

Chapter 5. Respiratory Physiology

Part 1 | Anatomy of the Respiratory System & Mechanics of Breathing

1 Learning Objectives

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

- 1. Map the entire respiratory tract from nares to alveolus, noting epithelium and support tissues at each level.
- 2. **Describe lung macro- and micro-anatomy**—lobes, segments, pleurae, acinus—and explain how structure facilitates gas exchange.
- 3. **Explain the mechanics of quiet and forced ventilation**, including pressure-volume relationships, compliance, surface tension and airway resistance.
- 4. **Apply anatomical and mechanical principles** to physiotherapy techniques such as positioning, breathing retraining, airway-clearance and exercise prescription.

2 Respiratory-Tract Architecture

Region	Sub-Structures	Epithelium & Special Features	Primary Functions	PT Relevance
Upper Tract	Nose, nasal cavity, sinuses, pharynx, larynx (to vocal folds)	Pseudostratified ciliated columnar with goblet cells; rich vascular plexus; stratified squamous in oropharynx	Warm, humidify, filter; phonation	Encourage nasal- diaphragmatic breathing for humidification & airway protection
Conducting Zone (Lower Tract to terminal bronchioles)	Trachea → main, lobar, segmental bronchi → bronchioles → terminal bronchioles	Gradual loss of cartilage; smooth-muscle layer 1; Clara (club) cells secrete surfactant-like fluid in bronchioles	Air conduit; mucociliary clearance; regulate flow via bronchomotor tone	Bronchodilator timing before PT improves airway calibre; manual percussion tracks segmental bronchi
Respiratory Zone	Respiratory bronchioles → alveolar ducts → alveolar sacs (~300 million alveoli)	Type I pneumocytes (gas diffusion) & Type II (surfactant); thin interstitium with capillary web	Gas exchange (O₂	Postural drainage positions target specific segments; deep-breathing spreads surfactant

Bronchopulmonary Segments

Right = 10, Left = 8-10; each fed by its own tertiary bronchus & artery—surgical and drainage units.

3 Lung & Pleural Anatomy

Item	Structure	Key Points	Clinical Angle
Lobes & Fissures	R: Superior, Middle, Inferior (horizontal + oblique fissure); L: Superior, Inferior (oblique only)	Right middle-lobe bronchus narrow—aspiration risk	Left side-lying drains right middle lobe
Pleurae	Visceral (lung surface), Parietal (thoracic wall) with $\sim\!15~\text{mL}$ serous fluid	Parietal innervated (pain); maintains – 4 mm Hg intrapleural P	Pleural effusion impedes expansion—thoracic expansion exs post-thoracentesis
Acinus	Terminal bronchiole + respiratory bronchioles + alveolar ducts/sacs	Functional gas-exchange unit	Emphysema destroys acini walls—teach pursed-lip breathing to prevent collapse

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4 Mechanics of Breathing

4.1 Pressure-Volume Relationships (Boyle's Law)

Event	Muscles Involved	Intrapleural P (P_pl)	Alveolar P (P_A)	Airflow
Quiet inspiration	Diaphragm ↓ 1.5 cm; external intercostals	-6 cm H₂O	-2 → 0	500 mL in
Quiet expiration	Passive recoil	-4 cm H ₂ O → -2	+2 → 0	500 mL out
Forced inspiration	+ SCM, scalenes, serratus	P_pl may reach -30 cm H₂O	Larger ΔP_A	Exercise, asthma
Forced expiration	Abdominals, internal intercostals	P_pl positive	P_A up to +30	Cough, huff techniques

4.2 Lung Compliance ($C = \Delta V/\Delta P$)

Normal combined ~ 200 mL cm⁻¹ H₂O.

- ↓ Compliance = fibrosis, edema → ↑ work of breathing (WoB).
- ↑ Compliance = emphysema → poor recoil, air-trapping.

4.3 Surface Tension & Surfactant

- Law of Laplace: P=2T/rP = 2T/rP=2T/r.
- Surfactant (DPPC from Type II cells) lowers T mostly in small alveoli → stability; deep breaths ↑ release → incentive spirometry rationale.

4.4 Airway Resistance (Raw)

Major component in 2 - 5 th gen bronchi; Raw $\propto 1/r^4$.

Bronchoconstriction (asthma) ↑ Raw—pre-treat with β₂-agonist before exercise.

5 Applied Physiotherapy Examples

Technique	Anatomy/Mechanic Target	Effect
Diaphragmatic breathing	Enlarge vertical thoracic dimension, improve compliance	↓ Accessory muscle work; better ventilation-perfusion
Positions (e.g., high Fowler)	Lower abdominal pressure, maximise diaphragm	Useful in COPD to reduce dyspnoea
PEP devices / Autogenic drainage	Raise P_pl during expiration to splint small airways	Mobilise secretions without excessive WoB
Manual percussion over posterior basal segments	Follows segmental bronchus orientation	Loosen secretions; combine with gravity drainage

6 Self-Check Quiz (answers below)

- 1. Which cell type produces pulmonary surfactant?
- 2. Why does the right lung have a higher aspiration incidence?
- 3. State Laplace's equation and its implication for alveolar stability.
- 4. Name two factors that decrease lung compliance.
- 5. During quiet breathing, what proportion of tidal volume is generated by diaphragmatic descent?

Answers:

1. Type II pneumocytes.

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- 2. Right main bronchus is wider, shorter and more vertical, creating a direct path for aspirated material.
- 3. P=2T/rP = 2T/rP=2T/r; without surfactant, smaller alveoli (small r) would require higher pressure and collapse into larger ones.
- 4. Pulmonary fibrosis, alveolar edema, atelectasis, surface-tension increase (loss of surfactant).
- 5. Approximately 75 %.

7 Key Take-Home Points

- Anatomical transitions—from cartilaginous bronchi to elastic alveoli—mirror functional shifts from conducting air to exchanging gases.
- **Pressure, compliance and resistance** dictate the mechanics of breathing; physiotherapists manipulate these via positioning, breathing techniques and mobilisations.
- Surfactant, pleural integrity and diaphragm function are critical—any compromise raises work of breathing and must be addressed in rehabilitation plans.

Part 2 | Gas Exchange & Transport

1 Learning Objectives

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

- 1. Explain the factors that govern diffusion of O₂ and CO₂ across the alveolar-capillary membrane.
- 2. Describe the three forms in which each gas travels in blood and quantify their relative contributions.
- 3. **Interpret the oxy- and carbamino-hemoglobin dissociation curves,** including Bohr and Haldane effects, and predict shifts during exercise or disease.
- 4. **Apply gas-transport principles to physiotherapy practice,** such as exercise prescription in COPD or oxygentitration during ambulation.

2 Alveolar-Capillary Gas Exchange

Factor (Fick's law)	Typical Value	Influence on PT
Surface area (A)	≈ 70 m²	Reduced in emphysema \rightarrow teach pursed-lip breathing to recruit alveoli
Membrane thickness (T)	≈ 0.5 µm	Edema/fibrosis $\uparrow T \rightarrow$ lower training intensities
Diffusion coefficient (D)	$CO_2 \approx 20 \times O_2$	CO ₂ elimination usually easier; focus on ventilation for hypoxemia
Partial-pressure gradient (ΔP)	O ₂ : 60 mm Hg CO ₂ : 6 mm Hg	Altitude ↓ PAO₂; supplemental O₂ may be required

Perfusion-limited gases (O_2 , CO_2): raising cardiac output (exercise) still delivers full equilibration in healthy lungs. Diffusion-limited (CO, diseased lungs): diffusion becomes the rate-limiting step.

3 Oxygen Transport

Form	% of Total	Details
Bound to hemoglobin	98.5 %	1 Hb binds 4 O2 (1.34 mL O2 \cdot g^-1). SaO2 reflects occupancy.
Dissolved in plasma	1.5 %	$0.003~\text{mL}\cdot\text{dL}^{-1}\cdot\text{mm Hg}^{-1}$ (basis of PaO ₂). Clinical for hyperbaric O ₂ .

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3.1 Oxyhemoglobin Dissociation Curve

Sigmoidal shape—co-operativity.

• P50 (PaO₂ at 50 % saturation) ≈ 26-27 mm Hg.

Shifts RIGHT (↓ affinity, ↑ unloading)

• ↑ CO₂ / ↓ pH (Bohr) • ↑ Temp • ↑ 2,3-DPG (chronic hypoxia, training)

Shifts LEFT (↑ affinity)

• Alkalosis • Hypothermia • Fetal Hb • CO poisoning

PT Pearl: Post-surgical patients with fever off-load O₂ to tissues more readily—monitor saturation but encourage early ambulation.

4 Carbon-Dioxide Transport

Form % of Total CO₂ Reaction

Bicarbonate (HCO₃⁻) \approx **70** % CO₂ + H₂CO₃ \rightleftharpoons HCO₃⁻ + H⁺ (carbonic anhydrase within RBC)

Carbamino compounds 20 % CO₂ binds globin of Hb (favoured by deoxy-Hb)

Dissolved CO₂ 10 % Henry's law; measured as PaCO₂

4.1 Haldane Effect

Deoxygenated Hb binds CO_2/H^+ more easily; venous blood carries more CO_2 . During pulmonary O_2 loading, CO_2 is released \rightarrow drives expiration.

5 Matching Delivery to Demand

Oxygen Delivery (DO₂)=CO×(1.34 × [Hb] × SaO₂+0.003×PaO₂)\text{Oxygen Delivery (DO₂)} = \text{CO} \times (\text{1.34 × [Hb] × SaO₂} + 0.003 × PaO₂)Oxygen Delivery (DO₂)=CO×(1.34 × [Hb] × SaO₂+0.003×PaO₂)

Exercise: \uparrow CO + small \downarrow mixed-venous saturation \rightarrow muscles extract more O₂. Anemia: Lower [Hb]—heart compensates with higher CO; monitor HR & fatigue.

6 Applied Physiotherapy Scenarios

Scenario	Underlying Gas Physiology	Intervention
COPD exacerbation	V/Q mismatch → PaO ₂ ↓ ; chronic PaCO ₂ ↑ (blunted chemoreflex)	Target SpO ₂ 88–92 %. Teach diaphragmatic breathing to reduce dead space.
High-altitude trek	↓ Barometric pressure → ↓ PAO_2 → right shift via 2,3-DPG	Gradual ascent, interval breathing drills.
Pulmonary fibrosis	Thickened membrane (\uparrow T) diffusion-limited O_2	Interval training with O ₂ ; monitor desaturation carefully.
HIIT session for athlete	Large $\Delta CO_2,$ temp 1, pH \downarrow \rightarrow Bohr right shift aids unloading	Active recovery keeps ventilation \uparrow to clear CO ₂ .

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7 Self-Check Quiz (answers below)

- 1. What percentage of total oxygen content is carried dissolved in plasma at sea level?
- 2. State two conditions that shift the oxyhemoglobin curve left.
- 3. Why is carbonic anhydrase essential for rapid CO2 transport?
- 4. During heavy exercise PaO₂ remains steady yet SaO₂ may drop slightly—why?
- 5. Explain the Haldane effect in one sentence.

Answers:

- 1. Roughly 1.5 %.
- 2. Alkalosis, hypothermia (also fetal Hb, low CO₂).
- 3. It catalyses CO₂ hydration to bicarbonate inside RBCs, vastly accelerating the reaction and allowing large CO₂ carriage.
- 4. Mixed-venous saturation falls, temperature & acidity rise—curve shifts right, causing slightly lower SaO₂ despite constant PaO₂.
- 5. Oxygen unloading at tissues increases Hb affinity for CO_2/H^+ , whereas oxygen binding in lungs promotes CO_2 release.

8 Key Take-Home Points

- Gas transport hinges on diffusion at the alveolus and hemoglobin chemistry in the blood.
- Curve shifts are your physiotherapy allies or enemies—know what moves them.
- Interventions that alter ventilation, temperature, pH or cardiac output immediately influence O₂/CO₂ exchange—adjust dosage accordingly.

Part 3 | Regulation of Breathing & Pathophysiology

1 Learning Objectives

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

- 1. **Describe the neural hierarchy that generates and modulates the breathing rhythm**, including medullary, pontine, cortical and peripheral inputs.
- 2. **Explain chemical control of ventilation**, detailing the role of central and peripheral chemoreceptors, blood-gas feedback and reflex loops.
- 3. **Predict ventilatory responses during exercise, sleep, altitude and disease** by integrating neural and chemical mechanisms.
- 4. **Relate dysregulation of these systems to common respiratory disorders** (e.g., COPD, asthma, sleep apnoea, restrictive lung disease) and outline implications for physiotherapy.

2 Neural Control of Breathing

Level	Nuclei / Pathways	Core Function	Key Points for PT
Medullary rhythm generator	Dorsal respiratory group (DRG) Ventral respiratory group	Sets basic eupnoeic rate (12-16 breaths min ⁻¹); DRG drives diaphragm via phrenic; VRG adds accessory muscles for forceful breathing	Spinal-cord injury above C3—lose phrenic output → ventilatory support

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Level	Nuclei / Pathways	Core Function	Key Points for PT
Pontine centres	Pneumotaxic (Kölliker- Fuse)Apneustic	Fine-tunes inspiratory off- switch (IT < 2 s) and pattern smoothing	Damage → apneustic breathing (prolonged inspiratory gasps)
Cortical override	Motor cortex → corticospinal tracts	Voluntary breath-hold, speech, singing	Pulmonary rehab uses paced speech counting for rhythm control
Subcortical	Limbic, hypothalamus	Emotion, temperature effects	Anxiety → hyperventilation; teach slow diaphragmatic breathing

3 Chemical Regulation

Receptor	Location	Stimulus	Ventilatory Effect	Clinical Example
Central chemoreceptors	Ventrolateral medulla (brain-ECF)	↑ PaCO ₂ (via ↑ H+ in CSF)	Powerful ↑MV (↑ depth & rate) within seconds	Chronic CO ₂ retention in COPD blunts response—rely on peripheral hypoxic drive
Peripheral chemoreceptors	Carotid bodies (glomus cells, CN IX) > Aortic bodies (CN X)	<pre></pre>	Rapid ↑MV, sympathetic spill-over ↑HR	High-altitude hypoxia stimulates ventilation; acclimatisation over days
Pulmonary stretch (Hering-Breuer)	Smooth-muscle of bronchi/bronchioles → vagus	Lung inflation > 800 mL	Terminates inspiration, prolongs expiration	Important during mechanical ventilation; explains sigh reflex
Irritant & J- receptors	Airway epithelium; alveolar walls	Smoke, dust, edema	Bronchoconstriction, cough, rapid shallow breathing	Asthma trigger avoidance; IPF rapid- shallow pattern

4 Integrated Ventilatory Responses

Situation	Neural/Chemical Integration	Resultant Pattern	PT Notes
Dynamic exercise	Central command (motor cortex) + mechano-metaboreceptors (muscle/joint) -> feed-forward	Phase I fast rise, Phase I exponential, Phase III steady	I Anticipate VE plateau at 60–80 % VO2max in healthy; COPD reaches ventilatory ceiling early
Sleep	↓ Wakeful cortical drive, ↑ PaCO₂ setpoint	\downarrow VT & RR \rightarrow \downarrow MV 10-15 %	Sleep apnoea: pharyngeal collapse + impaired chemoreflex arousal
Pregnancy	Progesterone ↑ central CO₂ sensitivity	Mild chronic hypocapnia (~30 mm Hg)	Consider when interpreting ABG; avoid hyperventilating during Lamaze
High altitude (acute)	Hypoxic peripheral drive 1, hypocapnic alkalosis inhibits central	Periodic Cheyne-Stokes; then renal HCO₃ loss resets pH	Graded ascent, acetazolamide aids acclimatisation

5 Respiratory Disorders - Physiological Basis

Disorder	Dysregulated Mechanism	Physiological Impact	Physiotherapy Considerations
COPD (chronic hypercapnia)	Central chemoreceptor dampening; reliance on hypoxic drive	High FiO ₂ > 0.60 may suppress ventilation \rightarrow CO ₂ narcosis	Target SpO ₂ 88-92 %; pacing, PLB to lower dynamic hyperinflation
Asthma	Hyper-responsive airway smooth-muscle; irritant receptor over-activity	Variable airflow obstruction; ↑ airway resistance	Pre-exercise β_2 agonist; prolonged warm-up; monitor PEFR
Restrictive lung disease (IPF)	Thickened alveolar membrane ↓ compliance; J-receptor stimulation	Rapid shallow breathing, high work	Interval training at low VT; inspiratory muscle training

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Disorder	Dysregulated Mechanism	Physiological Impact	Physiotherapy Considerations
Obstructive sleep apnoea	Pharyngeal dilator muscle collapse; blunted chemoreflex arousal	Intermittent hypoxia → sympathetic surges	CPAP adherence; daytime aerobic & weight-loss programmes
Central hypoventilation (e.g., brain-stem stroke)	Medullary drive failure	Persistent hypercapnia, hypoxemia	Diaphragmatic pacing if intact phrenic; careful monitoring during mobilisation

6 Self-Check Quiz (answers below)

- 1. Which chemoreceptor type is most sensitive to acute changes in PaCO2 and why?
- 2. Describe the neural pathway of the Hering-Breuer inflation reflex.
- 3. What pattern of breathing is typical of severe metabolic acidosis, and which centres drive it?
- 4. Why can high-dose oxygen precipitate CO₂ retention in chronic COPD?
- 5. List two physiological adjustments that occur during acclimatisation to high altitude.

Answers:

- 1. Central chemoreceptors—lack blood-brain barrier to CO₂; CO₂ diffuses → H⁺ in CSF, powerful stimulus.
- 2. Lung stretch receptors → vagus nerve afferent → medullary DRG → inhibits inspiratory neurons, ending inspiration.
- 3. **Kussmaul breathing**—deep, rapid respirations driven by peripheral chemoreceptor stimulation of medullary centres.
- 4. Oxygen abolishes hypoxic drive and worsens V/Q mismatch, leading to further rise in PaCO₂ (Haldane effect + hypoventilation).
- 5. Hyperventilation with renal HCO₃⁻ loss (pH compensation), ↑ 2,3-DPG shifting Hb curve right, ↑ erythropoietin → polycythaemia.

7 Key Take-Home Points

- Breathing rhythm is automatic yet plastic, shaped by brain-stem pacemakers, cortical input and robust chemo-/mechano-feedback loops.
- CO₂ is the main chemical driver; O₂ takes command only when dangerously low.
- Respiratory disorders often reflect **maladapted regulation**—physiotherapists can modulate drive via positioning, pacing, airway-clearance and education.

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