Clinical Education

Interpretation of ABGs

Interpretation of Arterial Blood Gases (ABGs)

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Introduction:

Interpreting an arterial blood gas (ABG) is a crucial skill for physicians, nurses, respiratory therapists, and other health care personnel. ABG interpretation is especially important in critically ill patients.

The following six-step process helps ensure a complete interpretation of every ABG. In addition, you will find tables that list commonly encountered acid-base disorders.

Many methods exist to guide the interpretation of the ABG. This discussion does not include some methods, such as analysis of base excess or Stewart's strong ion difference. A summary of these techniques can be found in some of the suggested articles. It is unclear whether these alternate methods offer clinically important advantages over the presented approach, which is based on the "anion gap."

6-step approach:

Step 1: Assess the internal consistency of the values using the Henderseon-Hasselbach equation:

$$[H+] = \frac{24(PaCO_2)}{[HCO_3-]}$$

If the pH and the [H+] are inconsistent, the ABG is probably not valid.

pH	Approximate [H+] (mmol/L)
7.00	100
7.05	89
7.10	79
7.15	71
7.20	63
7.25	56
7.30	50
7.35	45
7.40	40
7.45	35
7.50	32
7.55	28
7.60	25
7.65	22

Step 2: Is there alkalemia or acidemia present?

pH < 7.35 acidemia
pH > 7.45 alkalemia

- This is usually the primary disorder
- *Remember:* an acidosis or alkalosis may be present even if the pH is in the normal range (7.35 – 7.45)
- You will need to check the $PaCO_2$, HCO_3- and anion gap

Step 3: Is the disturbance respiratory or metabolic? What is the relationship between the direction of change in the pH and the direction of change in the $PaCO_2$? In primary respiratory disorders, the pH and PaCO2 change in *opposite* directions; in metabolic disorders the pH and $PaCO_2$ change in the same direction.

Acidosis	Respiratory	pH ↓	$PaCO_2$ ↑
Acidosis	Metabolic&	pH ↓	$PaCO_2$ ↓
Alkalosis	Respiratory	pH ↑	$PaCO_2$ ↓
Alkalosis	Metabolic	pH ↑	$PaCO_2$ ↑

Step 4: Is there appropriate compensation for the primary disturbance? Usually, compensation does not return the pH to normal (7.35 – 7.45).

Disorder	**Expected compensation**	**Correction factor**
Metabolic acidosis	$PaCO_2 = (1.5 \times [HCO_3\text{-}]) + 8$	± 2
Acute respiratory acidosis	Increase in $[HCO_3\text{-}] = \Delta PaCO_2/10$	± 3
Chronic respiratory acidosis (3-5 days)	Increase in $[HCO_3\text{-}] = 3.5(\Delta PaCO_2/10)$	
Metabolic alkalosis	Increase in $PaCO_2 = 40 + 0.6 (\Delta HCO_3\text{-})$	
Acute respiratory alkalosis	Decrease in $[HCO_3\text{-}] = 2(\Delta PaCO_2/10)$	

Chronic respiratory alkalosis	Decrease in $[HCO_3^-] = 5(\Delta PaCO_2/10)$ to $7(\Delta PaCO_2/10)$

If the observed compensation is not the expected compensation, it is likely that more than one acid-base disorder is present.

Step 5: Calculate the anion gap (if a metabolic acidosis exists): $AG = [Na^+]-([Cl^-] + [HCO_3^-])-12 \pm 2$

- A normal anion gap is approximately 12 meq/L.
- In patients with hypoalbuminemia, the normal anion gap is lower than 12 meq/L; the "normal" anion gap in patients with hypoalbuminemia is about 2.5 meq/L lower for each 1 gm/dL decrease in the plasma albumin concentration (for example, a patient with a plasma albumin of 2.0 gm/dL would be approximately 7 meq/L.)
- If the anion gap is elevated, consider calculating the osmolal gap in compatible clinical situations.
    - Elevation in AG is not explained by an obvious case (DKA, lactic acidosis, renal failure
    - Toxic ingestion is suspected
- OSM gap = measured OSM – (2[Na+] - glucose/18 – BUN/2.8
    - The OSM gap should be < 10

Step 6: If an increased anion gap is present, assess the relationship between the increase in the anion gap and the decrease in $[HCO_3^-]$.

Assess the ratio of the change in the anion gap (ΔAG) to the change in [HCO3-] ($\Delta[HCO_3^-]$): $\Delta AG/\Delta[HCO_3^-]$

This ratio should be between 1.0 and 2.0 if an uncomplicated anion gap metabolic acidosis is present.

If this ratio falls outside of this range, then another metabolic disorder is present:

- If $\Delta AG/\Delta[HCO_3^-] < 1.0$, then a concurrent non-anion gap metabolic acidosis is likely to be present.
- If $\Delta AG/\Delta[HCO_3^-] > 2.0$, then a concurrent metabolic alkalosis is likely to be present.

It is important to remember what the expected "normal" anion gap for your patient should

be, by adjusting for hypoalbuminemia (see **Step 5**, above.)

Table 1: Characteristics of acid-base disturbances

Disorder	pH	Primary problem	Compensation
Metabolic acidosis	↓	↓ in HCO_3-	↓ in $PaCO_2$
Metabolic alkalosis	↑	↑ in HCO_3-	↑ in $PaCO_2$
Respiratory acidosis	↓	↑ in $PaCO_2$	↑ in [HCO_3-]
Respiratory alkalosis	↑	↓ in $PaCO_2$	↓ in [HCO_3-]

Table 2: Selected etiologies of respiratory acidosis

- Airway obstruction
  - Upper
  - Lower
    - COPD
    - asthma
    - other obstructive lung disease
- CNS depression
- Sleep disordered breathing (OSA or OHS)
- Neuromuscular impairment
- Ventilatory restriction
- Increased CO2 production: shivering, rigors, seizures, malignant hyperthermia, hypermetabolism, increased intake of carbohydrates
- Incorrect mechanical ventilation settings

Table 3: Selected etiologies of respiratory alkalosis

- CNS stimulation: fever, pain, fear, anxiety, CVA, cerebral edema, brain trauma, brain tumor, CNS infection

- Hypoxemia or hypoxia: lung disease, profound anemia, low FiO2
- Stimulation of chest receptors: pulmonary edema, pleural effusion, pneumonia, pneumothorax, pulmonary embolus
- Drugs, hormones: salicylates, catecholamines, medroxyprogesterone, progestins
- Pregnancy, liver disease, sepsis, hyperthyroidism
- Incorrect mechanical ventilation settings

Table 4: Selected causes of metabolic alkalosis

- Hypovolemia with Cl- depletion
  - GI loss of H+
    - Vomiting, gastric suction, villous adenoma, diarrhea with chloride-rich fluid
  - Renal loss H+
    - Loop and thiazide diuretics, post-hypercapnia (especially after institution of mechanical ventilation)
- Hypervolemia, Cl- expansion
  - Renal loss of H+: edematous states (heart failure, cirrhosis, nephrotic syndrome), hyperaldosteronism, hypercortisolism, excess ACTH, exogenous steroids, hyperreninemia, severe hypokalemia, renal artery stenosis, bicarbonate administration

Table 5: Selected etiologies of metabolic acidosis

- Elevated anion gap:
  - Methanol intoxication
  - Uremia
  - Diabetic ketoacidosis[a], alcoholic ketoacidosis, starvation ketoacidosis
  - Paraldehyde toxicity
  - Isoniazid
  - Lactic acidosis[a]
    - Type A: tissue ischemia
    - Type B: Altered cellular metabolism
  - Ethanol[b] or ethylene glycol[b] intoxication

- Salicylate intoxication

[a] Most common causes of metabolic acidosis with an elevated anion gap

[b] Frequently associated with an osmolal gap

- Normal anion gap: will have increase in [Cl-]
  - GI loss of HCO_3-
    - Diarrhea, ileostomy, proximal colostomy, ureteral diversion
  - Renal loss of HCO_3-
    - proximal RTA
    - carbonic anhydrase inhibitor (acetazolamide)
  - Renal tubular disease
    - ATN
    - Chronic renal disease
    - Distal RTA
    - Aldosterone inhibitors or absence
    - NaCl infusion, TPN, NH_4+ administration

Table 6: Selected mixed and complex acid-base disturbances

Disorder	Characteristics	Selected situations
Respiratory acidosis with metabolic acidosis	↓in pH ↓ in HCO_3 ↑ in $PaCO_2$	• Cardiac arrest • Intoxications • Multi-organ failure
Respiratory alkalosis with metabolic alkalosis	↑in pH ↑ in HCO_3- ↓ in $PaCO_2$	• Cirrhosis with diuretics • Pregnancy with vomiting • Over ventilation of COPD

Respiratory acidosis with metabolic alkalosis	pH in normal range ↑ in $PaCO_2$, ↑ in HCO_3-	• COPD with diuretics, vomiting, NG suction • Severe hypokalemia
Respiratory alkalosis with metabolic acidosis	pH in normal range ↓ in $PaCO_2$ ↓ in HCO_3	• Sepsis • Salicylate toxicity • Renal failure with CHF or pneumonia • Advanced liver disease
Metabolic acidosis with metabolic alkalosis	pH in normal range HCO_3- normal	• Uremia or ketoacidosis with vomiting, NG suction, diuretics, etc.

Suggested additional reading:

- Rose, B.D. and T.W. Post. *Clinical physiology of acid-base and electrolyte disorders*, 5th ed. New York: McGraw Hill Medical Publishing Division, c2001.
- Fidkowski, C And J. Helstrom. Diagnosing metabolic acidosis in the critically ill: bridging the anion gap, Stewart and base excess methods. *Can J Anesth* 2009;56:247-256.
- Adrogué, H.J. and N.E. Madias. Management of life-threatening acid-base disorders—first of two parts. *N Engl J Med* 1998;338:26-34.
- Adrogué, H.J. and N.E. Madias. Management of life-threatening acid-base disorders—second of two parts. *N Engl J Med* 1998;338:107-111.