Arterial Blood Gas (ABG) Analysis

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ABG analysis is an indispensable test for critically ill patients or patients treated in an ICU. Interpretation of ABG analysis seems complicated; however, an understanding of the basic physiology can ease the interpretation. In this article, the basic physiology behind ABG analysis and a stepwise approach to interpreting ABG analysis will be discussed in detail.

Overview of ABG Analysis

Analysis of arterial blood gases (ABG) is an essential investigation in the management of critically ill patients as it provides important **information about alveolar ventilation, oxygenation, and acid-base balance**.

Measurement of ABG includes **measurement of three parameters, i.e., pH, $PaCO_2$, and PaO_2**; however, measurement of bicarbonate (**HCO_3^-**) and electrolytes is almost always done simultaneously to complete the whole picture of acid-base & electrolyte balance/imbalance.

Important values

pH

- Normal blood pH: 7.36–7.44
- Acidemia: pH < 7.35
- Alkalemia: pH > 7.45

$PaCO_2$ & $PaC0_2$

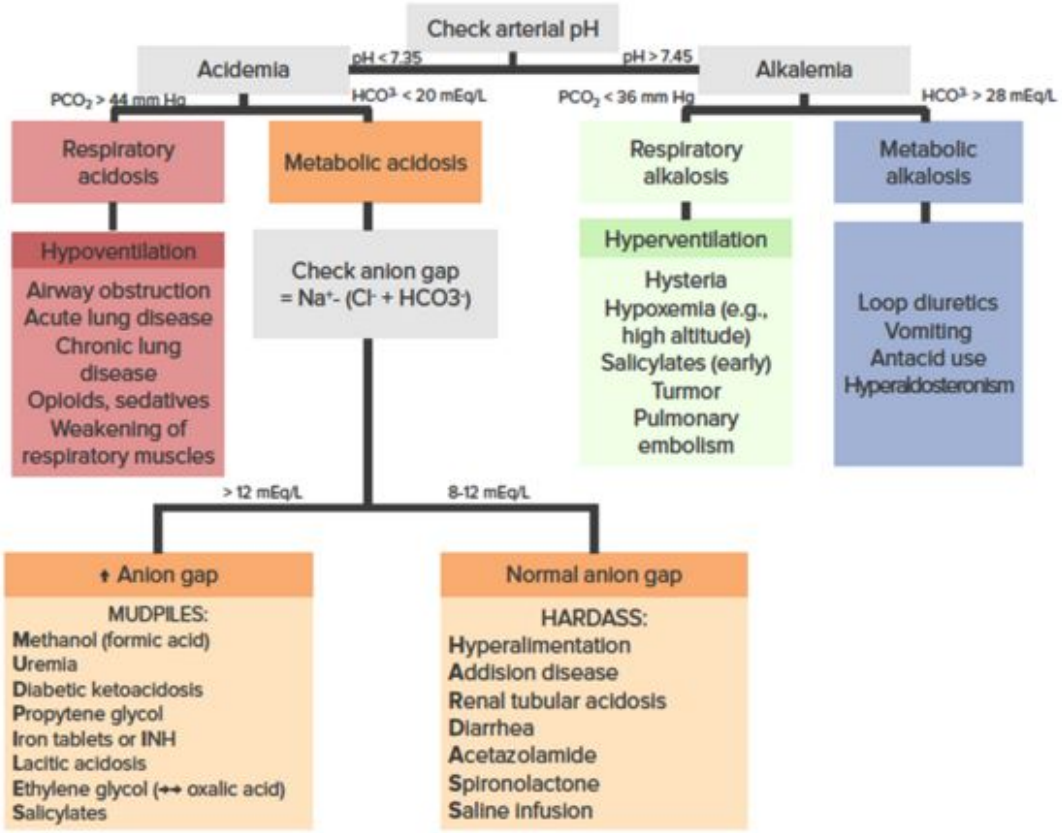
- Normal PaO_2: 80–100 mm Hg
- Normal $PaCO_2$: 36–44 mm Hg
- Respiratory acidosis: Acidemia with $PaCO_2$ > 44 mm Hg
- Respiratory alkalosis: Alkalemia with $PaCO_2$ < 36 mm Hg

HCO_3^-

- Normal HCO_3^-: 20–28 mEq/L
- Metabolic acidosis: Acidemia with HCO_3^- < 20 mEq/L
- Metabolic alkalosis: Alkalemia with HCO_3^- > 28 mEq/L
- Normal base excess/deficit: 0 ± 2 mEq/L

Anion Gap

- Normal Anion Gap (AG): 12 mEq/L
- Anion Gap Metabolic Acidosis (AGMA): Metabolic acidosis with AG > 12
- Non-Anion Gap Metabolic Acidosis (NAGMA): Metabolic acidosis with normal AG



"Acidosis and Alkalosis" by Kapil Pal

Basic Physiological Concepts

$PaCO_2$ is the best parameter that **reflects alveolar ventilation**; high (> 44 mm Hg) and low (< 36 mm Hg) $PaCO_2$ levels suggest alveolar hypoventilation and alveolar hyperventilation respectively.

The **status of oxygenation is affected mainly by PaO_2, FiO_2, and hemoglobin content** along with its affinity and saturation with oxygen. The presence of hypoxemia (PaO_2 < 80 mm Hg at sea level while breathing room air) does not necessarily mean the

presence of hypoxia.

Blood pH is maintained in the narrow range (7.36–7.44) by lungs, kidneys and blood buffers. In general, the blood buffer system (H_2CO_3/HCO_{3^-}) acts within a fraction of seconds, the respiratory system takes about 1–15 minutes, and kidneys may take many minutes to many days to adjust H^+ ions concentration.

$PaCO_2$ is mainly **controlled by lungs** and is a **marker of respiratory acid-base disturbance**. HCO_{3^-} is mainly controlled by kidneys and blood buffers, but it does not always purely indicate metabolic acid-base disturbance as it is affected by hydrolysis effect.

Plasma bicarbonate can be calculated using the **Henderson-Hasselbalch equation**; while standard bicarbonate is plasma bicarbonate obtained after blood has been equilibrated at 37°C with a $PaCO_2$ of 40 mm Hg.

Henderson-Hasselbalch equation: pH = 6.1 + log10 ([HCO3-]/0.03 x PaCO2)

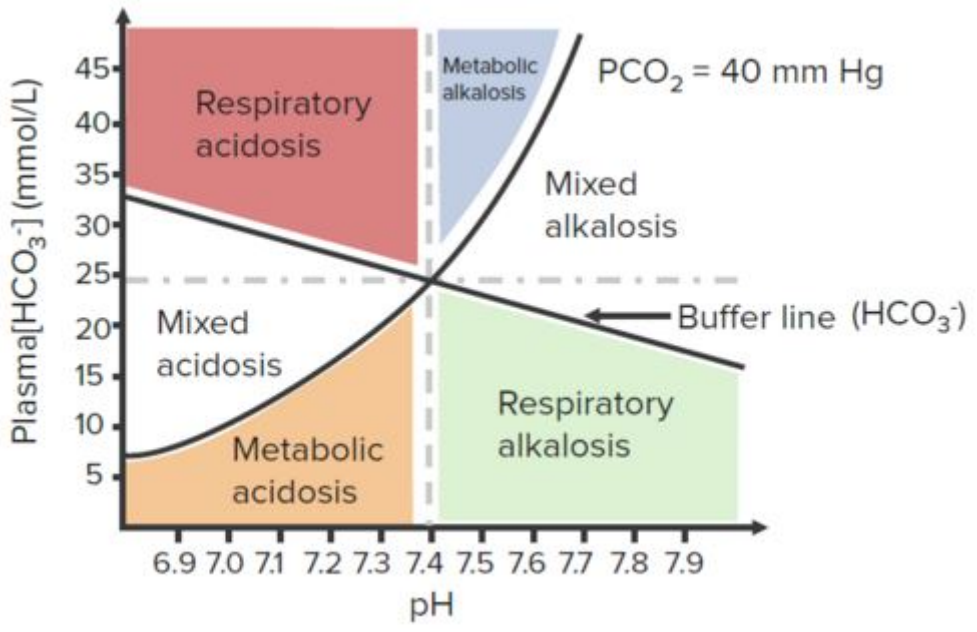
The difference between the measured bicarbonate and the calculated bicarbonate is known as **base excess** (if positive value) or **base deficit** (if negative value).

In acidemia, an excess of H^+ ions enter intracellular space, hence, K^+ ions move out from intracellular space into the plasma to maintain electroneutrality. Thus, acidemia is associated with hyperkalemia.

In distal renal tubular cells, K^+ or H^+ ions are exchanged for Na^+ ions. In alkalemia, as H^+ ions are retained, K^+ ions are exchanged for Na^+ ions, leading to hypokalemia.

Compensation: In response to primary acid-base disturbance, the respiratory system or kidneys attempt to maintain normal pH by the compensatory response. Compensation "moves the pH towards normal", but compensation can never "overshoot" the normal pH.

Respiratory compensation for a primary metabolic derangement is immediate and usually improves over the course of hours, while renal compensation for a primary respiratory derangement is gradual and improves over days as it takes time for the kidneys to generate/eliminate HCO_3.



"Davenport (Acid/Base) Diagram" by Kapil Pal

Anion Gap (AG)

Calculation of anion gap is very important for metabolic acidosis. In electroneutrality:

Unmeasured anions + Cl^- + HCO_3– = Unmeasured cations + Na^+

Unmeasured anions are usually proteins, SO_4, PO_4, organic anions, etc. and unmeasured cations are K^+, Ca^{++}, Mg^{++}, etc. Therefore:

Anion Gap (AG) = Na^+ – (Cl^- + HCO_3–) = Unmeasured anions – Unmeasured cations

An elevated anion gap refers to an increase in unmeasured anions. **Normal AG: 12 mEq/L**

Respiratory Acidosis

Key features: pH < 7.35 with $PaCO_2$ > 44 mm Hg

Causes: Basically, causes of respiratory acidosis are those that cause CO_2 retention or hypoventilation.

- Upper airway disorders such as obstruction, laryngospasm, obstructive sleep apnea
- Lower airway disorders such as COPD, asthma
- Thoracic cage abnormalities restricting respiratory movements such as flail chest, kyphoscoliosis
- Neuromuscular disorders affecting ventilation such as AIDP
- CNS depression due to CNS disorders, opioids, sedatives, etc.
- Respiratory muscle fatigue following any prolonged/severe respiratory disease

Respiratory Alkalosis

Key features: pH > 7.45 with $PaCO_2$ < 36 mm Hg

Causes: Basically, causes of respiratory alkalosis are those that cause CO_2 washout or hyperventilation.

- Hyperventilation due to pain, anxiety, hysteria, CNS tumor/disorder, pulmonary embolism, etc.
- Hypoxemia due to high altitude or any other cause
- Early salicylate poisoning

Metabolic Acidosis

Key features: pH < 7.35 with HCO_3– < 20 mEq/L

Metabolic acidosis is further sub-classified on the basis of the anion gap.

Anion Gap (AG) = Na^+ – (Cl^- + HCO_3-)/p>

Causes of high Anion Gap Metabolic Acidosis (AGMA) (AG > 12 mEq/L):

- **M**ethanol (formic acid) poisoning
- **U**remia
- **D**iabetic ketoacidosis, **D**rugs

- **P**ropylene glycol poisoning
- **I**ron poisoning, **I**soniazid
- **L**actic acidosis
- **E**thylene glycol (→ oxalic acid) poisoning, Ethanol poisoning (Alcoholic ketoacidosis)
- **S**alicylate poisoning

⇒ **Mnemonic: MUDPILES**

OR

- **L**actic acidosis
- **U**remia
- **S**alicylate poisoning
- Iron poisoning, **I**soniazid
- **D**iabetic ketoacidosis, **D**rugs
- **Alcohol** poisoning (methanol, ethanol, ethylene glycol, propylene glycol)

⇒ Mnemonic: **LUSID Alcohol**

Causes of Normal Anion Gap Metabolic Acidosis (NAGMA):

- **H**yperalimentation (TPN)
- **A**ldosterone related (Addison disease, spironolactone, RTA type IV)
- **A**cetazolamide
- **R**enal tubular acidosis
- **D**iarrhea, ureteral **D**iversion (ileal loop), after **D**KA treatment
- **F**luids (after volume resuscitation with low HCO_3– fluids such as normal saline)

⇒ Mnemonic: **HAARD Fluids**

Metabolic Alkalosis

Key features: pH > 7.45 with HCO_3– > 28 mEq/L

Metabolic alkalosis is further sub-classified into **saline-responsive** (generally low urine Cl^-) and **saline-resistant** (generally high urine Cl^-) metabolic alkalosis. Causes of saline-responsive metabolic alkalosis:

- **G**astrointestinal losses (emesis, nasogastric tube aspiration, villous adenoma, etc)
- **D**iuretics
- **P**ost-hypercapnia

⇒ Mnemonic: **GDP**

Causes of saline-resistant metabolic alkalosis:

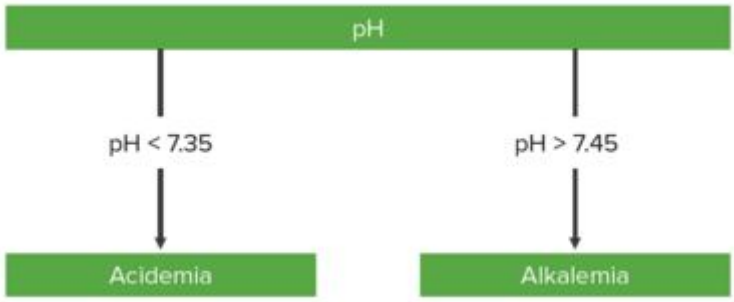
- **Li**corice ingestion
- **Mi**neralocorticoid excess (hyperaldosteronism, Cushing's syndrome, exogenous administration)
- **B**artter's syndrome
- Exogenous **A**lkali load
- **G**itelman's syndrome

⇒ Mnemonic: **LiMi BAG**

Stepwise Approach to Interpret ABG Analysis

Step 1: Is there acidemia or alkalemia?

- If pH < 7.35 → Acidemia
- If pH > 7.45 → Alkalemia



"Step 1" by Kapil Pal

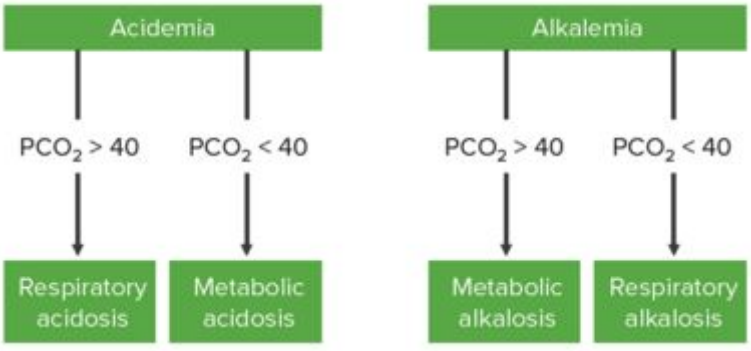
Step 2: Use PCO_2 to determine the primary derangement

In acidemia:

- If PCO_2 > 40 mm Hg → Respiratory acidosis
- If PCO_2 < 40 mm Hg → Metabolic acidosis

In alkalemia:

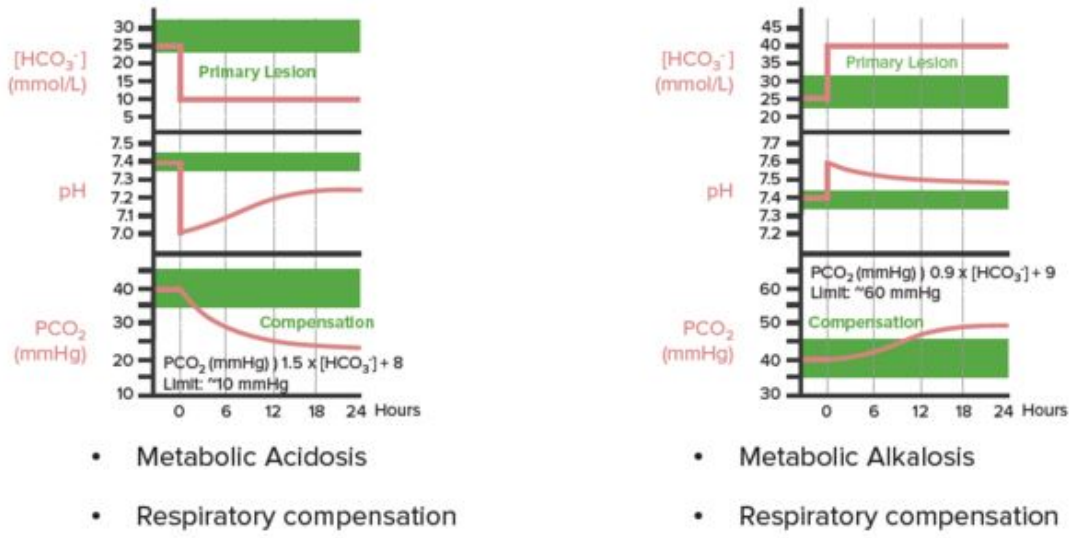
- If PCO_2 > 40 mm Hg → Metabolic alkalosis
- If PCO_2 < 40 mm Hg → Respiratory alkalosis



"Step 2" by Kapil Pal

Step 3: Determine if compensation is adequate

For **primary metabolic derangement**, respiratory compensation is usually immediate and improves over the course of hours. For **primary respiratory derangement**, renal compensation is usually gradual and improves over days. It also depends on whether the condition is acute or chronic.



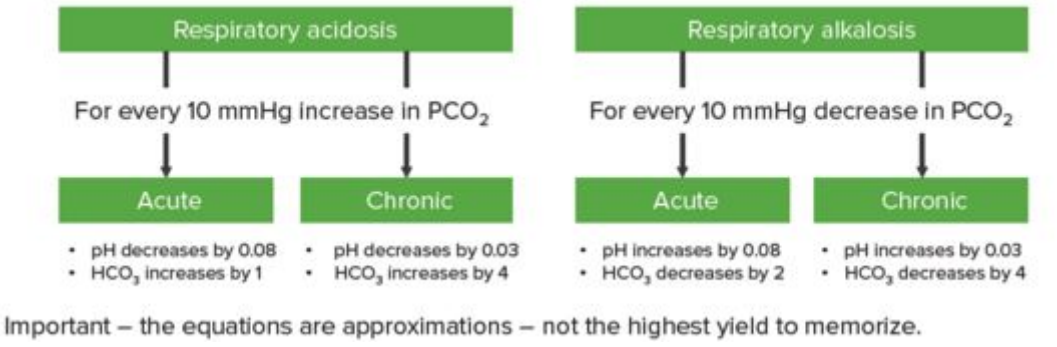
"Arterial Blood Gases" by Kapil Pal

In respiratory acidosis, for every 10 mm Hg increase in $PaCO_2$:

- In acute condition: pH decreases by 0.08 and HCO_3– increases by 1
- In chronic condition: pH decreases by 0.03 and HCO_3– increases by 4

In respiratory alkalosis, for every 10 mm Hg decrease in $PaCO_2$:

- In acute condition: pH increases by 0.08 and HCO_3– decreases by 2
- In chronic condition: pH increases by 0.03 and HCO_3– decreases by 4



"Step 3" by Kapil Pal

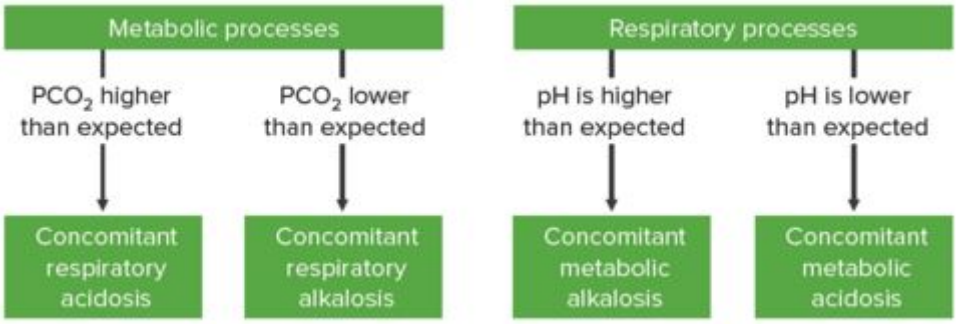
Step 3a: If the expected value for the level of compensation does not correlate with the measured value (pH or HCO_3-), then there is likely a mixed disorder present

In metabolic derangements:

- If PCO2 is higher than expected → Concomitant respiratory acidosis
- If PCO2 is lower than expected → Concomitant respiratory alkalosis

In respiratory derangements:

- If pH is higher than expected → Concomitant metabolic alkalosis
- If pH is lower than expected → Concomitant metabolic acidosis

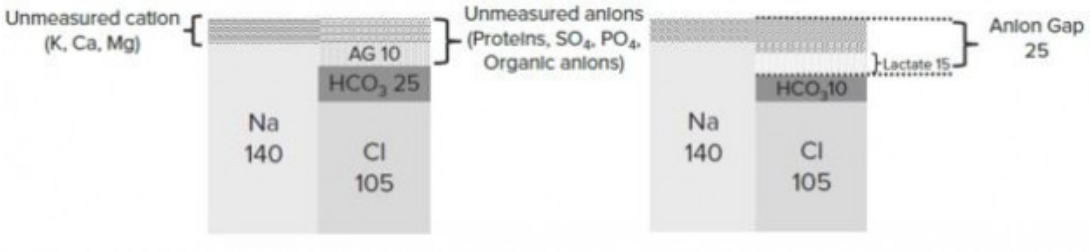


"Step 3a" by Kapil Pal

Step 4: For all metabolic acidosis, calculate the Anion Gap

Anion gap = Na^+ – (Cl- + HCO_3-) (Normal: 12 mEq/L)

AG should be corrected for hypoalbuminemia: For every 1 mg/dL below 4 → subtract 3 to the "normal" anion gap.



"Step 4" by Kapil Pal

For elevated anion gap metabolic acidosis (AGMA):

Important work up for AGMA includes urine ketones, renal function tests, serum lactate, toxin screen and osmolar(OG). (See the causes.)

- OG = Measured osmolality – Calculated osmolality
- Calculated osmolality = 2(Na^+) + Glucose/18 + BUN/2.8
- If OG > 10 → suggests the possibility of ingestion as a cause of AGMA

Another important consideration is to calculate ΔAG (change in AG) and ΔHCO_3– (change in bicarbonate).

ΔAG = Patient's AG – Normal AG

- If ΔAG = ΔHCO_3– → pure AGMA
- If Δ AG > Δ HCO_3– → concomitant metabolic alkalosis
- If Δ AG < Δ HCO_3– → concomitant non-anion gap metabolic acidosis or chronic respiratory acidosis

Example 1: HCO_3– = 16, AG = 20

Unmeasured anions Δ AG = 20 – 12 = 8 (normal AG = 12)
Δ HCO_3– = 24 – 16 = 8 (normal HCO_3– = 24)

Thus, ΔHCO_3– = Δ AG = 8 → pure AGMA

Example 2: HCO_3– = 10, AG = 20

Unmeasured anions Δ AG = 20 – 12 = 8
ΔHCO_3– = 24 – 10 = 14

Thus, Δ HCO< $_{3- \neq \Delta AG}$

8 mEq of change in HCO_3– is accounted for by AG acidosis; additional (14 – 8=) 6 mEq of change in HCO_3– must have been caused by non-AG acidosis → combined AG and non-AG acidosis.

For normal anion gap metabolic acidosis (NAGMA):

Check Urine Anion Gap: UNa^+ + UK^+ – UCl^-

- If urine AG is positive, it implies HCO_3– loss by the kidneys as in renal tubular acidosis.
- If urine AG is negative, it implies HCO_3– loss from an external source as in gastrointestinal losses.

Step 5: Review differential diagnosis

Refer to causes of acid-base disorders described above and correlate them with the conclusion before making a final diagnosis.

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Notes