conditions form the core of clinical respiratory practice and their management is directly applicable to encounters in Nigeria.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 4.1** describe the pathogenesis, clinical features, diagnosis, and management of asthma including acute severe asthma (Linked to Part 4.1),
- **SLO 4.2** describe the aetiology, pathophysiology, clinical features, diagnosis, and management of COPD and COPD exacerbations (Linked to Part 4.2),
- **SLO 4.3** describe the causes, investigation, and management of pleural effusion, pneumothorax, and empyema (Linked to Part 4.3), and
- **SLO 4.4** describe the clinical features, diagnosis, and management of idiopathic pulmonary fibrosis, pulmonary embolism, and pulmonary hypertension (Linked to Part 4.4).

4.1 Asthma

4.1.1 Epidemiology, pathogenesis, and classification of asthma

Asthma is a chronic inflammatory airway disease characterised by variable, reversible airflow obstruction. The underlying inflammation is predominantly **eosinophilic (type 2)** in atopic individuals, driven by IgE-mediated mast cell degranulation and Th2 cytokines (IL-4, IL-5, IL-13); non-eosinophilic (neutrophilic or paucigranulocytic) phenotypes also exist.

Asthma is classified by control (well-controlled: no daytime symptoms, no night waking, no rescue inhaler use above twice per week, no activity limitation; partly controlled: 1 to 2 criteria not met; uncontrolled: 3 to 4 not met). Common triggers include allergens (house dust mite, pollen, animal dander), exercise, cold air, respiratory infections, NSAIDs, and occupational sensitisers. Atopy is the strongest risk factor.

Fill in the blank: Asthma is characterised by variable reversible airflow obstruction; eosinophilic inflammation is driven by Th2 cytokines; triggers include allergens, NSAIDs, and exercise; _____ is the strongest risk factor.

4.1.2 Clinical features, diagnosis, and monitoring of asthma

Clinical features include episodic wheeze (polyphonic, expiratory), dyspnoea with nocturnal or early-morning diurnal variation, chest tightness, and dry cough (cough variant asthma). Between episodes, examination may be normal. During an attack: tachypnoea, accessory muscle use, polyphonic expiratory wheeze, prolonged expiration, and hyperinflation.

Diagnosis is supported by spirometry showing reversible obstruction (FEV1 improvement above 12 percent and above 200 mL after bronchodilator), PEFR diary showing diurnal variability above 20 percent, and elevated **FeNO above 40 ppb** (eosinophilic airway inflammation). Bronchial challenge testing (methacholine) is used when spirometry is normal but asthma is still suspected.

Fill in the blank: Asthma diagnosis: reversible obstruction (FEV1 improvement above 12 percent and 200 mL); PEFR diurnal variability above 20 percent; FeNO above 40 ppb; bronchial _____ testing when spirometry is normal.

4.1.3 Management of stable and acute severe asthma

Stable asthma management follows a GINA stepwise approach: Step 1 (SABA as reliever on demand); Step 2 (add low-dose ICS); Step 3 (add LABA to ICS); Step 4 (increase ICS dose, add montelukast); Step 5 (oral corticosteroids or biologics: anti-IgE omalizumab; anti-IL-5 mepolizumab for severe eosinophilic asthma).

Acute severe asthma features: PEFR 33 to 50 percent, SpO2 below 92 percent, RR above 25, HR above 110, inability to complete sentences. Life-threatening features: PEFR below 33 percent, silent chest, cyanosis, bradycardia, confusion. Management: high-flow oxygen (SpO2 94 to 98 percent), nebulised salbutamol 5 mg, ipratropium 500 micrograms, IV magnesium sulphate 1.2 to 2 g over 20 minutes, systemic corticosteroids. ICU and intubation for life-threatening features.

Fill in the blank: Acute severe asthma: PEFR 33 to 50 percent, SpO2 below 92 percent; life-threatening: PEFR below 33 percent, silent chest, cyanosis; manage with oxygen, nebulised salbutamol, ipratropium, IV _____ sulphate, and corticosteroids.

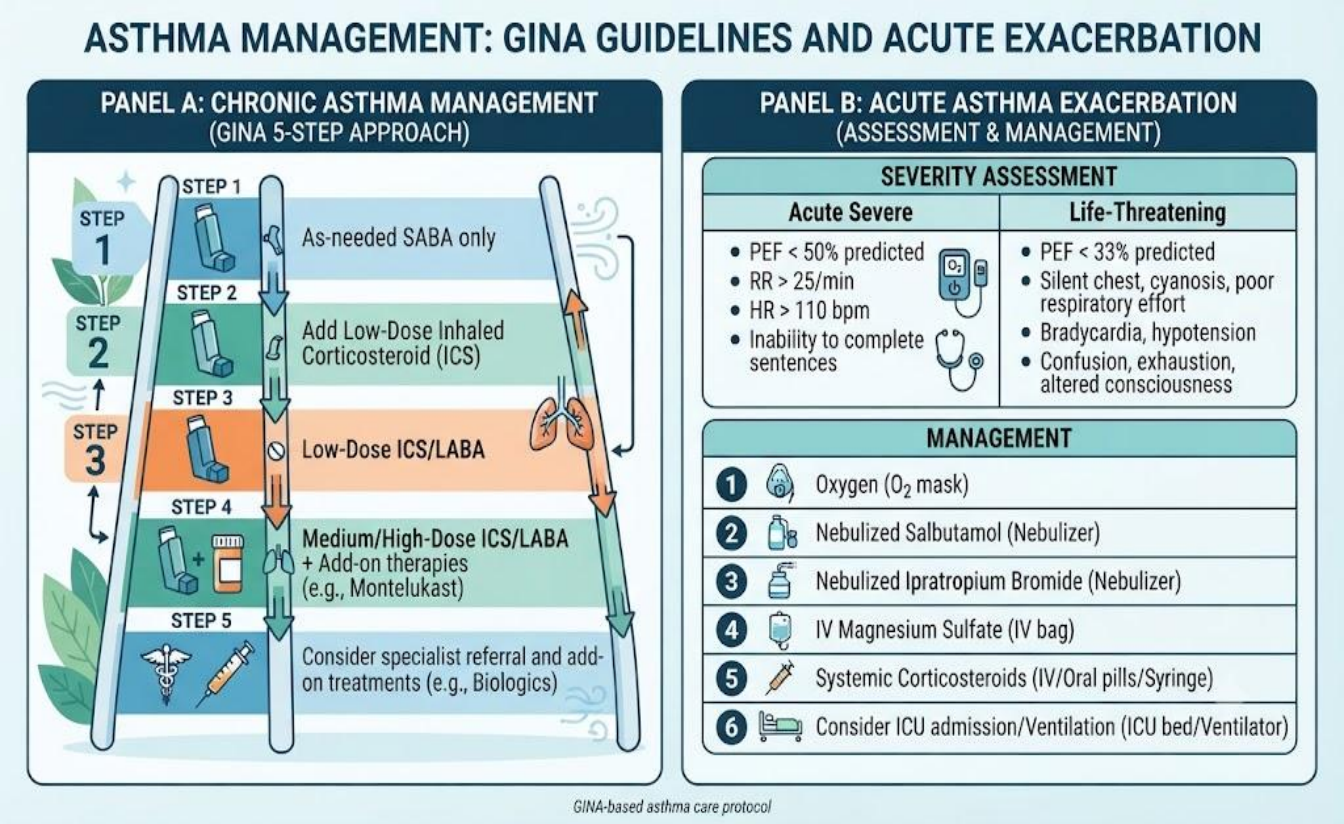


Figure 4.1: Asthma stepwise management and acute severe asthma assessment and treatment

Pilot questions 4.1

1. Describe the pathogenesis of eosinophilic asthma. (Linked to SLO 4.1)
2. Describe the clinical features of asthma. (Linked to SLO 4.1)
3. Describe the diagnostic tests used in asthma. (Linked to SLO 4.1)
4. Describe the GINA stepwise approach to stable asthma management. (Linked to SLO 4.1)
5. Describe the features of life-threatening asthma and its immediate management. (Linked to SLO 4.1)

4.2 Chronic Obstructive Pulmonary Disease

4.2.1 Aetiology and pathophysiology of COPD

COPD is characterised by persistent, not fully reversible, airflow limitation from chronic inflammation in response to noxious particles or gases. The leading cause globally is cigarette

smoking. In Nigeria, **indoor air pollution from biomass fuel combustion** (cooking fires, kerosene stoves) is a major cause, particularly in women who cook indoors. Occupational dust, outdoor air pollution, and recurrent childhood respiratory infections are additional causes.

Pathophysiology involves neutrophilic airway inflammation, mucus hypersecretion and goblet cell hyperplasia (chronic bronchitis), **emphysema** (neutrophil elastase destroys alveolar walls, reducing gas exchange surface area and elastic recoil), and air trapping (dynamic hyperinflation from loss of elastic recoil and small airway collapse, increasing RV and TLC).

Fill in the blank: COPD is caused by smoking globally and biomass fuel combustion in Nigerian women; pathophysiology includes neutrophilic inflammation, mucus hypersecretion, emphysema (destruction of alveolar _____), and air trapping.

4.2.2 Clinical features and diagnosis of COPD

Clinical features include progressive dyspnoea (the hallmark symptom; MRC Grade 2 or above), chronic productive cough (chronic bronchitis: cough for more than 3 months in 2 consecutive years), and wheeze. In advanced COPD: barrel chest, pursed-lip breathing, accessory muscle use, hyperinflation on CXR, and cor pulmonale (right heart failure from pulmonary hypertension: elevated JVP, peripheral oedema, hepatomegaly).

Diagnosis requires post-bronchodilator spirometry showing **FEV1/FVC ratio below 0.70** (fixed obstruction). GOLD staging classifies severity by FEV1: GOLD 1 (above 80 percent predicted: mild), GOLD 2 (50 to 79 percent: moderate), GOLD 3 (30 to 49 percent: severe), GOLD 4 (below 30 percent: very severe). CXR shows hyperinflation, flattened diaphragms, increased retrosternal airspace, and bullae in emphysema.

Fill in the blank: COPD diagnosis: post-bronchodilator FEV1/FVC below 0.70; GOLD 1: FEV1 above 80 percent; GOLD 4: FEV1 below 30 percent (very severe); CXR shows hyperinflation, flattened diaphragms, and _____ in emphysema.

4.2.3 Management of stable COPD and COPD exacerbations

Stable COPD management (GOLD guidelines): smoking cessation and removal of biomass fuel exposure (the most important intervention). Pharmacotherapy: SABA or SAMA (ipratropium) as reliever; LABA or LAMA (tiotropium) as maintenance; ICS added for FEV1 below 60 percent with frequent exacerbations; triple therapy (LABA/LAMA/ICS) for severe symptomatic COPD. Pulmonary rehabilitation improves exercise capacity and quality of life.

COPD exacerbation management: controlled low-flow oxygen (target SpO2 88 to 92 percent), nebulised bronchodilators (salbutamol plus ipratropium), systemic corticosteroids (prednisolone 30 to 40 mg oral for 5 days), antibiotics (amoxicillin or doxycycline for increased sputum purulence), and **NIV (non-invasive ventilation: BiPAP)** for type 2 respiratory failure (pH below 7.35). NIV reduces intubation rates and mortality.

Fill in the blank: Stable COPD: smoking cessation most important; LABA or LAMA maintenance; triple therapy for severe disease; exacerbation: controlled O2 (88 to 92 percent), bronchodilators, prednisolone 5 days, antibiotics, _____ for type 2 respiratory failure.

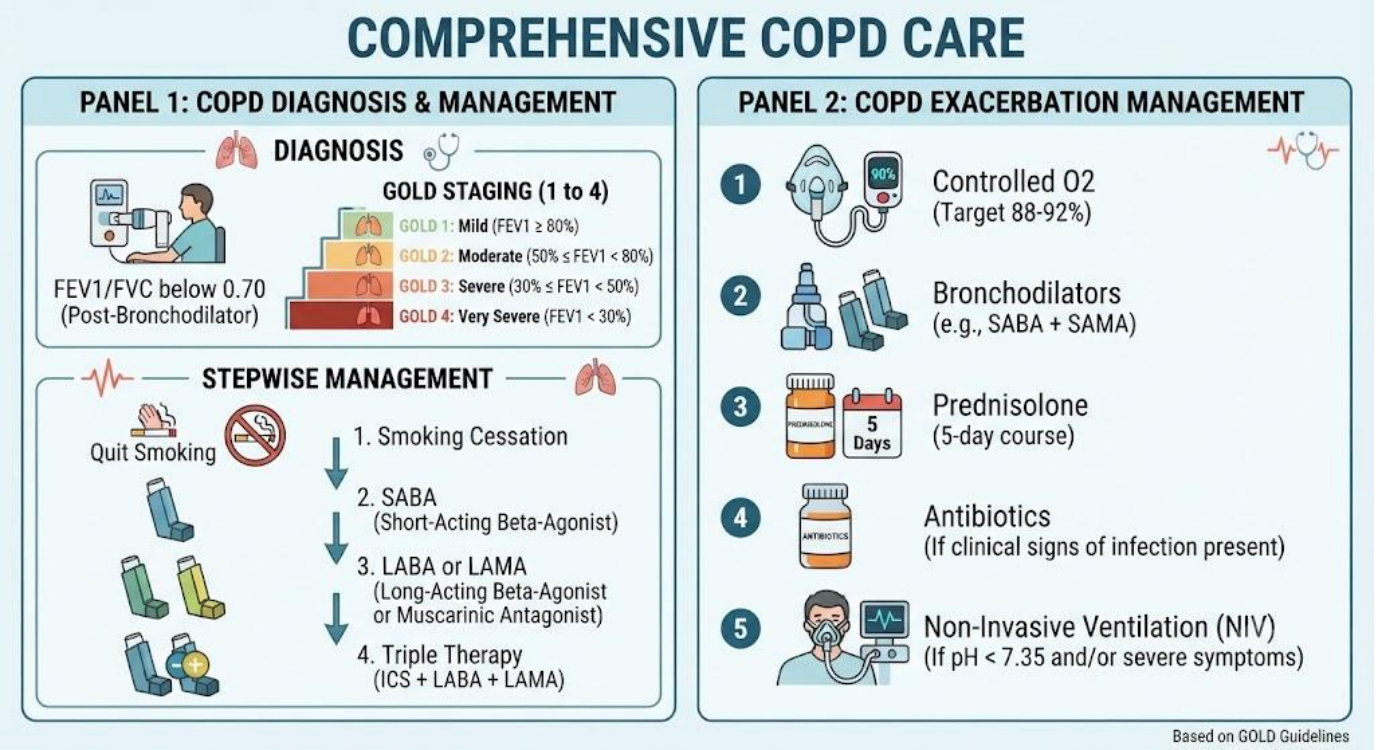


Figure 4.2: COPD -- GOLD staging, stepwise stable management, and exacerbation treatment

Pilot questions 4.2

1. Describe the role of biomass fuel in COPD in Nigeria. (Linked to SLO 4.2)
2. Describe the clinical features of COPD. (Linked to SLO 4.2)
3. Describe the spirometric criteria for diagnosing COPD and the GOLD staging system. (Linked to SLO 4.2)
4. Describe the stepwise pharmacological management of stable COPD. (Linked to SLO 4.2)
5. Describe the management of a COPD exacerbation in hospital. (Linked to SLO 4.2)

4.3 Pleural Effusion and Pneumothorax

4.3.1 Pleural effusion: causes and management

Pleural effusion is an abnormal accumulation of fluid in the pleural space. It is classified as **transudate** (protein below 25 g/L; caused by altered hydrostatic or oncotic pressure: heart

failure is the most common cause globally; also cirrhosis, nephrotic syndrome, hypoalbuminaemia) or **exudate** (protein above 30 g/L; increased capillary permeability: pneumonia, TB, malignancy, PE, connective tissue disease). Light's criteria distinguish borderline cases (protein 25 to 35 g/L).

Management depends on the cause. Transudates from heart failure or cirrhosis are managed by treating the underlying cause. Diagnostic thoracocentesis (20 to 50 mL) should be performed for any unexplained effusion. Therapeutic aspiration (up to 1500 mL per session) or intercostal chest tube drainage is used for large or exudative effusions. **Pleurodesis** (talc instillation) is used for recurrent malignant effusion.

Fill in the blank: Transudate: protein below 25 g/L; heart failure most common; exudate: protein above 30 g/L; pneumonia, TB, malignancy; therapeutic aspiration up to 1500 mL per session; _____ (talc) is used for recurrent malignant effusion.

4.3.2 Pneumothorax

Primary spontaneous pneumothorax (PSP) occurs in young tall thin men without underlying lung disease, from rupture of apical pleural blebs. **Secondary spontaneous pneumothorax (SSP)** occurs in patients with underlying lung disease (COPD, asthma, TB, pulmonary fibrosis) and is more dangerous as respiratory reserve is limited. **Tension pneumothorax** is a life-threatening emergency: air accumulates under pressure causing progressive mediastinal shift, compressing the contralateral lung and great vessels, with cardiovascular collapse.

Management by type: small PSP (below 2 cm rim of air) with minimal symptoms: discharge and outpatient follow-up. Large PSP (above 2 cm) or symptomatic: aspiration with a 16G cannula; if unsuccessful, intercostal drain. All SSP regardless of size: intercostal drain. Tension pneumothorax: immediate needle decompression (14G cannula, second intercostal space, midclavicular line) before CXR.

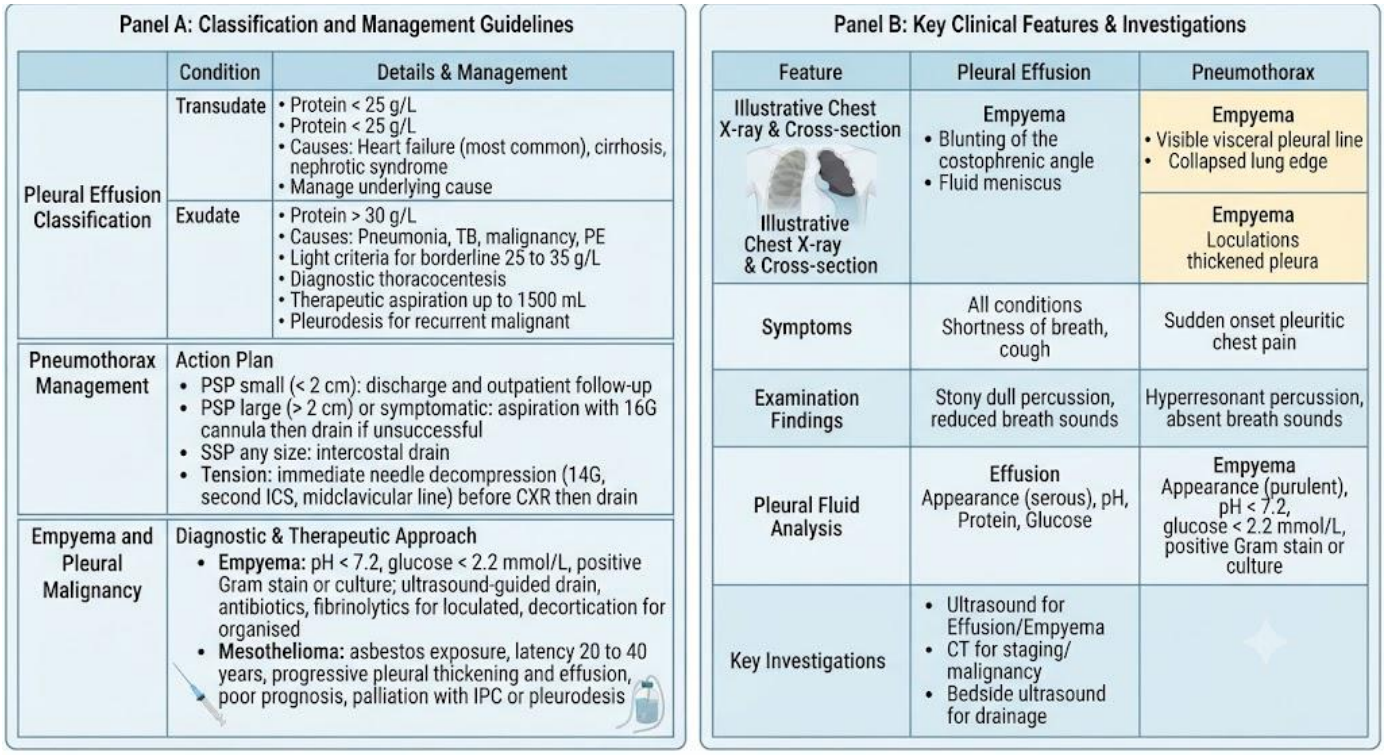
Fill in the blank: Tension pneumothorax causes cardiovascular collapse; treat immediately with needle decompression (14G, second ICS, _____ line) before CXR; all SSP require intercostal drain regardless of size.

4.3.3 Empyema and pleural malignancy

Empyema (pus in the pleural space) is indicated by pleural pH below 7.2, glucose below 2.2 mmol/L, and positive Gram stain or culture. Management requires early ultrasound-guided intercostal chest tube drainage plus appropriate antibiotics. Intrapleural fibrinolytics (streptokinase or tissue plasminogen activator) are used for loculated empyema. Decortication is required for organised empyema.

Pleural malignancy most commonly presents as malignant pleural effusion from metastatic carcinoma (primary sites: lung, breast, ovary, lymphoma) or **malignant pleural mesothelioma** (from asbestos exposure; latency 20 to 40 years; progressive dyspnoea, weight loss, and unilateral pleural thickening on CXR; poor prognosis). Palliation includes repeated aspiration, indwelling pleural catheter, or pleurodesis.

Fill in the blank: Empyema: pH below 7.2; manage with intercostal drain, antibiotics, and fibrinolytics for loculated disease; malignant mesothelioma is caused by _____ exposure; palliation with indwelling pleural catheter or pleurodesis.



Panel A: Classification and Management Guidelines

	Condition	Details & Management
Pleural Effusion Classification	Transudate	• Protein < 25 g/L • Protein < 25 g/L • Causes: Heart failure (most common), cirrhosis, nephrotic syndrome • Manage underlying cause
	Exudate	• Protein > 30 g/L • Causes: Pneumonia, TB, malignancy, PE • Light criteria for borderline 25 to 35 g/L • Diagnostic thoracocentesis • Therapeutic aspiration up to 1500 mL • Pleurodesis for recurrent malignant
Pneumothorax Management	Action Plan	• PSP small (< 2 cm): discharge and outpatient follow-up • PSP large (> 2 cm) or symptomatic: aspiration with 16G cannula then drain if unsuccessful • SSP any size: intercostal drain • Tension: immediate needle decompression (14G, second ICS, midclavicular line) before CXR then drain
Empyema and Pleural Malignancy	Diagnostic & Therapeutic Approach	• **Empyema:** pH < 7.2, glucose < 2.2 mmol/L, positive Gram stain or culture; ultrasound-guided drain, antibiotics, fibrinolytics for loculated, decortication for organised • **Mesothelioma:** asbestos exposure, latency 20 to 40 years, progressive pleural thickening and effusion, poor prognosis, palliation with IPC or pleurodesis

Panel B: Key Clinical Features & Investigations

Feature	Pleural Effusion	Pneumothorax
Illustrative Chest X-ray & Cross-section	Empyema • Blunting of the costophrenic angle • Fluid meniscus	Empyema • Visible visceral pleural line • Collapsed lung edge
Illustrative Chest X-ray & Cross-section		Empyema Loculations thickened pleura
Symptoms	All conditions Shortness of breath, cough	Sudden onset pleuritic chest pain
Examination Findings	Stony dull percussion, reduced breath sounds	Hyperresonant percussion, absent breath sounds
Pleural Fluid Analysis	Effusion Appearance (serous), pH, Protein, Glucose	Empyema Appearance (purulent), pH < 7.2, glucose < 2.2 mmol/L, positive Gram stain or culture
Key Investigations	• Ultrasound for Effusion/Empyema • CT for staging/ malignancy • Bedside ultrasound for drainage	

Figure 4.3: Pleural effusion, pneumothorax, and empyema -- classification and management

Pilot questions 4.3

1. Classify pleural effusion and list two causes of each type. (Linked to SLO 4.3)
2. Describe the management of a large exudative pleural effusion. (Linked to SLO 4.3)
3. Distinguish between PSP and SSP and describe the management of each. (Linked to SLO 4.3)
4. Describe the clinical presentation and management of tension pneumothorax. (Linked to SLO 4.3)
5. Describe the features and management of empyema. (Linked to SLO 4.3)

4.4 Pulmonary Fibrosis and Pulmonary Embolism

4.4.1 Idiopathic pulmonary fibrosis

Idiopathic pulmonary fibrosis (IPF) is the most common and severe idiopathic interstitial pneumonia, predominantly affecting men above 60 years with a smoking history. Clinical

features include progressive exertional dyspnoea, dry cough, bilateral fine end-inspiratory crackles (Velcro crackles) at the lung bases, and clubbing.

HRCT shows the UIP pattern: bilateral basal subpleural honeycombing with traction bronchiectasis and reticular shadowing, with minimal ground-glass opacity. Spirometry shows a **restrictive pattern** (FEV1 and FVC reduced; FEV1/FVC normal); DLCO is markedly reduced. Median survival after diagnosis is 3 to 5 years. Antifibrotic drugs (pirfenidone and nintedanib) slow FVC decline but do not reverse fibrosis. Lung transplantation is the only curative option.

Fill in the blank: IPF: progressive dyspnoea, Velcro crackles, clubbing; HRCT shows UIP pattern (honeycombing, traction bronchiectasis); restrictive spirometry; markedly reduced _____; antifibrotic drugs (pirfenidone, nintedanib) slow progression.

4.4.2 Pulmonary embolism

Pulmonary embolism (PE) occurs when thrombus (usually from DVT) lodges in the pulmonary arterial tree. Risk factors: immobility, surgery, malignancy, oral contraceptive pill, pregnancy, thrombophilia. Clinical features: acute dyspnoea, pleuritic chest pain, haemoptysis, tachycardia, and tachypnoea. Massive PE causes syncope, hypotension, and cardiovascular collapse.

Investigation (after pre-test probability scoring with Wells score): D-dimer (negative excludes PE in low to intermediate probability), CTPA (definitive: filling defects in pulmonary arteries), ECG (sinus tachycardia; S1Q3T3), and ABG (type 1 respiratory failure). Management: anticoagulation (LMWH then DOAC: rivaroxaban or apixaban). Thrombolysis (IV alteplase) for massive PE with haemodynamic compromise.

Fill in the blank: PE investigation: Wells score, D-dimer (negative excludes PE in low probability), CTPA (filling defects; definitive); management: LMWH then DOACs; IV _____ (alteplase) for massive PE with haemodynamic compromise.

4.4.3 Pulmonary hypertension

Pulmonary hypertension (PH) is defined as mean pulmonary arterial pressure above 20 mmHg at rest. It is classified into 5 groups: Group 1 (pulmonary arterial hypertension, PAH: idiopathic,

connective tissue disease, HIV, portal hypertension), Group 2 (left heart disease: the most common cause), Group 3 (lung disease or hypoxia: COPD, IPF), Group 4 (chronic thromboembolic: CTEPH; potentially curable by pulmonary endarterectomy), and Group 5 (miscellaneous).

Clinical features include exertional dyspnoea, fatigue, exertional syncope, and signs of right heart failure (elevated JVP, loud P2, peripheral oedema). Echocardiography screens for PH; right heart catheterisation confirms it. Group 1 PAH is treated with pulmonary vasodilators: **endothelin receptor antagonists** (bosentan, ambrisentan), **phosphodiesterase-5 inhibitors** (sildenafil, tadalafil), and **prostanoids** (epoprostenol).

Fill in the blank: PH Group 2 (left heart disease: most common) and Group 3 (COPD, IPF) are managed by treating the underlying condition; Group 1 PAH is treated with endothelin receptor antagonists, PDE5 _____ (sildenafil), and prostanoids.

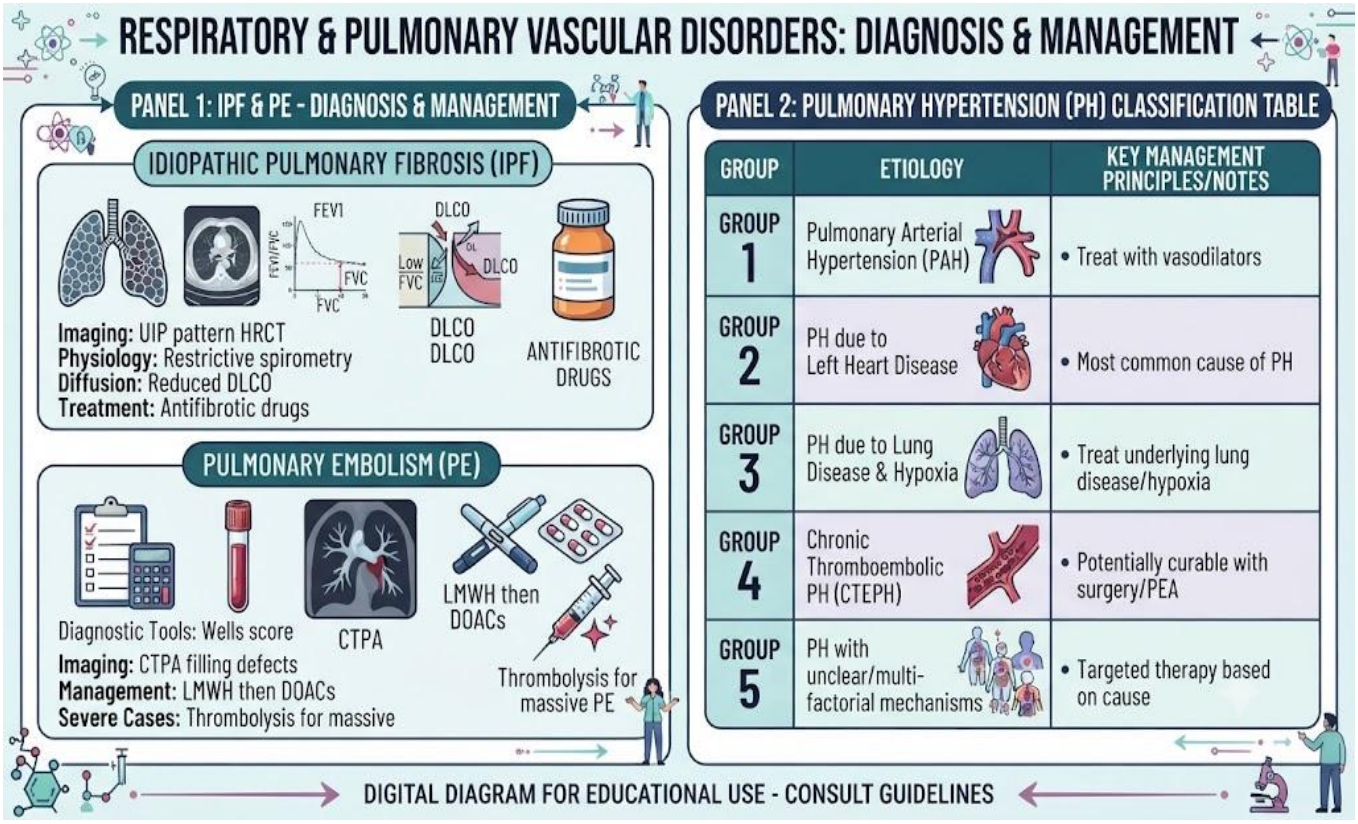


Figure 4.4: IPF, pulmonary embolism, and pulmonary hypertension -- features, investigation, and management

Pilot questions 4.4

1. Describe the clinical features, HRCT findings, and management of IPF. (Linked to SLO 4.4)
2. Describe the risk factors and clinical features of pulmonary embolism. (Linked to SLO 4.4)
3. Describe the investigation pathway for suspected pulmonary embolism. (Linked to SLO 4.4)
4. Describe the five-group classification of pulmonary hypertension. (Linked to SLO 4.4)
5. Describe the treatment of Group 1 pulmonary arterial hypertension. (Linked to SLO 4.4)

Series Summary

Asthma is a chronic eosinophilic airway disease with variable reversible obstruction; atopy is the strongest risk factor; triggers include allergens, NSAIDs, and exercise. Diagnosis: reversible obstruction (FEV1 improvement above 12 percent and 200 mL), PEFR diurnal variability above 20 percent, elevated FeNO. GINA stepwise treatment: SABA, add ICS, add LABA, increase ICS plus montelukast, biologics. Acute severe asthma: PEFR 33 to 50 percent, SpO2 below 92 percent; life-threatening: silent chest, PEFR below 33 percent; manage with oxygen, nebulised salbutamol, ipratropium, IV magnesium, corticosteroids, ICU. COPD: smoking and biomass fuel (Nigerian women); neutrophilic inflammation, emphysema, air trapping. Diagnosis: post-bronchodilator FEV1/FVC below 0.70; GOLD 1 to 4 by FEV1; CXR hyperinflation and bullae. Management: smoking cessation; LABA or LAMA; triple therapy; exacerbation: controlled O2 (88 to 92 percent), bronchodilators, prednisolone 5 days, antibiotics, NIV for pH below 7.35.

Pleural effusion: transudate (protein below 25 g/L; heart failure most common) or exudate (protein above 30 g/L; pneumonia, TB, malignancy); treat cause for transudate; drain for large or exudative; pleurodesis for recurrent malignant. Pneumothorax: PSP (young tall thin men; blebs); SSP (underlying lung disease; more dangerous; always drain); tension (cardiovascular collapse; immediate needle decompression, second ICS, midclavicular line). Empyema: pH below 7.2; drain plus antibiotics; fibrinolytics for loculated. Mesothelioma: asbestos exposure; poor prognosis. IPF: Velcro crackles, clubbing; UIP on HRCT; restrictive spirometry; reduced DLCO; antifibrotic therapy; 3 to 5-year median survival. PE: acute dyspnoea, pleuritic pain, tachycardia; D-dimer (low probability), CTPA (definitive); LMWH then DOACs; thrombolysis for massive PE. PH: five groups; Group 2 most common; Group 1 PAH: endothelin antagonists, sildenafil, prostanoids.

Glossary of Terms

- **FeNO:** fractional exhaled nitric oxide; a non-invasive marker of eosinophilic airway inflammation; above 40 ppb supports asthma diagnosis and predicts response to inhaled corticosteroids.

- **ICS:** inhaled corticosteroid; the cornerstone of asthma controller therapy; reduces airway inflammation and prevents exacerbations.
- **LABA:** long-acting beta-2 agonist; bronchodilator used as maintenance therapy in asthma (with ICS) and COPD; examples include salmeterol and formoterol.
- **LAMA:** long-acting muscarinic antagonist; bronchodilator used as maintenance therapy in COPD; tiotropium is the prototype.
- **GOLD staging:** Global Initiative for COPD staging by post-bronchodilator FEV1: Grade 1 (above 80 percent) to Grade 4 (below 30 percent).
- **Tension pneumothorax:** a life-threatening pneumothorax where air accumulates under pressure causing progressive mediastinal shift and cardiovascular collapse; treated with immediate needle decompression.
- **Empyema:** pus in the pleural space; indicated by pleural pH below 7.2, glucose below 2.2 mmol/L, or positive Gram stain; requires drainage.
- **Pleurodesis:** obliteration of the pleural space by instillation of an irritant (usually talc) to cause pleural adhesion; used for recurrent malignant pleural effusion.
- **UIP pattern:** usual interstitial pneumonia; the HRCT pattern of IPF: bilateral basal subpleural honeycombing with traction bronchiectasis and minimal ground-glass opacity.
- **CTPA:** CT pulmonary angiography; the definitive investigation for pulmonary embolism; demonstrates filling defects within the pulmonary arterial tree.
- **D-dimer:** a fibrin degradation product elevated in PE; a negative D-dimer effectively excludes PE in patients with low to intermediate pre-test probability.
- **Sildenafil:** a phosphodiesterase-5 inhibitor used as a pulmonary vasodilator in Group 1 pulmonary arterial hypertension.

Concept Check Questions [Series 4]

Objective Questions

1. Asthma is characterised by variable _____ airflow obstruction; eosinophilic inflammation is driven by Th2 cytokines; atopy is the strongest risk factor. (Linked to SLO 4.1)
2. Life-threatening asthma features include PEFR below 33 percent, _____ chest (airways too tight for air movement), cyanosis, and confusion. (Linked to SLO 4.1)
3. COPD is diagnosed by post-bronchodilator FEV1/FVC below 0.70; GOLD 4 is very severe COPD with FEV1 below _____ percent predicted. (Linked to SLO 4.2)
4. Tension pneumothorax requires immediate needle decompression at the second ICS _____ line before CXR. (Linked to SLO 4.3)
5. IPF shows a UIP pattern on HRCT and is treated with antifibrotic drugs including pirfenidone and _____. (Linked to SLO 4.4)
6. A negative D-dimer excludes PE in low to intermediate pre-test probability; definitive diagnosis uses _____. (Linked to SLO 4.4)

Short-Answer Questions

7. Describe the GINA stepwise management of stable asthma. (Linked to SLO 4.1)
8. Describe the management of a COPD exacerbation. (Linked to SLO 4.2)
9. Classify pneumothorax and describe the management of each type. (Linked to SLO 4.3)

Long-Answer Questions

10. Describe the aetiology, pathophysiology, clinical features, diagnosis, and management of COPD. (Linked to SLO 4.2)
11. Describe the clinical features, investigation, and management of pulmonary embolism. (Linked to SLO 4.4)

Answers to Concept Check Questions [Series 4]

Objective Questions

1. Reversible.
2. Silent.
3. 30.
4. Midclavicular.
5. Nintedanib.
6. CTPA.

Short-Answer Questions

7. Step 1: SABA on demand. Step 2: add low-dose ICS. Step 3: add LABA to ICS (combination inhaler). Step 4: increase ICS; add montelukast. Step 5: oral corticosteroids or biologics (omalizumab for allergic; mepolizumab for eosinophilic). Step up if uncontrolled; step down if controlled for 3 months.
8. Controlled low-flow oxygen (SpO2 88 to 92 percent). Nebulised salbutamol plus ipratropium. Oral prednisolone 30 to 40 mg for 5 days. Antibiotics (amoxicillin or doxycycline) if increased sputum purulence. NIV (BiPAP) for type 2 respiratory failure with pH below 7.35; reduces intubation and mortality.
9. PSP (young tall thin men; no underlying lung disease): small (below 2 cm): discharge and outpatient review; large (above 2 cm) or symptomatic: aspiration with 16G cannula, then drain if unsuccessful. SSP (underlying lung disease: always more dangerous): intercostal drain regardless of size. Tension pneumothorax: immediate 14G needle decompression (second ICS midclavicular line) before CXR, then drain.

Long-Answer Questions

10. **Basic response:** Aetiology: cigarette smoking (80 to 90 percent globally); biomass fuel combustion (major cause in Nigerian women cooking indoors); occupational dust; outdoor air pollution; childhood respiratory infections impairing lung development. Pathophysiology: neutrophilic airway inflammation; mucus hypersecretion and goblet cell hyperplasia (chronic bronchitis); emphysema (neutrophil elastase destroys alveolar walls, reduces elastic recoil and gas exchange surface; air trapping from loss of elastic

recoil and small airway collapse). Clinical features: progressive dyspnoea (MRC Grade 2 or above: hallmark); chronic productive cough (3 months per year for 2 consecutive years: chronic bronchitis definition); wheeze; reduced exercise tolerance; advanced: barrel chest, pursed-lip breathing, cor pulmonale (right heart failure: elevated JVP, oedema, hepatomegaly). Diagnosis: post-bronchodilator FEV1/FVC below 0.70; GOLD 1 (FEV1 above 80 percent) to GOLD 4 (below 30 percent); CXR: hyperinflation, flattened diaphragms, bullae. Management: smoking cessation (most effective); remove biomass fuel exposure; SABA or SAMA as reliever; LABA or LAMA maintenance; ICS added for FEV1 below 60 percent with frequent exacerbations; triple therapy (LABA/LAMA/ICS) for severe symptomatic disease; pulmonary rehabilitation; long-term oxygen for PaO2 below 7.3 kPa.

Value-added response: In Nigeria, recognition of biomass fuel-related COPD in non-smoking women is as clinically important as recognising smoking-related COPD in men; the history must always include cooking fuel type and kitchen ventilation.

11. **Basic response:** Risk factors: immobility, recent surgery, malignancy, oral contraceptive pill, pregnancy, thrombophilia, obesity. Clinical features: acute onset dyspnoea (most common), pleuritic chest pain, haemoptysis, tachycardia, tachypnoea, low-grade fever; massive PE: syncope, hypotension, cardiovascular collapse. Investigation: assess pre-test probability (Wells score); low to intermediate probability and negative D-dimer: PE excluded; positive D-dimer or high probability: CTPA (filling defects in pulmonary arteries: definitive); ECG (sinus tachycardia most common; S1Q3T3: deep S in lead I, Q wave and T inversion in lead III); ABG (type 1 respiratory failure). Management: anticoagulation with LMWH (enoxaparin) for minimum 5 days bridging to DOAC (rivaroxaban or apixaban: first-line; no INR monitoring needed); anticoagulation duration: 3 months for provoked PE; 6 months or longer for unprovoked; indefinite for malignancy-associated PE. Thrombolysis (IV alteplase 100 mg over 2 hours) for massive PE with haemodynamic compromise.

    Value-added response: DOACs are now preferred over warfarin for PE because they require no INR monitoring and have fixed dosing; in Nigeria where INR monitoring infrastructure is limited, this is a particularly important practical advantage.

Answers to Pilot Questions [Series 4]

Part 4.1

1. IgE antibodies against allergens bind to mast cells. Re-exposure cross-links IgE causing mast cell degranulation and release of histamine, prostaglandins, and leukotrienes (early-phase: bronchospasm within minutes). Th2 lymphocytes release IL-4, IL-5, and IL-13, driving eosinophil recruitment (late-phase: 4 to 8 hours; airway inflammation and remodelling). Chronic eosinophilic inflammation causes airway hyperresponsiveness, mucosal oedema, mucus hypersecretion, and structural remodelling.
2. Episodic wheeze (polyphonic, expiratory), nocturnal and early-morning dyspnoea, chest tightness, and dry cough (cough variant asthma). Symptoms are variable, episodic, and triggered (allergens, exercise, cold air, NSAIDs). Between episodes the examination may be normal. During an attack: tachypnoea, accessory muscle use, polyphonic expiratory wheeze, prolonged expiration, and hyperinflation.
3. Spirometry with bronchodilator reversibility (FEV1 improvement above 12 percent and above 200 mL after salbutamol 400 micrograms confirms variable obstruction). PEFR diary over 2 to 4 weeks (diurnal variability above 20 percent supports asthma). FeNO above 40 ppb indicates eosinophilic inflammation and predicts ICS response. Bronchial challenge test (methacholine: fall in FEV1 above 20 percent at below 8 mg/mL confirms airway hyperresponsiveness) when spirometry is normal.
4. Step 1: SABA on demand. Step 2: add low-dose ICS (beclometasone or fluticasone). Step 3: add LABA to ICS (combination inhaler: do not give LABA without ICS). Step 4: increase ICS to medium dose; add leukotriene receptor antagonist (montelukast 10 mg once daily). Step 5: specialist referral; add oral prednisolone or biologic (omalizumab for severe allergic; mepolizumab for severe eosinophilic).
5. Life-threatening features: PEFR below 33 percent, SpO2 below 92 percent, PaO2 below 8 kPa, silent chest (the most ominous sign), cyanosis, bradycardia, hypotension, confusion. Management: sit upright; high-flow oxygen (SpO2 94 to 98 percent); call for

senior help and ICU; continuous nebulised salbutamol 5 mg; ipratropium 500 micrograms nebulised; IV magnesium sulphate 1.2 to 2 g over 20 minutes; IV hydrocortisone 100 mg; prepare for intubation.

Part 4.2

1. Biomass fuel combustion (wood, charcoal, animal dung, crop residues) produces particulate matter, carbon monoxide, and volatile organic compounds. Indoor cooking without adequate ventilation exposes the cook (predominantly women in Nigeria) to concentrations far exceeding WHO limits. Chronic inhalation causes the same pathological changes as cigarette smoking. Studies show that COPD prevalence in non-smoking Nigerian women using biomass fuel approaches that in male smokers. Clinically, every woman with progressive dyspnoea and airflow obstruction must be asked about cooking fuel and kitchen ventilation.
2. Progressive dyspnoea (the hallmark; MRC Grade 2 or above), chronic productive cough (chronic bronchitis: 3 months per year for 2 consecutive years), wheeze, and reduced exercise tolerance. Advanced COPD: barrel chest (increased AP diameter from hyperinflation), pursed-lip breathing, accessory muscle use, reduced breath sounds. Cor pulmonale (right heart failure from pulmonary hypertension): elevated JVP, peripheral oedema, hepatomegaly, cyanosis.
3. Post-bronchodilator spirometry (20 minutes after 400 micrograms salbutamol or 80 micrograms ipratropium): FEV1/FVC ratio below 0.70 with minimal bronchodilator reversibility. GOLD staging by FEV1 as percent of predicted: GOLD 1 (above 80 percent: mild), GOLD 2 (50 to 79 percent: moderate), GOLD 3 (30 to 49 percent: severe), GOLD 4 (below 30 percent: very severe). Combined assessment also incorporates symptom burden (CAT score or mMRC) and exacerbation history.
4. SABA (salbutamol) as reliever for all patients. Low symptom burden (GOLD A): SAMA (ipratropium) or SABA as needed. Moderate symptoms or exacerbation risk (GOLD B and E): add LABA or LAMA; LABA/LAMA combination preferred for GOLD B. High exacerbation burden (GOLD E): LABA/LAMA combination; if frequent exacerbations and blood eosinophil count above 300: add ICS (triple therapy LABA/LAMA/ICS).

5. Controlled low-flow oxygen: 24 to 28 percent Venturi mask; target SpO2 88 to 92 percent (avoid high-flow oxygen: suppresses hypoxic drive and worsens hypercapnia). Nebulised bronchodilators: salbutamol 5 mg plus ipratropium 500 micrograms; repeat 4 to 6 hourly. Systemic corticosteroids: prednisolone 30 to 40 mg oral for 5 days. Antibiotics: amoxicillin or doxycycline if increased sputum purulence. NIV (BiPAP): for pH below 7.35 with type 2 respiratory failure; reduces intubation rates and mortality.

Part 4.3

1. Transudate (protein below 25 g/L): heart failure (most common globally), cirrhosis, nephrotic syndrome, hypoalbuminaemia. Exudate (protein above 30 g/L; Light's criteria for borderline): parapneumonic effusion (pneumonia), TB pleuritis (lymphocytic; low glucose; elevated ADA), malignancy (metastatic carcinoma or mesothelioma), pulmonary embolism.
2. Confirm the effusion clinically and by CXR. Perform ultrasound-guided diagnostic thoracocentesis (20 to 50 mL: protein, LDH, glucose, pH, cytology, microbiology, AFB). For large or symptomatic exudative effusion: therapeutic aspiration up to 1500 mL per session (avoid more to prevent re-expansion pulmonary oedema). If cytology is inconclusive and malignancy is suspected: thoracoscopic pleural biopsy (sensitivity above 90 percent). For recurrent malignant effusion: indwelling pleural catheter or talc pleurodesis.
3. PSP: young (20 to 30 years), tall, thin male; no underlying lung disease; rupture of apical blebs; sudden pleuritic chest pain and dyspnoea; management depends on size and symptoms. SSP: pre-existing lung disease (COPD most common, asthma, TB, pulmonary fibrosis); more dangerous due to reduced respiratory reserve; even a small pneumothorax may cause severe respiratory failure; always managed with intercostal drain regardless of size; higher mortality and recurrence rates.
4. Presentation: sudden severe dyspnoea, cyanosis, hypotension, tachycardia, tracheal deviation away from the affected side, absent breath sounds on the affected side, distended neck veins. This is a clinical diagnosis: do not wait for CXR. Immediate management: 14G cannula into the second intercostal space midclavicular line on the

affected side; a rush of air confirms decompression; followed by formal intercostal chest tube drainage when the patient is stabilised.

5. Empyema features: pleural pH below 7.2, glucose below 2.2 mmol/L, positive Gram stain or culture, frank pus on aspiration. Management: early ultrasound-guided small-bore (14 French) intercostal drain plus antibiotics (co-amoxiclav for community-acquired; piperacillin-tazobactam for hospital-acquired). Intrapleural fibrinolytics (streptokinase or tissue plasminogen activator plus DNase) for loculated empyema not draining adequately. Surgical decortication for organised fibrothorax trapping the lung.

Part 4.4

1. Clinical features: progressive exertional dyspnoea, dry cough, bilateral fine end-inspiratory Velcro crackles at lung bases, clubbing in approximately 50 percent. HRCT: UIP pattern (bilateral basal subpleural honeycombing with traction bronchiectasis; minimal ground-glass opacity). Spirometry: restrictive (FEV1 and FVC both reduced; FEV1/FVC normal); DLCO markedly reduced. Prognosis: median survival 3 to 5 years. Treatment: antifibrotic drugs (pirfenidone 801 mg three times daily or nintedanib 150 mg twice daily) slow FVC decline; oxygen for hypoxaemia; pulmonary rehabilitation; lung transplantation is the only curative option.
2. Risk factors: Virchow's triad of venous stasis (immobility, bed rest, long-haul travel), hypercoagulability (malignancy, thrombophilia, oral contraceptive pill, pregnancy), and endothelial injury (surgery, trauma, central venous catheter). Clinical features: acute dyspnoea (most common), pleuritic chest pain (peripheral PE with infarction), haemoptysis, tachycardia (HR above 100 bpm), tachypnoea, low-grade fever, signs of DVT (calf pain and swelling). Massive PE: syncope, hypotension, cardiovascular collapse, raised JVP.
3. Assess pre-test probability (Wells score). Low to intermediate probability: D-dimer (ELISA; negative below 500 ng/mL; if negative: PE excluded; if positive: proceed to CTPA). High probability: proceed directly to CTPA (filling defects in pulmonary arteries). ECG: sinus tachycardia most common; S1Q3T3; right bundle branch block. ABG: type 1 respiratory failure (low PaO2, low or normal PaCO2). Echo: RV dilatation in massive PE. If CTPA is contraindicated: V/Q scan.

4. Group 1 PAH: idiopathic, heritable, drug-induced, or associated with connective tissue disease, HIV, portal hypertension, congenital heart disease. Group 2: left heart disease (LV systolic or diastolic dysfunction, valvular disease: the most common overall cause of PH). Group 3: lung disease or hypoxia (COPD, IPF, obstructive sleep apnoea). Group 4: CTEPH (chronic thromboembolic; persistent clot after acute PE; potentially curable by pulmonary endarterectomy). Group 5: miscellaneous (sickle cell disease, sarcoidosis, Gaucher disease).
5. Three targeted pathways for Group 1 PAH: Endothelin pathway (endothelin receptor antagonists: bosentan, ambrisentan, macitentan; block vasoconstriction and vascular remodelling; monitor LFTs). Nitric oxide pathway (phosphodiesterase-5 inhibitors: sildenafil 20 mg three times daily or tadalafil; inhibit cGMP breakdown prolonging vasodilation). Prostacyclin pathway (prostanoids: IV epoprostenol for severe PAH; inhaled iloprost; subcutaneous treprostinil; oral selexipag). Combination therapy (ERA plus PDE5 inhibitor) is standard. Lung transplantation for refractory disease.

References and Attributions [Series 4]

Textual References

- Global Initiative for Asthma (GINA). (2023). Global Strategy for Asthma Management and Prevention. Available at: www.ginasthma.org.
- Global Initiative for Chronic Obstructive Lung Disease (GOLD). (2023). Global Strategy for the Diagnosis, Management, and Prevention of COPD. Available at: www.goldcopd.org.
- British Thoracic Society. (2010). BTS Pleural Disease Guideline 2010. Thorax, 65(Suppl 2), ii1 to ii76.
- Kumar, P. and Clark, M. (2017). Kumar and Clark's Clinical Medicine (9th ed.). Edinburgh: Elsevier.

Figures, Plates, Tables, and Boxes

- Figure 4.1: Asthma stepwise management and acute severe asthma. AI prompt: "Two-panel diagram: left GINA five-step ladder (SABA, add ICS, ICS/LABA, increase ICS plus montelukast, biologics); right severity assessment and management of acute severe and life-threatening asthma; clean professional infographic." OER search: "asthma GINA stepwise management acute severe asthma treatment diagram".
- Figure 4.2: COPD staging and management. AI prompt: "Two-panel diagram: GOLD staging and stepwise stable management; exacerbation management (O2, bronchodilators, prednisolone, NIV); clean infographic." OER search: "COPD GOLD staging stepwise management exacerbation treatment NIV diagram".
- Figure 4.3: Pleural effusion, pneumothorax, and empyema. AI prompt: "Three-row table: pleural effusion (transudate vs exudate), pneumothorax (PSP, SSP, tension), empyema and mesothelioma; clean infographic." OER search: "pleural effusion pneumothorax empyema classification management diagram".
- Figure 4.4: IPF, PE, and pulmonary hypertension. AI prompt: "Two-panel diagram: IPF (UIP HRCT, antifibrotic drugs) and PE (D-dimer, CTPA, DOACs, thrombolysis); PH five-group classification; clean infographic." OER search: "IPF pulmonary embolism CTPA pulmonary hypertension classification management diagram".

Series 5: Risk Factors and Preventive Strategies in Respiratory Health

- **Part 5.1: Risk Factors for Respiratory Disease**
    - Sub Part 5.1.1: Tobacco smoking and air pollution
    - Sub Part 5.1.2: Occupational and indoor air exposures
    - Sub Part 5.1.3: Infections and host factors
- **Part 5.2: Prevention of Respiratory Infections**
    - Sub Part 5.2.1: Prevention of pneumonia
    - Sub Part 5.2.2: Prevention and control of tuberculosis
    - Sub Part 5.2.3: Prevention of influenza and other respiratory viruses
- **Part 5.3: Prevention of Chronic Airway and Parenchymal Disease**
    - Sub Part 5.3.1: Prevention of asthma and occupational lung disease
    - Sub Part 5.3.2: Prevention and management of COPD risk
    - Sub Part 5.3.3: Prevention of lung cancer
- **Part 5.4: Health Promotion and Respiratory Rehabilitation**
    - Sub Part 5.4.1: Smoking cessation strategies
    - Sub Part 5.4.2: Pulmonary rehabilitation
    - Sub Part 5.4.3: Community-level respiratory health promotion in Nigeria

Introduction

Respiratory diseases are among the most preventable causes of morbidity and mortality in Nigeria. Tobacco smoking, indoor air pollution from biomass fuel combustion, occupational exposures, and infections including tuberculosis, pneumonia, and influenza are all modifiable risk factors. Prevention strategies operating at individual, community, and health-system levels can substantially reduce the burden of both infectious and non-communicable respiratory diseases.

This Series examines the major risk factors for respiratory disease in the Nigerian context, from tobacco and air pollution through occupational exposures, infections, and host susceptibility factors. Prevention strategies are then covered for each major disease category: respiratory infections, chronic airway diseases, and lung cancer. The Series concludes with smoking cessation strategies, pulmonary rehabilitation, and community-level health promotion applicable to Nigeria.

Preventive respiratory medicine is not a secondary concern; for diseases like COPD, pulmonary fibrosis, and lung cancer, prevention is more effective than any treatment available once the disease is established.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 5.1** describe the major risk factors for respiratory disease including tobacco, air pollution, occupational exposures, infections, and host factors (Linked to Part 5.1),
- **SLO 5.2** describe the prevention strategies for pneumonia, tuberculosis, and respiratory viral infections (Linked to Part 5.2),
- **SLO 5.3** describe the prevention of asthma, occupational lung disease, COPD, and lung cancer (Linked to Part 5.3), and

- **SLO 5.4** describe evidence-based smoking cessation strategies, pulmonary rehabilitation, and community-level respiratory health promotion in Nigeria (Linked to Part 5.4).

5.1 Risk Factors for Respiratory Disease

5.1.1 Tobacco smoking and air pollution

Tobacco smoking is the single most important preventable cause of respiratory disease globally. It causes COPD, lung cancer, and accelerates TB progression by impairing mucociliary clearance, activating neutrophilic airway inflammation, and directly destroying alveolar walls. Nigeria has a lower smoking prevalence than high-income countries, but rates are rising in young men.

Outdoor air pollution (PM2.5 and PM10, nitrogen oxides, ozone) from traffic, industry, and waste burning causes COPD, respiratory infections, and lung cancer. **Indoor air pollution** from biomass fuel combustion (wood, charcoal, animal dung, crop residues) is a major respiratory risk factor in Nigeria, causing COPD, pneumonia, and lung cancer predominantly in women and children. The WHO estimates 3.8 million deaths per year from indoor air pollution.

Fill in the blank: Tobacco smoking causes COPD, lung cancer, and emphysema; indoor air pollution from _____ fuel combustion is a major COPD and pneumonia risk factor for Nigerian women and children.

5.1.2 Occupational and indoor air exposures

Occupational lung diseases result from inhalation of dusts, fumes, or biological agents at work. Major conditions include: **pneumoconiosis** (coal dust: coal workers' pneumoconiosis; silica: silicosis; asbestos: asbestosis), **occupational asthma** (isocyanates, grain dust, animal allergens), **hypersensitivity pneumonitis** (mouldy hay: farmer's lung; avian proteins: bird fancier's lung), and **mesothelioma** (asbestos; latency 20 to 40 years).

In Nigeria, occupational exposures of significance include silica in quarrying, grain dust in agriculture, chemical fumes in manufacturing, and biomass combustion affecting women and

children at home. Identifying an occupational cause is important because removal from exposure is the most effective treatment for occupational lung disease.

Fill in the blank: Occupational lung diseases include pneumoconiosis (coal, silica, asbestos), occupational asthma (isocyanates, grain dust), hypersensitivity pneumonitis (mouldy hay: _____ lung), and mesothelioma (asbestos).

5.1.3 Infections and host factors

Repeated childhood respiratory infections impair lung development, reducing peak lung function and predisposing to COPD and bronchiectasis in adulthood. Pulmonary TB causes permanent structural damage (cavitation, fibrosis, bronchiectasis) predisposing to aspergilloma, secondary bacterial infection, and progressive respiratory failure.

Host factors increasing respiratory disease susceptibility include: **HIV infection** (markedly increases risk of TB, PCP, and bacterial pneumonia), **malnutrition** (impairs cell-mediated immunity), **diabetes mellitus** (increases TB reactivation risk and pneumonia severity), **sickle cell disease** (functional asplenia increases pneumococcal pneumonia risk), and **alpha-1 antitrypsin deficiency** (early-onset emphysema).

Fill in the blank: Host risk factors include HIV (TB, PCP), malnutrition (impairs cell-mediated immunity), diabetes (TB reactivation), sickle cell disease (pneumococcal pneumonia from functional _____), and alpha-1 antitrypsin deficiency (emphysema).

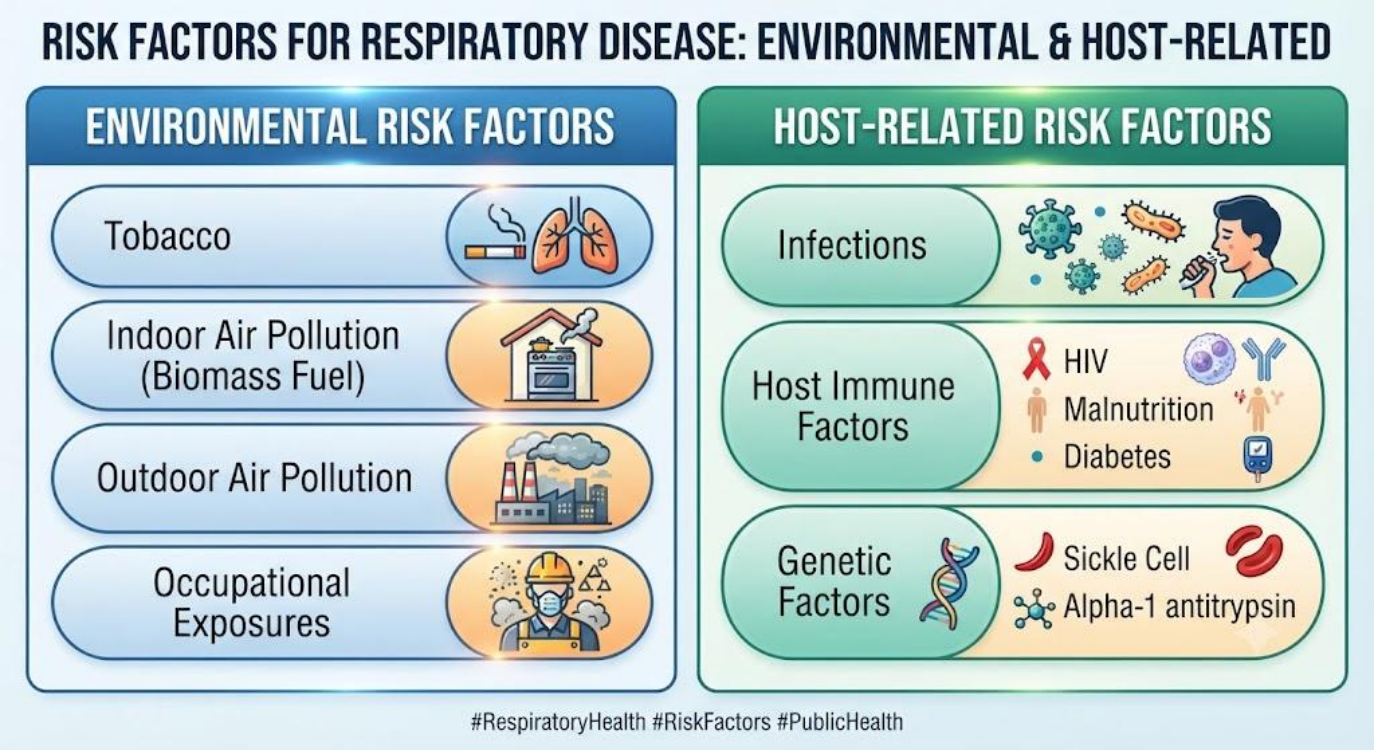


Figure 5.1: Risk factors for respiratory disease in Nigeria: environmental, occupational, infection-related, and host factors

Pilot questions 5.1

1. Describe how tobacco smoking causes COPD and lung cancer. (Linked to SLO 5.1)
2. Describe the respiratory effects of indoor air pollution from biomass fuel in Nigeria. (Linked to SLO 5.1)
3. List four occupational lung diseases and state the causative exposure for each. (Linked to SLO 5.1)
4. Describe how HIV infection increases the risk of respiratory disease. (Linked to SLO 5.1)
5. List the host factors that increase susceptibility to respiratory infection. (Linked to SLO 5.1)

5.2 Prevention of Respiratory Infections

5.2.1 Prevention of pneumonia

Pneumococcal conjugate vaccine (PCV13) is recommended for all children under 2 years (primary series at 6, 10, and 14 weeks in Nigeria's EPI) and adults above 65 years or with chronic disease; it reduces pneumococcal pneumonia by 45 to 75 percent. **Haemophilus influenzae type b (Hib) vaccine** prevents a major cause of childhood pneumonia and meningitis and is included in the Nigerian EPI as part of the pentavalent vaccine.

Additional preventive measures include exclusive breastfeeding for the first 6 months (reduces childhood pneumonia incidence by approximately 15 percent), adequate nutrition (vitamin A and zinc supplementation in malnourished children), reduction of indoor air pollution, and daily cotrimoxazole prophylaxis in HIV-positive patients with CD4 below 200 cells per microlitre.

Fill in the blank: Pneumonia prevention: PCV13 (children under 2 and adults above 65 in Nigerian EPI), Hib vaccine (EPI), exclusive breastfeeding (reduces pneumonia by 15 percent), _____ prophylaxis in HIV patients with CD4 below 200.

5.2.2 Prevention and control of tuberculosis

BCG vaccination is given at birth in Nigeria and provides approximately 80 percent protection against severe childhood TB (miliary TB and TB meningitis), though protection against adult pulmonary TB is variable.

TB control measures include prompt identification and treatment of smear-positive cases (each treated case prevents 10 to 15 new infections per year), **directly observed therapy (DOT)** (ensures adherence and prevents acquired resistance), **contact tracing** (exposes individuals for testing and preventive therapy), **TB preventive therapy (TPT)** (isoniazid 6 months or isoniazid-rifapentine 3 months for PLHIV and close contacts of smear-positive cases), and infection control in health facilities (natural ventilation, UV irradiation, N95 respirators).

Fill in the blank: TB control: BCG at birth; prompt treatment of smear-positive cases; DOT; contact tracing; TB preventive therapy (_____ 6 months for PLHIV and close contacts); infection control in health facilities.

5.2.3 Prevention of influenza and other respiratory viruses

Annual influenza vaccination is recommended for adults above 65 years, pregnant women, healthcare workers, and patients with chronic respiratory or cardiac disease; it reduces influenza-related hospitalisations by approximately 50 to 60 percent. Coverage in Nigeria remains low; advocacy for inclusion in the EPI is ongoing.

Non-pharmacological prevention includes hand hygiene (soap and water or alcohol-based hand rub: reduces respiratory virus transmission by 30 to 40 percent), respiratory etiquette (covering the mouth and nose when coughing or sneezing), face masks (surgical masks reduce droplet transmission; N95 respirators protect against airborne pathogens), and adequate ventilation in crowded spaces.

Fill in the blank: Influenza vaccination (annual) is recommended for adults above 65, pregnant women, and healthcare workers; hand hygiene reduces transmission by 30 to 40 percent; N95 respirators protect against _____ pathogens.

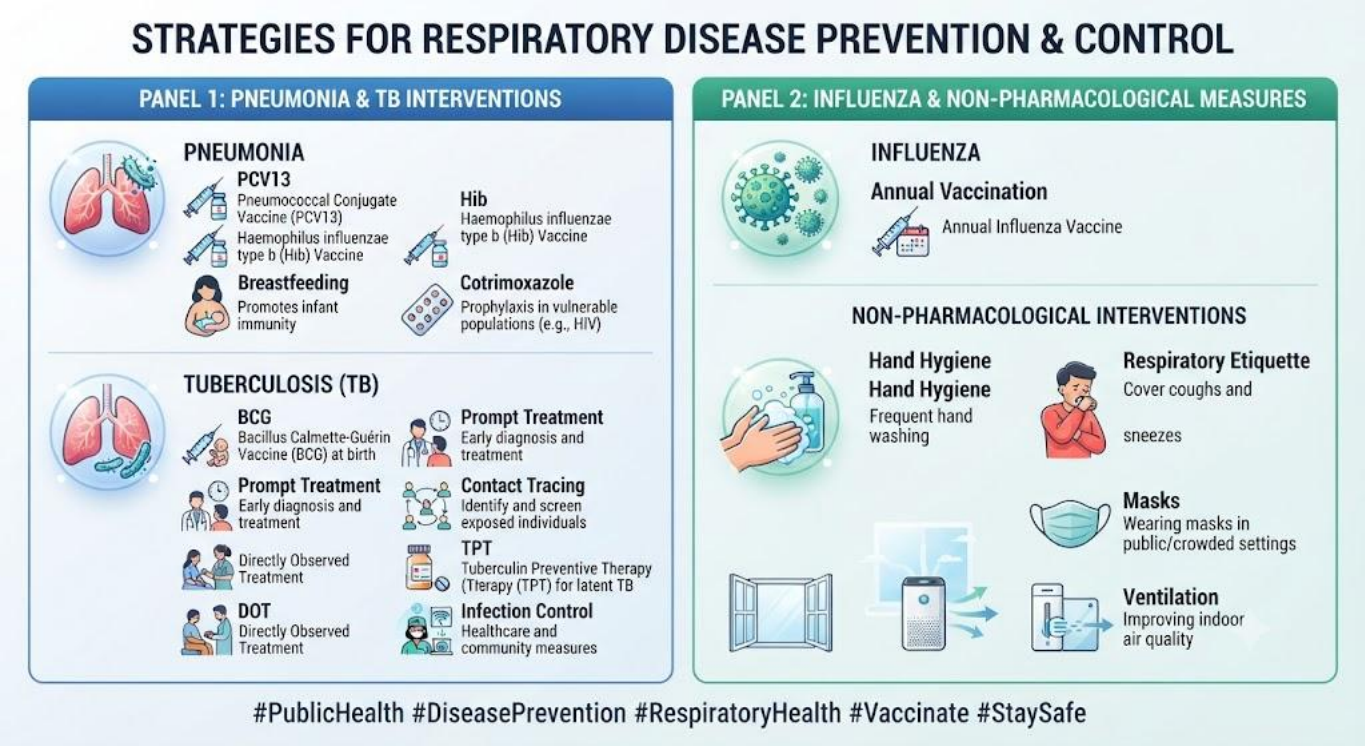


Figure 5.2: Prevention of respiratory infections: pneumonia, TB, influenza, and respiratory viruses

Pilot questions 5.2

1. Describe the vaccines used in pneumonia prevention and their target populations. (Linked to SLO 5.2)
2. Describe the role of BCG vaccination in TB prevention. (Linked to SLO 5.2)
3. Describe the components of TB control beyond vaccination. (Linked to SLO 5.2)
4. Describe the prevention of influenza including vaccination and non-pharmacological measures. (Linked to SLO 5.2)
5. Explain the role of TB preventive therapy and state the indications for its use. (Linked to SLO 5.2)

5.3 Prevention of Chronic Airway and Parenchymal Disease

5.3.1 Prevention of asthma and occupational lung disease

Primary prevention of asthma includes avoidance of prenatal tobacco smoke exposure, exclusive breastfeeding (reduces early respiratory infections), reduction of indoor allergen load (house dust mite reduction: mattress covers, reducing humidity), and avoiding early antibiotic overuse.

Prevention of occupational lung disease follows the hierarchy of controls: **elimination or substitution** (replace the hazardous substance), **engineering controls** (local exhaust ventilation to capture dust and fumes at source), **administrative controls** (job rotation, limit exposure duration, health surveillance with regular spirometry and CXR), and **personal protective equipment (PPE)** (dust respirators: last resort). Removal from exposure halts progression of most occupational lung diseases.

Fill in the blank: Occupational lung disease prevention: hierarchy of controls (elimination, engineering controls, administrative controls, _____: last resort); workplace health surveillance detects early disease before irreversible damage.

5.3.2 Prevention and management of COPD risk

Smoking cessation is the single most effective intervention to prevent COPD and slow its progression. Annual FEV1 decline slows from approximately 60 mL per year (continuing smokers) to 30 mL per year (ex-smokers), approaching that of non-smokers. Benefits occur at any stage of disease.

Reduction of biomass fuel exposure through cleaner cooking fuels (liquefied petroleum gas, solar cooking) and improved cookstove designs (which reduce particulate matter by 50 to 80 percent) is the primary COPD prevention strategy for Nigerian women. Childhood lung health is also important: low birth weight, preterm birth, and recurrent childhood infections impair lung development and increase COPD risk.

Fill in the blank: Smoking cessation slows FEV1 decline from 60 to 30 mL per year; reduction of _____ fuel exposure through LPG promotion and improved cookstoves is the primary COPD prevention strategy for Nigerian women.

5.3.3 Prevention of lung cancer

Lung cancer prevention is primarily through tobacco control, as tobacco causes approximately 85 percent of lung cancer cases. Tobacco control strategies include taxation (10 percent price increase reduces consumption by approximately 4 percent in adults and 7 percent in young people), plain packaging, advertising bans, smoke-free laws, and health warning labels.

Low-dose CT (LDCT) screening is recommended in high-risk populations (age 50 to 80 years, 20 or more pack-year history) and reduces lung cancer mortality by approximately 20 percent (NLST and NELSON trials); it is not yet widely available in Nigeria. Other carcinogens requiring control include **asbestos** (strict banning of mining and use) and **radon gas** (second most common cause of lung cancer in non-smokers: test homes in high-risk geological areas).

Fill in the blank: Tobacco control (taxation, advertising bans, smoke-free laws) is primary lung cancer prevention; LDCT screening reduces mortality by 20 percent in high-risk patients; asbestos banning and _____ gas testing are additional measures.

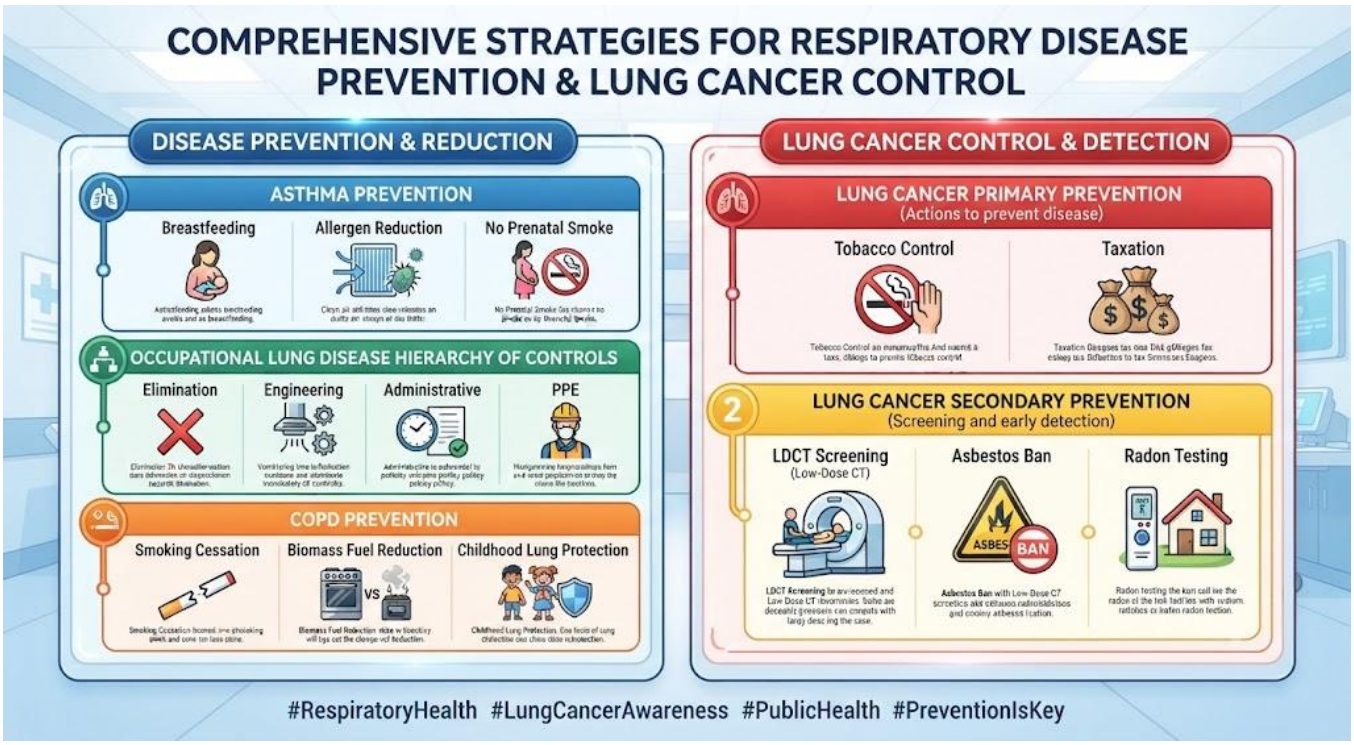


Figure 5.3: Prevention of asthma, occupational lung disease, COPD, and lung cancer

Pilot questions 5.3

1. Describe the primary prevention strategies for asthma. (Linked to SLO 5.3)
2. Describe the hierarchy of controls for occupational lung disease prevention. (Linked to SLO 5.3)
3. Explain why smoking cessation is the most important intervention in COPD prevention. (Linked to SLO 5.3)
4. Describe the role of biomass fuel reduction in COPD prevention in Nigeria. (Linked to SLO 5.3)
5. Describe the prevention strategies for lung cancer. (Linked to SLO 5.3)

5.4 Health Promotion and Respiratory Rehabilitation

5.4.1 Smoking cessation strategies

First-line pharmacotherapy for smoking cessation: **nicotine replacement therapy (NRT)** (patches, gum, lozenge, inhaler: doubles quit rates compared with placebo), **varenicline** (partial nicotinic receptor agonist: the most effective single pharmacotherapy; NNT approximately 5; caution in patients with psychiatric history), and **bupropion** (alternative when varenicline is contraindicated). Combined pharmacotherapy and behavioural support achieves quit rates of 20 to 30 percent at 12 months.

The 5 A's framework for clinical smoking cessation: Ask (ask about tobacco use at every visit), Advise (give clear, strong advice to quit), Assess (assess readiness to quit), Assist (offer pharmacotherapy and behavioural support), and Arrange (arrange follow-up within 1 to 2 weeks of quit date). Brief physician advice (3 minutes) increases the quit rate from 2 to 3 percent even without pharmacotherapy.

Fill in the blank: NRT doubles quit rates; varenicline is most effective (NNT approximately 5); 5 A's framework: Ask, Advise, Assess, _____, Arrange; combined pharmacotherapy and behavioural support achieves 20 to 30 percent quit rate.

5.4.2 Pulmonary rehabilitation

Pulmonary rehabilitation (PR) is a multidisciplinary programme of supervised exercise training, education, and behavioural support for patients with chronic respiratory disease, particularly COPD and interstitial lung disease. Supervised aerobic and resistance exercise training (at least 8 weeks) is the core component.

Evidence for PR is strong: it reduces dyspnoea and fatigue, improves 6-minute walk distance by approximately 50 metres, reduces hospital admissions by approximately 30 percent, and improves health-related quality of life. PR is indicated for COPD patients with MRC dyspnoea Grade 3 or above and should be offered within 4 weeks of a COPD exacerbation. In Nigeria, community-based walking programmes and physiotherapy-led outpatient programmes are feasible adaptations.

Fill in the blank: Pulmonary rehabilitation improves 6-minute walk distance by approximately 50 metres and reduces admissions by approximately 30 percent; indicated for MRC Grade _____ or above; offered within 4 weeks of a COPD exacerbation.

5.4.3 Community-level respiratory health promotion in Nigeria

Community-level interventions must address the major local risk factors. Indoor air pollution reduction: promotion of LPG and cleaner cooking fuels, distribution of improved biomass cookstoves, health education campaigns targeting women on respiratory risks of biomass combustion. TB control: community sensitisation to TB symptoms (cough above 2 weeks, night sweats, weight loss), stigma reduction, training community health workers to identify and refer presumptive TB cases, and DOT community support programmes.

Respiratory health promotion at the community level also includes vaccination campaigns (ensuring EPI completion: PCV13, Hib, influenza in priority groups), tobacco control education in schools and communities, occupational health education in high-risk workplaces, and training primary healthcare workers to identify and manage common respiratory conditions. Integration of respiratory health into existing HIV, maternal and child health, and NCD programmes maximises reach and minimises cost.

Fill in the blank: Community respiratory health promotion: indoor air pollution reduction (LPG, improved cookstoves), TB sensitisation (cough above 2 weeks), vaccination campaigns (PCV13, Hib), tobacco education in schools, integration with _____ and NCD programmes.

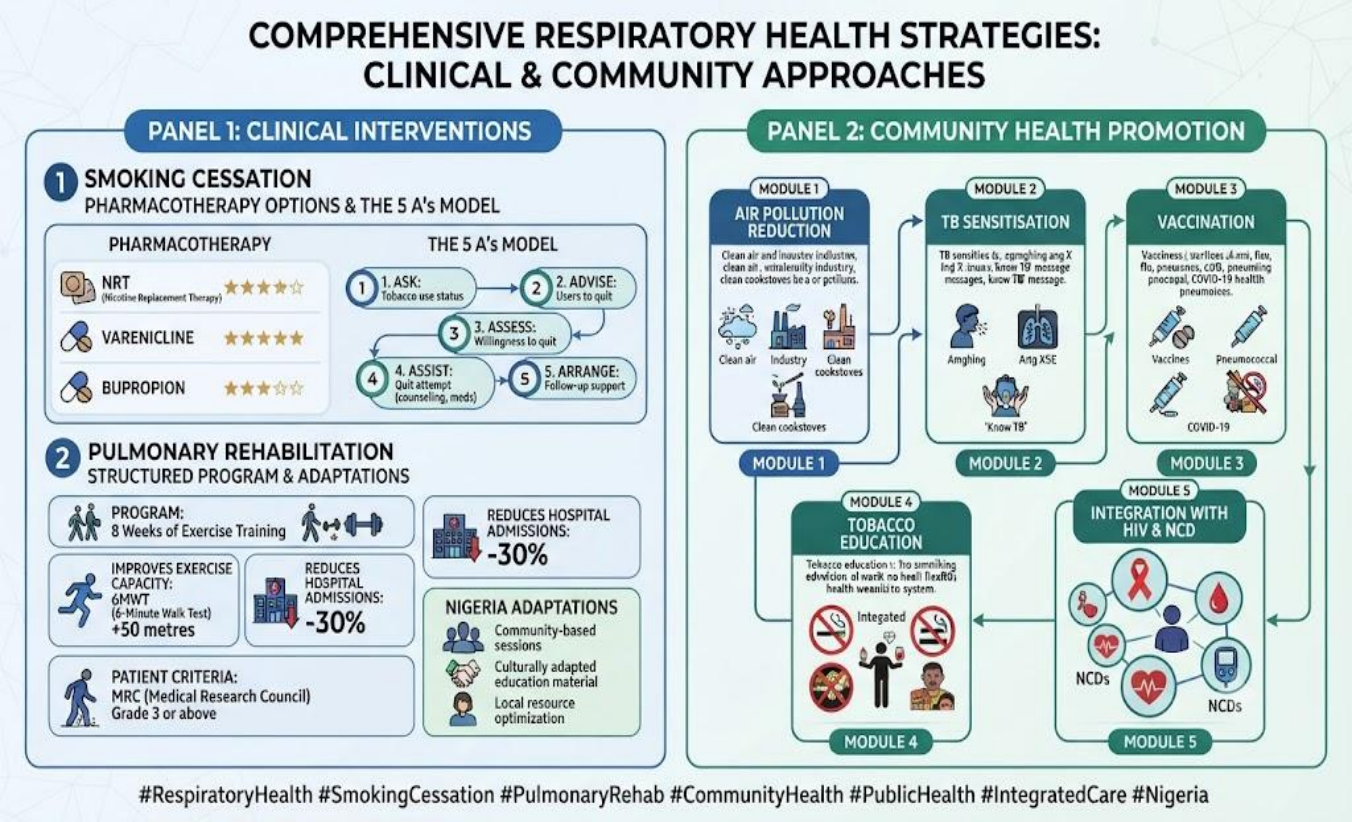


Figure 5.4: Smoking cessation, pulmonary rehabilitation, and community-level respiratory health promotion in Nigeria

Pilot questions 5.4

1. Describe the pharmacological options for smoking cessation and their relative effectiveness. (Linked to SLO 5.4)
2. Describe the 5 A's framework for smoking cessation counselling. (Linked to SLO 5.4)
3. Describe the evidence base and components of pulmonary rehabilitation. (Linked to SLO 5.4)
4. Describe community-level interventions to reduce indoor air pollution in Nigeria. (Linked to SLO 5.4)
5. Describe how TB prevention and control can be strengthened at the community level in Nigeria. (Linked to SLO 5.4)

Series Summary

Major respiratory risk factors: tobacco smoking (COPD, lung cancer, emphysema; rising in young Nigerian men), indoor air pollution from biomass fuel (COPD, pneumonia, lung cancer; 3.8 million deaths per year globally; major risk for Nigerian women and children), outdoor air pollution (PM2.5; COPD, infections), occupational exposures (pneumoconiosis: coal, silica, asbestos; occupational asthma: isocyanates, grain dust; farmer's lung: mouldy hay; mesothelioma: asbestos). Host factors: HIV (TB, PCP), malnutrition (cell-mediated immunity impairment), diabetes (TB reactivation and pneumonia severity), sickle cell disease (pneumococcal pneumonia from functional asplenia), alpha-1 antitrypsin deficiency (early emphysema). Pneumonia prevention: PCV13 (children under 2 and adults above 65), Hib vaccine (both in EPI), exclusive breastfeeding (reduces pneumonia by 15 percent), cotrimoxazole prophylaxis in HIV with CD4 below 200. TB control: BCG at birth; prompt treatment of smear-positive cases; DOT; contact tracing; TPT (isoniazid 6 months for PLHIV and close contacts); infection control in facilities. Influenza: annual vaccination (adults above 65, pregnant women, healthcare workers); hand hygiene reduces transmission 30 to 40 percent; N95 respirators for airborne pathogens.

Asthma prevention: reduce prenatal tobacco exposure, breastfeeding, allergen reduction, avoid antibiotic overuse. Occupational lung disease: hierarchy of controls (elimination, engineering, administrative, PPE); workplace surveillance. COPD prevention: smoking cessation (slows FEV1 decline from 60 to 30 mL per year); biomass fuel reduction (LPG, improved cookstoves) for Nigerian women. Lung cancer: tobacco control (taxation most effective: 10 percent increase reduces consumption 4 percent); LDCT screening reduces mortality 20 percent in high-risk patients; asbestos ban; radon testing. Smoking cessation: NRT doubles quit rate; varenicline most effective (NNT 5); 5 A's (Ask, Advise, Assess, Assist, Arrange); combined pharmacotherapy and support achieves 20 to 30 percent quit rate. Pulmonary rehabilitation: improves 6-minute walk distance by 50 metres; reduces admissions by 30 percent; indicated for MRC Grade 3 or above; offered within 4 weeks of COPD exacerbation. Community: LPG and improved cookstoves; TB sensitisation; EPI campaigns; tobacco education in schools; integration with HIV and NCD programmes.

Glossary of Terms

- **Biomass fuel:** organic material (wood, charcoal, animal dung, crop residues) burned for cooking; a major source of indoor air pollution and a leading cause of COPD and pneumonia in Nigerian women and children.
- **Pneumoconiosis:** occupational lung diseases caused by inhalation of mineral dusts: coal workers' pneumoconiosis (coal dust), silicosis (silica), and asbestosis (asbestos fibres).
- **Occupational asthma:** asthma caused by workplace exposure to sensitising agents including isocyanates, grain dust, and animal allergens; resolves or improves with removal from exposure.
- **Hypersensitivity pneumonitis (extrinsic allergic alveolitis):** inflammatory lung disease from repeated inhalation of organic antigens; farmer's lung (mouldy hay) and bird fancier's lung (avian proteins) are the classic forms.
- **BCG vaccine:** Bacillus Calmette-Guerin vaccine; given at birth in Nigeria; provides approximately 80 percent protection against severe childhood TB; less protective against adult pulmonary TB.
- **TB preventive therapy (TPT):** treatment of latent TB infection to prevent progression to active TB; standard regimen: isoniazid daily for 6 months; recommended for PLHIV and close contacts of smear-positive cases.
- **Pulmonary rehabilitation:** a multidisciplinary programme of supervised exercise training, education, and behavioural support for chronic respiratory disease; improves exercise capacity, dyspnoea, and quality of life.
- **Varenicline:** a partial nicotinic receptor agonist for smoking cessation; the most effective single pharmacotherapy; NNT approximately 5; caution in patients with psychiatric history.
- **5 A's framework:** a structured smoking cessation approach: Ask, Advise, Assess, Assist, Arrange.
- **Low-dose CT (LDCT) screening:** annual CT scan in high-risk individuals (age 50 to 80, 20 or more pack-year history) to detect early-stage lung cancer; reduces lung cancer mortality by approximately 20 percent.

- **Alpha-1 antitrypsin deficiency:** a genetic disorder causing deficiency of a serine protease inhibitor; predisposes to early-onset emphysema, particularly in smokers.
- **Nicotine replacement therapy (NRT):** pharmacotherapy delivering controlled nicotine via patch, gum, lozenge, or inhaler to reduce withdrawal symptoms; doubles quit rates compared with placebo.

Concept Check Questions [Series 5]

Objective Questions

1. Indoor air pollution from _____ fuel combustion causes COPD and pneumonia predominantly in Nigerian women and children. (Linked to SLO 5.1)
2. Farmer's lung from mouldy hay is caused by _____ pneumonitis (extrinsic allergic alveolitis); mesothelioma is caused by asbestos exposure with a 20 to 40-year latency. (Linked to SLO 5.1)
3. BCG vaccination at birth protects against severe childhood TB; TB preventive therapy using isoniazid for _____ months is recommended for PLHIV and close contacts. (Linked to SLO 5.2)
4. _____ is the most effective single pharmacotherapy for smoking cessation with NNT approximately 5; NRT doubles quit rates compared with placebo. (Linked to SLO 5.4)
5. Pulmonary rehabilitation improves 6-minute walk distance by approximately 50 metres and reduces admissions by approximately 30 percent; indicated for MRC Grade _____ or above. (Linked to SLO 5.4)
6. LDCT screening reduces lung cancer mortality by approximately 20 percent; tobacco _____ (a 10 percent price increase reduces consumption by 4 percent) is the most effective population-level prevention strategy. (Linked to SLO 5.3)

Short-Answer Questions

7. Describe the respiratory effects of indoor air pollution from biomass fuel in Nigeria. (Linked to SLO 5.1)
8. Describe the components of TB control beyond BCG vaccination. (Linked to SLO 5.2)
9. Describe the 5 A's framework for smoking cessation. (Linked to SLO 5.4)

Long-Answer Questions

10. Describe the major risk factors for respiratory disease in Nigeria and the preventive strategies for each. (Linked to SLOs 5.1 to 5.3)
11. Describe pulmonary rehabilitation and community-level respiratory health promotion in Nigeria. (Linked to SLO 5.4)

Answers to Concept Check Questions [Series 5]

Objective Questions

1. Biomass.
2. Hypersensitivity.
3. 6.
4. Varenicline.
5. 3.
6. Taxation.

Short-Answer Questions

7. Biomass combustion produces PM2.5, CO, and volatile organic compounds. Chronic inhalation causes neutrophilic airway inflammation, mucus hypersecretion, emphysema (COPD), increased pneumonia susceptibility, and lung cancer. Predominantly affects women and children cooking indoors without adequate ventilation.
8. Prompt treatment of smear-positive cases (prevents 10 to 15 new infections per treated case). DOT (ensures adherence; prevents acquired drug resistance). Contact tracing (identifies exposed individuals). TB preventive therapy: isoniazid 6 months for PLHIV and close contacts. Infection control in health facilities: natural ventilation, UV irradiation, N95 respirators.
9. Ask: enquire about tobacco use at every clinical visit. Advise: give clear, strong advice to quit. Assess: determine readiness to quit. Assist: offer pharmacotherapy (NRT, varenicline, or bupropion) and behavioural support. Arrange: schedule follow-up within 1 to 2 weeks of the quit date.

Long-Answer Questions

10. **Basic response:** Tobacco smoking: causes COPD, lung cancer, emphysema; prevention through taxation, smoke-free laws, and cessation support. Biomass fuel: causes COPD, pneumonia, lung cancer in Nigerian women and children; prevention through LPG promotion, improved cookstoves, and health education. Occupational exposures: pneumoconiosis (coal, silica, asbestos), occupational asthma (isocyanates), mesothelioma (asbestos); prevention through hierarchy of controls and workplace surveillance.

Infections: childhood infections impair lung development; TB causes structural damage; prevention through BCG, prompt treatment, DOT, TPT, and infection control. Host factors: HIV (cotrimoxazole prophylaxis and ART), malnutrition (nutritional support), diabetes (glucose control reduces TB reactivation risk), sickle cell disease (PCV13 vaccination).

Value-added response: In Nigeria, the highest-impact prevention investments are biomass fuel reduction for women, TB case-finding and treatment, PCV13 vaccination in children, and smoking cessation support in young men.

11. **Basic response:** Pulmonary rehabilitation: multidisciplinary programme for COPD and ILD patients; core component is supervised aerobic and resistance exercise for at least 8 weeks; education (disease understanding, inhaler technique, energy conservation) and psychological support are additional components. Evidence: improves 6-minute walk distance by 50 metres, reduces dyspnoea and fatigue, reduces hospital admissions by 30 percent, and improves quality of life; indicated for MRC Grade 3 or above; offered within 4 weeks of COPD exacerbation; Nigeria adaptations include community walking programmes and physiotherapy-led outpatient programmes. Community health promotion: indoor air pollution reduction (LPG promotion, improved cookstoves, kitchen ventilation education targeting women); TB sensitisation (community health worker training to recognise cough above 2 weeks, night sweats, weight loss; stigma reduction; DOT community support); vaccination campaigns (PCV13 and Hib for children; influenza for priority groups); tobacco control education in schools; integration with HIV, maternal and child health, and NCD platforms to maximise reach.

    Value-added response: Community-based pulmonary rehabilitation and respiratory health promotion, delivered through Nigeria's primary healthcare system and community health workers, offers the most scalable and cost-effective approach to reducing the respiratory disease burden at the population level.

Answers to Pilot Questions [Series 5]

Part 5.1

1. Tobacco smoke damages bronchial epithelium, impairs mucociliary clearance, and activates neutrophils causing chronic airway inflammation and mucus hypersecretion

(chronic bronchitis). Neutrophil elastase destroys alveolar walls causing emphysema (COPD). Carcinogens (polycyclic aromatic hydrocarbons, nitrosamines) cause DNA damage in bronchial epithelium leading to lung cancer. Smoking also impairs alveolar macrophage function, increasing TB susceptibility.

2. Biomass fuel combustion produces PM2.5, CO, nitrogen oxides, and volatile organic compounds at concentrations exceeding WHO indoor air quality guidelines. Chronic inhalation causes neutrophilic airway inflammation, emphysema, and COPD; impairs mucociliary clearance predisposing to pneumonia (increased child mortality under 5); and causes lung cancer from carcinogenic combustion products. Predominantly affects Nigerian women and children who cook indoors. COPD prevalence in non-smoking biomass fuel users approaches that in male smokers.
3. Coal dust: coal workers' pneumoconiosis. Silica: silicosis. Asbestos: asbestosis and mesothelioma (latency 20 to 40 years). Isocyanates (spray painting, polyurethane manufacturing): occupational asthma.
4. HIV depletes CD4 T-lymphocytes, impairing cell-mediated immunity. Consequences: 20 to 30-fold increased risk of TB reactivation; susceptibility to PCP (Pneumocystis jirovecii pneumonia); increased severity of bacterial pneumonia from impaired opsonisation; pulmonary Kaposi sarcoma in advanced AIDS.
5. HIV (TB, PCP, bacterial pneumonia). Malnutrition (impairs T-lymphocyte function and macrophage activation; increases TB and pneumonia risk). Diabetes mellitus (impairs neutrophil function; increases TB reactivation 2 to 3-fold). Sickle cell disease (functional asplenia increases invasive pneumococcal disease risk; PCV13 vaccination essential). Alpha-1 antitrypsin deficiency (impairs neutrophil elastase inhibition; accelerates emphysema, especially with smoking).

Part 5.2

1. PCV13: targets 13 pneumococcal serotypes; for all children under 2 years (primary series at 6, 10, 14 weeks in Nigerian EPI) and adults above 65 or with chronic disease; reduces pneumococcal pneumonia by 45 to 75 percent. Hib vaccine: targets Haemophilus influenzae type b; given as pentavalent vaccine in Nigerian EPI; prevents pneumonia, meningitis, and epiglottitis.

2. BCG provides approximately 80 percent protection against severe childhood TB (miliary TB and TB meningitis) by limiting haematogenous dissemination. It does not prevent initial infection or significantly protect against adult pulmonary TB. Its protective efficacy varies geographically and is less effective in tropical regions. Nigeria gives BCG at birth through the EPI.
3. Prompt treatment of smear-positive cases (prevents 10 to 15 new infections per year). DOT (ensures adherence; prevents acquired resistance). Contact tracing (screens household contacts; offers TPT). TPT: isoniazid 6 months or isoniazid-rifapentine 3 months weekly for PLHIV and close contacts. Infection control: natural ventilation in health facilities, UV irradiation in waiting areas, N95 respirators for healthcare workers.
4. Influenza vaccination: annual for high-risk groups (adults above 65, pregnant women, immunocompromised, chronic respiratory or cardiac disease, healthcare workers); reduces influenza hospitalisations by 50 to 60 percent; updated annually to match circulating strains. Non-pharmacological: hand hygiene (soap and water or alcohol hand rub: reduces transmission 30 to 40 percent); respiratory etiquette (cover mouth and nose; use tissues); face masks (surgical: droplet protection; N95: airborne pathogens); ventilation (open windows in crowded spaces).
5. TPT treats latent TB infection to prevent progression to active disease. Indications: PLHIV (regardless of TST result in high-burden settings: 20 to 30-fold increased reactivation risk), close household contacts of smear-positive cases (especially children under 5), and immunosuppressed patients starting TNF inhibitors. Regimens: isoniazid 300 mg daily for 6 months (most widely used in Nigeria) or isoniazid-rifapentine once weekly for 3 months. Screen for active TB before starting.

Part 5.3

1. Avoidance of prenatal tobacco smoke exposure (impairs lung development and increases asthma risk). Exclusive breastfeeding for 6 months (reduces early respiratory infections and atopic sensitisation). Reduction of indoor allergen load (house dust mite: mattress covers, reduce humidity). Avoidance of antibiotic overuse in infancy (preserves gut microbiome diversity, reducing allergic sensitisation).

2. Elimination or substitution: replace the hazardous substance (most effective; e.g., replace asbestos with safer alternatives). Engineering controls: enclose the process; use local exhaust ventilation to capture dust and fumes at source. Administrative controls: job rotation to limit exposure duration; health surveillance with regular spirometry and CXR to detect early disease. Personal protective equipment: dust respirators as last resort; least reliable because it depends on correct use at all times.
3. Smoking cessation slows the accelerated annual FEV1 decline in susceptible smokers from approximately 60 mL per year (continuing smokers) to approximately 30 mL per year (ex-smokers), approaching the rate in non-smokers. Since COPD is defined by FEV1/FVC below 0.70, slowing FEV1 decline delays or prevents reaching this threshold. Cessation at any GOLD stage slows further decline and reduces exacerbation frequency, cardiovascular risk, and all-cause mortality.
4. Biomass fuel combustion produces PM2.5 at concentrations comparable to or exceeding tobacco smoke in poorly ventilated kitchens. Prevention: promotion of LPG and cleaner fuels (most effective but limited by cost and availability); improved biomass cookstoves (rocket stoves, gasifier stoves: reduce PM2.5 by 50 to 80 percent); health worker education to identify non-smoking women with COPD and enquire about cooking fuel; government policies supporting fuel subsidies.
5. Primary prevention: tobacco control (taxation most effective: 10 percent price increase reduces adult consumption by 4 percent and youth by 7 percent; plain packaging; advertising bans; smoke-free laws; graphic health warnings). Secondary prevention: LDCT screening annually in age 50 to 80 years with 20 or more pack-year history; NLST and NELSON trials demonstrate 20 to 24 percent reduction in lung cancer mortality; not yet widely available in Nigeria. Additional: strict asbestos banning; radon gas testing in high-risk areas.

Part 5.4

1. NRT (patches, gum, lozenge, inhaler): reduces withdrawal symptoms; doubles quit rates vs placebo (approximately 10 to 15 percent vs 5 to 7 percent at 12 months); can combine patch plus short-acting NRT; safest in pregnancy and cardiovascular disease. Varenicline: partial nicotinic receptor agonist; most effective single agent (20 to 30 percent quit rate at

12 months; NNT approximately 5); caution in psychiatric history. Bupropion: alternative when varenicline is contraindicated; 15 to 20 percent quit rate. Combined pharmacotherapy plus behavioural support achieves highest quit rates.

2. Ask: ask every patient about tobacco use at every clinical visit. Advise: give clear, strong, personalised advice to quit. Assess: ask about readiness to make a quit attempt. Assist: help set a quit date; prescribe pharmacotherapy; provide or refer for behavioural support. Arrange: follow-up within 1 to 2 weeks of the quit date to review progress and manage relapse.
3. Components: supervised aerobic and resistance exercise training (at least 8 weeks: the core); education (disease understanding, inhaler technique, energy conservation, self-management); psychological support; smoking cessation. Evidence: improves 6-minute walk distance by approximately 50 metres (clinically meaningful threshold 25 to 30 metres), reduces hospital admissions by approximately 30 percent, reduces dyspnoea and fatigue, and improves quality of life. Indications: COPD with MRC Grade 3 or above; offered within 4 weeks of a COPD exacerbation.
4. LPG promotion (government subsidies, community distribution networks). Improved biomass cookstoves: distribution of rocket stoves and gasifier stoves (reduce PM2.5 by 50 to 80 percent); training on correct use and maintenance. Education campaigns: community health worker visits targeting women on respiratory risks of biomass fuel; radio and social media campaigns. Kitchen ventilation education: importance of opening windows and doors while cooking; advocacy for ventilation standards in new building construction.
5. Community sensitisation: training community health workers to recognise presumptive TB (cough above 2 weeks, night sweats, weight loss, haemoptysis) and refer for GeneXpert testing. Stigma reduction: community dialogue sessions in churches, mosques, and community centres to address TB stigma (which delays care-seeking by 2 to 3 months). DOT support: community-based DOT using trained community members to improve completion rates above 90 percent. Contact tracing: household investigation of all smear-positive cases; offer TPT to contacts with latent TB. Integration with HIV services: intensified TB case-finding in all HIV clinics; HIV testing in all TB patients.

References and Attributions [Series 5]

Textual References

- World Health Organization. (2021). WHO Global Air Quality Guidelines. Geneva: WHO.
- World Health Organization. (2022). WHO Consolidated Guidelines on Tuberculosis. Module 1: Prevention. Geneva: WHO.
- Global Initiative for Chronic Obstructive Lung Disease (GOLD). (2023). Global Strategy for COPD. Available at: www.goldcopd.org.
- Kumar, P. and Clark, M. (2017). Kumar and Clark's Clinical Medicine (9th ed.). Edinburgh: Elsevier.

Figures, Plates, Tables, and Boxes

- Figure 5.1: Risk factors for respiratory disease in Nigeria. AI prompt: "Two-panel diagram: left four-row table (tobacco, indoor air pollution biomass fuel, outdoor air pollution, occupational exposures); right three-row table (infections, host immune factors: HIV and malnutrition and diabetes, genetic factors: sickle cell disease and alpha-1 antitrypsin deficiency); clean professional infographic." OER search: "risk factors respiratory disease Nigeria tobacco biomass fuel occupational infection host diagram".
- Figure 5.2: Prevention of respiratory infections. AI prompt: "Two-panel diagram: left (pneumonia: PCV13, Hib, breastfeeding, cotrimoxazole; TB: BCG, prompt treatment, DOT, TPT, infection control); right (influenza vaccination; non-pharmacological: hand hygiene, masks, ventilation); clean infographic." OER search: "prevention pneumonia TB influenza vaccination hand hygiene respiratory infection diagram".
- Figure 5.3: Prevention of chronic respiratory disease and lung cancer. AI prompt: "Two-panel diagram: left (asthma, occupational lung disease hierarchy of controls, COPD: smoking cessation and biomass reduction); right (lung cancer: tobacco control and taxation; LDCT screening; asbestos ban; radon testing); clean infographic." OER search: "prevention asthma occupational lung disease COPD lung cancer tobacco control LDCT screening diagram".
- Figure 5.4: Smoking cessation, pulmonary rehabilitation, and community health promotion. AI prompt: "Two-panel diagram: left (smoking cessation: NRT, varenicline, bupropion; 5 A's framework; pulmonary rehabilitation: 8 weeks exercise, 50 metres

6MWT, 30 percent admissions reduction); right (community health promotion: air pollution reduction, TB sensitisation, vaccination, tobacco education, integration with HIV and NCD); clean infographic." OER search: "smoking cessation varenicline NRT pulmonary rehabilitation community health promotion Nigeria diagram".