PIPADOR REVISION

MRCOG Part 1 Revision Notes

Acid–Base Balance

BIOCHEMISTRY • LECTURE 235

VISUAL EXAMINATION REVISION NOTES

2026 Edition

235

MRCOG PART 1 REVISION NOTES | BIOCHEMISTRY

Acid–Base Balance

Buffers and Henderson–Hasselbalch, respiratory and renal regulation, blood gas interpretation, anion gap and causes of disorders

7.35–7.45	4.7–6.0 kPa	22–26	8–16
Arterial pH (H^+ ≈ 40 nmol/L)	$PaCO_2$	HCO_3^- (mmol/L)	Anion gap (mmol/L; without K^+)

■ KEY FACTS: Learning objectives

- Describe pH, buffers and the Henderson–Hasselbalch equation.
- Describe respiratory and renal regulation of acid–base balance.
- Interpret arterial blood gases, compensation and the anion gap.
- Describe causes of acid–base disorders, including obstetric examples.

235.1 Principles

■ KEY EQUATIONS

Henderson–Hasselbalch: pH = 6.1 + log [HCO_3^-] ÷ (0.23 × $PaCO_2$ kPa) — pH depends on the **ratio** of HCO_3^- (kidney) to $PaCO_2$ (lungs).

$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$ (carbonic anhydrase).

Anion gap = $Na^+ - (Cl^- + HCO_3^-)$ ≈ 8–16 mmol/L (unmeasured anions — albumin, phosphate, lactate, ketones); corrected for low albumin (↓ ≈ 2.5 per 10 g/L).

Buffer system	Location	Notes
Bicarbonate / CO_2	ECF	Most important — open system (CO_2 exhaled, HCO_3^- regulated by kidney)
Haemoglobin	Red cells	Histidine imidazole groups; deoxy-Hb better buffer (Haldane)
Proteins (albumin)	Plasma, ICF	
Phosphate	ICF, urine	Titratable acid excretion
Ammonia/ammonium	Urine	Main adaptive renal acid excretion (glutamine)
Bone	—	Chronic acidosis buffering (calcium loss)

Regulator	Speed	Mechanism
Chemical buffers	Seconds	Immediate
Lungs	Minutes–hours	Central/peripheral chemoreceptors adjust ventilation → $PaCO_2$
Kidneys	Hours–days	Reabsorb filtered HCO_3^- (PCT), generate new HCO_3^- via NH_4^+ and titratable acid (distal) — Lecture 151

235.2 Interpreting a blood gas

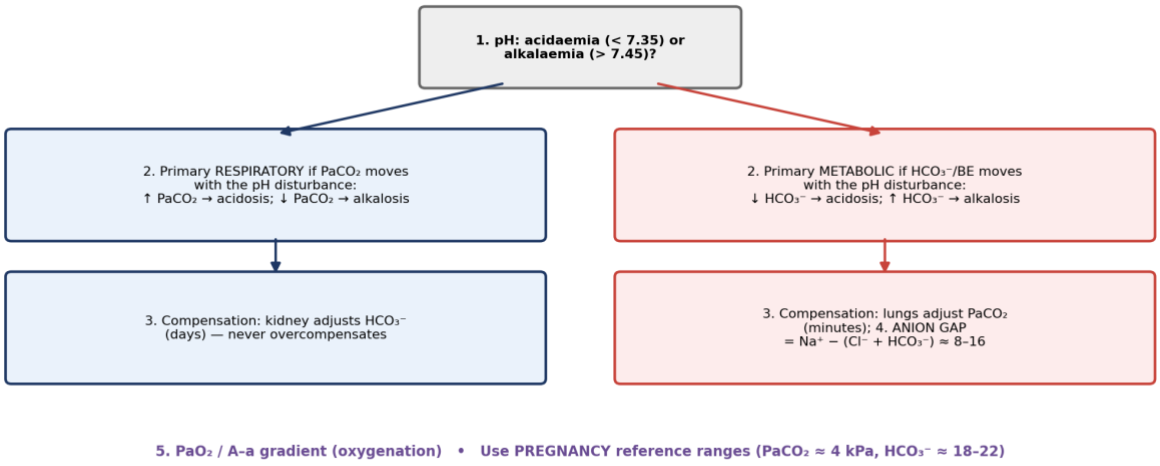


Figure 235.1 — Stepwise ABG interpretation

Disorder	Primary change	Compensation	Causes (O&G examples)
Respiratory acidosis	↑ $PaCO_2$	↑ HCO_3^- (renal)	Hypoventilation: opioids, $MgSO_4$ toxicity, exhaustion in severe asthma, neuromuscular disease, high spinal, obesity
Respiratory alkalosis	↓ $PaCO_2$	↓ HCO_3^-	Normal pregnancy, pain/anxiety in labour, PE, sepsis (early), salicylates, hypoxia
Metabolic acidosis — ↑ anion gap	↓ HCO_3^-	↓ $PaCO_2$ (Kussmaul)	Lactate (sepsis, PPH/shock, AFLP), ketones (DKA, starvation/hyperemesis), renal failure, toxins (methanol, ethylene glycol, salicylate)
Metabolic acidosis — normal anion gap (hyperchloraemic)	↓ HCO_3^-	↓ $PaCO_2$	Large volumes of 0.9% saline, diarrhoea, renal tubular acidosis, acetazolamide, ureteric diversion
Metabolic alkalosis	↑ HCO_3^-	↑ $PaCO_2$ (limited)	Vomiting (hyperemesis) — H^+ and Cl^- loss, volume contraction; diuretics, hyperaldosteronism, antacid excess

■ CLINICAL CORRELATION

- **Mixed disorders**: compensation is never complete/overcompensating — a 'normal' pH with abnormal $PaCO_2$ and HCO_3^- suggests two processes (e.g. salicylate: respiratory alkalosis + metabolic acidosis; hyperemesis with ketosis: metabolic alkalosis + acidosis).
- **Hyperkalaemia** accompanies acidosis (H^+/K^+ shift); **alkalosis ↓ ionised Ca^{2+}** (tetany).

235.3 Must-know summary

■ EXAM TIP

- pH set by HCO_3^- : $PaCO_2$ ratio; bicarbonate is the key buffer; lungs act in minutes, kidneys in days.
- Step 1 pH → step 2 which parameter explains it → step 3 compensation → step 4 anion gap → step 5 oxygenation.
- High-AG acidosis: lactate, ketones, renal failure, toxins; normal-AG: saline, diarrhoea, RTA.
- Vomiting → **hypochloraemic metabolic alkalosis**; opioids/$MgSO_4$ → respiratory acidosis.

■ MNEMONIC / MEMORY AID

High anion gap — 'MUDPILES': Methanol, Uraemia, DKA (ketones), Paracetamol/Propylene glycol, Iron/Isoniazid, Lactate, Ethylene glycol, Salicylates.