

## Chapter 11. Respiratory System

### Part 1 | Anatomy of the Respiratory Tract & Lungs

#### 1 Learning Objectives

After studying this part you will be able to ...

1. **Identify all structures of the upper and lower respiratory tracts**, listing their histological specialisations and functional roles.
2. **Describe the gross and microscopic anatomy of the lungs**, including lobes, segments, pleurae and the bronchial tree.
3. **Explain the mechanics of ventilation and gas exchange** in relation to airway and alveolar structure.
4. **Relate respiratory anatomy to common physiotherapy applications** such as airway-clearance techniques, breathing retraining and postural drainage.

### 2 Upper vs. Lower Respiratory Tract

Level	Structures	Epithelial Lining	Primary Functions	PT Significance
<b>Upper</b>	<ul style="list-style-type: none"> <li>• <b>Nose &amp; Nasal cavity</b></li> <li>• Paranasal sinuses</li> <li>• <b>Pharynx:</b> Nasopharynx, Oropharynx, Laryngopharynx</li> <li>• <b>Larynx</b> (to vocal folds)</li> </ul>	Mostly <b>pseudostratified ciliated columnar</b> with goblet cells; oropharynx—stratified squamous	Filtration, humidification, warming; resonance & phonation	Teach nasal-breathing to enhance humidification; voice conservation post-laryngeal surgery
<b>Trachea → Primary bronchus → Bronchial tree → Alveoli</b>		Gradual transition: ciliated columnar → cuboidal → <b>simple squamous (Type I pneumocytes)</b>	Air conduction, mucociliary clearance, gas exchange	Manual percussion aligns with segmental bronchi; pursed-lip breathing targets small airways

### 3 Airway Anatomy in Detail

#### 3.1 Conducting Zone (Dead-Space Airway)

Generation	Key Features	Cartilage? Smooth Muscle?
<b>Trachea</b>	~11 cm; C-shaped hyaline rings; carina at T4/5	✓ "C" rings Few
<b>Main (Primary) Bronchi</b>	Right wider, shorter & more vertical (aspiration risk)	✓ Plates ↑
<b>Lobar (Secondary) Bronchi</b>	3 on right, 2 on left	✓ Plates ↑↑
<b>Segmental (Tertiary) Bronchi</b>	10 segments (R), 8-10 (L)	✓ Plates ↑↑
<b>Bronchioles (&lt; 1 mm)</b>	No cartilage; clara/club cells secrete surfactant-like fluid	X ✓✓
<b>Terminal Bronchioles</b>	End of conducting zone; last with cilia	X ✓✓

#### 3.2 Respiratory Zone

Structure	Function	Clinical Note
<b>Respiratory bronchioles</b>	Start of gas exchange; occasional alveoli in walls	Site of early emphysematous change
<b>Alveolar ducts &amp; sacs</b>	Lined almost entirely by alveoli	Postural drainage positions target these segments

<b>Structure</b>	<b>Function</b>	<b>Clinical Note</b>
<b>Alveoli (<math>\approx 300</math> million)</b>	Type I cells for diffusion; Type II produce surfactant; alveolar macrophages for defence	Surfactant deficiency $\rightarrow$ neonatal RDS; incentive spirometry prevents collapse

#### 4 Gross Lung Anatomy

<b>Aspect</b>	<b>Right Lung</b>	<b>Left Lung</b>	<b>Functional Angle</b>
<b>Lobes</b>	3 – Superior, Middle, Inferior	2 – Superior, Inferior	Right middle lobe drains best in left side-lying
<b>Fissures</b>	Oblique + Horizontal	Oblique only	Auscultation landmarks for segmental percussion
<b>Bronchopulmonary Segments</b>	10	8-10	Surgical resection units; PT can isolate by positioning
<b>Hilum Contents</b>	Pulmonary artery (anterior), veins (inferior), main bronchus (posterior)	Artery superior to bronchus	Endotracheal suction depth awareness
<b>Pleurae</b>	<b>Visceral</b> (adheres to lung) + <b>Parietal</b> (thoracic cavity) with potential space	Pain from parietal only (phrenic/intercostal nerves)	Educate splinted breathing for pleuritic pain

#### Neurovascular Supply

- **Bronchial arteries** (systemic) nourish lung tissue; pulmonary arteries carry deoxygenated blood for gas exchange.
- **Parasympathetic (vagus)  $\rightarrow$  bronchoconstriction / mucus  $\uparrow$ ; Sympathetic  $\rightarrow$  bronchodilation / mucus  $\downarrow$**  — basis for inhaler pharmacology.

#### 5 Mechanics of Ventilation (Quick Recap)

- **Inspiration:** Diaphragm (75 %) + external intercostals enlarge thoracic volume  $\rightarrow$  intrapleural pressure falls from  $-2$  to  $-6$  mm Hg  $\rightarrow$  lungs expand.
- **Expiration:** Passive recoil (quiet); abdominal & internal intercostals (forced).
- **Compliance ( $\Delta V/\Delta P$ )** highest at FRC; fibrosis  $\downarrow$  compliance, emphysema  $\uparrow$  compliance – exercise prescription differs.

#### 6 Structure-Function-Clinical Correlations

<b>Anatomy Feature</b>	<b>Physiological Benefit</b>	<b>PT Application</b>
Mucociliary escalator (goblet + cilia)	Clears particles 5-10 $\mu\text{m}$	Flutter device & active cycle of breathing aid clearance
Right main bronchus vertical	Facilitates aspiration	Side-lying right 30° post feeding in neuro-patients
Segmental anatomy	Localises infection / collapse	Specific postural drainage + manual techniques
Type II pneumocytes	Surfactant reduces surface tension	Deep-breathing exercises recruit surfactant release
Pleural recesses	Costodiaphragmatic recess drains fluid	Thoracic expansion exercises promote re-expansion post-thoracentesis

#### 7 Self-Check Quiz (with Answers)

1. **Which airway generation marks the end of cartilage and the start of substantial smooth muscle?**

**Answer: Bronchioles** (following segmental bronchi).

2. **Name the only pain-sensitive layer of the lung's pleural covering.**

**Answer: Parietal pleura.**

3. **Why is the right lung more prone to aspiration pneumonia?**

**Answer:** The **right main bronchus** is wider, shorter, and more vertical than the left, so aspirated material follows gravity into right lower lobe segments.

4. **Which cells produce pulmonary surfactant and what is one physiotherapy technique that can stimulate its distribution?**

**Answer: Type II pneumocytes;** deep-breathing / incentive spirometry promotes surfactant spread.

5. **During quiet breathing, what percentage of the tidal volume is contributed by the diaphragm?**

**Answer:** Approximately 75 %.

## 8 Suggested Lab / Practical Activities

Activity	Outcome
<b>Airway Model Dissection</b>	Trace trachea to alveoli; identify histological changes with hand-lenses.
<b>Lung-Segment Positioning Drill</b>	Students place peers in correct drainage postures for each segment.
<b>Spirometry &amp; Flow-Volume Loop Lab</b>	Correlate obstructive vs restrictive patterns with anatomical sites.
<b>Cilia Beat Experiment (video microscopy)</b>	Visualise mucociliary action; discuss impact of smoking.

## 9 Key Take-Home Points

- The respiratory tract transitions from **rigid, cartilage-supported conduits to delicate gas-exchange membranes**; each segment has distinct vulnerabilities and therapeutic targets.
- Bronchopulmonary segments** permit selective physiotherapy techniques, surgical resections, and precise auscultation.
- Understanding pleural anatomy** aids in managing pain, preventing atelectasis, and guiding breathing exercises.
- Physiotherapists leverage airway structure knowledge to design **airway-clearance, breathing retraining, and positioning protocols** tailored to pathology.

## Part 2 | Mechanics of Breathing, Gas Exchange & Applied Physiology

### 1 • Learning Objectives

After completing this part you should be able to ...

- List all primary and accessory muscles of inspiration and expiration**, their origins/insertions, nerve supply and kinesiologic actions.
- Explain the pressure-volume relationships** (Boyle's law) that drive airflow during quiet and forced ventilation.
- Define static and dynamic lung volumes and capacities**, and relate them to spirometric patterns in obstructive vs restrictive disease.
- Describe alveolar-capillary gas exchange** using Fick's law, including diffusion-limitation vs perfusion-limitation concepts.
- Outline oxygen and carbon-dioxide transport mechanisms** (Hb dissociation curve, Haldane & Bohr effects).
- Apply these principles clinically** to breathing retraining, airway-clearance, positioning and exercise prescription.

### 2 • Respiratory Muscles

Group	Muscles	Origin → Insertion	Innervation	Phase & Action	PT Relevance
<b>Primary Inspiratory</b>	<b>Diaphragm</b>	Sternum, lower 6 ribs, L1-L3 crura → central tendon	C3-5 phrenic	Quiet inspiration (75 % tidal volume) - dome descends 1.5 cm	Diaphragmatic breathing retrains efficient pattern; C-spine injury $\geq$ C3 threatens ventilation
	External intercostals	Inferior border rib n → superior border rib n+1	T1-T11 intercostals	Bucket-handle & pump-handle rib lift	Segmental expansion cue ("sniff test")
<b>Accessory Inspiratory</b> (recruited during exertion/obstruction)	SCM, scalenes, upper trap, serratus anterior/posterior, pectoralis minor/major (fixed arms), erector spinae	Various	CN XI, C2-8	Elevate sternum/ribs, extend spine	Overactivity → apical breathing; manual facilitation in spinal cord lesions
<b>Quiet Expiratory</b>	— (passive elastic recoil)	—	—	—	Loss of recoil in emphysema prolongs expiration - pursed-lip breathing slows collapse
<b>Forced Expiratory</b>	Internal intercostals, <b>abdominals</b> (rectus, obliques, transversus), serratus posterior inferior	T6-L1 ventral rami	—	Compress thorax, ↑ intra-abdominal pressure	Huff & cough techniques require strong abs; abdominal binder in high SCI

### 3 • Ventilatory Mechanics

#### 3.1 Pressure Dynamics (Quiet Breathing)

- **End-expiration:** Intra-alveolar ( $P_A$ ) = atmospheric ( $P_B$ ), trans-pulmonary ( $P_{TP} = P_A - P_{pl}$ )  $\approx +4$  cm  $H_2O$  holds lungs open.
- **Inspiration:** Diaphragm  $\downarrow$  → intrapleural ( $P_{pl}$ ) drops to  $\sim -6$  cm  $H_2O$  →  $P_A$  falls  $\sim -2$  cm  $H_2O$  → air flows in until  $P_A = P_B$ .
- **Expiration:** Relax →  $P_{pl}$  rises, elastic recoil  $\uparrow P_A$  to  $+2$  cm  $H_2O$  → air flows out.

#### 3.2 Lung Volumes & Capacities

Static volume	Avg adult (m)	Description	Clinical Interpretation
Tidal Volume (VT)	500 mL	Quiet breath in/out	$\downarrow$ in pain, neuromuscular weakness
Inspiratory Reserve (IRV)	3000 mL	Max extra inspiration	$\downarrow$ in restrictive disease
Expiratory Reserve (ERV)	1100 mL	Max extra expiration	$\downarrow$ in COPD (air-trapping)
Residual Volume (RV)	1200 mL	Air never exhaled	$\uparrow$ in emphysema
Vital Capacity (VC = VT+IRV+ERV)	4600 mL	Max movable air	Measured in spirometry
Total Lung Capacity (TLC)	5800 mL	VC + RV	$\uparrow$ in hyperinflation; $\downarrow$ fibrosis

- **FEV<sub>1</sub> / FVC ratio**  $< 70\%$  = obstruction; normal/high with  $\downarrow$  volumes = restriction.

### 4 • Gas Exchange Physiology

#### 4.1 Alveolar-Capillary Diffusion

**Fick's Law:**  $V'_{\text{gas}} = A \cdot D \cdot (P_1 - P_2) / T$  dot{V}\_{\text{gas}} = A \cdot D \cdot (P\_1 - P\_2) / T

- $A = 70 \text{ m}^2$ ;  $T \approx 0.5 \mu\text{m}$ ;  $D$  depends on solubility ( $\text{CO}_2 \times 20 > \text{O}_2$ ).

$\text{O}_2$ :

- Alveolar  $\text{PO}_2 \approx 100 \text{ mm Hg} \rightarrow$  arterial  $95 \text{ mm Hg}$ .

$\text{CO}_2$ :

- Alveolar  $\text{PCO}_2 \approx 40 \text{ mm Hg} \rightarrow$  venous  $46 \text{ mm Hg}$ .

**Diffusion-limited** (e.g.,  $\text{CO}$ , fibrosis); **perfusion-limited** ( $\text{O}_2, \text{CO}_2$ ). Exercise  $\uparrow$  cardiac output  $\rightarrow$  perfusion-limitation more prominent.

#### 4.2 Ventilation-Perfusion (V/Q) Matching

- Ideal  $V/Q \approx 0.8$  (4 L air / 5 L blood  $\text{min}^{-1}$ ).
- **Apices:**  $V/Q > 1$  (dead-space like)  $\rightarrow \uparrow \text{PAO}_2, \downarrow \text{PACO}_2$ .
- **Bases:**  $V/Q < 0.6$  (shunt-like)  $\rightarrow \downarrow \text{PAO}_2, \uparrow \text{PACO}_2$ .
- Therapeutic positioning (e.g., unilateral lung disease) places healthier lung **down** to optimise  $V/Q$ .

#### 4.3 Oxygen & $\text{CO}_2$ Transport

Mode	%	Mechanism	Clinically Relevant Curve
<b><math>\text{O}_2</math> bound to Hb</b>	98.5 %	1.34 $\text{mL O}_2 \cdot \text{g}^{-1} \text{ Hb}$ ; $\text{SaO}_2$ curve (sigmoid)	<b>Bohr shift:</b> $\uparrow \text{CO}_2, \uparrow \text{H}^+, \uparrow \text{temp}, \uparrow 2,3\text{-DPG} \rightarrow$ curve right (unloading)
<b><math>\text{O}_2</math> dissolved</b>	1.5 %	0.003 $\text{mL} \cdot \text{dL}^{-1} \cdot \text{mm Hg}^{-1}$	Basis of $\text{P}_{\text{aO}_2}$ reading — hypoxemia if $< 80 \text{ mm Hg}$
<b><math>\text{CO}_2</math> dissolved</b>	10 %	Direct plasma solution	Hyperventilation $\downarrow \text{PaCO}_2$
<b>Carbamino-Hb</b>	20 %	Binds globin	<b>Haldane effect:</b> $\text{O}_2$ unloading $\uparrow \text{CO}_2$ carriage
<b>Bicarbonate (<math>\text{HCO}_3^-</math>)</b>	70 %	Carbonic anhydrase in RBC	Respiratory acidosis/alkalosis management

#### 5 • Structure-Function-Clinical Correlations

Phenomenon	Anatomical Basis	Physiotherapy Note
<b>Diaphragmatic descent</b> increases vertical thoracic diameter	Central tendon anchored to pericardium $\rightarrow$ can affect venous return	Abdominal breathing aids venous return in HF
<b>Bucket-handle rib motion</b> widens transverse diameter	External intercostals pivot on costotransverse joints	Lateral costal expansion cue post-thoracotomy
<b>Collateral ventilation (pores of Kohn)</b> opens at deep breaths	Alveolar pores / canals of Lambert	Incentive spirometry prevents atelectasis
<b>Dynamic airway compression</b> during forced expiration	Intrapleural pressure exceeds airway pressure distal to equal-pressure point	COPD teach pursed-lip breathing to move EPP distally and keep airways splinted

#### 6 • Self-Check Quiz (Answers below)

1. **Which abdominal muscle is most active during a forceful cough?**
2. **Explain why  $\text{FEV}_1$  is reduced more than  $\text{FVC}$  in obstructive disease.**
3. **What positional strategy improves oxygenation in unilateral pneumonia and why?**
4. **Describe the Bohr effect in simple terms.**
5. **During vigorous exercise, which variable—diffusing capacity or cardiac output—limits arterial  $\text{O}_2$  content first in a healthy adult?**

#### Answers

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1. **Rectus abdominis** (with internal oblique) generates high intra-abdominal pressure.
2. **Airflow limitation** (small-airway collapse) prolongs expiration, so the volume exhaled in first second (FEV<sub>1</sub>) drops disproportionately compared with total exhaled volume (FVC).
3. **“Good lung down”**—placing healthy lung in the dependent position maximises perfusion matching to better-ventilated alveoli through gravity-directed blood flow.
4. Rising **CO<sub>2</sub>/H<sup>+</sup> shifts the haemoglobin-O<sub>2</sub> dissociation curve right**, enabling easier O<sub>2</sub> unloading to tissues.
5. **Cardiac output/perfusion** becomes limiting; diffusing capacity rises (recruitment) and usually exceeds demand in healthy lungs.

## 7 • Suggested Practical / Lab Activities

Activity	Skill Gained
<b>Surface EMG of respiratory muscles</b> during quiet vs pursed-lip breathing	Muscle recruitment analysis
<b>Spirometry workshop</b> – perform, interpret FVC, FEV <sub>1</sub> , MVV	Identify obstructive vs restrictive patterns
<b>Incentive-spirometer &amp; flow-volume loop simulation</b>	Teach patient coaching cues
<b>Blood-Gas Case Scenarios</b>	Diagnose respiratory vs metabolic acidosis, devise breathing strategies

## 8 • Key Take-Home Points

- **Diaphragm dominates quiet inspiration**; accessory muscles signal increased load or dysfunction.
- **Breathing mechanics hinge on pressure gradients** created by thoracic and abdominal muscle action plus lung compliance.
- **Gas exchange efficiency depends on intact alveolar-capillary membrane, optimal V/Q matching and Hb capacity**.
- Physiotherapists manipulate **positioning, breathing patterns, airway-clearance techniques and exercise intensity** to optimise these variables across a wide spectrum of cardiorespiratory conditions.

## Part 3 | Common Respiratory Disorders — Asthma, COPD & Pneumonia

### 1 Learning Objectives

By the end of this part you should be able to ...

1. **Describe the pathophysiology, hallmark clinical signs, and diagnostic criteria** for bronchial asthma, chronic obstructive pulmonary disease (COPD), and pneumonia.
2. **Differentiate obstructive from restrictive spirometry patterns** and recognise red-flag features that warrant urgent referral.
3. **Outline evidence-based physiotherapy interventions**—airway-clearance, breathing retraining, exercise prescription, and patient education—for each disorder.
4. **Apply infection-control and safety precautions** relevant to acute respiratory infections.

### 2 Disorder Snapshots

Feature	Asthma	COPD (Chronic Bronchitis &/or Emphysema)	Pneumonia

Feature	Asthma	COPD (Chronic Bronchitis &/or Emphysema)	Pneumonia
<b>Core Pathology</b>	Chronic airway inflammation → hyper-responsiveness, reversible bronchoconstriction	Progressive, largely irreversible airflow limitation; chronic inflammation + parenchymal destruction	Acute infection of distal airways & alveoli (bacterial, viral, fungal)
<b>Key Triggers / Risks</b>	Allergens, exercise, cold air, irritants, viral URTI	Tobacco smoke, biomass fuel, pollution, $\alpha$ -1 antitrypsin deficiency	Age < 5 / > 65, chronic disease, aspiration, immobility
<b>Typical Symptoms</b>	Episodic wheeze, cough (night/early AM), chest tightness, prolonged expiration	Chronic cough, sputum, exertional dyspnoea, wheeze, weight loss	Fever, productive cough, pleuritic pain, dyspnoea, fatigue
<b>Spirometry</b>	Obstructive; FEV <sub>1</sub> /FVC < 70 % but <b>reversibility &gt; 12 % &amp; 200 mL</b> post-bronchodilator	Obstructive; FEV <sub>1</sub> /FVC < 70 % with < 12 % reversibility	Often restrictive (↓ VC) + diffusion defect during acute phase
<b>Radiology</b>	Usually normal or hyper-inflated on attack	Hyper-inflation, flattened diaphragm, bullae	Lobar/segmental consolidation or interstitial pattern
<b>Blood Gases</b>	Mild hypoxemia during attack; PaCO <sub>2</sub> ↓ or normal	Chronic compensated hypercapnia; hypoxemia	Hypoxemia ± hypercapnia depending on severity
<b>Clinical Red Flags</b>	Silent chest, SpO <sub>2</sub> < 90 %, PEFR < 33 % predicted	Acute exacerbation with drowsiness, cyanosis, RR > 30	Rapid RR > 30, SpO <sub>2</sub> < 92 % on air, sepsis criteria

### 3 Physiotherapy Management Framework

Stage	Asthma	COPD	Pneumonia
<b>Acute (exacerbation / hospitalization)</b>	<ul style="list-style-type: none"> <li>High-Fowler position.</li> <li>Teach <b>pursed-lip breathing</b> (PLB).</li> <li>Gentle thoracic expansion with hold for collateral ventilation.</li> <li>Short bouts of UL supported positioning (tripod).</li> </ul>	<ul style="list-style-type: none"> <li>PLB + <b>paced breathing</b> with activity.</li> <li><b>Active Cycle of Breathing Technique (ACBT)</b> avoiding fatigue.</li> <li>Early mobilisation (sit ↔ stand).</li> </ul>	<ul style="list-style-type: none"> <li>Ensure adequate <b>oxygen therapy</b> &amp; SpO<sub>2</sub> monitoring.</li> <li><b>Thoracic expansion exercises</b> to improve ventilation.</li> <li><b>Supported cough / huff</b> to clear secretions; splint incision if post-op.</li> <li>Mobilise as tolerated to prevent deconditioning.</li> <li>Gradual re-conditioning post-infection; monitor for desaturation.</li> <li>Segmental breathing + incentive spirometry to prevent atelectasis.</li> <li>Airway-clearance if residual sputum.</li> <li>Education on hydration and early mobilisation.</li> </ul>
<b>Sub-acute / Stable</b>	<ul style="list-style-type: none"> <li>Identify triggers; breathing retraining (diaphragmatic, nasal).</li> <li>Aerobic training 3-5 d·wk<sup>-1</sup>, 40-60 % HRR.</li> <li>Inspiratory muscle training (IMT) if reduced PI<sub>max</sub>.</li> <li>Patient education: inhaler technique, PEFR diary.</li> </ul>	<ul style="list-style-type: none"> <li><b>Pulmonary rehabilitation</b> 6-12 wk: endurance + strength (upper &amp; lower limb), IMT.</li> <li>Postural drainage &amp; manual techniques for chronic bronchitis.</li> <li>Energy-conservation and pacing strategies.</li> <li>Education: smoking cessation, nutrition, vaccine update.</li> </ul>	
<b>Long-term Goals</b>	Symptom-free daily life; maintain airway health; prevent remodeling.	Slow decline of FEV <sub>1</sub> ; ↓ hospitalisations; enhance QOL.	Restore premorbid function; prevent recurrence / complications (VTE, de-conditioning).

### 4 Key Physiological Concepts for PT Intervention

#### 1. Dynamic Hyperinflation (COPD):

Air trapping ↑ end-expiratory lung volume → flattened diaphragm & ↓ inspiratory capacity.  
PLB & interval training allow longer expiratory time & ↓ hyperinflation.

#### 2. Asthmatic Airway Resistance:

Bronchospasm + mucus plugs narrow radius (Poiseuille:  $R \propto 1/r^4$ ).

Bronchodilator use + warm-up can attenuate exercise-induced bronchoconstriction.

**3. V/Q Mismatch in Pneumonia:**

Consolidated alveoli perfused but not ventilated → shunt.

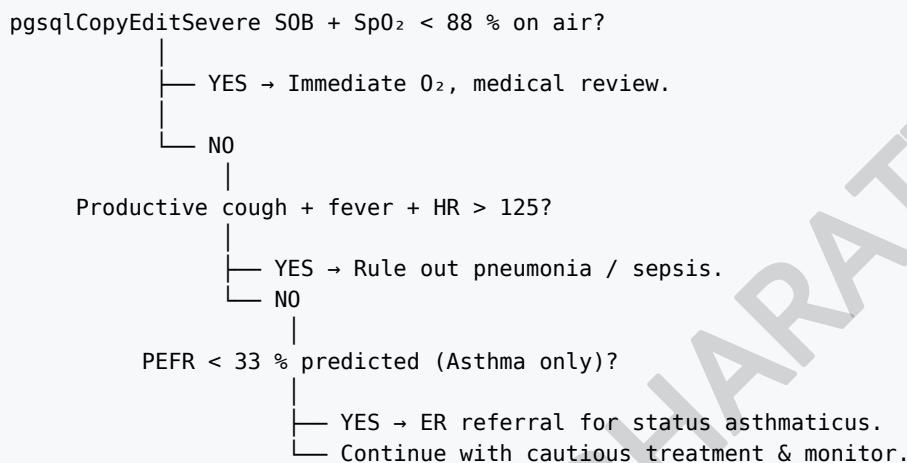
Position with affected lung **uppermost** to improve overall V/Q.

**4. Oxygen-Hb Dissociation (Acute Exacerbation):**

↑  $CO_2$  + ↓ pH shift curve right (Bohr), aiding  $O_2$  unloading but risking hypoxemia.

Controlled  $O_2$  (target 88–92 %) in COPD prevents  $CO_2$  narcosis.

## 5 Red-Flag Decision Tree for the Physio



## 6 Self-Check Quiz (answers below)

1. What spirometric criterion distinguishes reversible airway obstruction in asthma from COPD?
2. Why might administering high-flow oxygen ( $FiO_2 > 0.4$ ) to a patient with chronic hypercapnic COPD precipitate  $CO_2$  retention?
3. State two breathing-control strategies useful during an acute asthmatic attack.
4. Which lung segments are most prone to aspiration pneumonia when a supine patient aspirates, and how would you position them for drainage?
5. List three absolute contraindications to chest percussion.

## Answers

1. A post-bronchodilator increase in  $FEV_1 \geq 12\% \text{ and } \geq 200 \text{ mL}$  denotes reversibility typical of asthma.
2. High  $FiO_2$  suppresses the **hypoxic respiratory drive** and worsens V/Q mismatch by reversing hypoxic pulmonary vasoconstriction, leading to  **$CO_2$  narcosis**.
3. **Pursed-lip breathing, forward-leaning with arm support ("tripod")**, controlled diaphragmatic breathing.
4. **Posterior segments of upper lobes and superior segments of lower lobes**; position patient in **prone with head-down 15–30°** to drain these areas.
5. **Undrained pneumothorax, severe osteoporosis, rib fracture, unstable haemodynamics, or recent thoracic surgery incision** without clearance.

## 7 Key Take-Home Points

- **Asthma is reversible; COPD is progressive and largely irreversible; pneumonia is infectious.** Each requires distinct but overlapping PT strategies.



- **Accurate assessment (vitals, spirometry, auscultation)** enables safe progression of therapy and early recognition of exacerbations.
- **Breathing retraining, airway-clearance, targeted positioning, and exercise** are cornerstone interventions—tailored to pathophysiology and patient tolerance.
- Red-flag knowledge and timely referral are critical for patient safety.

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