FINAL MBBS · GENERAL MEDICINE · LONG CASE

A E T C M

COPD Case Sheet

A Complete Respiratory Case — from History to Viva

A fully worked case sheet of chronic obstructive pulmonary disease in acute exacerbation with cor pulmonale and hypercapnic respiratory failure, written the way a final-year MBBS student should record and present it in the university examination — with the reasoning behind every question asked and every sign elicited.

History of presenting illness › | Relevant negatives › | Smoking & biomass exposure › | General examination ›
Cor pulmonale & CO_2 retention › | Inspection — barrel chest › | Percussion & auscultation › | Signs of hyperinflation ›
Final diagnosis & GOLD staging › | Differential diagnosis › | Spirometry (PFT) › | Chest X-ray, CT & ECG › | ABG ›
Acute exacerbation & NIV › | Stable COPD — inhalers › | Oxygen, rehab & vaccines › | Model presentation ›
Viva questions ›

TAP A TOPIC TO JUMP TO IT

HISTORY smoking · biomass · exacerbations › **EXAMINATION** hyperinflation · cor pulmonale › **PFT & IMAGING** FEV_1/FVC · X-ray · ABG › **MANAGEMENT** acute · stable · oxygen

SEE THE SIGNS ONLINE — TAP TO OPEN IMAGE / X-RAY / AUDIO

Barrel chest ↗ | Pursed-lip breathing ↗ | Tripod position ↗ | Hoover's sign ↗ | Central cyanosis ↗ | Clubbing ↗
Wheeze (audio) ↗ | COPD chest X-ray ↗ | CT emphysema ↗ | Flow–volume loop ↗ | ECG in COPD ↗
All signs — visual atlas ›

AETCM
Academy for Emergency, Trauma & Critical care Medicine

By

Dr. Gireesh Kumar K.P.

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▶ Respiratory exam & viva | ▶ Respiratory system examination | ▶ Breathlessness

For final-year MBBS students, interns and teachers of clinical medicine

COPD Case Sheet

A complete respiratory long case for final MBBS preparation and presentation

Dr. Gireesh Kumar K.P.
Academy for Emergency, Trauma & Critical care Medicine (AETCM) · First edition · October 2026

How to use this case sheet

Chronic obstructive pulmonary disease (COPD) is the commonest respiratory long case in the final MBBS General Medicine examination. It tests whether a student can take a careful exposure history, elicit and **explain** the signs of hyperinflation, recognise cor pulmonale and carbon-dioxide retention, interpret spirometry and an arterial blood gas, and manage both the acute exacerbation and the stable disease. This booklet presents one complete, fully worked case exactly as it should be written in the case record and presented at the bedside.

Each section has three layers: the **case record** itself (what you write), **green boxes** that summarise and interpret (what you conclude), and **blue boxes** with examiner's tips (what you are judged on). Tables of signs, phenotypes, GOLD grading, differential diagnosis and drug therapy turn the single case into a revision of the whole topic. The book ends with a **model presentation** to practise aloud and **38 viva questions** with short answers.

New in this case sheet — tap to see the sign

Wherever a sign, X-ray, CT, ECG or spirometry finding is described, a small button opens a free online image, radiograph or sound recording: ▶ Photo clinical sign ▶ X-ray imaging ▶ Listen breath-sound audio. A one-page **visual atlas** with QR codes is provided for the printed copy.

The patient described is a composite teaching case; details have been chosen to illustrate the commonest examination scenario. Examine your own patient carefully and record only what you find. Drug doses follow GOLD 2026 and standard Indian practice — always check local protocols.

Contents

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SECTION A · HISTORY

Case Sheet — COPD (Long Case, Respiratory)

Final MBBS General Medicine · Case-taking and presentation format

1. Particulars of the patient

Name	Mr. V. M. (initials only — always respect confidentiality)
Age / Sex	64 years / Male
Occupation	**Worked 22 years in a roofing-tile factory (kiln smoke and clay dust)**; now an agricultural labourer — always record the **occupation in full**, past and present
Address	Ottapalam, Palakkad district, Kerala (rural; house with a wood-fired kitchen)
Religion / Education	Hindu / Studied up to 7th standard
Informant	Patient and his son — **reliable** (patient was drowsy on admission; history re-checked on day 3 when alert)
Date of admission / examination	Admitted through casualty; examined on day 3 of admission

2. Chief complaints (in chronological order, in the patient's own words)

- **Cough with expectoration** — 8 years; increased in amount and became yellowish-green — 5 days
- **Breathlessness on exertion** — 6 years; increased, now even at rest — 5 days
- **Wheezing (noisy breathing)** — 3 years; worse — 5 days
- **Swelling of both legs** — 10 days
- **Low-grade fever** — 4 days
- **Headache on waking and drowsiness** — 2 days

> **Examiner's tip — two time-lines**
>
> In every COPD case separate the **chronic baseline illness** (years) from the **acute exacerbation** (days) that brought the patient to hospital. Write symptoms, never a diagnosis: ‘breathlessness on exertion × 6 years’ is right; ‘COPD × 6 years’ is wrong.

3. History of presenting illness

The chronic illness. The patient was apparently well 8 years ago, when he developed **cough with mucoid, whitish expectoration** (about 1–2 tablespoons a day), worst in the early morning and during the monsoon months. The cough was present on most days for **more than 3 months in each year for 2 consecutive years** — the clinical definition of **chronic bronchitis**. There was no haemoptysis.

Six years ago he noticed **breathlessness on exertion** — first only while climbing the hill to his field. It was **insidious in onset and slowly progressive**: 2 years ago he had to stop after walking about 100 m on level ground (**mMRC grade 3**), which has been his baseline since. Breathlessness is worse with exertion, cold air, smoke from the kitchen and colds, and is partly relieved by rest, sitting forward and his inhaler. There is **no diurnal or seasonal variation** typical of asthma, no sneezing or atopy, and he never had breathlessness as a child.

For 3 years he has had **wheezing** — noisy breathing heard by his wife, worse during colds, partially relieved by his blue inhaler. He has had **two hospital admissions in the past 12 months** for similar worsening — one needed a ‘mask machine’ (NIV) — and one outpatient course of tablets and antibiotics. He has never been intubated.

The present exacerbation. Five days ago, after a cold with running nose and sore throat, his breathlessness worsened over 2 days until he was **breathless at rest** and could speak only in short phrases. He could not lie flat and slept sitting up, leaning on a table (no episodes of waking up gasping — i.e. no PND). The **sputum increased** to about half a cup a day and became **thick and yellowish-green**. He had **low-grade fever** without chills for 4 days. He used his reliever inhaler 8–10 times a day with little relief.

Ten days ago both legs began to **swell**, starting at the ankles and rising to mid-shin, with heaviness in the right upper abdomen and reduced urine output. For 2 days before admission he had **headache on waking, daytime drowsiness and, one night, confusion** — his son noticed his hands ‘twitching’. He was brought to casualty, where his SpO_2 was 82 % on room air. He was given controlled oxygen, nebulisation, steroids and antibiotics, and **non-invasive ventilation (BiPAP) for 18 hours**. He is now on 1 L/min oxygen by nasal prongs, alert, and feels much better.

Breathlessness — grade it, always

mMRC grade	Description	In this patient
0	Breathless only with strenuous exercise	
1	Breathless when hurrying on level ground or walking up a slight hill	
2	Walks slower than people of the same age because of breathlessness, or has to stop for breath when walking at own pace	
3	**Stops for breath after walking about 100 m or after a few minutes on level ground**	**Baseline (stable state)**
4	Too breathless to leave the house, or breathless when dressing or undressing	During the present exacerbation

The mMRC grade and the **CAT score** (8 items, 0–40) ▶ Tool are used to classify symptom burden in GOLD. Grade the **stable baseline**, not the exacerbation. This patient's CAT score at the last stable visit was **24** (high impact).

History of presenting illness — relevant positives and negatives

In every case of COPD the history must answer five questions: **(1) Is it chronic airflow obstruction? (2) What caused it** — tobacco, biomass, occupation, previous TB? **(3) Is this an exacerbation, and what triggered it? (4) Are there complications** — cor pulmonale, CO_2 retention, polycythaemia? **(5) Is there another disease mimicking or accompanying it?**

Question asked	Answer in this patient	Why it is asked
Childhood wheeze, allergic rhinitis, eczema, diurnal variation, symptom-free periods?	No	**Bronchial asthma** — variable, reversible, younger onset
Large volumes of sputum daily (cupfuls), foul-smelling, all through the year; recurrent pneumonias?	Sputum only half a cup during exacerbation; mucoid otherwise	**Bronchiectasis**
Haemoptysis?	No	Tuberculosis, lung cancer, bronchiectasis, PE
Evening fever, night sweats, weight loss, TB contact, previous anti-TB treatment?	**Weight loss 4 kg in 1 year**, appetite reduced; no night sweats; no previous TB	**Pulmonary TB / post-TB obstructive airway disease** (common in India); COPD cachexia
Hoarseness of voice, dysphagia, bone pain, change in cough?	No	**Lung cancer** — shares the same risk factor
Sudden sharp chest pain with sudden worsening of breathlessness?	No	**Pneumothorax** (ruptured bulla), pulmonary embolism
Orthopnoea, PND, angina, palpitations?	Sits up to breathe; **no PND**, no angina, no palpitations	Left ventricular failure, IHD, arrhythmia (AF, MAT)
Leg swelling, abdominal distension, right-upper-abdominal heaviness, reduced urine?	**Yes — 10 days**	**Cor pulmonale** (right heart failure)
Morning headache, drowsiness, confusion, tremor of hands?	**Yes — 2 days before admission**	**Hypercapnia (CO_2 narcosis)** — type 2 respiratory failure
Loud snoring, witnessed pauses in breathing, daytime sleepiness?	Snores; no witnessed apnoea	**Overlap syndrome** (COPD + obstructive sleep apnoea)
Progressive dry cough, joint pains, dust or bird exposure?	Clay dust at work only	Interstitial lung disease, pneumoconiosis
Calf pain or swelling of one leg, recent immobility?	No (both legs swollen equally)	DVT/PE — common, often missed, in AECOPD
Drugs — β-blockers (propranolol), aspirin, ACE inhibitor cough?	Amlodipine only	Drug-induced bronchospasm or cough
Bowel, bladder, sleep?	Sleep disturbed (sits up at night)	Nocturnal hypoxaemia, OSA; urinary retention with anticholinergics

4. Past history

- **Hypertension** — 5 years; on amlodipine 5 mg OD, regular.
- **Pneumonia** treated with admission 3 years ago. Two COPD hospitalisations in the past year (one with NIV). Never intubated.
- No history of pulmonary tuberculosis, asthma, diabetes, coronary artery disease, chest injury or chest surgery.

5. Treatment history — and how he uses his inhalers

Drug	How he actually takes it
Tiotropium 18 µg rotacap OD (LAMA)	**Irregular** — stops when he feels better; 'it is costly'
Salbutamol pMDI 100 µg, 2 puffs as needed	Uses **without a spacer**; fires the puff before breathing in; no breath-hold; 8–10 times a day this week
Doxofylline 400 mg tablets	Bought from the local pharmacy when breathless
Oral prednisolone	**3 short courses in the last year** (steroid side-effects — weight gain absent; check glucose, bones)
Home oxygen, pulmonary rehabilitation, vaccinations	None — **never vaccinated** against influenza or pneumococcus

Examiner's tip — ask him to show you his inhaler

Up to two-thirds of patients use their inhaler wrongly. 'Ask the patient to demonstrate the technique' is the single most useful step before escalating therapy — and examiners love to see it. Also record **adherence, cost and device**, the number of steroid courses and antibiotic courses, and vaccination status.

6. Personal history

Smoking	**Bidi smoker — 20 bidis a day from age 18 to 60 (42 years); stopped 4 years ago** after his first admission
Smoking index	20 bidis/day × 42 years = **840** (>300 = heavy smoker) · ≈ **42 pack-years** (20 sticks = 1 pack)
Biomass smoke	Wife cooks on a **firewood chulha** in a kitchen without a chimney — household exposure for over 40 years
Occupational	Tile-factory kiln smoke and clay dust × 22 years, without a mask
Alcohol	Occasional toddy in the past; none for 10 years
Diet / appetite	Mixed diet; **appetite reduced**; 4 kg weight loss in 1 year
Sleep / bowel / bladder	Sleep disturbed by breathlessness; bowel and bladder normal

Smoking index and pack-years — how to calculate

Pack-years = (number of cigarettes per day ÷ 20) × years smoked. ≥10 pack-years is a significant risk for COPD; ≥20 pack-years qualifies for lung-cancer screening. The **smoking index** (used in India, includes bidis) = number of bidis/cigarettes per day × years smoked: <100 light, 101–300 moderate, >300 heavy. One bidi delivers as much or more tar and carbon monoxide as a cigarette. Also ask about hookah, chillum, chewing tobacco (not a COPD risk) and **passive smoking**.

7. Family and socio-economic history

Father, a chronic smoker, died at 68 of 'lung disease'. No family history of asthma, early emphysema, liver disease or tuberculosis contact (**α_1-antitrypsin deficiency** is suspected when emphysema occurs before 45 years, in a non-smoker, with basal panacinar emphysema, or with a family history). He lives with his wife, son and grandchildren in a two-room house; lower socio-economic class (modified Kuppuswamy scale); covered under a state health-insurance scheme. Daily wage income lost during each exacerbation — relevant when choosing affordable inhalers.

Risk factors for COPD in this patient

Present	Absent / to be checked	Indian context
Tobacco — bidi, 42 pack-years · household biomass smoke · occupational kiln smoke and dust · age >40 · recurrent chest infections · low socio-economic status	Previous TB (none) · α_1-antitrypsin deficiency (not suspected) · childhood asthma (none) · low birth weight / childhood infections (unknown)	About half of COPD in India is in **non-smokers** — biomass fuel (especially women), outdoor air pollution and **post-tuberculosis lung disease** are major causes.

8. Summary of history

Summary (say this before you start the examination)

A **64-year-old man**, an **ex-bidi smoker (42 pack-years, stopped 4 years ago)** with long-standing **household biomass** and **occupational kiln-smoke exposure**, a known hypertensive, presents with **chronic productive cough for 8 years**, **progressive exertional breathlessness for 6 years (baseline mMRC 3)** and **wheeze for 3 years**, with **two hospitalisations for exacerbations in the past year**. For **5 days** after an upper respiratory infection he has had **increased breathlessness, increased and purulent sputum and low-grade fever**, with **bilateral leg swelling for 10 days** and **morning headache, drowsiness and confusion** before admission. There is no haemoptysis, history of TB, atopy, chest pain or PND. He uses his inhalers irregularly and with poor technique, and has never been vaccinated.

9. Provisional diagnosis from history

Chronic obstructive pulmonary disease — chronic bronchitis predominant — in acute exacerbation, probably infective (Anthonisen type 1), with features of chronic cor pulmonale and hypercapnic (type 2) respiratory failure, in an ex-bidi smoker with biomass and occupational exposure; frequent exacerbator.

How the history leads to the diagnosis — the reasoning

Clue from history	Inference
Age >40, heavy smoking, biomass and dust exposure	The setting for **COPD** — risk factor + symptoms is the indication for spirometry
Productive cough >3 months a year for 2 consecutive years	**Chronic bronchitis** (a clinical definition — airflow obstruction must still be proved by spirometry)
Slowly progressive, persistent exertional breathlessness, no symptom-free periods	**Fixed (persistent) airflow limitation** rather than variable asthma
No childhood onset, atopy or diurnal variation	Asthma less likely (though asthma and COPD can coexist)
Worse breathlessness + more sputum + purulent sputum after a cold	**Acute exacerbation** — all three cardinal symptoms = **Anthonisen type 1** → antibiotics indicated
Bilateral leg swelling, right-upper-abdominal heaviness	**Right heart failure due to lung disease — cor pulmonale**
Morning headache, drowsiness, confusion, twitching hands	**CO_2 retention** (hypercapnic encephalopathy, asterixis)
Two admissions in 12 months, one needing NIV	**Frequent exacerbator; GOLD group E** — high risk of death and future exacerbations
Weight loss, poor appetite	Advanced COPD (cachexia, poor prognosis) — but **exclude TB and lung cancer** first

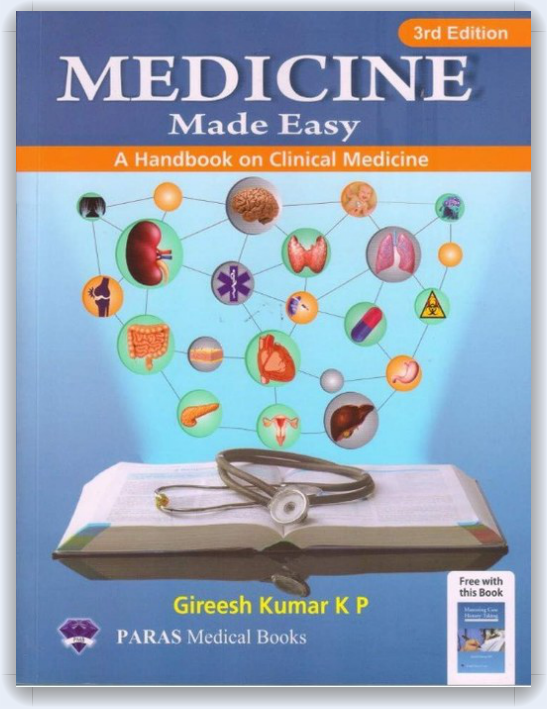
Examiner's tip — what the examiner looks for in the history

(1) **Smoking quantified** in pack-years / smoking index, and biomass and occupational exposure. (2) **mMRC grade** at baseline. (3) **Number of exacerbations and hospitalisations** in the last year, and any NIV or intubation. (4) Symptoms of **cor pulmonale and CO_2 retention**. (5) Negatives for **asthma, bronchiectasis, TB and lung cancer**. (6) **Inhaler use and technique**, steroid courses, vaccination.

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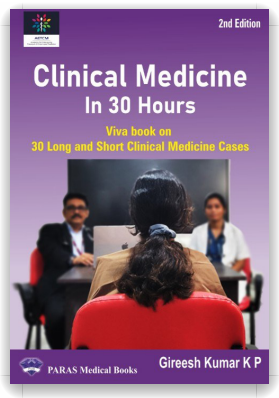
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SECTION B · EXAMINATION

General Physical Examination

Look for respiratory distress, hypoxia, CO_2 retention, cor pulmonale and the clues to another diagnosis

Consciousness / orientation	Conscious, alert and oriented today (drowsy on admission — GCS 13)
Position (decubitus)	**Sitting up, leaning forward with hands on the knees — tripod position** ▶ Photo fixes the shoulder girdle so that the accessory muscles can work
Respiratory distress	Breathless at rest; speaks in short sentences; **pursed-lip breathing** ▶ Photo; accessory muscles active
Built / nourishment	Thin; height 162 cm, weight 51 kg, **BMI 19.4 kg/m²** (<21 = poor prognosis in COPD — the 'B' of BODE); mid-arm circumference 22 cm
Hands	**Tar (nicotine) staining** of right index and middle fingers; warm peripheries; no clubbing; no flap today

Sign	Finding	Relevance
Cyanosis	**Central cyanosis present** — tongue, lips (mild); peripheral also present ▶ Photo	Hypoxaemia — reduced Hb >5 g/dL; easier to see with polycythaemia
Clubbing	**Absent** — Schamroth's window preserved ▶ Photo ▶ Schamroth	**COPD does not cause clubbing** — if present, look for lung cancer, bronchiectasis, ILD
Pallor / plethora	No pallor; **plethoric, suffused conjunctivae**	Secondary polycythaemia due to chronic hypoxia
Icterus	Absent	Mild icterus may occur with congestive hepatopathy
Lymph nodes	Not enlarged (supraclavicular, cervical, axillary)	Lung cancer, TB
Oedema	**Bilateral pitting pedal oedema up to mid-shin; mild sacral oedema** ▶ Photo	Cor pulmonale; hypoxia and hypercapnia cause salt and water retention
JVP	**Raised — 8 cm above the sternal angle, prominent 'v' waves**; hepatojugular reflux positive ▶ Article	Right heart failure with functional tricuspid regurgitation
Flapping tremor	Absent today; **present on admission** ($PaCO_2$ 62 mmHg) ▶ Video/Article	CO_2 narcosis (also hepatic and uraemic encephalopathy)
Neck	Prominent **sternocleidomastoids and scaleni**; **tracheal tug**; no goitre	Accessory muscles in action; hyperinflation
Eyes / fundus	Conjunctival suffusion; fundus — dilated retinal veins, **no papilloedema**	Hypercapnia dilates vessels (severe — papilloedema)
Skin	Thin skin, bruises over forearms	Repeated corticosteroid courses

Vital signs

Vital	Finding	Interpretation
Pulse	104/min, regular, **bounding (high volume)**; all peripheral pulses felt	Tachycardia (hypoxia, salbutamol, fever); bounding pulse of CO_2 retention. Irregular → AF or multifocal atrial tachycardia
Pulsus paradoxus	8 mmHg	>10 mmHg = severe airflow obstruction
Blood pressure	136/84 mmHg (right arm, sitting)	Controlled hypertension
Respiratory rate	**26/min, I:E ratio 1:3**, prolonged expiration	Tachypnoea; prolonged expiration = obstruction. RR >30 → severe exacerbation
Temperature	37.9 °C	Infective exacerbation
SpO_2	**90 % on 1 L/min O_2** (82 % on room air at admission)	Target in COPD is **88–92 %** — not 100 %

Hypoxia, CO_2 retention and cor pulmonale — signs to look for at the bedside

Group	Signs	In this patient
Hypoxia	Central cyanosis, tachycardia, tachypnoea, restlessness, confusion; polycythaemia (chronic)	Cyanosis, plethora, tachycardia

Group	Signs	In this patient
CO_2 retention (hypercapnia)	Warm, flushed peripheries; **bounding pulse**; dilated hand veins; **flapping tremor**; morning headache; drowsiness → coma; miosis; papilloedema (severe)	Warm hands, bounding pulse; flap and drowsiness on admission
Cor pulmonale (right heart failure due to lung disease)	Raised JVP with prominent v waves; **left parasternal heave / epigastric pulsation**; **loud P_2**; right ventricular S_3; pansystolic murmur of TR (louder on inspiration — Carvallo's sign); tender hepatomegaly; ascites; pitting oedema	All present except ascites and S_3
Respiratory distress	Tripod position, pursed lips, accessory muscles, intercostal and supraclavicular recession, tracheal tug, paradoxical (Hoover's) movement, inability to complete sentences	Present

Why is clubbing so important in a COPD case?

Uncomplicated COPD never causes clubbing. If you find clubbing in a smoker with COPD, you must actively look for another disease: **bronchogenic carcinoma** (same risk factor — look for lymph nodes, hoarseness, Horner's), **bronchiectasis** (copious sputum, coarse crackles), **interstitial lung disease** (fine end-inspiratory crackles), **lung abscess / empyema**, or **post-tuberculous fibrosis with bronchiectasis**. Test with Schamroth's window — the diamond-shaped gap between opposed nails is lost in clubbing. ▶ Photo

Examiner's tip — 'Show me the signs of CO_2 retention'

Feel the hands (warm), the pulse (bounding), look at the dorsal veins (dilated), ask the patient to stretch out the arms with wrists dorsiflexed and fingers spread for 30 seconds (asterixis), and look at the fundus. Then say: '**I would like to confirm with an arterial blood gas.**'

Pink puffer and blue bloater — the two classical phenotypes

Feature	Pink puffer (emphysema-predominant)	Blue bloater (chronic bronchitis-predominant)
Build	Thin, cachectic	Stocky, often overweight
Dominant symptom	Breathlessness, little sputum	Productive cough, recurrent infections
Breathing	Pursed-lip breathing, accessory muscles, tachypnoea	Less distressed at rest
Cyanosis / oedema	Absent (PaO_2 near-normal, normocapnic) — 'pink'	Cyanosis, oedema (cor pulmonale) — 'blue and bloated'
Respiratory drive	High — keeps $PaCO_2$ normal	Low — hypercapnia (type 2 failure)
Chest X-ray	Marked hyperinflation, bullae, small vertical heart	Prominent bronchovascular markings, cardiomegaly, enlarged pulmonary arteries
DLCO	Reduced	Normal or mildly reduced
Polycythaemia	Uncommon	Common

Most patients — including this one (thin but cyanosed, oedematous and hypercapnic) — show a **mixed picture**. GOLD no longer uses these labels for treatment, but examiners still ask them.

SECTION B · RESPIRATORY SYSTEM

Examination of the Respiratory System

Examine in this order: upper respiratory tract → inspection → palpation → percussion → auscultation — front, then back

Upper respiratory tract: nose — no polyps, no deviated septum; oral cavity — no candidiasis (inhaled steroids), teeth fair, no sinus tenderness. Pharynx — mildly congested.

a) Inspection

Feature	Finding	Interpretation
Shape of chest	**Barrel-shaped** — AP diameter increased; **AP : transverse ratio ≈ 0.9** (normal ≈ 0.7, i.e. 5:7) ▶ Photo	Hyperinflation — the chest is held near total lung capacity
Ribs and subcostal angle	Ribs **horizontal**; subcostal angle **widened (obtuse)**; mild kyphosis	Hyperinflation; kyphosis adds to the barrel look
Trachea / suprasternal notch	Central; **cricosternal distance 2 finger-breadths** (normal 3–4); **tracheal tug** — trachea descends with each inspiration (Campbell's sign)	Low-set, pulled-down larynx in hyperinflation
Movements	Bilaterally **reduced**, symmetrical; mainly upward 'pump-handle' movement of the whole thorax	Limited excursion of an already-inflated chest
Accessory muscles	Sternocleidomastoids, scaleni and trapezius active; **indrawing** of supraclavicular fossae and lower intercostal spaces on inspiration	Increased work of breathing
Hoover's sign	**Positive** — paradoxical **inward** movement of the lower lateral rib margins on inspiration ▶ Article	Flattened diaphragm pulls the lower ribs inward instead of lifting them out
Respiratory pattern	Rate 26/min; **prolonged expiration**; pursed-lip breathing; abdominal muscles contract in expiration (active expiration)	Expiratory flow limitation
Apex impulse / other	Apex not visible; **epigastric pulsation** present; no scars, sinuses, dilated veins or Harrison's sulcus	RV hypertrophy beneath a low diaphragm

b) Palpation

Feature	Finding	Interpretation
Position of trachea	Central (tested by three-finger method)	Deviation → collapse, fibrosis, pneumothorax, effusion
Apex beat	**Not palpable**	Hyperinflated lung covers the heart
Chest expansion	**Reduced — 2 cm** at the 4th intercostal space (normal ≥5 cm); symmetrical	Bilateral, symmetrical disease
Vocal fremitus	Bilaterally **diminished**, symmetrical	Air-filled lung transmits vibrations poorly
Parasternal heave	Grade 1 left parasternal heave; **subxiphoid RV impulse**; palpable P_2	RV hypertrophy and pulmonary hypertension
Tenderness / crepitus	None; no subcutaneous emphysema	Pneumothorax, rib fracture after coughing

c) Percussion

Area	Finding	Interpretation
Lung fields — front, sides, back	**Hyperresonant** bilaterally, symmetrical	Increased air in the lungs
Cardiac dullness	**Obliterated**	Hyperinflated lung overlies the heart
Upper border of liver dullness	**Lowered to the 7th intercostal space** in the right mid-clavicular line (normal 5th)	Diaphragm pushed down — **so a palpable liver may not be enlarged; always measure the span**
Tidal percussion (diaphragmatic excursion)	Reduced — 1 cm (normal 3–5 cm)	Flat, fixed diaphragm
Traube's space, Kronig's isthmus	Resonant; Kronig's isthmus widened	Hyperinflation of the apices

d) Auscultation

Feature	Finding	Interpretation
Breath sounds	**Vesicular with prolonged expiration**; intensity **diminished** bilaterally	Airflow limitation; reduced conduction through emphysematous lung
Added sounds — wheeze	**Bilateral polyphonic expiratory wheeze (rhonchi)**, more in the lower zones ▶ Listen	Narrowing of many airways of different sizes. **Monophonic** fixed wheeze → single obstruction (tumour)
Added sounds — crackles	**Coarse early-inspiratory crackles** at both bases that **change or clear after coughing** ▶ Listen	Secretions in airways (bronchitis). Fine, late, persistent crackles → ILD or LVF
Vocal resonance	Bilaterally diminished, symmetrical; no bronchophony or whispering pectoriloquy	No consolidation
Forced expiratory time (FET)	**9 seconds** (stethoscope over the trachea, forced expiration from full inspiration)	FET >6 s strongly suggests obstruction (a bedside spirometer)
Snider's match test	Unable to blow out a lit match held 15 cm away with mouth open	FEV_1 likely <1.5 L (rough bedside test)
Pleural rub	Absent	Pneumonia, infarction

Conclusion — respiratory system

Bilateral, symmetrical **hyperinflation** (barrel chest, low trachea with tracheal tug, Hoover's sign, hyperresonance, obliterated cardiac dullness, low liver dullness) with **diffuse airflow obstruction** (prolonged expiration, polyphonic wheeze, FET 9 s) and **secretions** (coarse basal crackles that clear with coughing). No signs of consolidation, effusion, collapse, fibrosis or pneumothorax.

Signs of hyperinflation — learn this table

Sign	Mechanism / meaning
Barrel chest ▶ Photo	Chest fixed in inspiratory position; AP diameter approaches transverse
Reduced cricosternal distance (<3 finger-breadths)	Sternum lifted and larynx pulled down by a low diaphragm
Tracheal tug (Campbell's sign)	Downward jerk of the trachea with each inspiration — the flattened diaphragm pulls on the mediastinum
Hoover's sign ▶ Article	Flat diaphragm contracts horizontally and pulls the lower ribs inward
Intercostal and supraclavicular indrawing	Large negative intrathoracic pressure during inspiration
Hyperresonance, loss of cardiac dullness, low liver dullness	Excess air; lung overlies heart; diaphragm displaced downwards
Apex beat impalpable; epigastric pulsation	Heart covered by lung and vertical; RV felt below the xiphisternum
Palpable liver without true enlargement	Liver pushed down — measure the span (normal 12–15 cm in the MCL)

Examination of other systems

System	Findings	Relevance in COPD
Cardiovascular	Apex not palpable; **left parasternal heave** and **epigastric pulsation**; heart sounds best heard in the epigastrium; **loud P_2**; **pansystolic murmur at the left lower sternal border, louder on inspiration** (functional TR); no S_3; no pericardial rub	**Pulmonary hypertension and cor pulmonale**; heart sounds are soft over a hyperinflated chest
Abdomen	**Liver palpable 3 cm below the right costal margin, tender, smooth, firm; span 14 cm** (true congestive hepatomegaly); hepatojugular reflux positive; no ascites; spleen not palpable	Congestive hepatomegaly of right heart failure — always measure the span, because the liver is pushed down by hyperinflation
Nervous system	Conscious, oriented; no flap today; no focal deficit; fundus — venous congestion, no papilloedema	Hypercapnic encephalopathy resolved
Musculoskeletal	Wasting of the small muscles of the hands and quadriceps (disuse); kyphosis; no bone tenderness	Skeletal-muscle dysfunction, osteoporosis (steroids), lung cancer metastases

Functional assessment — record it in every COPD case

Scale	Score in this patient	Meaning
mMRC dyspnoea scale (0–4)	**3 (stable)**	Severe breathlessness — 'more symptoms' group
CAT score (0–40) ▶ Tool	**24**	High impact (>20); ≥10 = 'more symptoms'
Six-minute walk distance	**240 m (done at the last stable visit)**	Reduced exercise capacity; desaturation to 84 % during walk
Exacerbations in last 12 months	**2 hospitalised + 1 moderate**	High-risk — **GOLD group E**
BODE index (0–10)	**7 (BMI 1 + FEV_1 2 + mMRC 2 + 6MWD 2)**	Highest quartile (7–10) — high mortality; consider for advanced therapies
DECAF score (admission)	**1 (eMRCD 5a)**	Low risk of in-hospital death (0–1 low, 2 intermediate, 3–6 high)

Examiner's tip — expose, sit up, compare

Examine with the patient **sitting up** and the chest fully exposed; compare **both sides at the same level** at every step; examine the **back and axillae**; and always finish with 'I would like to see the sputum cup, the temperature chart, the oxygen prescription and the inhaler technique.' A **sputum cup at the bedside** is a free investigation — describe its colour, volume and consistency.

Sputum at the bedside — what it tells you

Sputum	Suggests
Mucoid, white, small amount	Stable chronic bronchitis
Purulent (yellow/green), increased volume	Bacterial exacerbation (or eosinophils in asthma)
Copious (>1 cup/day), layering in three, foul	Bronchiectasis, lung abscess
Rusty	Pneumococcal pneumonia
Blood-streaked	TB, carcinoma, bronchiectasis, infective exacerbation
Pink, frothy	Pulmonary oedema (LVF)

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SECTION C · DIAGNOSIS

Summary and Final Clinical Diagnosis

Disease → phenotype → severity → exacerbation → complications → aetiology → comorbidities

Summary of examination

Say this after your history summary

On examination, he is conscious and oriented, sitting in the **tripod position** with **pursed-lip breathing**, thin (BMI 19.4), with **central cyanosis**, plethora, **bilateral pitting pedal oedema** and a **raised JVP with prominent v waves**. There is **no clubbing**. Pulse 104/min, bounding; RR 26/min with prolonged expiration; SpO_2 90 % on 1 L/min oxygen. The chest is **barrel-shaped** with reduced cricosternal distance, tracheal tug, use of accessory muscles and a **positive Hoover's sign**; expansion is reduced bilaterally; the percussion note is **hyperresonant** with obliterated cardiac dullness and liver dullness lowered to the 7th space; breath sounds are vesicular with **prolonged expiration**, with **bilateral polyphonic wheeze** and coarse basal crackles that change with coughing. Forced expiratory time is 9 seconds. There is a **left parasternal heave, loud P_2, a TR murmur and tender congestive hepatomegaly**.

Arriving at the diagnosis — step by step

Question	Answer	Conclusion
Is there airflow obstruction?	Prolonged expiration, wheeze, FET 9 s, hyperinflation (to be confirmed by spirometry)	**Obstructive airway disease**
Is it fixed or variable?	Late onset, progressive, no symptom-free intervals, smoker	**Persistent — COPD**
Which phenotype?	Chronic productive cough + hyperinflation	**Chronic bronchitis with emphysema (mixed)**
Is there an exacerbation?	↑ dyspnoea, ↑ sputum volume, purulence, fever over 5 days (<14 days)	**Acute exacerbation — infective**
How severe is the exacerbation?	Hospitalised; hypercapnic acidosis needing NIV	**Severe**
Complications?	Raised JVP, RV heave, loud P_2, TR, hepatomegaly, oedema; cyanosis, flap	**Cor pulmonale; type 2 respiratory failure; polycythaemia**
Cause?	Bidi smoking, biomass, occupational dust	**Tobacco + biomass related**

Final clinical diagnosis

Write it in this format

Chronic obstructive pulmonary disease [disease] — **chronic bronchitis with emphysema (mixed phenotype)** [phenotype] — **GOLD grade 3 (severe airflow limitation), group E**, mMRC 3, CAT 24 [severity — confirmed by spirometry] — in **severe acute exacerbation, probably bacterial (Anthonisen type 1)** [current event] — complicated by **acute-on-chronic type 2 (hypercapnic) respiratory failure, chronic cor pulmonale in right heart failure and secondary polycythaemia** [complications] — in an **ex-bidi smoker (42 pack-years) with household biomass and occupational kiln-smoke exposure** [aetiology] — with **hypertension** as comorbidity; **frequent exacerbator, poor inhaler technique, unvaccinated** [management issues].

Phenotype and severity at a glance

Classification	Basis	This patient
Spirometric grade (GOLD 1–4)	Post-bronchodilator FEV_1 % predicted (only if FEV_1/FVC <0.70)	**GOLD 3 — FEV_1 39 %**
GOLD group (A, B, E)	Exacerbation history + symptoms (mMRC/CAT)	**E (≥2 moderate or ≥1 hospitalisation)**
Blood eosinophils (stable)	Guides inhaled corticosteroid use	**320/µL — ICS likely to help**
Exacerbation severity	Mild (short-acting bronchodilator only) · moderate (+ steroids/antibiotics) · severe (hospital/ED)	**Severe**

Classification	Basis	This patient
Respiratory failure	ABG: type 1 (PaO_2 <60) or type 2 (PaO_2 <60 + $PaCO_2$ >45)	**Type 2, acute-on-chronic**

Differential diagnosis — for and against

Condition	Points in favour	Points against
COPD with exacerbation (most likely)	Age, heavy smoking + biomass, chronic cough, progressive dyspnoea, hyperinflation, cor pulmonale	— (confirm with post-bronchodilator spirometry when stable)
Bronchial asthma	Wheeze, partial response to salbutamol, eosinophils 320/µL	Onset after 50, no atopy, no variability or symptom-free periods, persistent obstruction; 'asthma + COPD' can coexist
Bronchiectasis	Chronic productive cough, coarse crackles, recurrent infections	Sputum small except during exacerbation; no clubbing; no haemoptysis; CT not suggestive
Post-TB obstructive airway disease / active TB	Common in India; weight loss, cough	No past TB; no night sweats; sputum CBNAAT negative; no apical fibrosis
Congestive cardiac failure (LVF)	Breathlessness, orthopnoea, oedema, basal crackles	No PND, no S_3, crackles clear on coughing, normal LV function on echo, NT-proBNP only mildly raised (RV strain)
Bronchogenic carcinoma	Smoker, weight loss	No haemoptysis, hoarseness, clubbing, lymph nodes; no mass on X-ray/CT
Interstitial lung disease / pneumoconiosis	Dust exposure, dyspnoea	No fine end-inspiratory crackles, no clubbing; obstructive (not restrictive) pattern; no reticulation on CT
Pneumothorax complicating COPD	Sudden worsening possible with bullae	No sudden pleuritic pain, symmetrical signs, X-ray normal
Pulmonary embolism	Hypoxia, tachycardia, RV strain	Clear infective trigger; no unilateral leg swelling; low Wells score — keep in mind if not improving
Obliterative bronchiolitis, diffuse panbronchiolitis	Fixed obstruction	Rare; non-smokers; rheumatoid arthritis, transplant

COPD versus bronchial asthma (a favourite viva question)

Feature	COPD	Asthma
Onset	Middle age, after 40	Usually childhood / young adult
Risk factor	Smoking, biomass, occupational dust	Atopy, family history, allergens
Symptoms	Persistent, slowly progressive; productive cough	Episodic, variable, worse at night / early morning; symptom-free periods
Inflammation	Neutrophils, $CD8^+$ T cells, macrophages	Eosinophils, $CD4^+$ (Th2) cells, mast cells
Airflow limitation	Persistent; post-bronchodilator FEV_1/FVC <0.70	Variable; significant reversibility (≥12 % and ≥200 mL); PEF variability >10 %
DLCO	Reduced in emphysema	Normal or raised
Response to steroids	Limited (better if eosinophils high)	Excellent — ICS is the cornerstone
Natural history	Progressive decline in FEV_1	Usually non-progressive when controlled
Key initial therapy	Long-acting bronchodilators (LAMA + LABA)	ICS-containing reliever/controller; **never LABA alone**

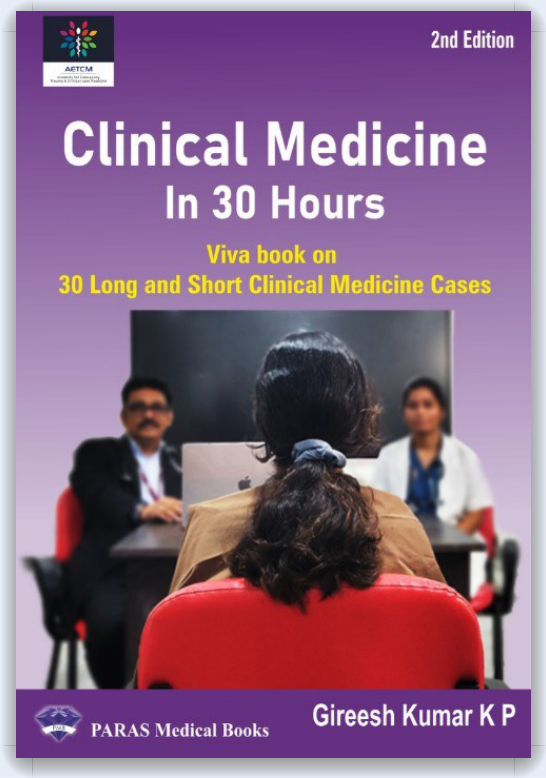
Causes of chronic airflow limitation — classification

Group	Causes
Exposures	Tobacco (cigarette, bidi, hookah, passive), household biomass smoke, outdoor pollution, occupational dusts, fumes and gases
Genetic	α_1-antitrypsin deficiency (Pi ZZ) — panacinar, basal, young; cutis laxa
Infection-related	Post-tuberculosis lung disease, childhood infections, HIV, bronchiectasis
Developmental	Prematurity, low birth weight, abnormal lung growth
Other airway diseases	Chronic asthma with remodelling, obliterative bronchiolitis, cystic fibrosis, primary ciliary dyskinesia

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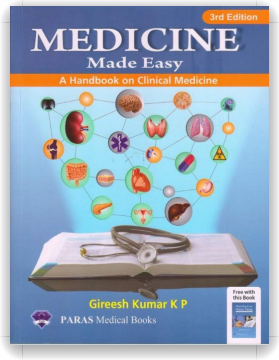
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SECTION D · INVESTIGATIONS

Investigations — Plan, Rationale and Results

Confirm obstruction, grade severity, assess the exacerbation, detect complications and exclude mimics

Investigation	Why	Result in this patient
Pulse oximetry, ABG (on admission, then 1 h after NIV)	Type of respiratory failure, acidosis, need for NIV	pH 7.31, $PaCO_2$ 62, PaO_2 52 mmHg (room air), HCO_3^- 30 mmol/L → after 1 h NIV pH 7.35, $PaCO_2$ 55
Complete blood count	Infection, polycythaemia, eosinophils (ICS decision), anaemia	Hb 17.2 g/dL, **PCV 52 %**, TLC 13 800/µL (N 82 %), eosinophils 180/µL (admission), platelets 2.4 lakh
CRP / procalcitonin	Bacterial infection, guide antibiotics	CRP 36 mg/L
Sputum Gram stain, culture; AFB / CBNAAT ×2	Organism and sensitivity; exclude TB (India)	Gram-negative coccobacilli — ***Haemophilus influenzae***, sensitive to amoxicillin–clavulanate; CBNAAT negative
Chest X-ray PA (+ lateral)	Hyperinflation, pneumonia, pneumothorax, effusion, mass, cardiomegaly	Hyperinflation, enlarged pulmonary arteries, no consolidation ▶ X-ray
ECG	P pulmonale, RVH, RAD, arrhythmia, ischaemia	Sinus tachycardia, P pulmonale, RAD ▶ ECG
2D echocardiography	Pulmonary hypertension, RV function, LV function	Dilated RA/RV, TAPSE 15 mm, **PASP 52 mmHg**, moderate functional TR, LVEF 60 %
Renal function, electrolytes	Hypokalaemia (β_2-agonists, steroids, diuretics), hyponatraemia	Urea 38, creatinine 1.0 mg/dL, Na^+ 134, **K^+ 3.4** mmol/L (corrected)
Blood glucose, LFT	Steroid hyperglycaemia; congestive hepatopathy	RBS 168 mg/dL on steroids; bilirubin 1.4, ALT 52 U/L
NT-proBNP, troponin	Heart failure / myocardial injury as cause or companion of exacerbation	NT-proBNP 640 pg/mL (RV strain); troponin normal
Spirometry (post-bronchodilator)	**Gold standard — diagnosis and grade**; done when stable (≥4–6 weeks after exacerbation)	FEV_1/FVC 0.46; FEV_1 39 % predicted (last stable visit) ▶ PFT
Lung volumes, DLCO, 6-min walk	Hyperinflation, emphysema, exercise capacity, desaturation	TLC 118 %, RV 182 %, RV/TLC 0.56; DLCO 52 %; 6MWD 240 m
CT thorax (non-contrast, HRCT)	Emphysema type and distribution, bullae, bronchiectasis, nodules; pre-surgery / valves	Upper-lobe centrilobular emphysema, bronchial wall thickening; no bronchiectasis or mass ▶ CT
Selected cases	α_1-antitrypsin level (young, non-smoker, basal emphysema), polysomnography (overlap syndrome), D-dimer / CTPA (suspected PE)	Polysomnography planned (snoring)

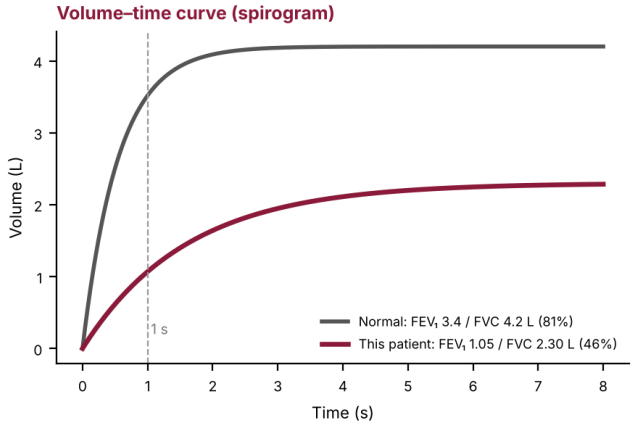
Examiner's tip — no spirometry during an exacerbation

Spirometry is not done to diagnose COPD during an acute exacerbation — the values are falsely low and the patient cannot perform it. Quote the last **stable** post-bronchodilator test, or plan it 4–6 weeks after recovery. During the exacerbation, the **ABG, chest X-ray and ECG** are the key tests.

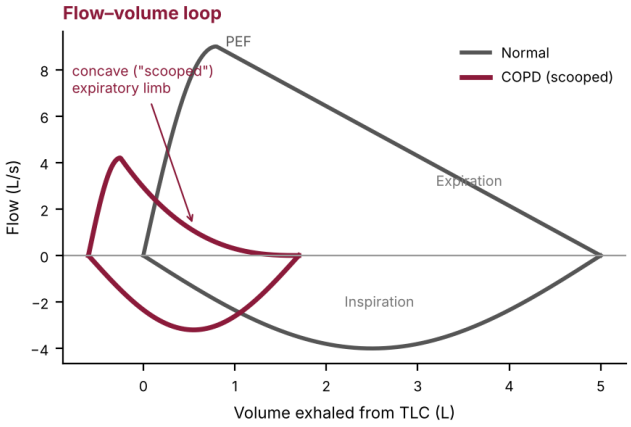
Other investigation results — quick interpretation

Finding	Interpretation and action
PCV 52 % (Hb 17.2)	Secondary polycythaemia; venesection only if haematocrit >55 % with symptoms; LTOT corrects it
Eosinophils 180/µL (admission) · 320/µL (stable)	≥300/µL when stable → strong case for ICS in a frequent exacerbator; eosinophils ≥2 % at admission predict good steroid response
K^+ 3.4 mmol/L	Due to nebulised salbutamol, steroids, diuretics — correct (arrhythmia risk)
PASP 52 mmHg, dilated RV	Pulmonary hypertension due to lung disease (group 3) — treat hypoxia; vasodilators not routinely recommended
DLCO 52 %	Emphysema component; also predicts exercise desaturation
RV 182 %, RV/TLC 0.56	Gas trapping and static hyperinflation — target for bronchodilators and lung-volume reduction

Spirometry — the investigation that makes the diagnosis



Volume–time curve: the COPD curve rises slowly and does not plateau for >6 s.



Flow–volume loop: low peak flow and a concave ('scooped') expiratory limb. ▶ See real loop ▶ How to read

Parameter (post-BD)	Predicted	Observed	% predicted	Interpretation
FVC	3.38 L	2.30 L	68 %	Reduced — air-trapping (pseudo-restriction)
FEV_1	2.70 L	1.05 L	**39 %**	**Severe airflow limitation — GOLD 3**
FEV_1/FVC	0.78	**0.46**	—	**<0.70 → obstruction confirmed**
Bronchodilator response	—	+90 mL	+4 %	Not significant (needs ≥12 % **and** ≥200 mL) — persistent obstruction
$FEF_{25–75}$	2.60 L/s	0.42 L/s	16 %	Small-airway disease

GOLD grade	Post-BD FEV_1 (with FEV_1/FVC <0.70)	Severity
GOLD 1	≥80 % predicted	Mild
GOLD 2	50–79 %	Moderate
GOLD 3	**30–49 %**	**Severe — this patient**
GOLD 4	<30 %	Very severe

Obstructive versus restrictive pattern

Test	Obstructive (COPD, asthma)	Restrictive (ILD, chest wall, neuromuscular)
FEV_1 / FVC	↓ / normal or ↓	↓ / ↓
FEV_1/FVC ratio	**<0.70**	Normal or **increased** (>0.80)
TLC, RV, RV/TLC	↑ (hyperinflation, air trapping)	↓
DLCO	↓ in emphysema; normal in chronic bronchitis and asthma	↓ in ILD; normal in chest-wall disease
Flow–volume loop	Scooped expiratory limb	Narrow, 'witch's hat' loop

Pitfalls of the fixed 0.70 ratio

The ratio falls with age, so a fixed 0.70 cut-off may **over-diagnose** COPD in the elderly and **under-diagnose** it in the young; the lower limit of normal (LLN, z-score < −1.645) is more accurate but GOLD retains 0.70 for simplicity. People with symptoms and normal ratio but reduced FEV_1 ('PRISm') or structural changes on CT ('pre-COPD') need follow-up.

Chest X-ray in COPD ▶ X-ray: hyperinflation ▶ Case: PA + lateral

Feature	In this patient
Hyperinflation — >6 anterior ribs or >10 posterior ribs above the diaphragm in the mid-clavicular line	7 anterior / 11 posterior ribs visible

Feature	In this patient
Flat, low diaphragm — dome height <1.5 cm above a line joining the costophrenic and cardiophrenic angles; blunted costophrenic angles from flattening	Flattened both domes
Long, narrow, vertical ('tubular') heart; increased retrosternal air space >2.5 cm on lateral view	Present on lateral film
Hypertranslucent lung fields with pruning of peripheral vessels; **bullae** ▶ X-ray/CT	Upper-zone translucency; no large bulla
Prominent pulmonary arteries — right descending pulmonary artery >16 mm; enlarged RV in cor pulmonale ▶ X-ray	RDPA 18 mm; mild RV enlargement
Sabre-sheath trachea — intrathoracic trachea narrowed side-to-side ▶ X-ray/CT	Not seen
Complications to exclude: **pneumothorax**, pneumonia, effusion, mass	None

CT thorax

Centrilobular emphysema — small lucencies without walls in the centre of secondary lobules, **upper-lobe** predominant, typical of smokers ▶ CT. **Panacinar emphysema** — diffuse, **lower-lobe**, in α_1-antitrypsin deficiency ▶ CT. **Paraseptal** emphysema — subpleural, can rupture → pneumothorax. CT also shows bronchial wall thickening, bronchiectasis, nodules and the suitability for lung-volume reduction.

ECG in COPD ▶ ECG examples

Feature	Mechanism
P pulmonale — tall peaked P >2.5 mm in II, III, aVF (here 2.8 mm)	Right atrial enlargement
Right axis deviation (here +110°); clockwise rotation, poor R-wave progression	Vertical heart, RV dominance
Low-voltage QRS in limb leads	Hyperinflated lungs insulate the heart
RVH — dominant R in V_1, RV strain; right bundle branch block	Cor pulmonale
Arrhythmias — sinus tachycardia, **multifocal atrial tachycardia** (≥3 P morphologies), AF	Hypoxia, β-agonists, theophylline, RA stretch

Arterial blood gas — step-by-step interpretation

Values (room air, admission)	pH **7.31** · $PaCO_2$ **62 mmHg** · PaO_2 **52 mmHg** · HCO_3^- **30 mmol/L** · SaO_2 84 % · lactate 1.2
Step 1 — oxygenation	PaO_2 <60 mmHg = **respiratory failure**; with $PaCO_2$ >45 = **type 2**
Step 2 — pH	7.31 = **acidaemia**
Step 3 — primary process	$PaCO_2$ ↑ in the same direction as the acidaemia → **respiratory acidosis**
Step 4 — acute or chronic?	Expected HCO_3^-: acute = 24 + 0.1 × 22 = **26**; chronic = 24 + 0.35 × 22 = **32**. Observed 30 lies between → **acute-on-chronic respiratory acidosis**
Step 5 — A–a gradient	PAO_2 = 0.21 × (760 − 47) − 62/0.8 = 150 − 78 = 72; A–a = 72 − 52 = **20 mmHg** (normal for age ≈ 20) → hypoxaemia mostly from **hypoventilation** with V/Q mismatch
Conclusion	**Acute-on-chronic type 2 respiratory failure with uncompensated respiratory acidosis** — pH ≤7.35 with $PaCO_2$ ≥45 → **indication for NIV**

Course of ABG in this patient

On controlled oxygen via a 28 % Venturi mask the PaO_2 rose to 62 mmHg; BiPAP (IPAP 14, EPAP 5 cmH_2O) was started; at 1 hour pH 7.35, $PaCO_2$ 55; at 4 hours pH 7.38, $PaCO_2$ 50, HCO_3^- 29. NIV was weaned over 18 hours. Today on 1 L/min: pH 7.40, $PaCO_2$ 50, PaO_2 61 — his 'stable' chronic compensated hypercapnia.

SECTION E · MANAGEMENT

Management of COPD

Acute exacerbation · ventilatory support · stable disease · oxygen · rehabilitation · prevention

Definition — exacerbation of COPD (GOLD)

An event characterised by **increased dyspnoea and/or cough and sputum that worsens over <14 days**, which may be accompanied by tachypnoea and/or tachycardia, and is often associated with increased local and systemic inflammation caused by infection, pollution or other insult to the airways. Commonest triggers: **viral infections** (rhinovirus, influenza, RSV) and **bacteria** (*H. influenzae*, *S. pneumoniae*, *M. catarrhalis*; *Pseudomonas* in severe disease). Always exclude **pneumonia, PE, heart failure, pneumothorax and arrhythmia**.

A. Acute exacerbation — what should be done

Step	Action
Assess severity	RR, HR, SpO_2, mental state, use of accessory muscles, ABG, chest X-ray, ECG. **Admit** if severe symptoms (sudden worsening of rest dyspnoea, RR >30, SpO_2 falling), acute respiratory failure, new cyanosis/oedema, confusion, failure of initial treatment, serious comorbidity or poor home support
Controlled oxygen	Target **SpO_2 88–92 %** using a **Venturi mask (24 → 28 %)** or nasal prongs 1–2 L/min; repeat ABG in 30–60 min. High-flow uncontrolled O_2 worsens hypercapnia
Short-acting bronchodilators	**Salbutamol 2.5–5 mg + ipratropium 500 µg** nebulised (with air, O_2 by nasal prongs) 4–6-hourly, or pMDI 4–8 puffs via spacer — equally effective
Systemic corticosteroid	**Prednisolone 40 mg orally once daily for 5 days** (IV hydrocortisone 100 mg 6-hourly only if unable to swallow); no taper needed. Faster recovery, fewer relapses. Watch glucose
Antibiotics	When sputum is **purulent + increased dyspnoea or volume** (Anthonisen type 1 or 2), or the patient needs ventilation. 5–7 days: **amoxicillin–clavulanate**, doxycycline or azithromycin; anti-pseudomonal (e.g. piperacillin–tazobactam) if risk factors. Guided by sputum culture and CRP/procalcitonin
NIV (BiPAP)	**pH ≤7.35 and $PaCO_2$ ≥45 mmHg**, or severe dyspnoea with fatigue/accessory muscles despite therapy, or persistent hypoxaemia. Start IPAP 10–12 → titrate to 15–20, EPAP 4–5 cmH_2O; ABG at 1 h and 4 h. Reduces intubation and **mortality**
Invasive ventilation	NIV failure or intolerance; respiratory or cardiac arrest; pH <7.25 with worsening despite NIV; reduced consciousness or agitation not controlled; massive aspiration; haemodynamic instability; life-threatening arrhythmia
Supportive	Fluids and nutrition; correct K^+; **DVT prophylaxis** (enoxaparin 40 mg SC OD); diuretic for oedema of cor pulmonale (cautious — metabolic alkalosis depresses breathing); treat hypertension, diabetes; **avoid sedatives and opioids**; chest physiotherapy for secretions
Before discharge	Start/optimise **long-acting bronchodilator maintenance**; check **inhaler technique**; oxygen assessment; smoking cessation; vaccines; **pulmonary rehabilitation within 4 weeks**; follow-up in 1–4 weeks and at 12–16 weeks

In this patient: 28 % Venturi mask → nasal prongs 1 L/min; nebulised salbutamol + ipratropium 6-hourly; prednisolone 40 mg × 5 days; amoxicillin–clavulanate 625 mg TDS × 5 days (*H. influenzae*, sensitive); **BiPAP for 18 h**; furosemide 40 mg IV OD × 3 days for oedema; enoxaparin; potassium supplement.

NIV — indications and contraindications

Indications (any)	Contraindications
Respiratory acidosis: **pH ≤7.35 and $PaCO_2$ ≥45 mmHg** Severe dyspnoea with fatigue, accessory muscle use, paradoxical abdominal motion Persistent hypoxaemia despite oxygen	Respiratory arrest; cardiovascular instability; impaired consciousness / unable to protect the airway or clear secretions; facial trauma or burns; recent upper-GI or facial surgery; vomiting, upper GI bleed; untreated pneumothorax; uncooperative patient

B. Stable COPD — GOLD 2026 approach

Goals: reduce symptoms (dyspnoea, exercise intolerance, health status) and reduce future risk (exacerbations, progression, death). GOLD 2026 describes the aim as reaching a **low disease-activity state** and stresses that even a single exacerbation raises the risk of the next. ▶ Guideline

GOLD group	Definition	Initial pharmacological therapy
A	0–1 moderate exacerbation (not hospitalised) and mMRC 0–1 / CAT <10	A bronchodilator (long-acting preferred)
B	0–1 moderate exacerbation and mMRC ≥2 / CAT ≥10	**LABA + LAMA** (single inhaler preferred)
E	**≥2 moderate or ≥1 exacerbation leading to hospitalisation**	**LABA + LAMA**; **consider LABA + LAMA + ICS if blood eosinophils ≥300/µL** — this patient

Follow-up — escalate according to the main problem

Problem on treatment	Next step
Persistent dyspnoea	Check technique and adherence; LABA + LAMA if on one; consider switching device/molecule; look for other causes (heart failure, anaemia, deconditioning); **pulmonary rehabilitation**; ensifentrine (PDE3/4 inhibitor) where available
Exacerbations on LABA + LAMA	Eosinophils ≥100/µL → **add ICS (triple therapy)**. Eosinophils <100 → **roflumilast** (FEV_1 <50 % and chronic bronchitis) or **azithromycin** (preferably in former smokers)
Exacerbations on triple therapy	Eosinophils ≥300/µL → **dupilumab** (with chronic bronchitis features) or **mepolizumab**; otherwise roflumilast / azithromycin; consider stopping ICS if pneumonia or no benefit

Blood eosinophils (stable)	Meaning for inhaled corticosteroids
<100/µL	Little benefit — avoid ICS
100–300/µL	Consider, especially with exacerbations
≥300/µL	**Strong support for ICS** (also if history of asthma or hospitalisation)
Against ICS	Repeated pneumonia, mycobacterial infection (TB — relevant in India), eosinophils <100

Drug classes — names you must know

SABA: salbutamol, levosalbutamol · **SAMA:** ipratropium · **LABA:** formoterol, salmeterol, indacaterol, olodaterol, vilanterol · **LAMA:** tiotropium, glycopyrronium, umeclidinium, aclidinium · **ICS:** budesonide, fluticasone, beclomethasone · **PDE-4 inhibitor:** roflumilast · **PDE-3/4:** ensifentrine · **Methylxanthines:** theophylline, doxofylline (narrow window; not first-line) · **Mucolytics/ antioxidants:** N-acetylcysteine, carbocisteine (reduce exacerbations in some) · **Biologics:** dupilumab (anti-IL-4Rα), mepolizumab (anti-IL-5). **ICS alone or LABA + ICS alone are not recommended in COPD.**

Inhaler devices — match the device to the patient

Device	Key points	Suits
pMDI + spacer	Slow, deep breath; coordination not needed with a spacer; cheap	Most patients, including during exacerbations
Dry-powder inhaler (rotacap, multidose)	Needs a **fast, forceful** inspiration (≥30–60 L/min)	Patients with good inspiratory flow
Soft-mist inhaler	Slow aerosol, less coordination	Weak inspiratory effort
Nebuliser	No technique; time-consuming; for severe/acute or those unable to use devices	Elderly, cognitively impaired, acute setting

C. Non-pharmacological management — as important as drugs

Measure	Details
Smoking cessation	The only measure (with LTOT) shown to **slow FEV_1 decline and improve survival**. 5 A's (Ask, Advise, Assess, Assist, Arrange); **varenicline** 0.5 mg OD × 3 days → 0.5 mg BD × 4 days → 1 mg BD for 12 weeks; NRT patch + gum; bupropion. Biomass: improved cook-stoves, LPG, chimney, ventilation
Vaccination	**Influenza** yearly · **pneumococcal** (PCV20 or PCV21 once; or PCV15 followed by PPSV23) · **COVID-19** · **RSV** (GOLD 2026: adults ≥50 with COPD) · **Tdap** if not vaccinated in adolescence · herpes zoster ≥50
Pulmonary rehabilitation	Exercise training (endurance + resistance), breathing techniques (**pursed-lip, diaphragmatic**) ▶ Technique, education, nutrition, psychosocial support; 6–12 weeks; start **within 4 weeks of a hospitalisation**. Improves dyspnoea, exercise capacity, quality of life and reduces readmissions. Give a **written action plan** (rescue pack: prednisolone + antibiotic)
Long-term oxygen therapy (LTOT)	Indicated when, at rest and stable (measure twice, 3 weeks apart): **PaO_2 ≤55 mmHg or SpO_2 ≤88 %**, or **PaO_2 56–59 mmHg with cor pulmonale, peripheral oedema or polycythaemia (haematocrit >55 %)**. Given **≥15 hours/day** (better 24 h) by concentrator, to SpO_2 ≥90 %. Improves survival (NOTT, MRC trials). Must not smoke (fire risk). Re-assess 60–90 days after discharge. **Home NIV** if $PaCO_2$ stays ≥52 mmHg after a hospitalisation

Measure	Details
Nutrition	BMI <21 is a poor prognostic sign; small frequent high-protein, high-calorie meals; vitamin D
Interventional	**Lung-volume reduction surgery** (upper-lobe emphysema, low exercise capacity); **endobronchial valves** (no collateral ventilation); **bullectomy** for giant bullae ▶ CT; **lung transplantation** (BODE 7–10, FEV_1 <20 %)
Palliative care	Low-dose oral morphine for refractory breathlessness, fan therapy, anxiety management, advance care planning

D. Discharge prescription (sample)

Drug	Dose	Remarks
Inh. LAMA + LABA + ICS (e.g. glycopyrronium + formoterol + budesonide) pMDI	2 puffs BD via spacer	Single-inhaler triple therapy — group E, eosinophils ≥300; **rinse mouth** after use
Inh. Salbutamol pMDI 100 µg	2 puffs as needed via spacer	Reliever; record frequency of use
Tab. Prednisolone	40 mg OD after food	To complete 5 days — stop; no taper
Tab. Amoxicillin–clavulanate	625 mg TDS	To complete 5 days
Tab. Furosemide	20 mg OD	For oedema; review weight, K^+ at 1 week
Tab. Amlodipine	5 mg OD	Continue; cardioselective β-blockers are safe if needed
Home oxygen (concentrator)	1 L/min ≥15 h/day	Interim; repeat ABG at 6–8 weeks to confirm LTOT need
Vaccines	Influenza, pneumococcal, RSV, COVID-19	Before discharge / at first follow-up
Referrals	Pulmonary rehabilitation; polysomnography	Review in 2 weeks; spirometry when stable; written action plan

E. Complications of COPD — prevent, detect and treat

- **Acute exacerbations** and acute-on-chronic respiratory failure
- **Cor pulmonale** and pulmonary hypertension
- **Secondary polycythaemia**; venous thromboembolism
- **Pneumothorax** (rupture of bullae)
- **Pneumonia** (more with ICS)
- **Lung cancer** — shared risk; screen with low-dose CT if eligible
- **Cardiovascular** — IHD, heart failure, arrhythmias (MAT, AF)
- **Skeletal muscle wasting, cachexia**, osteoporosis (steroids)
- **Anxiety and depression**; cognitive impairment
- **Sleep-disordered breathing** — overlap syndrome
- Comorbidities (GERD, diabetes, anaemia) and drug effects (candidiasis, tremor, hypokalaemia)

F. Prognosis

COPD is progressive but **treatable and preventable**. Poor prognostic factors in this patient: **frequent hospitalised exacerbations, hypercapnia needing NIV, cor pulmonale, FEV_1 39 %, low BMI, short 6-minute walk and BODE index 7** (BMI, Obstruction, Dyspnoea, Exercise; 0–10 — 7–10 is the highest-risk quartile). After a hospitalised exacerbation, about a quarter of patients are readmitted within 3 months and mortality over the next year is high. Favourable factors: he has stopped smoking, has good family support, and can benefit from triple therapy, vaccination, rehabilitation and oxygen. **DECAF** (Dyspnoea eMRCD, Eosinopenia, Consolidation, Acidaemia pH <7.30, AF) predicts in-hospital death in exacerbations.

Examiner's tip — answer 'How will you manage this patient?' in three parts

(1) **The acute exacerbation** — controlled oxygen to 88–92 % (high-flow O_2 worsens hypercapnia by V/Q mismatch and the Haldane effect — but never withhold oxygen), nebulised bronchodilators, steroids, antibiotics, NIV with ABG monitoring. (2) **The stable disease** — GOLD group-based inhaler, technique, eosinophil-guided ICS. (3) **Prevention and rehabilitation** — smoking/biomass cessation, vaccines, pulmonary rehabilitation, LTOT, nutrition, action plan. Mention **cor pulmonale** treatment (oxygen, diuretics) separately.

SECTION F · PRESENTATION

How to Present This Case in the Examination

A 5-minute presentation that impresses the examiner

Structure of the presentation

- **Introduce** — name (initials), age, sex, occupation (past and present), place.
- **Chief complaints** with duration — chronic baseline first, then the acute worsening.
- **HOPI** — cough and sputum, breathlessness with mMRC grade, wheeze, exacerbations in the past year, present trigger, cor pulmonale and CO_2 symptoms, relevant negatives.
- **Exposure history** — smoking in pack-years / smoking index, biomass, occupation; treatment and inhaler technique.
- **Summary of history** and what you think from it.
- **General examination** — distress, position, cyanosis, clubbing (present or absent), oedema, JVP, flap, vitals with SpO_2 and oxygen.
- **Respiratory system** — inspection, palpation, percussion, auscultation, FET; then CVS and abdomen for cor pulmonale.
- **Diagnosis** in the complete format; then differential diagnoses only if asked.

Model presentation (read aloud and practise until you can say it in 4–5 minutes)

Sir / Madam, I would like to present the case of…

"…Mr V. M., a **64-year-old ex-tile-factory worker and agricultural labourer from Ottapalam**, who presented with **cough with expectoration for 8 years, breathlessness on exertion for 6 years and wheeze for 3 years**, all **worsened for the past 5 days** with increased and purulent sputum and low-grade fever, along with **swelling of both legs for 10 days** and **morning headache and drowsiness for 2 days**.

His cough has been present for more than 3 months every year, initially mucoid. His breathlessness is insidious and progressive, and at baseline he stops after 100 metres — **mMRC grade 3**. There is no diurnal variation, atopy or childhood asthma. He has had **two hospitalisations in the last year, one requiring NIV**. This episode followed a cold. There is no haemoptysis, history of tuberculosis, chest pain or PND.

He was a **bidi smoker of 42 pack-years (smoking index 840), stopped 4 years ago**, with **household biomass and occupational kiln-smoke exposure**. He is a known hypertensive. He uses tiotropium irregularly and salbutamol without a spacer with poor technique, has had three steroid courses this year and has never been vaccinated.

On examination he is conscious and oriented, sitting in the **tripod position with pursed-lip breathing**, thin with a BMI of 19.4, **centrally cyanosed**, with **no clubbing**, **bilateral pitting pedal oedema** and a **JVP raised to 8 cm with prominent v waves**. Pulse 104 per minute, bounding; respiratory rate 26 with prolonged expiration; SpO_2 90 % on 1 litre of oxygen.

The chest is **barrel-shaped** with reduced cricosternal distance, tracheal tug, accessory-muscle use and a **positive Hoover's sign**. Expansion is reduced bilaterally, percussion note is **hyperresonant** with obliterated cardiac dullness and liver dullness in the 7th space. Breath sounds are vesicular with **prolonged expiration, bilateral polyphonic wheeze** and coarse basal crackles that change with coughing. Forced expiratory time is 9 seconds. There is a **parasternal heave, loud P_2, a TR murmur and tender hepatomegaly with a 14 cm span**.

So my diagnosis is **chronic obstructive pulmonary disease, mixed chronic bronchitis–emphysema phenotype, in severe acute infective exacerbation, with acute-on-chronic type 2 respiratory failure and chronic cor pulmonale in right heart failure, in an ex-bidi smoker with biomass exposure — a frequent exacerbator, GOLD group E**, to be graded by post-bronchodilator spirometry."

Do's and don'ts during presentation

Do	Don't
Quantify smoking and name biomass exposure	Say 'smoker' without amount and duration
Give the mMRC grade and the number of exacerbations	Diagnose COPD without mentioning that spirometry is needed
State clearly that **clubbing is absent** (and why it matters)	Report 'clubbing present — due to COPD'
Separate hyperinflation, obstruction and cor pulmonale findings	Read a long list of normal findings
Mention the oxygen flow with the SpO_2	Quote SpO_2 without saying whether it was on room air
Be ready to demonstrate Hoover's sign, FET, flap, JVP	Report a sign you have not elicited yourself

SECTION G · VIVA VOCE

Frequently Asked Viva Questions with Answers

Short, exam-ready answers

Q1 Define COPD.

A heterogeneous lung condition characterised by **chronic respiratory symptoms** (dyspnoea, cough, sputum, exacerbations) due to abnormalities of the airways (bronchitis, bronchiolitis) and/or alveoli (emphysema) that cause **persistent, often progressive, airflow obstruction** (GOLD). Diagnosis requires a **post-bronchodilator FEV_1/FVC <0.70**.

Q2 Define chronic bronchitis and emphysema.

Chronic bronchitis — a **clinical** definition: productive cough on most days for at least 3 months a year for 2 consecutive years, other causes excluded. **Emphysema** — a **pathological** definition: permanent enlargement of airspaces distal to the terminal bronchiole with destruction of their walls, without obvious fibrosis.

Q3 What are the types of emphysema?

Centriacinar (centrilobular) — respiratory bronchioles, upper lobes, smokers. **Panacinar** — whole acinus, lower lobes, α_1-antitrypsin deficiency. **Paraseptal (distal acinar)** — subpleural, causes pneumothorax in young adults. **Irregular (paracicatricial)** — around scars, e.g. post-TB.

Q4 Outline the pathogenesis of COPD.

Inhaled particles → chronic inflammation (macrophages, **neutrophils, $CD8^+$ T cells**) → **protease–antiprotease imbalance** (neutrophil elastase, MMPs vs α_1-antitrypsin) and **oxidative stress** → alveolar wall destruction (emphysema), mucous gland hyperplasia (bronchitis) and small-airway fibrosis → loss of elastic recoil and airway narrowing → airflow limitation, air trapping and hyperinflation.

Q5 Why is expiration prolonged? What is dynamic hyperinflation?

Loss of elastic recoil and narrowed airways that collapse during expiration slow emptying. If the next breath starts before the lung has emptied, air is trapped — **dynamic hyperinflation** — creating **intrinsic PEEP (auto-PEEP)**, which increases work of breathing, flattens the diaphragm and causes exertional dyspnoea, and may lower BP during ventilation.

Q6 How does pursed-lip breathing help?

It creates back-pressure (a small PEEP) in the airways during expiration, **splinting the small airways open**, slowing the respiratory rate, reducing air trapping and improving gas exchange and dyspnoea.

Q7 What is Hoover's sign? How is it produced?

Paradoxical **inward movement of the lower lateral rib margins during inspiration**. The flattened diaphragm, instead of descending, contracts horizontally and pulls the costal margin inwards. It indicates severe hyperinflation and correlates with severity. (Different from Hoover's sign in functional leg weakness.)

Q8 Name the signs of hyperinflation.

Barrel chest, horizontal ribs, reduced cricosternal distance, tracheal tug, Hoover's sign, intercostal indrawing, hyperresonance, obliterated cardiac dullness, lowered liver dullness, reduced diaphragmatic excursion, impalpable apex and epigastric pulsation.

Q9 Does COPD cause clubbing?

No. If clubbing is present in a COPD patient, suspect bronchogenic carcinoma, bronchiectasis, interstitial lung disease, lung abscess, empyema or post-TB fibrosis with bronchiectasis.

Q10 Pink puffer vs blue bloater?

Pink puffer — emphysema-predominant, thin, breathless, pursed lips, normal $PaCO_2$, no cyanosis or oedema. Blue bloater — bronchitis-predominant, cyanosed, hypercapnic, oedematous, cor pulmonale, polycythaemia. Most patients are mixed.

Q11 Define cor pulmonale. What is the mechanism in COPD?

Right ventricular hypertrophy and/or dilatation secondary to disease of the lungs, pulmonary vessels or chest wall, **excluding left heart disease**. In COPD: alveolar **hypoxia** → **hypoxic pulmonary vasoconstriction**, vascular remodelling, loss of capillary bed, polycythaemia and acidosis → pulmonary hypertension → RV failure.

Q12 What are the signs of CO_2 retention?

Warm peripheries, bounding pulse, dilated veins, flapping tremor, headache (morning), drowsiness, confusion, coma, miosis and papilloedema.

Q13 Why controlled oxygen in COPD? What is the target?

Target **SpO_2 88–92 %**. Uncontrolled high-flow oxygen raises $PaCO_2$ by worsening V/Q mismatch (reversal of hypoxic vasoconstriction), the Haldane effect and reduced hypoxic drive — causing respiratory acidosis and narcosis. Use a **Venturi mask** (24 %, 28 %) which delivers a fixed FiO_2 regardless of the patient's breathing pattern.

Q14 What is the principle of a Venturi mask?

Oxygen passes through a narrow jet at high velocity, creating a low pressure that entrains room air through side ports in a fixed ratio (Bernoulli/Venturi principle) — giving a **fixed, predictable FiO_2** at a high total flow.

Q15 What are the indications for NIV in an exacerbation?

Respiratory acidosis — **pH ≤7.35 and $PaCO_2$ ≥45 mmHg**; severe dyspnoea with signs of muscle fatigue; persistent hypoxaemia despite oxygen. Check ABG at 1 hour; failure to improve → consider intubation.

Q16 When will you intubate a patient with AECOPD?

NIV failure or intolerance, respiratory or cardiac arrest, worsening acidosis (pH <7.25) despite NIV, reduced consciousness or uncontrolled agitation, inability to clear secretions, massive aspiration, haemodynamic instability, life-threatening arrhythmias.

Q17 What are the Anthonisen criteria?

Three cardinal symptoms: **increased dyspnoea, increased sputum volume, increased sputum purulence**. Type 1 = all three; type 2 = two; type 3 = one + URTI, fever, wheeze or cough. Antibiotics benefit types 1 and 2 (when purulence is one of them).

Q18 Which organisms cause exacerbations?

Viruses — rhinovirus, influenza, RSV, parainfluenza, SARS-CoV-2. Bacteria — *Haemophilus influenzae*, *Streptococcus pneumoniae*, *Moraxella catarrhalis*; *Pseudomonas aeruginosa* in severe disease, frequent antibiotics or bronchiectasis. Non-infective — pollution, PE, heart failure.

Q19 What is the steroid dose in an exacerbation?

Prednisolone 40 mg/day for 5 days (REDUCE trial — as effective as 14 days). Speeds recovery of FEV_1 and oxygenation and reduces treatment failure. Greater benefit with blood eosinophils ≥2 %.

Q20 What is the GOLD spirometric grading?

With FEV_1/FVC <0.70: GOLD 1 FEV_1 ≥80 %; GOLD 2 50–79 %; GOLD 3 30–49 %; GOLD 4 <30 % predicted (post-bronchodilator).

Q21 Explain the GOLD ABE assessment.

Based on **exacerbations** and **symptoms**. E: ≥2 moderate or ≥1 hospitalised exacerbation in the past year. Otherwise A (mMRC 0–1, CAT <10) or B (mMRC ≥2 or CAT ≥10). Initial therapy: A — a bronchodilator; B — LABA+LAMA; E — LABA+LAMA, add ICS if eosinophils ≥300/µL.

Q22 What is the role of blood eosinophils in COPD?

A biomarker of ICS response: <100/µL — little benefit; 100–300 — consider; ≥300 — strong benefit. Also guides biologics (dupilumab, mepolizumab ≥300/µL) and predicts steroid response in exacerbations.

Q23 Why is ICS alone not used in COPD? Side effects of ICS?

ICS alone does not improve survival or lung function decline and increases pneumonia; it must be combined with long-acting bronchodilators. Side effects: **pneumonia**, oral candidiasis, dysphonia, skin bruising, osteoporosis, cataract, diabetes, reactivation of **TB** and non-tuberculous mycobacteria.

Q24 How do LABA and LAMA work? Name examples.

LABA — β_2-agonists, raise cAMP and relax airway smooth muscle (formoterol, salmeterol, indacaterol, olodaterol, vilanterol). LAMA — block M_3 receptors and vagal bronchoconstriction, reduce mucus (tiotropium, glycopyrronium, umeclidinium, aclidinium). LAMA is better than LABA at preventing exacerbations; the combination is better than either.

Q25 What is the place of theophylline / doxofylline?

Weak bronchodilators, used only when long-acting inhaled therapy is unavailable or unaffordable. Narrow therapeutic index (theophylline 5–15 mg/L); toxicity — nausea, tachyarrhythmia, seizures; interactions — ciprofloxacin, macrolides, smoking cessation raises levels. Doxofylline has fewer side effects.

Q26 When is roflumilast used?

Oral PDE-4 inhibitor (500 µg OD) for patients with **FEV_1 <50 %, chronic bronchitis and exacerbations** despite LABA+LAMA (±ICS). Side effects: diarrhoea, nausea, weight loss, psychiatric effects — avoid in underweight or depressed patients.

Q27 What is the role of long-term azithromycin?

250 mg daily or 500 mg three times a week for a year reduces exacerbations, mainly in **former smokers**. Risks — QT prolongation, hearing loss, macrolide resistance; exclude NTM infection first.

Q28 Which biologics are used in COPD?

Dupilumab (anti-IL-4Rα) and **mepolizumab** (anti-IL-5) for patients with eosinophils ≥300/µL who still exacerbate on triple therapy — dupilumab particularly with chronic bronchitis. They reduce exacerbations and improve symptoms and lung function.

Q29 What are the criteria for LTOT? What is its benefit?

Stable patient, at rest, twice 3 weeks apart: **PaO_2 ≤55 mmHg or SpO_2 ≤88 %**; or PaO_2 56–59 mmHg with **cor pulmonale, oedema or polycythaemia (Hct >55 %)**. Give ≥15 h/day to SpO_2 ≥90 %. Improves survival (NOTT and MRC trials), reduces polycythaemia and pulmonary pressure. Patient must not smoke.

Q30 What does pulmonary rehabilitation include?

Supervised exercise training (endurance and strength), breathing techniques, inhaler and self-management education, nutritional advice, psychological support and energy-conservation techniques — usually 6–12 weeks. Improves dyspnoea, exercise tolerance and quality of life and reduces readmission when started within 4 weeks of discharge.

Q31 What is the BODE index?

A multidimensional prognostic score (0–10): **B**MI, airflow **O**bstruction (FEV_1 %), **D**yspnoea (mMRC) and **E**xercise capacity (6-minute walk). Higher scores predict higher mortality; it is better than FEV_1 alone and is used for transplant referral.

Q32 When will you suspect α_1-antitrypsin deficiency?

Emphysema before 45 years, in a non-smoker or light smoker, **basal panacinar** emphysema, family history of emphysema or liver disease, unexplained liver cirrhosis, necrotising panniculitis, c-ANCA vasculitis. WHO recommends screening all COPD patients once, especially in high-prevalence areas.

Q33 What is post-tuberculosis obstructive airway disease?

Persistent airflow obstruction after treated pulmonary TB, due to bronchial stenosis, bronchiectasis, fibrosis and small-airway damage. Very common in India; presents in younger non-smokers with fixed obstruction and abnormal CXR. Always ask about past anti-TB treatment and look for apical fibrosis.

Q34 How do you differentiate COPD from asthma on spirometry?

Asthma — **significant reversibility** (FEV_1 ↑ ≥12 % and ≥200 mL after bronchodilator), normal post-BD ratio possible, PEF variability >10 %, normal DLCO. COPD — persistent post-BD FEV_1/FVC <0.70, small reversibility, reduced DLCO in emphysema. Very large reversibility (>400 mL) suggests asthma or asthma + COPD.

Q35 What are the causes of a hyperresonant percussion note?

Emphysema / hyperinflation (bilateral), pneumothorax (unilateral, with absent breath sounds), large bulla, large cavity near the surface, and severe acute asthma.

Q36 What is secondary polycythaemia? When will you do venesection?

Raised red-cell mass from chronic hypoxia-driven erythropoietin release (haematocrit >52 % in men). Treat hypoxia with LTOT and stop smoking; venesection only if **haematocrit >55 %** with symptoms of hyperviscosity (headache, plethora, thrombosis).

Q37 Which drugs should be avoided or used with care in COPD?

Sedatives, hypnotics and opioids (respiratory depression), non-selective β-blockers such as propranolol (bronchospasm), high-flow uncontrolled oxygen, excess diuretics (metabolic alkalosis). **Cardioselective β-blockers** (bisoprolol, metoprolol) are safe when indicated, e.g. after MI or in heart failure.

Q38 What is the overlap syndrome?

Coexistence of **COPD and obstructive sleep apnoea**. Causes more severe nocturnal hypoxaemia, hypercapnia, pulmonary hypertension and higher mortality than either alone. Suspect with snoring, obesity, daytime sleepiness and morning headache; treat with CPAP/BiPAP.

Last-minute checklist before you go to the examiner

✔ Smoking in pack-years · ✔ biomass & occupation · ✔ mMRC grade · ✔ exacerbations / admissions / NIV in last year · ✔ inhaler technique · ✔ vaccination · ✔ tripod & pursed lips · ✔ cyanosis · ✔ clubbing absent · ✔ JVP, oedema, flap · ✔ SpO_2 with O_2 flow · ✔ barrel chest, cricosternal distance, tracheal tug, Hoover's · ✔ hyperresonance, cardiac & liver dullness · ✔ prolonged expiration, wheeze, FET · ✔ parasternal heave, loud P_2, TR · ✔ complete diagnosis with GOLD grade & group · ✔ management in three parts.

VISUAL ATLAS

See the Signs — Tap or Scan

Free photographs, X-rays, CT, ECGs, spirometry loops and breath sounds — from Radiopaedia, LITFL, Wikipedia and other open sites

Barrel chest
▶ Photo
Increased AP diameter, horizontal ribs

Pursed-lip breathing
▶ Photo
Expiration through pursed lips

Tripod position
▶ Photo
Leaning forward, arms braced

Hoover's sign
▶ Article
Inward lower-rib movement on inspiration

Central cyanosis
▶ Photo
Bluish tongue and lips

Clubbing
▶ Photo
Loss of nail-fold angle — not due to COPD

Schamroth's sign
▶ Photo
Obliterated diamond window

Asterixis (CO_2 flap)
▶ Video/Article
Flapping tremor of outstretched hands

Raised JVP
▶ Article
Right heart failure — cor pulmonale

Pitting pedal oedema
▶ Photo
Cor pulmonale / RV failure

Wheeze (rhonchi)
▶ Listen
Polyphonic expiratory wheeze — audio

Coarse crackles
▶ Listen
Fine vs coarse crackles — audio

Chest X-ray — hyperinflation
▶ X-ray
Flat diaphragms, ↑ rib count, tubular heart

COPD chest X-ray case
▶ X-ray
Radiopaedia case with PA + lateral

Cor pulmonale
▶ X-ray
Enlarged pulmonary arteries, RV

Pulmonary bullae
▶ X-ray/CT
Air spaces >1 cm, avascular

Sabre-sheath trachea
▶ X-ray/CT
Coronal narrowing of intrathoracic trachea

Centrilobular emphysema
▶ CT
Upper-lobe lucencies — smokers

Panacinar emphysema
▶ CT
Lower-lobe, α1-antitrypsin deficiency

ECG in COPD
▶ ECG
P pulmonale, RAD, low voltage, MAT

Flow–volume loop
▶ PFT
Scooped expiratory limb

Spirometry & flow–volume loop
▶ PFT
How the loop is read — illustrated

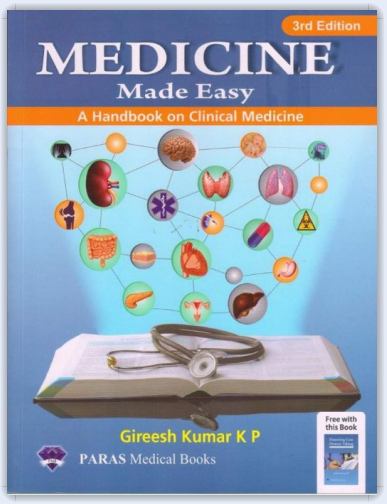
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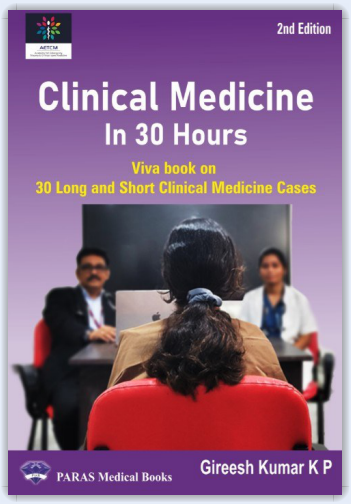
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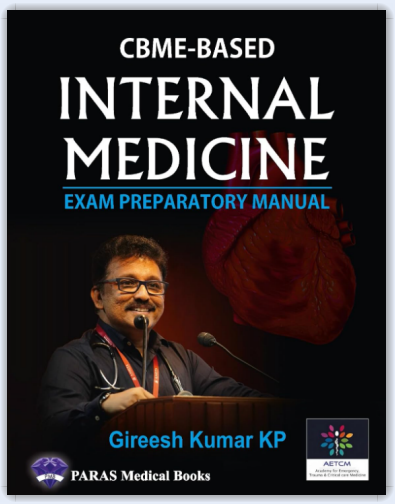
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