PIPADOR REVISION

MRCOG Part 1 Revision Notes

Respiratory Physiology

PHYSIOLOGY • LECTURE 130

VISUAL EXAMINATION REVISION NOTES

2026 Edition

130

MRCOG PART 1 REVISION NOTES | PHYSIOLOGY

Respiratory Physiology

Mechanics, lung volumes, ventilation, gas exchange, O_2 and CO_2 transport, acid–base and control of breathing

≈ 6 L	≈ 2.4 L	500 mL	≈ 6 L/min
Total lung capacity	FRC — the O_2 reservoir (ERV + RV)	Tidal volume (dead space ≈ 150 mL)	Minute ventilation (VT × RR 12)

■ **KEY FACTS: Learning objectives**

- Describe airway structure, the mechanics of breathing, compliance, surfactant and airway resistance.
- Define lung volumes and capacities and interpret spirometry.
- Apply the alveolar ventilation and alveolar gas equations; explain diffusion and V/Q matching.
- Describe O_2 and CO_2 transport, acid–base balance and the control of breathing.

130.1 Functional anatomy

Zone	Structures	Key facts
Conducting zone (generations 0–16)	Trachea → bronchi → bronchioles → terminal bronchioles	No gas exchange = anatomical dead space ≈ 150 mL (≈ 2 mL/kg); warms, humidifies, filters (mucociliary escalator)
Respiratory zone (generations 17–23)	Respiratory bronchioles → alveolar ducts → alveoli	≈ 300–500 million alveoli; ≈ 70 m^2; blood–gas barrier ≈ 0.3 µm

Type I	Type II	Macrophages
Pneumocytes: ≈ 95% of surface — gas exchange	Pneumocytes: SURFACTANT; progenitors for type I	Clear particles and microbes

130.2 Mechanics of breathing

Diaphragm contracts (phrenic C3–5) + external intercostals ▶ Thoracic volume ↑ ▶ **Intrapleural pressure** −5 → −8 cmH_2O ▶ Alveolar pressure < atmospheric → **air in** ▶ **Expiration passive** (elastic recoil) at rest

Concept	Key points
Compliance	$\Delta V/\Delta P$ ≈ 200 mL/cmH_2O (lung + chest wall ≈ 100). ↓ in fibrosis, pulmonary oedema, RDS, obesity, late pregnancy chest wall; ↑ in emphysema and with age
Surfactant	Type II pneumocytes; DPPC (dipalmitoyl-phosphatidylcholine) + SP-A to D. ↓ surface tension → ↑ compliance, stabilises small alveoli (Laplace P = 2T/r), keeps alveoli dry
Airway resistance	Greatest in medium-sized bronchi (not the small airways — huge parallel cross-section). Parasympathetic (M_3) constricts; β_2 dilates; ↓ as lung volume ↑
Work of breathing	Elastic + resistive; minimised by optimal RR (≈ 12–16/min)

130.3 Lung volumes and spirometry

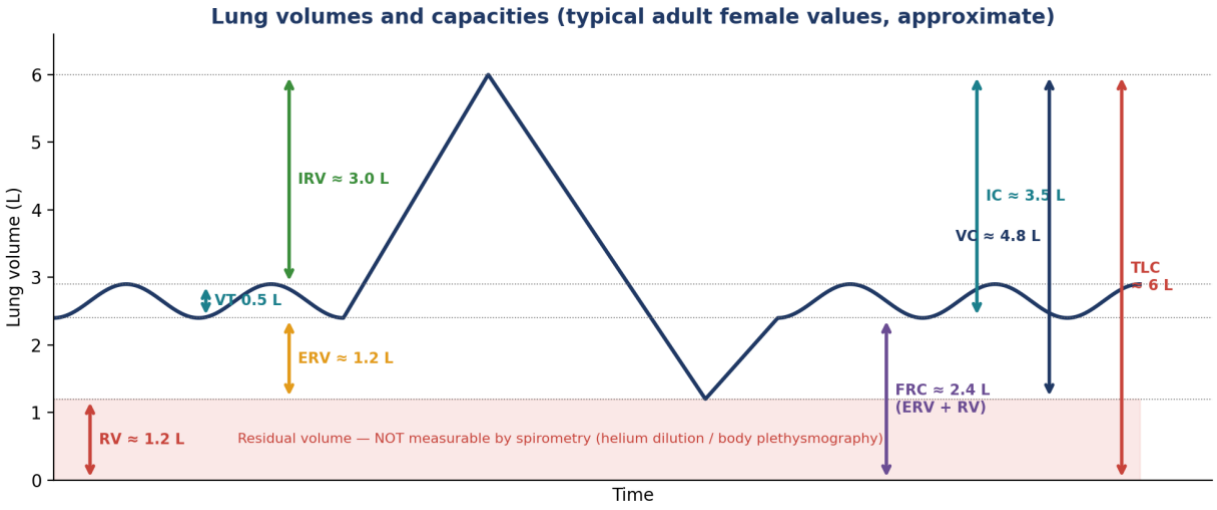


Figure 130.1 — Lung volumes and capacities

FRC	VC	TLC	FEV_1/FVC
= ERV + RV O_2 store during apnoea	= IRV + VT + ERV	= VC + RV	Normal ≥ 0.7 (< 0.7 = obstruction)

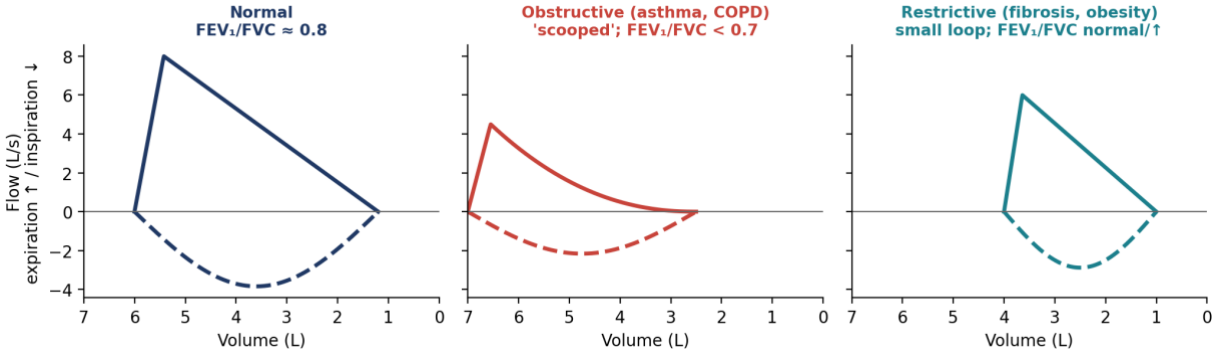


Figure 130.2 — Flow–volume loops: normal, obstructive and restrictive patterns

130.4 Ventilation, gas exchange and V/Q

■ KEY EQUATIONS

Minute ventilation VE = VT × RR; **Alveolar ventilation** VA = (VT − VD) × RR ≈ (500 − 150) × 12 ≈ 4.2 L/min

$PaCO_2 \propto VCO_2 \div VA$ — $PaCO_2$ is the index of alveolar ventilation (halve VA → $PaCO_2$ doubles)

Bohr (physiological dead space): $VD/VT = (PaCO_2 - PECO_2) \div PaCO_2 \approx 0.3$

Alveolar gas equation: $PAO_2 = FiO_2 (PB - PH_2O) - PaCO_2/R = 0.21 \times (101 - 6.3) - 5.3/0.8 \approx$ **13.3 kPa**; **A–a gradient** normally < 2 kPa

Fick's law of diffusion: $\dot{V}gas \propto (Area \times D \times \Delta P) \div thickness$. Blood takes ≈ 0.75 s to cross a capillary; O_2 equilibrates in ≈ 0.25 s (large reserve). Measure with **DLCO**

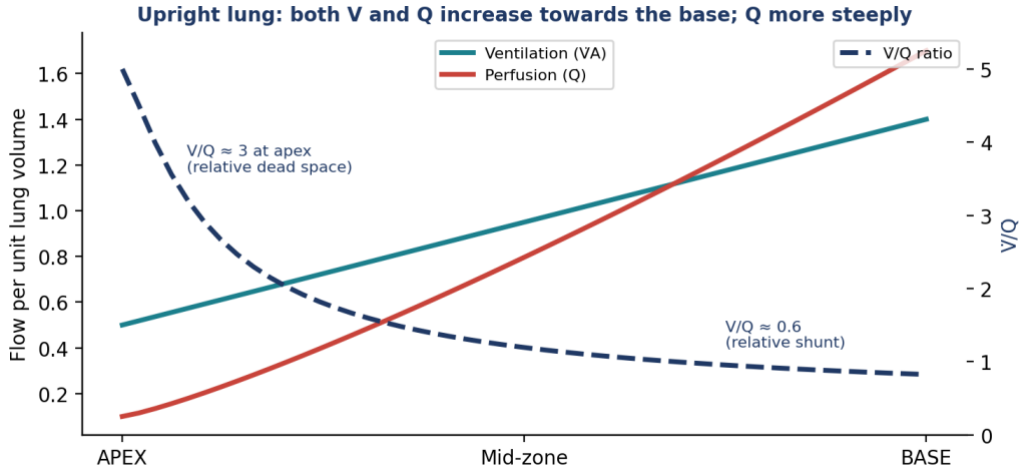


Figure 130.3 — Regional ventilation, perfusion and V/Q in the upright lung (overall V/Q ≈ 0.8)

Cause of hypoxaemia	A–a gradient	Responds to O_2?	Example
Low inspired PO_2	Normal	Yes	Altitude
Hypoventilation	Normal ($PaCO_2$ ↑)	Yes	Opioids, $MgSO_4$ toxicity, neuromuscular disease
Diffusion impairment	↑	Yes	Interstitial lung disease
V/Q mismatch (commonest)	↑	Yes	PE, pneumonia, asthma
Shunt (V/Q = 0)	↑	Poorly	Atelectasis, ARDS, cyanotic heart disease

Hypoxic pulmonary vasoconstriction diverts blood away from poorly ventilated alveoli — the opposite of the systemic response, and the reason fetal PVR is high.

130.5 Oxygen transport

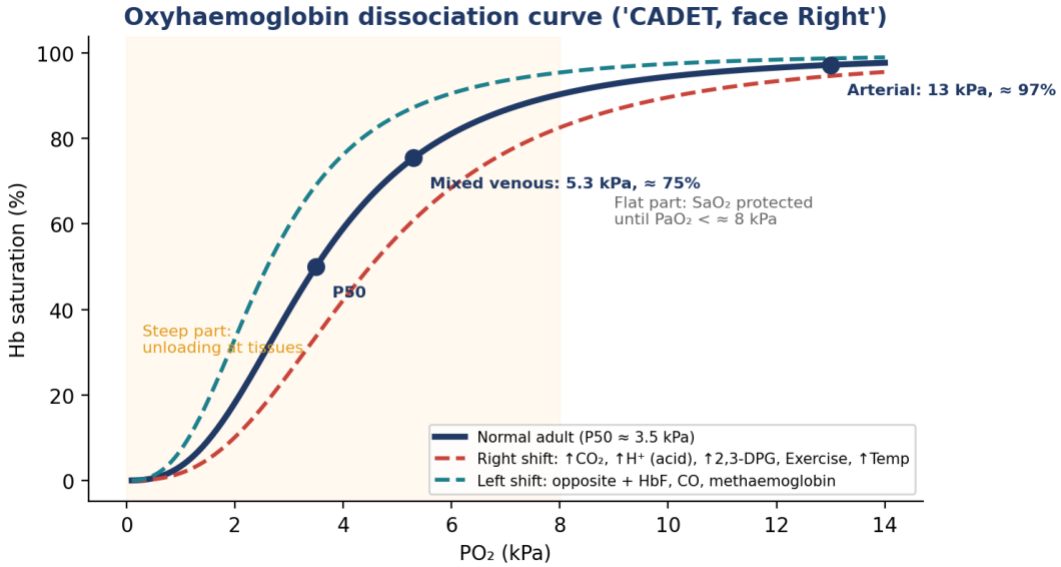


Figure 130.4 — Oxyhaemoglobin dissociation curve and its shifts

■ KEY EQUATIONS

CaO_2 = (1.34 × Hb × SaO_2) + (0.023 × PaO_2 kPa) ≈ 20 mL/dL; **DO_2 = CO × CaO_2** ≈ 1000 mL/min; VO_2 ≈ 250 mL/min

Bohr effect: ↑ CO_2 / H^+ lowers Hb O_2 affinity (right shift) → O_2 released in tissues. **Carbon monoxide**: affinity ≈ 240 × O_2; ↓ carrying capacity AND left-shifts the curve; SpO_2 falsely normal

130.6 Carbon dioxide transport and acid–base

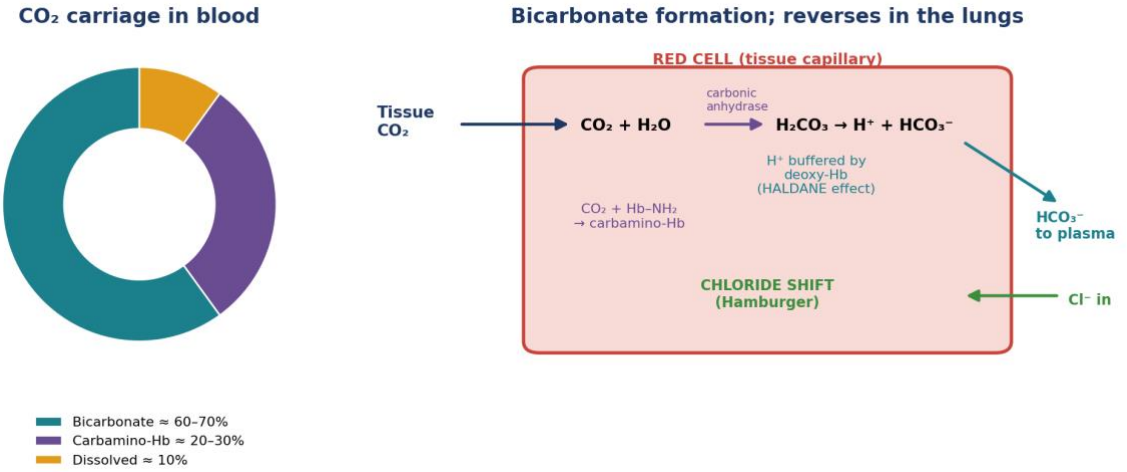


Figure 130.5 — CO_2 carriage, carbonic anhydrase and the chloride shift

> **■ DEFINITION**
>
> **Haldane effect**: deoxygenated Hb carries more CO_2 (better H^+ buffer, more carbamino) → CO_2 loading in tissues, unloading in lungs. **Henderson–Hasselbalch**: pH = 6.1 + log [HCO_3^-] ÷ (0.03 × PCO_2 mmHg).

Disorder	pH	$PaCO_2$	HCO_3^-	Obstetric examples
Respiratory acidosis	↓	↑	↑ (compensation)	Severe asthma, opioid/$MgSO_4$ respiratory depression
Respiratory alkalosis	↑	↓	↓ (compensation)	Normal pregnancy, pain/anxiety in labour, PE
Metabolic acidosis	↓	↓ (compensation)	↓	Sepsis, haemorrhage (lactate), DKA, eclampsia
Metabolic alkalosis	↑	↑ (compensation)	↑	Hyperemesis gravidarum (vomiting)

Normal non-pregnant ABG: pH 7.35–7.45; PaO_2 10.5–13.5 kPa; $PaCO_2$ 4.7–6.0 kPa; HCO_3^- 22–26 mmol/L; base excess ± 2.

130.7 Control of breathing

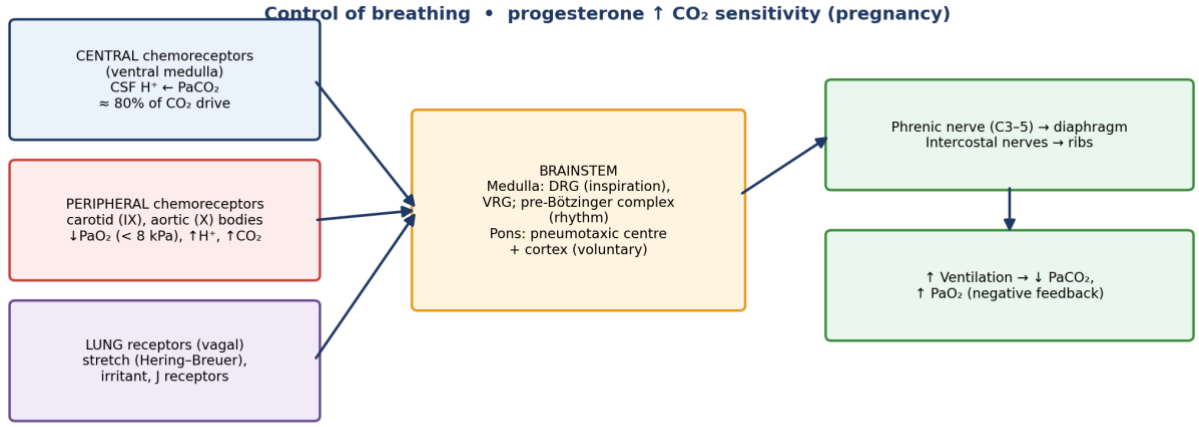


Figure 130.6 — Neural and chemical control of ventilation

Central	Peripheral	Hering–Breuer	Progesterone
Main drive: $PaCO_2$ via CSF H^+ (≈ 80%)	Carotid bodies: hypoxic drive (PaO_2 < 8 kPa)	Stretch → inhibits inspiration (vagal)	↑ CO_2 sensitivity → hyperventilation

130.8 Non-respiratory functions of the lung

ACE (pulmonary endothelium) converts angiotensin I → II and inactivates **bradykinin**; the lung metabolises **prostaglandin E_2** (relevant to ductus arteriosus closure), noradrenaline and serotonin; filters emboli; blood reservoir; heat and water loss; immune defence.

130.9 Must-know summary

■ **EXAM TIP**

- Anatomical dead space ≈ 150 mL; FRC = ERV + RV and cannot be measured by spirometry.
- $PaCO_2$ reflects **alveolar ventilation**; A–a gradient separates hypoventilation (normal) from lung disease (↑).
- V/Q mismatch is the commonest cause of hypoxaemia; **shunt does not correct with O_2**.
- **CO_2 is carried mainly as bicarbonate**; carbonic anhydrase in red cells; chloride shift.
- Central chemoreceptors respond to CO_2 (via H^+), not to hypoxia; peripheral (carotid) bodies sense hypoxia.

■ **MNEMONIC / MEMORY AID**

Right shift of the ODC — 'CADET, face Right': CO_2, Acid, 2,3-DPG, Exercise, Temperature. **Hypoxaemia causes — 'Low, Hypo, Diff, V/Q, Shunt'**.