

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 → terminal bronchioles	Gradual loss of cartilage; smooth-muscle layer ↑; 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 (O ₂ ⇌ CO ₂)	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 ~15 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

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 H ₂ O	-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 H ₂ O	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 = ΔV/ΔP)

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 ∝ 1/r⁴.

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.



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 oxygenation during ambulation.

2 Alveolar-Capillary Gas Exchange

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

Perfusion-limited gases (O₂, CO₂): 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 O ₂ (1.34 mL O ₂ · g ⁻¹). SaO ₂ reflects occupancy.
Dissolved in plasma	1.5 %	0.003 mL · dL ⁻¹ · mm Hg ⁻¹ (basis of PaO ₂). Clinical for hyperbaric O ₂ .



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 CO ₂	Reaction
Bicarbonate (HCO ₃ ⁻)	≈ 70 %	CO ₂ + H ₂ O ⇌ H ₂ CO ₃ ⇌ HCO ₃ ⁻ + H ⁺ (carbonic anhydrase within RBC)
Carbamino compounds	20 %	CO ₂ binds globin of Hb (favoured by deoxy-Hb)
Dissolved CO ₂	10 %	Henry's law; measured as PaCO ₂

4.1 Haldane Effect

Deoxygenated Hb binds CO₂/H⁺ more easily; venous blood carries more CO₂. During pulmonary O₂ loading, CO₂ is released → drives expiration.

5 Matching Delivery to Demand

Oxygen Delivery (DO₂)=CO×(1.34 × [Hb] × SaO₂+0.003×PaO₂)

$$\text{Oxygen Delivery (DO}_2\text{)} = \text{CO} \times (\text{1.34} \times [\text{Hb}] \times \text{SaO}_2 + 0.003 \times \text{PaO}_2)$$

Exercise: ↑ CO + small ↓ mixed-venous saturation → 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 (↑ T) diffusion-limited O ₂	Interval training with O ₂ ; monitor desaturation carefully.
HIIT session for athlete	Large ΔCO ₂ , temp ↑, pH ↓ → Bohr right shift aids unloading	Active recovery keeps ventilation ↑ to clear CO ₂ .

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 CO₂/H⁺, whereas oxygen binding in lungs promotes CO₂ 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	<ul style="list-style-type: none">• Pre-Bötzinger complex (pacemaker)• Dorsal respiratory group (DRG)• Ventral respiratory group (VRG)	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

Level	Nuclei / Pathways	Core Function	Key Points for PT
Pontine centres	<ul style="list-style-type: none"> • 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)	↑ PaCO₂ (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)	↓ PaO₂ (< 60 mm Hg); ↑ PaCO ₂ ; ↓ pH	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 II exponential, Phase III steady	Anticipate VE plateau at 60–80 % VO ₂ max in healthy; COPD reaches ventilatory ceiling early
Sleep	↓ Wakeful cortical drive, ↑ PaCO ₂ set-point	↓ VT & RR → ↓ MV 10–15 %	Sleep apnoea: pharyngeal collapse + impaired chemoreflex arousal
Pregnancy	Progesterone ↑ central CO ₂ sensitivity	Mild chronic hypocapnia (~30 mm Hg)	Consider when interpreting ABG; avoid hyperventilating during Lamaze
High altitude (acute)	Hypoxic peripheral drive ↑, hypocapnic alkalosis inhibits central	Periodic Cheyne-Stokes; then renal HCO ₃ ⁻ 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 → 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



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.