



Acid Base Disorders

> Objectives:

- To provide a simple, systematic approach to interpreting arterial blood gas (ABG) samples..

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Note: there won't be **calculations** in the exam so focus more on the theory part. [SUMMARY](#)

Acid Base Disorders

- Getting in the routine of performing these steps on each patient in which an ABG and electrolytes are performed will help decrease the rate of missed complex acid-base disturbances and hopefully improve patient care.

Five Steps of Acid-Base Analysis 1-5 (Understand the steps memorise none)

-Step 1: **Acidemia** (pH <7.38) or **alkalemia** (pH >7.42)

Acidosis is the process that induce academia, alkalosis is the process that induce alkalemia

Can a patient have acidosis without acidemia, how ?

yes. when the patient has a mix acid-base disorder he can present with normal pH and still has acidosis, and here the cause of normal PH is not compensation but the mix acid-base disorder so you can't trust the numbers, and follow the steps. compensation can make PH normal only in one case: **chronic respiratory alkalosis**

-Step 2: **Primary** respiratory or metabolic disturbance? Look at PCO₂ and pH.

-If pH and PCO₂ going in same direction (both increase or decrease) = **Metabolic**

-If pH and PCO₂ not going in same direction = **Respiratory**

-Step 3: Is there appropriate compensation for the primary disorder?

-Metabolic acidosis: $PCO_2 = [1.5 \times (\text{serum } HCO_3)] + 8 (\pm 2)$

Winter's formula/expected PCO₂:

calculated PCO₂ = the patient PCO₂ → appropriate response (compensation)

calculated PCO₂ ↓ than the patient PCO₂ → respiratory acidosis

calculated PCO₂ ↑ than the patient PCO₂ → respiratory alkalosis

-Metabolic alkalosis: $\uparrow PCO_2 = 0.6 \times \uparrow HCO_3 (\pm 2)$

-Respiratory acidosis: $\uparrow PCO_2$ 10, $\uparrow HCO_3$ by 1 (acute) or 4 (chronic)

-Respiratory alkalosis: $\downarrow PCO_2$ 10, $\downarrow HCO_3$ by 2 (acute) or 5 (chronic)

-Step 4: Is there an anion gap metabolic acidosis (AGMA)?

$AG = Na - (HCO_3 + Cl)$. If > 12, an AGMA is present (AKA high anion gap).

you do step 4 for all types, not only metabolic acidosis, because there might be metabolic acidosis blunted by a stronger disturbance

-Step 5: If metabolic acidosis, is there another concomitant metabolic disturbance?

If **AGMA**, then calculate $\Delta Gap = \Delta AG - \Delta HCO_3 = (AG - 12) - (24 - HCO_3)$

-If the ΔGap is > 6, there is a combined AGMA and **metabolic alkalosis**.

-If the ΔGap is < -6, there is a combined AGMA and **NAGMA**.

- If between -6 and 6, there is only AGMA

If **NAGMA**, for every 1 mEq/L $\uparrow Cl$, there should be a 1 mEq/L $\downarrow HCO_3 (+ 5)$.

-If HCO₃ decrease is less than predicted, then NAGMA and **metabolic alkalosis**.

Metabolic Acidosis

- In the presence of a pH < 7.38, metabolic acidosis is diagnosed as a primary condition when the pCO₂ is < 40 mmHg or the bicarbonate is < 24 mEq/L.
- Metabolic acidosis can be further classified based on the presence of an anion gap. The anion gap reflects the balance between positively and negatively charged particles in the blood.
- Sodium is the only significant positively charged particle that is measured, while the measured anions are chloride and bicarbonate. Therefore, the anion gap is calculated by the formula: $Na - (Cl + HCO_3)$.
k isn't used in the formula, if used the normal AG value is 20 instead of 12
- One potential pitfall in the measurement of the anion gap is patients with low albumin.
- Albumin has several negative charges on it and therefore, in a patient with a low albumin level, their "normal" anion gap might be much lower than 12.
- For every 1 gram drop in serum albumin level, the anion gap decreases by 2.5.
- A patient with a calculated anion gap of 10 and a 2 gram drop in their albumin may actually have an anion gap metabolic acidosis (recalculated AG 15).
- Detection of an AGMA is important because only a few conditions commonly cause it.
- In addition, in mixed acid-base disorders, an elevation in the anion gap may be the only signal that a metabolic acidosis is present.
- The causes of an AGMA are divided into four main categories:
 - renal failure.
 - ketoacidosis.
 - lactic acidosis.
 - toxins. toxic alcohols : Ethylene glycol, methanol.

Metabolic acidosis causes:

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- Analgesics (massive NSAID such as ASPIRIN , acetaminophen). Aspirin causes both metabolic acidosis (AGMA) and respiratory alkalosis, except in children, their respiratory center isn't well developed yet so they present with only AGMA.
- Cyanide, Carbon monoxide
- Arsenic, Alcoholic ketoacidosis.
- Toluene
- Methanol, Metformin
- Uremia
- Diabetic ketoacidosis
- Paraldehyde, Phenformin
- Iron, Isoniazid
- Lactic acidosis
- Ethylene glycol



In any patient with an AGMA, calculate an osmol gap:

-Osmol gaps are a clue to a potentially life-threatening toxic alcohol ingestion (ie. ethylene glycol and methanol).

-The osmol gap is determined by subtracting the calculated osmolality from the measured osmolality.

Calculated osmolality = $2(\text{Na}) + \text{Glc}/18 + \text{BUN}/2.4 + \text{ETOH}/4.6$

$2 \text{ Na} + \text{Glucose} + \text{Urea}$ (all in mmol/L). if more than 10 > high osmolar gap

Example:

-32 year old man with depression and alcohol abuse presents with altered mental status.

-ABG: pH 6.9, pCO₂ 29, pO₂ 100

-Metabolic panel: Na 140, Cl 101, HCO₃ 5

-Step 1: Acidosis

-Step 2: Metabolic

-Step 3: pCO₂ = $1.5(\text{HCO}_3) + 8 = 15$, but the patient's pCO₂ is higher than 15. Therefore, a respiratory acidosis is also present, possibly secondary to CNS depression.

-Step 4: AG = $140 - (101 + 5) = 34$ > AGMA

-Step 5: Delta gap = $(34-12) - (24-5) = 3$. between 6 and -6 just AGMA No additional metabolic disorders other than AGMA.

-Answer: Anion gap metabolic acidosis and respiratory acidosis. The patient had an osmol gap of 174 and a methanol level of 510 mg/dL.

Non-Anion Gap Metabolic Acidosis (NAGMA)

-A NAGMA is due to either GI or renal losses of bicarbonate.

-If desired, GI mediated and renally mediated losses can be distinguished by obtaining urine electrolytes (ie. Na, K, and Cl) and calculating the urine anion gap.

-The urine anion gap : $\text{Na} + \text{K} - \text{Cl}$

-The urine anion gap is the difference between the spot urine positive ions and spot urine negative ions.

-If an excess of negatively charged ions is present, the acidemia is due to the kidney

-Causes : renal tubular acidosis, Vesicoenteric fistulas, Diarrhea

Example:

-A 68 year old man who recently took antibiotics for a skin infection presents with 10 episodes of watery diarrhea per day for the last 5 days.

-ABG: pH 7.34, pCO₂ 34, pO₂ 80

-Metabolic panel: Na 135, Cl 108, HCO₃ 18

-Step 1: Acidosis

-Step 2: Metabolic

-Step 3: pCO₂ = $1.5(\text{HCO}_3) + 8 = 35$ > matches the patient pco₂ (± 2) > no other respiratory disorder

-Step 4: AG = $135 - (108 + 18) = 9$ > NAGMA

-Step 5: Cl \uparrow by 8 and HCO₃ \downarrow by 6; therefore there is no metabolic alkalosis.

-Answer: NAGMA due to diarrhea

Respiratory acidosis

-Respiratory acidosis is characterized by an **elevation in the pCO₂** and a **decrease in blood pH** due most commonly to **hypoventilation**.

-It results from conditions that decrease the ability of the lungs to excrete carbon dioxide at a rate to keep up with the body's production.

-The kidney compensates for primary respiratory acidosis by retaining bicarbonate.

-This compensation occurs over hours to days and is generally at a maximum within four days.

-The rate of onset of respiratory acidosis can be determined by the degree of renal compensation (increase in HCO₃) as listed above in step 3.

-Alternatively, the chronicity of the respiratory acidosis can be predicted by the change in the pH:

- In **acute** respiratory acidosis, the **pH decreases by 0.08 units** for each increase of 10 mmHg in the pCO₂ from its baseline of 40 mmHg.
- **Chronic** respiratory acidosis is marked by a **decrease in the pH of 0.03 units** for every increase of 10 mmHg in the pCO₂.
- Acute on top of chronic

-Differentiating acute from chronic respiratory conditions can have important clinical implications that may alert the clinician to a patient that is rapidly spiraling downward and might require emergent intubation, from a patient who has chronic disease, but is in less danger of imminent decompensation

examples of chronic respiratory acidosis are smoking and COPD > no need for urgent Rx

Differential diagnosis includes:

- Central nervous system depression (sedatives, CNS disease, sleep apnea)
- Pleural disease (large pneumothorax or pleural effusion)
- Lung disease (ARDS, COPD, pulmonary edema, severe pneumonia)
- Acute airway obstruction (laryngospasm, sleep apnea)
- Neuromuscular disorders (GBS, myasthenia gravis, botulism)
- Thoracic cage injury (flail chest)
- Ventilator dysfunction

Example:

-A 70 year-old smoker presents with an acute onset of shortness of breath.

-ABG: pH 7.30, pCO₂ = 60 mmHg, pO₂ 60 mmHg

-Metabolic panel: Na 135, Cl 100, HCO₃ 30

-Step 1: Acidosis

-Step 2: Respiratory

-Step 3: Acute on chronic. pCO₂ increased by 20, therefore the HCO₃ should increase by 2 if acute and 8 if chronic. Because the HCO₃ increased from 24 to 30 (6), an acute on chronic respiratory acidosis is present.

-Step 4: AG = 135 - (100 + 26) = 9. No anion gap metabolic acidosis

-Step 5: XX

-Answer: Acute on chronic respiratory acidosis due to COPD exacerbation.

Metabolic alkalosis

Metabolic alkalosis is characterized by an increase in the serum **bicarbonate concentration**.

The causes of metabolic alkalosis:

- Volume contraction (vomiting, NG suction, loop or thiazide diuretics).
- Excess glucocorticoids or mineralocorticoids (eg, Cushing's syndrome).
- Hypokalemia
- Bartter's syndrome.
- Alkali ingestion/infusion.
- Post-hypercapnic alkalosis

To differentiate the most common cause of metabolic alkalosis which is volume depletion from other causes you need to measure urine chloride.

- If urine chloride less than 10 this due to volume depletion (saline response)
- If urine chloride more than 10 this due to other causes (saline resistance)

Example:

-A 20 year old student presents with excessive vomiting after binge drinking.

-ABG: pH 7.50, pCO₂ 44, pO₂ 100

-Metabolic panel: Na 138, Cl 100, HCO₃ 30

-Step 1: Alkalosis

-Step 2: Metabolic both pH and pCO₂ high - metabolic

-Step 3: Increase in pCO₂ should equal 0.6 multiplied by the elevation of the HCO₃ ±2. The increase of the pCO₂ of 4 is within two of 6(0.6) or 3.6; therefore there is appropriate compensation.

-Step 4: AG = 138 - (100 + 30) = 8 here we stop, only metabolic alkalosis cause we have normal AG

-Step 5: XX

-Answer: Metabolic alkalosis secondary to vomiting

Respiratory alkalosis

-Respiratory alkalosis is characterized by a **decrease in the pCO₂** and an **elevation in the blood pH**.

-The pO₂ can be used to distinguish between disease of the lungs and other causes of hyperpnea (eg, fever)

Causes of a primary respiratory alkalosis :

- CNS disease (CVA)
- Toxins (Salicylates)
- High altitude
- Severe anemia
- Pregnancy
- Lung disease/hypoxia (asthma, pneumonia, PE, pulmonary edema, pulmonary fibrosis)
- Anxiety
- **Cirrhosis** of the liver
- Fever (Sepsis)
- Ventilator dysfunction

Example:

-A 22 year-old woman presents with 4 hours of numbness in both hands. typical of previous episodes of anxiety.

-ABG: pH 7.48, pCO₂ 30 mmHg, pO₂ 86 mmHg

-Metabolic panel: Na 140, Cl 110, HCO₃ 22

-Step 1: Alkalosis

-Step 2: Respiratory

-Step 3: Acute. Drop in the pCO₂ by 10 corresponds to a drop in the HCO₃ by 2 if acute and 5 if chronic. 24-22 = 2 and therefore, as would be expected by the clinical history, an acute disorder is diagnosed.

-Step 4: AG 140-(110+22) = 8

-Step 5: XX

-Answer: Acute respiratory alkalosis secondary to a panic attack

Practice Cases

CASE 1

-A diabetic presents with diarrhea and cough. CXR reveals an infiltrate.

-pH 7.31; pCO₂ 10

-Na 123; Cl 99; HCO₃ 5

ANS: Primary AGMA (DKA), respiratory alkalosis (pneumonia), NAGMA (diarrhea)

CASE 2

-An alcoholic presents with vomiting.

-pH 7.20; pCO₂ 25

-Na 130; Cl 80; HCO₃ 10

ANS: Primary AGMA (alcoholic ketoacidosis), metabolic alkalosis (vomiting)

CASE 3

-A man with arthritis presents with confusion, shortness of breath, and diaphoresis.

-pH 7.30; pCO₂ 18

-Na 147; Cl 108; HCO₃ 16

ANS: Primary AGMA and respiratory alkalosis (Salicylate toxicity—107 mg/dl)

CASE 4

-A patient with COPD presents with shortness of breath.

-pH 7.18; pCO₂ 80

-Na 135; Cl 93; HCO₃ 30

ANS: Primary respiratory acidosis—acute-on-chronic (COPD exacerbation)

CASE 5

-A woman with Crohn's disease presents with fever, vomiting, and diarrhea.

-pH 7.36; pCO₂ 22

-Na 147; Cl 121; HCO₃ 14

ANS: Primary NAGMA (diarrhea), respiratory alkalosis (fever), metabolic alkalosis (vomiting)

CASE 6

-A noncompliant patient with diabetes and cirrhosis presents with vomiting.

-pH 7.46; pCO₂ 17

-Na 133; Cl 84; HCO₃ 15

ANS: Primary chronic respiratory alkalosis (cirrhosis), AGMA (DKA), metabolic alkalosis

CASE 1:

step 1 : acidosis low PH

step 2 : metabolic why ? both PH and Pco₂ ↓

step 3 : $(1.5 \times 5) + 8 = 15.5$ (respiratory alkalosis) the patient's pCO₂ is lower than 15.5 Therefore, a respiratory alkalosis

step 4 : $AG = 123 - (99 + 5) = 19$ (AGMA) more than 12

step 5 : $(19 - 12) - (24 - 5) = -12$ (AGMA + NAGMA) If the ΔGap is < -6, there is a combined AGMA and NAGMA.

CASE 2:

step 1 : acidosis low PH

step 2 : metabolic both PH and Pco₂ ↓

step 3 : $(1.5 \times 10) + 8 = 23$ there is appropriate compensation.

step 4 : $AG = 130 - (80 + 10) = 40$ (AGMA) more than 12

step 5 : $(40 - 12) - (24 - 10) = 8$ If the ΔGap is > 6, there is a combined AGMA and metabolic alkalosis.

CASE 3:

step 1 : acidosis low PH

step 2 : metabolic both PH and Pco₂ ↓

step 3 : $(1.5 \times 16) + 8 = 32$ (respiratory alkalosis) the patient's pCO₂ is lower than 32 Therefore, a respiratory alkalosis

step 4 : $AG = 147 - (108 + 16) = 23$ (AGMA) more than 12

step 5 : $(23 - 12) - (24 - 16) = -1$ between 6 and -6 no more acid-base disorder

CASE 4:

step 1 : acidosis low PH

step 2 : respiratory. why ? PH low and Pco₂ high

step 3 : Pco₂ 80 and Hco₃ 30. ↑ PCO₂ 10, ↑ HCO₃ by 1 (acute) or 4 (chronic) . the patient is acute on chronic

step 4 : $AG = 135 - (93 + 30) = 12$ NAGMA

CASE 5:

step 1 : acidosis low PH

step 2 : metabolic both PH and Pco2 ↓

step 3 : $(1.5 \times 14) + 8 = 29$ (respiratory alkalosis) the patient's pCO₂ is lower than 29 Therefore, a respiratory alkalosis.

step 4 : $AG = 147 - (121 + 14) = 12$ NAGMA

step 5 : If NAGMA, for every 1 mEq/L ↑ Cl, there should be a 1 mEq/L ↓ HCO₃ (±5).

If HCO₃ decrease is less than predicted, then NAGMA and metabolic alkalosis.

CASE 6:

step 1 : alkalosis high PH

step 2 : respiratory PH high and Pco2 low

step 3 : Pco2 17 Hco3 15 (chronic)

step 4 : $AG = 133 - (84 + 15) = 34$ (AGMA) more than 12

step 5 : $(34 - 12) - (24 - 15) = 13$ (AGMA - metabolic alkalosis) more than 6.

SUMMARY of most important causes of acid-base disturbance:

	Acidosis		Alkalosis
Metabolic	AGMA: <ul style="list-style-type: none"> - Renal failure. - Ketoacidosis (diabetic/ alcoholic: methanol & ethylene glycol) - Lactic acidosis - Aspirin - Sepsis 	NAGMA: <ul style="list-style-type: none"> - renal tubular acidosis, - Vesicoenteric fistulas, - Diarrhea 	Metabolic Alkalosis: <ul style="list-style-type: none"> - vomiting
Respiratory	Respiratory acidosis: <ul style="list-style-type: none"> - Hypoventilation - Chronic = smoking or COPD - Acute on chronic = COPD exacerbation 		Respiratory Alkalosis: <ul style="list-style-type: none"> - Acute = Hyperventilation e.g. Panic attack, fever (sepsis) - Chronic = cirrhosis - Aspirin