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.

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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 ↑; 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.

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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

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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 H_2O	–2 → 0	500 mL in
Quiet expiration	Passive recoil	–4 cm H_2O → –2	+2 → 0	500 mL out
Forced inspiration	+ SCM, scalenes, serratus	P_pl may reach –30 cm H_2O	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} H_2O.

- ↓ 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 ∝ $1/r^4$.
Bronchoconstriction (asthma) ↑ Raw—pre-treat with β_2-agonist before exercise.

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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

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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 %**.

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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

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1 Learning Objectives

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

1. **Explain the factors that govern diffusion of O_2 and CO_2** 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 oxygen-titration during ambulation.

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2 Alveolar–Capillary Gas Exchange

Factor (Fick's law)	Typical Value	Influence on PT
Surface area (A)	≈ 70 m²	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 CO_2: 6 mm Hg	Altitude ↓ PAO_2; supplemental O_2 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.

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3 Oxygen Transport

Form	% of Total	Details
Bound to hemoglobin	**98.5 %**	1 Hb binds 4 O_2 (1.34 mL $O_2 \cdot g^{-1}$). SaO_2 reflects occupancy.
Dissolved in plasma	1.5 %	0.003 mL $\cdot$ $dL^{-1} \cdot$ mm Hg^{-1} (basis of PaO_2). Clinical for hyperbaric O_2.

3.1 Oxyhemoglobin Dissociation Curve

Sigmoidal shape—co-operativity.

- P50 (PaO_2 at 50 % saturation) ≈ 26–27 mm Hg.

Shifts RIGHT (↓ affinity, ↑ unloading)
• ↑ CO_2 / ↓ 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 O_2 to tissues more readily—monitor saturation but encourage early ambulation.

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4 Carbon-Dioxide Transport

Form	% of Total CO_2	Reaction
Bicarbonate (HCO_3^-)	**≈ 70 %**	$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons HCO_3^- + H^+$ (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 CO_2/H^+ more easily; venous blood carries more CO_2. During pulmonary O_2 loading, CO_2 is released → drives expiration.

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5 Matching Delivery to Demand

$\text{Oxygen Delivery } (DO_2) = CO \times (1.34 \times [Hb] \times SaO_2 + 0.003 \times PaO2)$

Exercise: ↑ CO + small ↓ mixed-venous saturation → muscles extract more O_2.
Anemia: Lower [Hb]—heart compensates with higher CO; monitor HR & fatigue.

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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.

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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 CO_2 transport?**
4. **During heavy exercise PaO_2 remains steady yet SaO_2 may drop slightly—why?**
5. **Explain the Haldane effect in one sentence.**

Answers:

1. Roughly **1.5 %**.
2. Alkalosis, hypothermia (also fetal Hb, low CO_2).
3. It catalyses CO_2 hydration to bicarbonate inside RBCs, vastly accelerating the reaction and allowing large CO_2 carriage.
4. Mixed-venous saturation falls, temperature & acidity rise—curve shifts right, causing slightly lower SaO_2 despite constant PaO_2.
5. **Oxygen unloading at tissues increases Hb affinity for CO_2/H^+, whereas oxygen binding in lungs promotes CO_2 release.**

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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 O_2/CO_2 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.

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2 Neural Control of Breathing

Level	Nuclei / Pathways	Core Function	Key Points for PT
Medullary rhythm generator	• **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	• **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)	↓ **PaO_2** (< 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_2max 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 hyper-inflation
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 $PaCO_2$ 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 CO_2 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 CO_2; CO_2 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 $PaCO_2$ (Haldane effect + hypoventilation).
5. Hyperventilation with renal HCO_3^- 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.
- **CO_2 is the main chemical driver**; O_2 takes command only when dangerously low.
- Respiratory disorders often reflect **maladapted regulation**—physiotherapists can modulate drive via positioning, pacing, airway-clearance and education.