

# Algorithm For Acid Base Disorders

*Before You Begin: Gather Data*

What lab tests do you need?

- ABG vs. VBG
- VBG can provide reliable estimation for pH (0.02-0.04 lower than ABG) and HCO<sub>3</sub> (1-2 mEq higher than ABG).
- pCO<sub>2</sub> on VBG is higher than arterial PaO<sub>2</sub> by 4-9mmHg.
- ABG is necessary to accurately assess PaCO<sub>2</sub> in hypercapnia or severe shock.
- BMP.
- Albumin.

Step 1: Determine Primary Acid-Base Disturbance.

- Remember that chronic acid-base disturbance(s) and/or mixed acute acid-base disturbances can be obscured by a normal pH.
- Consider the following **“normal”** values:
  - Arterial pH: 7.40
  - Arterial pCO<sub>2</sub>: 40
  - Bicarb (HCO<sub>3</sub>): 24
  - Anion gap: 12
- **Acidemia**
  - pH < 7.38
  - Primary disturbance
    - Respiratory acidosis = pCO<sub>2</sub> > 42
    - Metabolic acidosis = HCO<sub>3</sub> < 22
- **Alkalemia**
  - pH > 7.42
  - Primary disturbance
    - Respiratory alkalosis = pCO<sub>2</sub> < 38
    - Metabolic alkalosis = HCO<sub>3</sub> > 26

Step 2a: Calculate and Interpret the Anion Gap

- Anion gap (AG) =  $[Na] - [Cl] - [HCO_3]$ .
- Normal AG = 8-12 mEq/L with a serum albumin of 4g/dl.
- The normal AG depends on serum albumin. The expected "normal" range for AG can be calculated by  $2.5 \times \text{albumin (g/dl)} \pm 2$ .
- For severe hypoalbuminemia (2.0 g/dl), the expected gap would be  $5 \pm 2$ , and an AG of 8-12 would be abnormally elevated.
- Paraproteinemia (especially polyclonal or monoclonal IgG) can lower anion gap due to the abundance of cations added to the system; this should be accounted for when evaluating anion gap since it could obscure a truly elevated AG.
- For calculation,  $>12$  is generally used as the cutoff for a high AG. Because 12 is the upper limit of normal, keep in mind that approach might increase specificity for elevated AG at the cost of sensitivity. Because of person-to-person variation in "normal" AG, it can be useful to review a patient's previous values of AG to establish a baseline and give context to small, acute changes in AG.
- If the AG is elevated, there is an anion gap metabolic acidosis present. Proceed to step 2b.
- If the AG not elevated, there is not an anion gap metabolic acidosis present. Proceed to step 3.

### Step 2b: Calculate and Interpret the Excess Anion Gap

- If there is an anion gap, determine whether the excess in AG fully explains the drop in bicarbonate.
- For every 1 unit increase in AG ( $\Delta AG \uparrow$ ) from its upper limit of normal value (12), there is an expected 1 drop in  $HCO_3$  ( $\Delta HCO_3 \downarrow$ ) from its "normal" value (24).
- If the AG and  $HCO_3$  are balanced, ( $\Delta HCO_3 \pm 5 \text{ mmol/L} \approx \Delta AG$ ), there is no concurrent metabolic process.
- If the  $HCO_3$  is lower than expected ( $\Delta HCO_3 \pm 5 \text{ mmol/L} > \Delta AG$ ), there is a concurrent non-elevated anion gap metabolic acidosis as evidenced by a reduction of  $HCO_3$ .
- If the  $HCO_3$  is higher than expected ( $\Delta HCO_3 \pm 5 \text{ mmol/L} < \Delta AG$ ), there is a concurrent metabolic alkalosis as evidenced by an excess of  $HCO_3$ .
- The 5mmol/L correction accounts for the normal range of bicarbonate.
- Alternate approach to answer this question: "correcting" the bicarb.
- Corrected  $HCO_3 = HCO_3 + (AG - 12)$ .
- If the sum is greater than the usual bicarbonate range ( $> 28$ ), there is a concomitant metabolic alkalosis. If the sum is less than the usual bicarbonate range ( $< 22$ ), there is a concomitant non-elevated anion gap metabolic acidosis.
- Finally, there is a third approach to this same problem, "the delta-delta," which is not discussed here.

### Step 3: Evaluate for Compensation

Evaluate for physiologic compensation for the acid-base disorder.

Any values above or below expected suggest an additional acid-base disturbance; a compensation should not normalize or overcorrect the pH.

The following table gives a quick rule-of-thumb for evaluating compensation. More detailed formulas are listed below.

	Primary disturbance	Compensatory change
Metabolic acidosis	1 ↓ HCO <sub>3</sub>	1 ↓ pCO <sub>2</sub>
Metabolic alkalosis	10 ↑ HCO <sub>3</sub>	7 ↑ pCO <sub>2</sub>
Acute respiratory acidosis	10 ↑ pCO <sub>2</sub>	1 ↑ HCO <sub>3</sub>
Chronic respiratory acidosis	10 ↑ pCO <sub>2</sub>	4 ↑ HCO <sub>3</sub>
Acute respiratory alkalosis	10 ↓ pCO <sub>2</sub>	2 ↓ HCO <sub>3</sub>
Chronic respiratory alkalosis	10 ↓ pCO <sub>2</sub>	4 ↓ HCO <sub>3</sub>

### Common Questions in Metabolic Acidosis

- What is the pathophysiology of saline-induced normal anion gap (hyperchloremic) metabolic acidosis?

The normal anion gap metabolic acidosis resulting from large volume administration of normal saline (NS) can be explained as a dilutional effect on the existing bicarbonate level. This is because NS contains a supraphysiologic concentration of chloride (154 mEq/L) and no bicarbonate; thus it will raise serum chloride while diluting other anions - namely lowering bicarbonate - inducing a metabolic acidosis. This may be avoided by using a balanced intravenous solution such as Lactated Ringer's or Plasmalyte. These solutions have a lower [Cl<sup>-</sup>] concentration compared to NS and, most importantly, contain lactate and acetate respectively, which are rapidly metabolized to an equimolar amount of bicarbonate.

- What are the disadvantages of giving bicarbonate in the setting of acute metabolic acidosis?
  - Can cause an initial transient worsening of intracellular acidosis.
  - Can lead to generation of increased CO<sub>2</sub> which causes respiratory acidosis (especially in patients with respiratory failure such as in ARDS).
  - Can represent a large sodium load that can exacerbate hypervolemia.
  - Can worsen hypokalemia.
- The 2008 Surviving Sepsis guidelines recommend against its use in sepsis if pH > 7.15. One can consider temporary NaHCO<sub>3</sub> administration in the setting of severe metabolic acidosis (pH < 7.1) or to facilitate permissive hypercarbia. In most cases of normal anion gap metabolic acidosis, it is probably safe. Consider renal replacement therapy in cases of severe acidosis refractory to medical therapy.

### Key Points

- It is important to follow a systematic approach each time interpreting a blood gas. Establish the primary disorder. Calculate AG to reveal an anion gap metabolic acidosis (can be hidden when pH is normal but mixed disorders are present). Compare  $\Delta$ AG and  $\Delta$ HCO<sub>3</sub> to look for concurrent metabolic alkalosis or normal anion gap metabolic acidosis. Use a compensation chart/formula to reveal “overcompensation” or “undercompensation” which indicates the presence of another disorder.
- The presence of a normal pH with abnormal pCO<sub>2</sub> and bicarbonate suggests a mixed acid-base disorder with counterbalancing acidosis and alkalosis.
- The pCO<sub>2</sub> and serum bicarbonate typically move in parallel with an isolated acid-base disorder; both are high OR both are low. For example, a respiratory acidosis will have an increased pCO<sub>2</sub> with a compensatory increase in serum bicarbonate. If the pCO<sub>2</sub> and serum bicarbonate move in opposite directions (one high and one low), then you should consider the possibility of two simultaneous primary acid-base disorders (i.e., a mixed acid-base disorder).
- A mixed acid-based disorder consists of any combination of at least two disorders: two metabolic disturbances OR one respiratory and one metabolic. Triple acid-base disorders include one respiratory disorder (acidosis or alkalosis) with two metabolic disorders (high gap and normal gap metabolic acidosis OR high gap metabolic acidosis and metabolic alkalosis).

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