

# Mastering blood gas interpretation: A practical guide for primary care providers



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Accurate arterial blood gas (ABG) interpretation is essential for primary care providers (PCPs), especially in emergency and inpatient settings where timely, informed decisions can significantly impact patient outcomes. This review guides PCPs from basic to advanced interpretation through a systematic five-step approach for ABG analysis, focussing on oxygenation, pH status, and metabolic and respiratory disorders. Emphasising the recognition of complex acid-base disorders that may coexist even when pH appears normal, it incorporates tools such as delta and osmolar gap calculations to address multiple concurrent metabolic disturbances and clarify the interpretation of mixed acid-base conditions. The article also briefly considers the use of arterial and venous blood samples in clinical practice.

**Keywords:** blood gas interpretation; primary care providers; simple acid-base disorders; mixed acid-base disorders; five-step approach; anion gap; delta gap.

## Introduction

Arterial blood gas (ABG) analysis is essential for diagnosing and managing respiratory and metabolic conditions.<sup>1</sup> Accurate interpretation directly impacts clinical decisions, but can be challenging because of the interplay of various factors.<sup>2</sup> This article provides step-by-step guidance to enhance primary care providers' (PCPs) competence in ABG analysis, covering fundamental principles, common pitfalls and strategies for accurate interpretation.

Three primary methods exist for acid-base evaluation: the traditional (Boston), base excess (Copenhagen) and physicochemical (Stewart) methods.<sup>3</sup> These methods provide a framework for ABG interpretation, guiding the diagnosis and management of acid-base disorders. All three methods generally yield correct clinical interpretation when used properly.<sup>2,3</sup> Accurate interpretation is crucial to distinguish between simple acid-base disorders – where a primary disturbance and its compensation are present – and mixed disorders, where multiple primary disturbances occur simultaneously.<sup>2,3,4,5</sup>

Proper specimen collection and handling are vital, as errors like non-arterial samples, air bubbles, incorrect anticoagulant levels and delayed analysis can distort results.<sup>2</sup> When using a liquid heparin syringe, hold it vertically with the needle upward and expel excess heparin and air bubbles.<sup>6</sup> Air bubbles cause gas exchange with the blood, reducing PaCO<sub>2</sub> (partial pressure of carbon dioxide) and increasing PaO<sub>2</sub> (partial pressure of oxygen).<sup>6</sup> Samples should be placed on ice if immediate analysis is not possible to prevent oxygen metabolism by platelets and leukocytes. Analyse room-temperature samples within 15 min and iced samples within 1 h.<sup>6</sup> For detailed instructions on safe and accurate arterial blood sampling, refer to guidelines such as that from the World Health Organization (WHO).<sup>7</sup>

Venous blood samples are a practical alternative when arterial access is challenging (Table 1). While less precise for assessing oxygenation and ventilation, venous blood gas (VBG) analysis offers valuable pH and HCO<sub>3</sub><sup>-</sup> (bicarbonate) data and is less invasive, making it useful in conditions like diabetic ketoacidosis or when respiratory involvement is minimal. However, ABG remains essential for accurately evaluating oxygenation and ventilation, particularly in suspected or confirmed respiratory disorders or hypoxaemia.

## Starting the interpretation

To interpret an ABG report accurately, begin by verifying the internal consistency of the ABG values and obtaining relevant clinical information before applying the five-step approach.

**Note:** Additional supporting information may be found in the online version of this article as Online Appendix 1.

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**TABLE 1:** A comparison of arterial and venous blood gas analyses.

Parameter	ABG analysis	VBG analysis
Purpose	Assess oxygenation status, ventilation, and acid-base balance	Less invasive alternative to ABG; assess acid-base balance and venous CO <sub>2</sub> levels
Sample site	Radial, brachial or femoral artery	Peripheral vein in the arm
Key parameters (Normal values)	<p>pH: 7.35–7.45 (H<sup>+</sup> 35–45 nmol/L)</p> <p>PaO<sub>2</sub>: 80–100 mmHg (10.6–13.3 kPa)</p> <p>PaCO<sub>2</sub>: 35–45 mmHg (4.7–6.0 kPa)</p> <p>HCO<sub>3</sub><sup>-</sup>: 22–26 mmol/L</p> <p>BE: -2 to +2 mmol/L</p>	<p>pH: 7.32–7.43 (H<sup>+</sup> 37–48 nmol/L)</p> <p>PvO<sub>2</sub>: 25–40 mmHg (3.3–5.3 kPa)</p> <p>PvCO<sub>2</sub>: 41–50 mmHg (5.5–6.7 kPa)</p> <p>HCO<sub>3</sub><sup>-</sup>: 23–27 mmol/L</p> <p>BE: Similar to arterial values</p>
Limitations	Invasive and painful procedure. Risk of complications such as bleeding, haematoma and arterial injury	Less accurate for assessing oxygenation status, may not detect hypoxaemia. Differences in pH, PvO <sub>2</sub> and PvCO <sub>2</sub> require careful interpretation

ABG, arterial blood gas; VBG, venous blood gas; pH, potential of hydrogen; PaO<sub>2</sub>, partial pressure of oxygen in arterial blood; PaCO<sub>2</sub>, partial pressure of carbon dioxide in arterial blood; HCO<sub>3</sub><sup>-</sup>, bicarbonate; PvO<sub>2</sub>, partial pressure of oxygen in venous blood; PvCO<sub>2</sub>, partial pressure of carbon dioxide in venous blood; BE, base excess.

## Verify the consistency of the arterial blood gas report

Checking the internal consistency of ABG reports is essential to ensure that the measured parameters align with known physiological relationships.<sup>8</sup> This practice helps identify discrepancies that may arise from preanalytical and analytical errors. Internal consistency differs from calibration: calibration ensures that the machine provides accurate measurements, while internal consistency confirms that the reported values are logical and cohesive based on physiological norms. Common issues affecting internal consistency include improper sample collection, delays in processing and contamination. The *Henderson-Hasselbalch equation* verifies ABG consistency by relating pH, HCO<sub>3</sub><sup>-</sup> and PaCO<sub>2</sub>:

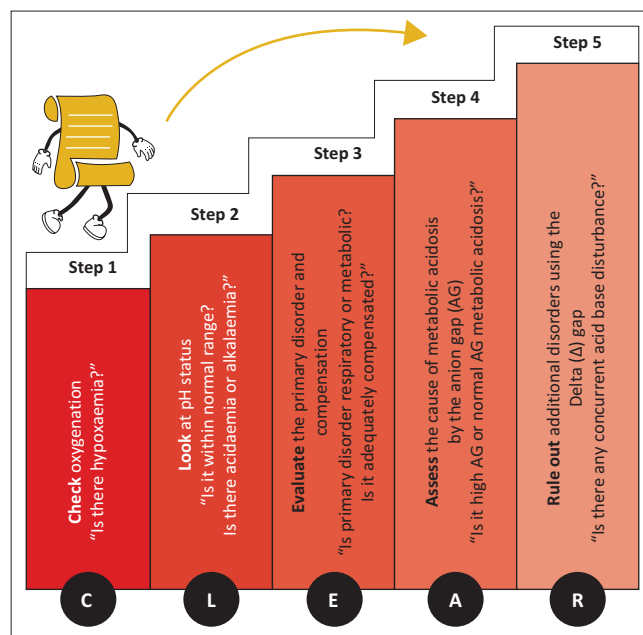
$$\text{pH} = 6.1 + \log (\text{HCO}_3^- / 0.03 \times \text{PaCO}_2) \quad [\text{Eqn } 1]$$

6.1 is the acid dissociation constant (pK<sub>a</sub>) for carbonic acid at body temperature, and 0.03 is the solubility coefficient of CO<sub>2</sub> in the blood (mmol/L per mmHg).

The equation requires a calculator with a logarithm function, available on most mobile devices. See Box 1 for a practical example. Alternatively, embedding the equation in a spreadsheet allows for automatic calculation of pH upon entering the values of HCO<sub>3</sub><sup>-</sup> and PaCO<sub>2</sub>. If the calculated pH differs from the reported pH by more than ± 0.05, suspect an error in one or more parameters and repeat the test, as such discrepancies are common.<sup>9</sup>

## Obtain clinical information

Gather focussed clinical history and examination findings to contextualise ABG results.<sup>2,9</sup> Determining whether an ABG abnormality is acute or chronic informs the urgency and type of treatment. For example, chronic respiratory acidosis may not require immediate intervention compared to acute cases. Serial blood gas measurements can monitor the patient's response to treatment and disease progression.

**FIGURE 1:** The CLEAR path.

## A five-step approach to ABG interpretation: The CLEAR path

The 'CLEAR' mnemonic provides a structured five-step framework for ABG interpretation (Figure 1) with a flow diagram (Figure 2) offering a visual summary.

### Step 1: Check oxygenation

- Check partial pressure of oxygen (PaO<sub>2</sub>)

*Normal PaO<sub>2</sub> (80 mmHg – 100 mmHg):* Proceed to step 2.

*Decreased PaO<sub>2</sub> (< 80 mmHg):* Suggests hypoxaemia. It should be interpreted in the context of fraction of inspired oxygen (FiO<sub>2</sub>). Estimate the expected PaO<sub>2</sub> by multiplying FiO<sub>2</sub> by 5, and use this to classify hypoxaemia as mild, moderate or severe:<sup>2</sup>

- Calculate expected PaO<sub>2</sub>:

$$\text{Expected PaO}_2 = \text{FiO}_2 \% \times 5 \quad [\text{Eqn } 2]$$

Age adjusted PaO<sub>2</sub> on room air = 100 mmHg – (0.3 × age in years).

- Classify hypoxaemia:

PaO<sub>2</sub> 60 mmHg – 79 mmHg: Mild hypoxaemia

PaO<sub>2</sub> 40 mmHg – 59 mmHg: Moderate hypoxaemia

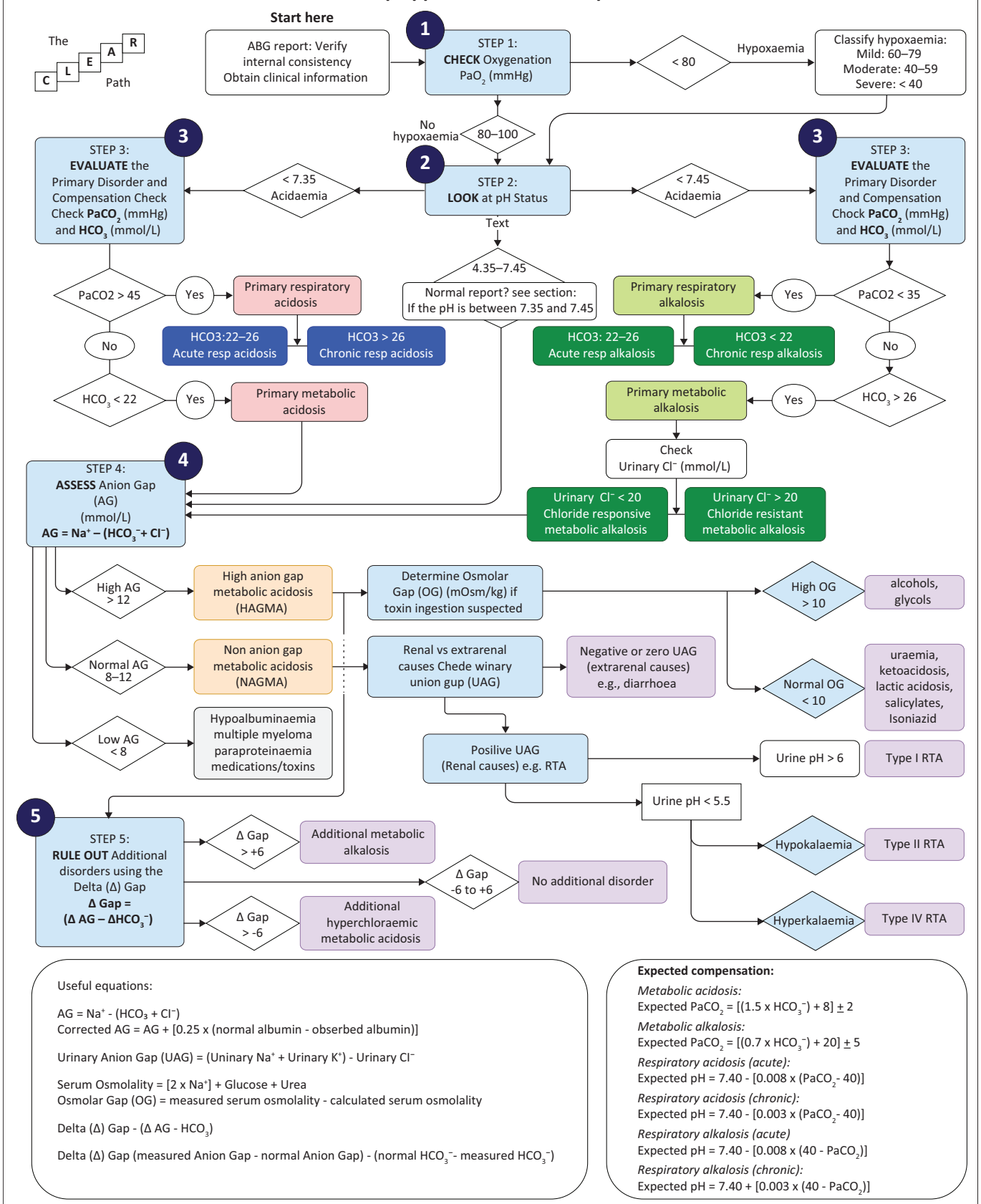
PaO<sub>2</sub> < 40 mmHg: Severe hypoxaemia

### Step 2: Look at pH status

- If the pH is between 7.35 and 7.45

Check if other parameters are within normal range (Table 1). A normal pH, PaCO<sub>2</sub> and HCO<sub>3</sub><sup>-</sup> does not exclude acid-base disorders.<sup>10</sup> If anion gap (AG) is increased, a patient can still have metabolic acidosis and alkalosis. When all the

## A five-step approach to ABG interpretation



Note: A printable PDF copy is available as Online Appendix 1.

ABG, arterial blood gas; resp, respiratory; UAG, urinary anion gap; OG, osmolar gap; RTA, renal tubular acidosis.

**FIGURE 2:** A five-step approach to arterial blood gas interpretation.

parameters, including AG, are within normal limits, the patient has a normal acid-base balance.

A normal pH with abnormal  $\text{PaCO}_2$  and  $\text{HCO}_3^-$  indicates a mixed acid-base disorder, with acidosis and alkalosis balancing each other.<sup>5</sup> Calculating the AG is essential in ABG interpretation to uncover hidden acid-base disorders.<sup>11</sup>

- **Acidaemia or alkalaemia?**
  - pH < 7.35: Acidaemia, suggesting either respiratory acidosis, metabolic acidosis or a combination. Proceed to Step - For acidaemia (pH < 7.35).
  - pH > 7.45: Alkalaemia, suggesting either respiratory alkalosis, metabolic alkalosis or a combination. Proceed to Step - For alkalaemia (pH > 7.45).

### Step 3: Evaluate the Primary Disorder and Compensation

- For acidaemia (pH < 7.35):

Check  $\text{PaCO}_2$  – *Elevated  $\text{PaCO}_2$  (> 45 mmHg):* Suggests *Primary respiratory acidosis*. For example, if an ABG report shows a pH of 7.30,  $\text{PaCO}_2$  of 50 mmHg, and  $\text{HCO}_3^-$  of 24 mmol/L, this indicates acidaemia. The elevated  $\text{PaCO}_2$  suggests that the primary cause is respiratory acidosis:

- **Assess  $\text{HCO}_3^-$  levels:**
  - Normal  $\text{HCO}_3^-$  (22 mmol/L – 26 mmol/L): Likely *acute respiratory acidosis* (no time for renal compensation).
  - Elevated  $\text{HCO}_3^-$  (> 26 mmol/L): Indicates *chronic respiratory acidosis* (renal compensation has occurred).
- **Calculate expected renal compensation:**
  - Refer to Table 2 for expected compensation values and determine if there is an additional metabolic disorder.

Check  $\text{HCO}_3^-$  – *Decreased  $\text{HCO}_3^-$  (< 22 mmol/L):* Suggests *Primary metabolic acidosis*:

- **Calculate expected respiratory compensation:**
  - Expected  $\text{PaCO}_2 = [(1.5 \times \text{HCO}_3^-) + 8] \pm 2$  (Table 2). For example, if measured  $\text{HCO}_3^-$  is 12 mmol/L, then expected  $\text{PaCO}_2$  will be  $[(1.5 \times 12) + 8] \pm 2 = 26 \pm 2$  mmHg. If the patient's actual  $\text{PaCO}_2$  is within 24 mmHg – 28 mmHg, compensation is appropriate. Values outside this range suggest a mixed disorder.
- **Proceed to Step 4.**
- **For alkalaemia (pH > 7.45):**

Check  $\text{PaCO}_2$  – *Decreased  $\text{PaCO}_2$  (< 35 mmHg):* Suggests *Primary respiratory alkalosis*:

- **Assess  $\text{HCO}_3^-$  levels:**
  - Normal  $\text{HCO}_3^-$  (22 mmol/L – 26 mmol/L): Likely *acute respiratory alkalosis*.
  - Decreased  $\text{HCO}_3^-$  (< 22 mmol/L): indicates *chronic respiratory alkalosis* (renal compensation).
- **Calculate expected renal compensation:**
  - Refer to Table 2 for expected compensation values and determine if there is an additional metabolic disorder.

Check  $\text{HCO}_3^-$  – *Elevated  $\text{HCO}_3^-$  (> 26 mmol/L):* Suggests *Primary metabolic alkalosis*:

- **Calculate expected respiratory compensation:**
  - Expected  $\text{PaCO}_2 = [(0.7 \times \text{HCO}_3^-) + 20] \pm 5$  (Table 2). For example, if measured  $\text{HCO}_3^-$  is 12 mmol/L, then expected  $\text{PaCO}_2$  will be  $[(0.7 \times 12) + 20] \pm 5 = 28.4 \pm 5$  mmHg. If the patient's actual  $\text{PaCO}_2$  is within 23.4 mmHg – 33.4 mmHg, compensation is appropriate. Values outside this range suggest a mixed disorder.

**TABLE 2:** Primary acid-base disorders and expected compensation.

Primary acid-base disorders	pH	$\text{HCO}_3^-$	$\text{PaCO}_2$	Expected compensation
<b>Metabolic</b>				
Acidosis	↓	↓		Expected $\text{PaCO}_2 = [(1.5 \times \text{HCO}_3^-) + 8] \pm 2$ Less precise: Expected $\text{PaCO}_2$ = last two digits of pH If $\text{PaCO}_2 >$ expected: Concomitant respiratory acidosis If $\text{PaCO}_2 <$ expected: Concomitant respiratory alkalosis
Alkalosis	↑	↑		Expected $\text{PaCO}_2 = [(0.7 \times \text{HCO}_3^-) + 20] \pm 5$ If $\text{PaCO}_2 >$ expected: Concomitant respiratory acidosis If $\text{PaCO}_2 <$ expected: Concomitant respiratory alkalosis
<b>Respiratory</b>				
<b>Acidosis</b>				
Acute†	↓		↑	Expected pH = $7.40 - [0.008 \times (\text{PaCO}_2 - 40)]$ $\text{HCO}_3^-$ will increase by 1 mmol/L for each 10 mmHg rise in $\text{PaCO}_2$ above 40 mmHg
Chronic‡	↓		↑	Expected pH = $7.40 - [0.003 \times (\text{PaCO}_2 - 40)]$ $\text{HCO}_3^-$ will increase by 4 mmol/L for each 10 mmHg rise in $\text{PaCO}_2$ above 40 mmHg
<b>Alkalosis</b>				
Acute†	↑		↓	Expected pH = $7.40 + [0.008 \times (40 - \text{PaCO}_2)]$ $\text{HCO}_3^-$ will decrease by 2 mmol/L for each 10 mmHg decrease in $\text{PaCO}_2$ below 40 mmHg
Chronic‡	↑		↓	Expected pH = $7.40 + [0.003 \times (40 - \text{PaCO}_2)]$ $\text{HCO}_3^-$ will decrease by 5 mmol/L for each 10 mmHg decrease in $\text{PaCO}_2$ below 40 mmHg

pH, potential of hydrogen;  $\text{PaCO}_2$ , partial pressure of carbon dioxide in arterial blood;  $\text{HCO}_3^-$ , serum bicarbonate concentration; mmol/L, millimoles per litre.

↑, increased; ↓, decreased.

†, Acute < 3–5 days; ‡, Chronic > 3–5 days.

- *Classify metabolic alkalosis – Metabolic alkalosis is divided into two groups based on urinary chloride:*

- *Chloride responsive:* (extravascular volume depletion because of vomiting, chronic diarrhoea, diuretic, post-hypercapnic).

Urinary chloride < 20 mmol/L. These patients respond to administered sodium chloride (NaCl) infusion.

- *Chloride resistant:* Urinary chloride > 20 mmol/L; typically seen in euvoletic or fluid overload states. These patients do not respond to NaCl infusion and need potassium chloride (KCl) to correct hypokalaemia. The condition often results from excessive mineralocorticoids, causing sodium retention and potassium excretion.

#### Step 4: Assess the cause of metabolic acidosis by the anion gap (AG)

- Calculate the anion gap.

$$AG = Na^+ - (HCO_3^- + Cl^-)$$

Normal AG range: 8 mmol/L – 12 mmol/L

- Adjust for Hypoalbuminaemia:

To correct the AG for hypoalbuminaemia, add 0.25 mmol/L to the AG for each 1 g/L drop in albumin below 40 g/L.<sup>10</sup>

$$\text{Corrected AG} = AG + 0.25 (\text{normal albumin} - \text{observed albumin})$$

- Interpret the anion gap.

#### BOX 1: Clinical case study – Application of the five-step approach to arterial blood gas interpretation.

##### Clinical scenario:

A 55-year-old man is brought to the emergency department with altered mental status, nausea, and vomiting. He has a history of alcoholism and was found near empty antifreeze (ethylene glycol) containers.

##### Vital signs:

Pulse: 110 bpm, BP: 90/60 mmHg, RR: 28 breaths/min, SpO<sub>2</sub>: 94% on room air

##### Exam:

Lethargic, Kussmaul respirations, dehydrated

##### Laboratory results:

- -ABG: pH: 7.12, PaCO<sub>2</sub>: 22 mmHg, PaO<sub>2</sub>: 85 mmHg, HCO<sub>3</sub><sup>-</sup>: 7 mmol/L
- Electrolytes: Na<sup>+</sup>: 140 mmol/L, Cl<sup>-</sup>: 100 mmol/L, Serum Osmolality: 360 mOsm/kg, Serum Albumin: 40 g/L

##### Starting the interpretation

Verify ABG report's internal consistency

- Method 1: Henderson-Hasselbalch Equation:  $pH = 6.1 + \log (HCO_3^- / 0.03 \times PaCO_2) = 6.1 + \log (7 / 0.03 \times 22) = 6.1 + \log (7 / 0.66) = 7.125$
- Calculated pH matches reported pH; ABG values are consistent.

Obtain clinical information

- History suggests ethylene glycol ingestion.
- Symptoms indicate metabolic acidosis.

##### Five-step approach

##### Step 1: Oxygenation status

- PaO<sub>2</sub>: 85 mmHg on room air (within normal range)
- Expected PaO<sub>2</sub>:  $21\% \times 5 \approx 100$  mmHg.
- Age adjusted PaO<sub>2</sub> =  $100 \text{ mmHg} - (0.3 \times \text{Age in years}) = 83.5$  mmHg
- Interpretation: No significant hypoxaemia.

##### Step 2: pH status

- pH 7.12: Indicates acidaemia.

##### Step 3: Determine primary disorder

- Low HCO<sub>3</sub><sup>-</sup> (7 mmol/L): Primary metabolic acidosis.
- Low PaCO<sub>2</sub> (22 mmHg): Respiratory compensation.
- Calculate Expected PaCO<sub>2</sub> (Winter's Formula):  $\text{Expected PaCO}_2 = (1.5 \times HCO_3^-) + 8 \pm 2 = (1.5 \times 7) + 8 \pm 2 = 18.5 \pm 2$  mmHg
- Expected range: 16.5–20.5 mmHg

Measured PaCO<sub>2</sub> (22 mmHg) is slightly above expected range; suggests inadequate respiratory compensation or concurrent respiratory acidosis.

##### Step 4: Calculate Anion Gap (AG)

- AG:  $(140 - [100 + 7]) = 33$  mmol/L

Elevated AG indicates high anion gap metabolic acidosis (HAGMA).

- Calculate Osmolar Gap (OG):

$$\text{Calculated Osmolality} = (2 \times 140) + 6 + 5 = 291 \text{ mOsm/kg}$$

$$\text{OG} = \text{measured serum osmolality} - \text{calculate serum osmolality} = 360 - 291 = 69 \text{ mOsm/kg}$$

Elevated OG suggests ethylene glycol poisoning.

##### Step 5: Assess for additional disorders (Δ Gap)

- $\Delta AG = 33 - 12 = 21$  mmol/L
- $\Delta HCO_3^- = 24 - 7 = 17$  mmol/L
- $\Delta \text{Gap} = \Delta AG - \Delta HCO_3^- = 21 - 17 = +4$  mmol/L

Delta gap is within normal range (-6 to +6); no additional acid-base disorder is revealed.

##### Conclusion

- Primary disorder: High anion gap metabolic acidosis because of ethylene glycol poisoning.
- Compensation: Inadequate respiratory compensation; possible concurrent respiratory acidosis.

ABG, arterial blood gas; pH, potential of hydrogen; HCO<sub>3</sub><sup>-</sup>, bicarbonate.



High anion gap ( $> 12$  mmol/L) This indicates *High Anion Gap Metabolic Acidosis* (HAGMA):

- AG is increased because of retention of unmeasured anion from the titrated strong acid. Bicarbonate is reduced through buffering of added strong acid.
- If AG is  $\geq 20$  mmol/L, then a *metabolic acidosis* is present regardless of the pH or serum  $\text{HCO}_3^-$  values.
- Determine *Osmolar Gap* (OG), if toxin ingestion is suspected. Osmolar Gap helps identify the presence of unmeasured osmotically active substances, for example alcohols, glycols, in the blood.

$\text{Osmolar Gap} = \text{measured serum osmolality} - \text{calculate serum osmolality}$

$\text{Serum Osmolality} = 2 \times \text{Na} + \text{Glucose} + \text{Urea}$

High OG ( $> 10$  mOsm/kg): methanol, ethylene glycol

Normal OG ( $< 10$  mOsm/kg): Uraemia, ketoacidosis, lactic acidosis, salicylates, isoniazid

- Proceed to Step 5.

Normal anion gap (8 mmol/L – 12 mmol/L) This suggests *Non-Anion Gap Metabolic Acidosis* (NAGMA) (Hyperchloremic metabolic acidosis):

- Mainly because of losses of bicarbonate (commonly from gastrointestinal tract), and/or increased chloride ingestion, and/or infusion of substances that release hydrochloric acid (such as NaCl).
- No anion gap is present because of the absence of unmeasured anion from titrated acid and secondary chloride retention with  $\text{HCO}_3^-$  loss. Kidneys fail to reabsorb or regenerate  $\text{HCO}_3^-$ .
- Differentiate renal from extrarenal causes in NAGMA – The urinary anion gap is used to differentiate renal from extrarenal causes.

$\text{Urinary anion gap (UAG)} = (\text{Urinary Na}^+ + \text{Urinary K}^+) - \text{Urinary Cl}^-$

A negative or zero UAG indicates an extrarenal cause, such as diarrhoea.

A positive UAG suggests a renal cause, such as renal tubular acidosis (RTA).

- A positive UAG with urine pH of  $> 6$  is suggestive of Type I RTA
- A positive UAG with urine pH of  $< 5.5$  with hypokalaemia is suggestive of Type II RTA; and with hyperkalaemia indicates Type IV RTA.

Low AG ( $< 8$  mmol/L)<sup>12</sup>:

- Check for hypoalbuminaemia.
- Assess for multiple myeloma and paraproteinaemia: Consider serum protein electrophoresis.
- Evaluate electrolytes: Look for hypercalcaemia or hypomagnesaemia.
- Review medications and toxins: Consider lithium toxicity; exposure to bromide or iodide.

#### Step 5: Rule out Additional Disorders using the Delta ( $\Delta$ ) Gap

- The  $\Delta$  Gap is a calculation used to uncover additional acid-base disorders in the context of HAGMA.<sup>11,13</sup> It

compares the change ( $\Delta$ ) in the AG to the change ( $\Delta$ ) in  $\text{HCO}_3^-$  levels.

$$\Delta \text{ Gap} = (\Delta \text{ AG} - \Delta \text{ HCO}_3^-)$$

$$\Delta \text{ Gap} = (\text{Measured Anion Gap} - \text{Normal Anion Gap}) - (\text{Normal HCO}_3^- - \text{Measured HCO}_3^-)$$

$$\Delta \text{ Gap} = (\text{AG} - 12) - (24 - \text{HCO}_3^-)$$

For every 1 mmol/L rise in AG,  $\text{HCO}_3^-$  should drop by 1 mmol/L in a simple acid-base disorder.

$\Delta$  Gap:  $-6$  to  $+6$  (normal) No additional disorder.

$\Delta$  Gap  $> +6$  suggests an additional metabolic alkalosis, since the rise in AG is more than the fall in  $\text{HCO}_3^-$ .

$\Delta$  Gap  $< -6$  suggests an additional hyperchloremic metabolic acidosis, because the rise in AG is less than the fall in  $\text{HCO}_3^-$ .

- Up to three disorders can coexist, and  $\Delta$  Gap calculations can help identify them. While two metabolic abnormalities can coexist, only one respiratory disorder can occur at a time, as a patient cannot simultaneously have both hypoventilation and hyperventilation.<sup>14</sup>

See Box 1 for a practical example of this five-step approach in practice.

## Recommendations

- Obtain ABG and/or VBG measurements when clinically indicated, interpret them confidently and adhere to a structured, stepwise approach.
- The printable PDF flowchart (supplementary file) with this article provides a quick-reference tool in clinical settings.
- Use apps and online calculators (e.g. MDCalc) for quick ABG analysis. Investigate emerging AI-driven tools while validating their outputs against clinical judgement.
- Reflect on challenging cases to identify learning opportunities and share with colleagues to promote collective learning.

## Conclusion

Blood gas interpretation is crucial for PCPs, enabling timely, impactful decisions. Mastering ABG fundamentals and using a systematic approach help clinicians confidently manage acid-base disorders. Practical knowledge and awareness of common pitfalls enhance diagnostic skills, empowering PCPs to deliver high-quality care. Ongoing learning, collaboration and reflective practice keep PCPs at the forefront of evolving healthcare.

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T.H. conceptualised the idea and wrote the first draft. A.N., S.M., H.S. and N.I. contributed to the article's critical evaluation and approved the final draft.

### Ethical considerations

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### Data availability

The authors confirm that the data supporting this article are available within the article and its references.

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