

## 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>Lower</b>	<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

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

#### 4 Gross Lung Anatomy

Aspect	Right Lung	Left Lung	Functional Angle
<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)** → **bronchoconstriction** / **mucus ↑**; **Sympathetic** → **bronchodilation** / **mucus ↓** — basis for inhaler pharmacology.

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

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

#### 6 Structure-Function-Clinical Correlations

Anatomy Feature	Physiological Benefit	PT Application
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 ...

1. **List all primary and accessory muscles of inspiration and expiration**, their origins/insertions, nerve supply and kinesiological actions.
2. **Explain the pressure-volume relationships** (Boyle's law) that drive airflow during quiet and forced ventilation.
3. **Define static and dynamic lung volumes and capacities**, and relate them to spirometric patterns in obstructive vs restrictive disease.
4. **Describe alveolar-capillary gas exchange** using Fick's law, including diffusion-limitation vs perfusion-limitation concepts.
5. **Outline oxygen and carbon-dioxide transport mechanisms** (Hb dissociation curve, Haldane & Bohr effects).
6. **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 ≥ 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<sub>2</sub>O holds lungs open.
- **Inspiration:** Diaphragm ↓ → intrapleural ( $P_{pl}$ ) drops to  $\sim -6$  cm H<sub>2</sub>O →  $P_A$  falls  $\sim -2$  cm H<sub>2</sub>O → air flows in until  $P_A = P_B$ .
- **Expiration:** Relax →  $P_{pl}$  rises, elastic recoil ↑  $P_A$  to  $+2$  cm H<sub>2</sub>O → air flows out.

#### 3.2 Lung Volumes & Capacities

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

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

### 4 • Gas Exchange Physiology



#### 4.1 Alveolar-Capillary Diffusion

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

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

$\text{O}_2$ :

- Alveolar  $\text{PO}_2 \approx 100 \text{ mm Hg}$  → arterial  $95 \text{ mm Hg}$ .  
 $\text{CO}_2$ :
- Alveolar  $\text{PCO}_2 \approx 40 \text{ mm Hg}$  → venous  $46 \text{ mm Hg}$ .

**Diffusion-limited** (e.g., CO, fibrosis); **perfusion-limited** ( $\text{O}_2$ ,  $\text{CO}_2$ ). Exercise ↑ cardiac output → 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) – ↑  $\text{PAO}_2$ , ↓  $\text{PACO}_2$ .
- **Bases:**  $V/Q < 0.6$  (shunt-like) – ↓  $\text{PAO}_2$ , ↑  $\text{PACO}_2$ .
- 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> ↑ $\text{CO}_2$ , ↑ $\text{H}^+$ , ↑ temp, ↑ 2,3-DPG → 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 ↓ $\text{PaCO}_2$
<b>Carbamino-Hb</b>	20 %	Binds globin	<b>Haldane effect:</b> $\text{O}_2$ unloading ↑ $\text{CO}_2$ 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 → 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 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

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, α-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 <b>&lt; 12 % reversibility</b>	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> </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 P<sub>Imax</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>	<ul style="list-style-type: none"> <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>Long-term Goals</b>	Symptom-free daily life; maintain airway health; prevent remodeling.	Slow decline of FEV <sub>1</sub> ; ↓ hospitalisations; enhance QOL.	Restore pre-morbid 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.

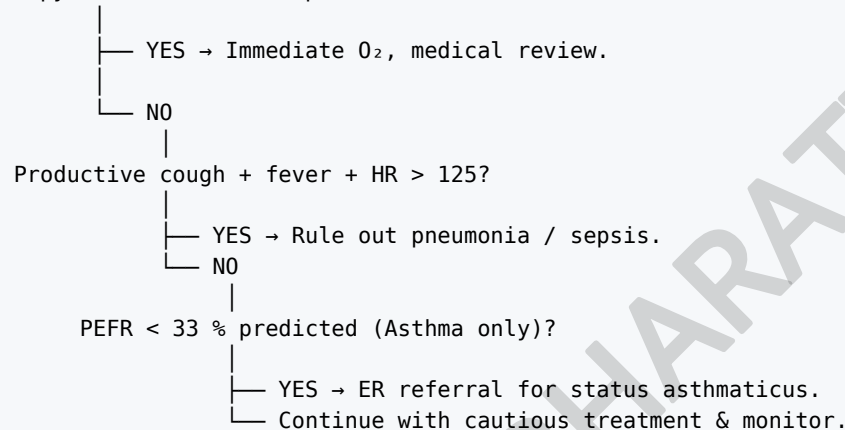
4. **Oxygen-Hb Dissociation (Acute Exacerbation):**

↑ CO<sub>2</sub> + ↓ pH shift curve right (Bohr), aiding O<sub>2</sub> unloading but risking hypoxemia.

Controlled O<sub>2</sub> (target 88-92 %) in COPD prevents CO<sub>2</sub> narcosis.

## 5 Red-Flag Decision Tree for the Physio

pgsqlCopyEditSevere SOB + SpO<sub>2</sub> < 88 % on air?



## 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<sub>2</sub> > 0.4) to a patient with chronic hypercapnic COPD precipitate CO<sub>2</sub> 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<sub>1</sub> ≥ 12 % and ≥ 200 mL** denotes reversibility typical of asthma.
2. High FiO<sub>2</sub> suppresses the **hypoxic respiratory drive** and worsens V/Q mismatch by reversing hypoxic pulmonary vasoconstriction, leading to **CO<sub>2</sub> 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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