



# Acid-Base Disorder

## Objectives:

- Develop an approach to acid base problems
- Identify the primary acid base disturbance
- Solve simple acid base cases

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**Resources:** 436 slides + 435 team + Davidson + kumar + Recall questions step up to medicine.

- [Editing file](#)
- [Feedback](#)

## Introduction

Helpful video: [3: 21 minutes](#)

### ★ Normal value

- Arterial blood pH = 7.35 – 7.45 while normal pH of venous blood is 7.31-7.41
- PaCO<sub>2</sub> = 35-45 → 40
- Serum HCO<sub>3</sub><sup>-</sup> = 22-26 → 24
- Anion gap = 8-12

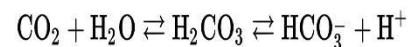
Normal values are different due to different references. Check the given reference in the test. maintaining normal pH is important to maintain the integrity of protein function (Enzyme function and membrane proteins).

### ★ Basic RECALL

- Acid-base balance is concerned with maintaining a normal hydrogen ion concentration in the body fluids. This balance is achieved by buffers utilization of in extracellular fluid and intracellular fluid, by respiratory mechanisms that excrete carbon dioxide, and by renal mechanisms that reabsorb bicarbonate and secrete hydrogen ions.
- Blood pH refers to the level of H<sup>+</sup> ions and maintained by several buffering systems.
  - A decrease in blood pH is called acidemia and is caused by acidosis.
  - An increase in blood pH is called alkalemia and is caused by alkalosis.
- Disturbances of acid-base balance are described as either metabolic or respiratory, depending on whether the primary disturbance is in HCO<sub>3</sub><sup>-</sup> or CO<sub>2</sub>.
- The assessment of acid base abnormalities is typically done using arterial blood gases (ABG)
- Given the ease of obtaining venous blood gases (VBG) and capillary blood gases (CBG) these are often used in clinical practice
- Always check the reference range in your local laboratory

### ★ Buffering

- A buffered solution resists a change in pH.
- Most importantly the **bicarbonate-carbonic acid buffer pair** that depends on the balance between bicarbonate ions and carbonic acid.



In compensation, Ph gets close to the normal value but does not reach normal. our bodies compensate by 3 mechanisms: Breathing in or out (if it is metabolic ), Excreting / retaining hydrogen (respiratory) and Buffers in blood.

- ★ In cases of normal Ph levels, look at the patient's health. if the patient has a disease with a normal pH level think of mixed acidosis and alkalosis. If the patient appears healthy with a normal pH they're most probably normal

# Respiratory Acidosis

★ **Definition**

- Increased **PaCO<sub>2</sub>** and decreased pH.

★ **Mechanism**

- Process that primarily causes elevation in PaCO<sub>2</sub> :
- Reduce effective ventilation e.g. **many chronic respiratory diseases** or drugs depressing the respiratory center.
- Alveolar Hypoventilation → Accumulation of CO<sub>2</sub> → Increases in PaCO<sub>2</sub> → Respiratory acidosis → pH decreases.
- HCO<sub>3</sub> will increase (Compensation) but it needs time (12 -24 h) as the kidney need time to compensate.

★ **Etiology Hypoventilation of any cause:**

<b>CNS</b>	Damage of the respiratory center in the brainstem Caused by: <b>Stroke</b> , Hemorrhage, Trauma, Tumor, Medication (Commonly sleeping pills, other: morphine, anesthetics and narcotics)
<b>Peripheral nervous system</b>	Demyelinating disease Of PNS ex. Guillain-Barre syndrome <sup>1</sup>
<b>Neuromuscular junction</b>	Myasthenia gravis
<b>Muscular disease</b>	Intercostal muscle atrophy, such as: ● Duchenne dystrophy ● Congenital muscle atrophy
<b>Chest wall</b>	Severe scoliosis
<b>Bronchial tree</b>	<b>COPD<sup>2</sup></b> → retain CO <sub>2</sub> → exchange gases lung defect→ leading to acute/chronic Respiratory acidosis)
<b>Other</b>	<b>Drowning</b> , Sleep apnea and Morbid obesity.

<sup>1</sup> usually follow diarrhea or flu like illness → followed by ascending paralysis from legs going up (reach respiratory muscles).

<sup>2</sup> Any disorder that reduces CO<sub>2</sub> clearance (i.e., inhibits adequate ventilation) can lead to respiratory acidosis.

★ **Clinical Features:**

- **Symptoms:** Somnolence<sup>3</sup>, confusion, myoclonus with asterixis (Flapping tremors)
- **Signs of acute CO<sub>2</sub> retention:** headaches, confusion, and papilledema<sup>4</sup>.

★ **Classification:**

Each of the simple respiratory disorders has two ranges of expected values, one for the acute disorder and one for the chronic disorder. The acute disorder is present before renal compensation has occurred, and, therefore, values for blood pH tend to be more abnormal. The chronic disorder is present once renal compensation has occurred, which takes several days (starts within 24 hours). Renal mechanisms increase the excretion of H<sup>+</sup> within 24 hours and may correct the resulting acidosis caused by chronic retention of CO<sub>2</sub> to a certain extent. Because of the compensatory process, values for blood pH tend to be more normal in the chronic phase.

	<b>Acute Respiratory Acidosis</b>	<b>Chronic Respiratory Acidosis</b>
<b>Causes</b>	<ol style="list-style-type: none"> <li>1. Respiratory: airway obstruction, severe pneumonia, chest trauma/pneumothorax</li> <li>2. Acute drug intoxication: narcotics, sedatives.</li> <li>3. Residual neuromuscular blockade.</li> <li>4. CNS disease (head trauma)</li> </ol>	<ol style="list-style-type: none"> <li>1. Chronic lung disease (COPD)</li> <li>2. Neuromuscular disease</li> <li>3. Extreme obesity</li> <li>4. Chest wall deformity</li> <li>5. Muscular e.g. Duchenne dystrophy</li> </ol>
<b>pH</b>	<b>LOW</b>	<b>Almost NORMAL due compensatory mechanism.</b>
<b>Compensation</b>	<ul style="list-style-type: none"> <li>• Immediate compensatory ↑ of HCO<sub>3</sub>.</li> <li>• HCO<sub>3</sub> ↑ by 1 mEq/l for every 10 mmHg ↑ in PaCO<sub>2</sub>.</li> </ul>	HCO <sub>3</sub> ↑ by 3-3.5 mEq/l for every 10 mmHg ↑ in PaCO <sub>2</sub> (Due to renal adaptation)

<sup>3</sup> Drowsiness or sleepiness

<sup>4</sup> Papilledema is optic disc swelling that is secondary to elevated intracranial pressure.

Pathophysiology: Increased PaCO<sub>2</sub> causes increased cerebral blood flow which increases CSF pressure → Resulting in generalized CNS depression.

### ★ Treatment:

- Verify patency of airways.
- Give supplemental oxygen: If PaO<sub>2</sub> is low (<60 mmHg), Oxygen is contraindicated in COPD patients (CO<sub>2</sub> retention) as it can exacerbate symptoms. The explanation is that hypoxia drives breathing, so when the patient is no longer hypoxic, hypoventilation can result and hypercapnia worsens, causing a respiratory acidosis.
- Treat underlying cause.
- Intubation and mechanical ventilation might be required for:
  - Severe acidosis (PH <7).
  - PaCO<sub>2</sub> > 60 or inability to increase PaO<sub>2</sub>.
  - Mental deterioration.
  - Impending respiratory fatigue.

## Respiratory Alkalosis

### ★ Definition:

Decreased PaCO<sub>2</sub> and increased pH.

### ★ Mechanism:

- Process that primarily causes reduction in PaCO<sub>2</sub> :
- Increase ventilation e.g.in response to hypoxia or secondary to metabolic acidosis.
- Alveolar hyperventilation → increased wash out CO<sub>2</sub> → decrease in PaCO<sub>2</sub> → increased pH.
- **Compensation:** HCO<sub>3</sub><sup>-</sup> will decrease after (12 -24 h).

### ★ Etiology Hyperventilation<sup>5</sup> of any Cause:

- Overaggressive mechanical ventilation.
  - **Anxiety** (most common), **Fever** (not severe) 2nd most common.
  - Pain, Sepsis, Pregnancy<sup>6</sup>, Hepatic failure (cirrhosis)
  - Hypoxemia, Restrictive lung disease
  - Medication (salicylate toxicity e.g. aspirin overdose<sup>7</sup>)
  - Severe congestive heart failure, Thyrotoxicosis.
  - Pulmonary embolism, asthma, pneumonia.
- Respiratory alkalosis can be caused by anything that causes hyperventilation (anxiety causing panic attack)

<sup>5</sup> Any disorder that increases the respiratory rate inappropriately can lead to respiratory alkalosis

<sup>6</sup> increase serum prostaglandin → Hyperventilation.

<sup>7</sup> overstimulation of respiratory center → Hyperventilation.

Aspirin can cause both respiratory alkalosis and metabolic acidosis.

★ **Clinical Features:**

- (lightheadedness, dizziness, anxiety, paresthesia, and perioral numbness)<sup>8</sup>
- Tetany<sup>9</sup>, Arrhythmias, Trousseau’s sign and Chvostek’s sign may be positive

**Classification:**

Acute Respiratory Alkalosis	Chronic Respiratory Alkalosis
HCO <sub>3</sub> ↓ by 2 mEq/l for every 10 mmHg ↓ in PaCO <sub>2</sub> .	HCO <sub>3</sub> ↓ by 4-5 mEq/l for every 10 mmHg ↓ in PaCO <sub>2</sub> .

★ **Treatment:**

- Treat the underlying cause.
- Sometimes this does not need to be treated (e.g., in the case of pregnancy).
- Breathe into paper bag to recycle the exhaled CO<sub>2</sub> (especially who have anxiety).
- 

**Metabolic Acidosis**

★ **Definition:**

Loss of [HCO<sub>3</sub>] or addition of [H<sup>+</sup>] and decreased pH.

★ **Mechanism:**

- Process that primarily reduce bicarbonate:
- Excessive H<sup>+</sup> formation e.g. lactic acidosis, ketoacidosis.
- Reduce H<sup>+</sup> excretion e.g. renal failure.
- Excessive HCO<sub>3</sub><sup>-</sup> loss e.g. diarrhea.
- Increase acid gaining either Exogenous Intake or Endogenous production. Or Decrease acid excretion. Or Loss or decrease production of bicarbonate.
- Compensation<sup>10</sup>: Hyperventilation → decrease PCO<sub>2</sub> immediately.
- If the kidneys are intact and the primary cause of acidosis is not renal in origin, the kidney can gradually increase acid secretion over days to weeks and restore a new steady state

<sup>8</sup> Symptoms are mostly related to decreased cerebral blood flow (vasoconstriction).

<sup>9</sup> Indistinguishable from hypocalcemia

<sup>10</sup> Metabolic problems always show compensation.

here's a helpful [video](#) to explain the Anion gap!

- **The Anion gap:**

The difference between primary measured cations (Na<sup>+</sup> and K<sup>+</sup>) and the primary measured anions (Cl<sup>-</sup> and HCO<sub>3</sub><sup>-</sup>) in serum:

- Anion gap = cations - anions → AG= ([Na<sup>+</sup>] + [K<sup>+</sup>]) - ([Cl<sup>-</sup>] + [HCO<sub>3</sub><sup>-</sup>])  
Or
- Anion gap = Sodium - (Chloride + Bicarbonate) →AG = [Na<sup>+</sup>] - ([Cl<sup>-</sup>] + [HCO<sub>3</sub><sup>-</sup>]).

- It is helpful in determining the cause of a **metabolic acidosis**

- ★ **Classification & Etiology:**

It's mainly divided into normal Anion gap acidosis and High Anion gap acidosis

**Causes of High Anion Gap Metabolic Acidosis (MUD PILES):**

- Methanol
- Uremia
- **DKA (check glucose)**
- Propylene glycol (not paraldehyde)
- INH (impaired hepatic clearance of lactate)
- Lactic acidosis
- Ethanol/Ethylene Glycol
- Salicylates

Problem		Causes
Increased Endogenous production	Lactic acidosis	<ul style="list-style-type: none"> <li>• <b>Low tissue perfusion</b> (decreased oxygen delivery to tissues)</li> <li>• <b>Shock</b> states (septic, cardiogenic, hypovolemic)</li> </ul>
	<b>Diabetic Ketoacidosis</b>	DM, Prolonged starvation and prolonged alcohol abuse
	Uremia	Renal failure—decreased NH <sub>4</sub> + excretion (thus decreasing net acid)—Decreased excretion of organic anions, sulfate, and phosphate increases AG.
Increased Exogenous Intake	Oxalic acid	Ethylene glycol overdose/intoxication (manifestations include cardiopulmonary failure, calcium oxalate crystals and renal failure)
	Formic acid	Methanol overdose (manifestations include blurred vision)
	Other	Salicylates <sup>11</sup> (Aspirin overdose), aldehyde, Acetaminophen, alcohol

<sup>11</sup> Salicylate overdose causes both primary respiratory alkalosis and primary metabolic acidosis.

- **Normal AG Acidosis** → The low  $\text{HCO}_3^-$  is associated with high  $\text{Cl}^-$ , so that the AG remains normal.

<b>GI</b>	loss of $\text{HCO}_3^-$	<b>Diarrhea</b> , fistula in intestine or pancreas, Ureterosigmoidostomy: (colon secretes $\text{HCO}_3^-$ in urine in exchange for $\text{Cl}^-$ )
<b>RENAL</b> <sup>12</sup>	↓ $\text{HCO}_3^-$ reabsorption.	<b>Proximal Tubular Acidosis (RTA Type 2)</b>
	↓ production of $\text{HCO}_3^-$	<b>Distal Tubular Acidosis (RTA Type 1)</b>
	Carbonic anhydrase inhibition	Due to diuretics as acetazolamide.
<b>Other</b>	Post- hypocapnia	Respiratory alkalosis → renal wasting of $\text{HCO}_3^-$ → rapid correction of respiratory alkalosis → transient Acidosis until $\text{HCO}_3^-$ regenerated
	Dilutional	Due to rapid infusion of bicarbonate - free IV fluids.

- ★ One of the causes of metabolic acidosis with normal anion gap is Urinary diversion procedure. In this procedure the ureter is taken from kidney and attached to gut, urine is excreted with stool. With that, there is retention of chloride from urine leading to hyperchloremic acidosis

In summary:

- 1) Gain acid from A) Outside: alcohol “ethanol, methanol” or B) Inside: renal failure, lactic acidosis, ketoacidosis
- 2) Loss  $\text{HCO}_3^-$  from diarrhea or RTA

★ **REMEMBER!**

**1- High Anion Gap**

- a. Endogenous:
  - +ve Ketones: ketoacidosis because of starvation or diabetic ketoacidosis
  - -ve ketones: lactic acidosis because of ischemia or hypoxia, or uremia in renal failure.
- b. Exogenous: (e.g. alcohol, ethanol, methanol, paraldehyde and aspirin overdose)

**2- Normal Anion Gap**

Diarrhea and Renal tubular acidosis.

<sup>12</sup> To distinguish between RTA & Diarrhea we perform Urine Anion Gap (UAG= Sodium - Chloride):

- In RTA there is a defect in acid secretion → so less  $\text{Cl}^-$  into urine → result of UAG positive number.
- In Diarrhea Excretion acid is intact →  $\text{H}^+$  is excreted with  $\text{Cl}^-$  in urine → UAG negative number.



## ★ Clinical Features:

- Hyperventilation (deep rhythmic breathing) also called Kussmaul respiration.
- Decreased in Cardiac output and tissue perfusion.

- a. Occurs with severe metabolic acidosis (blood pH < 7.2)
- b. Acidosis diminishes tissue responsiveness to catecholamines. This can lead to an undesirable chain of events: poor tissue perfusion → lactic acidosis → decreased cardiac output → hypotension → further decrease in tissue perfusion.

## ★ Treatment:

1. Treat the underlying cause.
2. Sodium bicarbonate is sometimes used in severe acidosis (esp. in normal AG acidosis).
3. Mechanical ventilation might be needed if the patient is fatigued (esp. in DKA)

## Metabolic Alkalosis

### ★ Definition:

Addition of  $[\text{HCO}_3^-]$  or loss of  $[\text{H}^+]$  and increase pH

### ★ Mechanism:

- Process that primarily raises bicarbonate:
- Extracellular fluid volume loss e.g. due to vomiting or diuretics
- Excessive potassium loss with subsequent hyperaldosteronism
- Initiating metabolic alkalosis by either:
  - Gaining of  $\text{HCO}_3^-$
  - Or Loss of acid ( $\text{H}^+$ ) ex: from vomiting.
- Maintaining Metabolic alkalosis due to the kidney inability to excrete the excess  $\text{HCO}_3^-$ <sup>13</sup>
- Compensation: Hypoventilation → increased  $\text{PCO}_2$  (respiratory Acidosis) immediately ( $\text{PaCO}_2 \uparrow$  by 0.6 mmHg for every 1 mEq/l  $\uparrow$  in  $\text{HCO}_3^-$ ).

<sup>13</sup> Uncomplicated metabolic alkalosis is typically transient, because kidney can normally excrete the excess  $\text{HCO}_3^-$

★ **Classification & causes:**

	<b>Saline Responsive Urine (cl-) &lt;20</b>	<b>Saline resistant Urine (cl-) &gt;20</b>
<b>Definition</b>	Metabolic alkalosis with ECF contraction (due to → fluid loss).	Metabolic alkalosis with ECF volume expansion (no fluid loss)
<b>Causes</b>	<ul style="list-style-type: none"> <li>● <b>Gastric loss of H<sup>+</sup></b> and generation of HCO<sub>3</sub> such as vomiting (HCl loss), <u>NGT drainage</u></li> <li>● <b>Diuretic use</b> → These decrease the ECF volume, body HCO<sub>3</sub> content is normal, but plasma HCO<sub>3</sub> increases due to ECF contraction.</li> <li>● <b>Volume depletion:</b> Loss of sodium and fluid leads to hypovolemia and secondary hyperaldosteronism, triggering proximal sodium bicarbonate reabsorption and additional acid secretion by the distal tubule Hypokalemia occurs due to potassium loss in the vomitus and by the kidney as the result of secondary hyperaldosteronism, and itself is a stimulus to acid secretion.</li> <li>● Post-hypercapnia</li> <li>● Villous adenoma of colon, diarrhea with high chloride content</li> </ul>	<p>Hypertensive:</p> <ul style="list-style-type: none"> <li>● <b>Primary Hyperaldosteronism</b> either</li> <li>● Non-mineralocorticoid, Cushing Syndrome</li> </ul> <p>Hypo/normo tensive:</p> <ul style="list-style-type: none"> <li>● Exogenous alkali load either IV or oral sodium bicarbonate</li> <li>● Bartter's syndrome &amp; Gitelman's syndrome, Severe hypokalemia</li> </ul>
<b>Treatment</b>	Treat by saline due to volume depletion	According to Etiology

★ **Clinical Features:**

There are no characteristic signs and symptoms (most imp. Hx)

★ **Treatment:**

- Treat the underlying cause.
- Give normal saline plus potassium in saline responsive.
- Spironolactone (K<sup>+</sup> sparing diuretic) might be considered in saline resistant.
- 

**Steps in Acid-Base Analysis**

Helpful Video: [8:38 min](#)

★ **Normal value**

- Arterial blood pH = 7.35 – 7.45 → 7.4
- PaCO<sub>2</sub> = 35-45 → 40
- Serum HCO<sub>3</sub><sup>-</sup> = 22-26 → 24
- Anion gap = 8-12

### ★ Step 1:

**Take a thorough history and physical examination, look for clues that may lead to the abnormalities in pH**

For example: Vomiting (metabolic alkalosis), Diarrhea (metabolic acidosis), COPD → CO<sub>2</sub> → Hypoventilation (respiratory acidosis), hyperventilation → Respiratory alkalosis, Medications: laxatives, diuretics, etc → (metabolic alkalosis), Diabetes (DKA) → metabolic acidosis

- Vomiting
- Diarrhea
- Hypoventilation
- Respiratory disease
- Medications (laxatives, diuretics, etc)
- Diabetes
- etc

### ★ Step 2:

**Look at the pH:**

**Determine if this is**

- Normal 7.35 – 7.45 (No abnormality or mixed acidosis and alkalosis)
- Low <7.35 (acidemic)
- High >7.45 (alkalemic)
- pH 7.35-7.45 → Normal or mixed acidosis and alkalosis

### ★ Step 3-a:

**Determine the primary abnormality that is causing the abnormal pH**

- If the pH is acidemic (<7.35), then look for  
Low HCO<sub>3</sub> (Metabolic) or High PCO<sub>2</sub> (Respiratory)
- If the pH is alkalemic (>7.45), then look for  
High HCO<sub>3</sub> (Metabolic) or Low PCO<sub>2</sub> (Respiratory)
- To determine whether the disturbance affects primarily the arterial PaCO<sub>2</sub> or the serum HCO<sub>3</sub>.
- Primary disturbance is in CO<sub>2</sub> → Respiratory (normal value 35-45)
- Primary disturbance is in HCO<sub>3</sub> → Metabolic (normal value 22-26)
- As mentioned earlier, Compensation will not return the pH to the normal range, it's just a mechanism which the body trying to reduce the impact.

### ★ Step 3-b:

**If pH is normal, that doesn't rule out mixed acidosis and alkalosis (Determine what is being mixed)**

- Look for high or low  $\text{PCO}_2$
- Look for high or low  $\text{HCO}_3$
- **Low**  $\text{PCO}_2$  suggests **respiratory alkalosis**
- **High**  $\text{PCO}_2$  suggests **respiratory acidosis**
- **Low**  $\text{HCO}_3$  suggests **metabolic acidosis**
- **High**  $\text{HCO}_3$  suggests **metabolic alkalosis**

### Is the respiratory disturbance acute or chronic?

- Acute respiratory acidosis:  $\text{HCO}_3$  increase by 1 mEq/l for every 10 mmHg increase in  $\text{PaCO}_2$ .
- Chronic respiratory acidosis:  $\text{HCO}_3$  increase by 3-3.5 mEq/l for every 10 mmHg increase in  $\text{PaCO}_2$ .
- Acute respiratory alkalosis:  $\text{HCO}_3$  decrease by 2 mEq/l for every 10 mmHg decrease in  $\text{PaCO}_2$ .
- Chronic respiratory alkalosis:  $\text{HCO}_3$  decrease by 4-5 mEq/l for every 10 mmHg decrease in  $\text{PaCO}_2$ .

### ★ Step 4:

**After determining the primary abnormality, check for compensation:**

**Compensation is the mechanism by which the body adapts to either acidosis or alkalosis, it will not fully correct the abnormality, For example:**

- A patient has diabetic ketoacidosis, pH is 7.29,  $\text{HCO}_3$  is 15
- Expected  $\text{PCO}_2$  by using Winter's formula
- $\text{PCO}_2 = 1.5 \times \text{HCO}_3 + 8 (\pm 2)$   
 $= 1.5 \times 15 + 8 = 30.5$
- So you expect the  $\text{PCO}_2$  in this patient to be in the range of 28.5– 32.5
- If the  $\text{PCO}_2$  in this patient is higher than 32.5 → consider additional respiratory acidosis
- If the  $\text{PCO}_2$  in the patient is lower than 28.5 → consider additional respiratory alkalosis

### Compensation calculations:

Memorize one compensation equation for each acid base abnormality.

Example: If patient has metabolic acidosis, calculate PCO<sub>2</sub>, Higher or lower than expected means it is Mixed.

If the PCO<sub>2</sub> in this patient is high → (mixed metabolic and respiratory acidosis)

If the PCO<sub>2</sub> in the patient is low → mixed metabolic acidosis respiratory alkalosis

Primary Disorder	Expected Compensation
Metabolic Acidosis	$\downarrow PaCO_2 = 1.2 \times \Delta HCO_3$ or $PaCO_2 = 1.5 \times HCO_3 + 8 \pm 2$ or $PaCO_2 \sim$ last two digits of pH.
Metabolic Alkalosis	$\uparrow PaCO_2 = 0.7 \times \Delta HCO_3$
Acute Respiratory Acidosis	$\uparrow HCO_3 = 0.1 \times \Delta PaCO_2$
Chronic Respiratory Acidosis	$\uparrow HCO_3 = 0.35 \times \Delta PaCO_2$ also $\downarrow pH = 0.003 \times \Delta PaCO_2$
Acute Respiratory Alkalosis	$\downarrow HCO_3 = 0.2 \times \Delta PaCO_2$
Chronic Respiratory Alkalosis	$\downarrow HCO_3 = 0.4 \times \Delta PaCO_2$

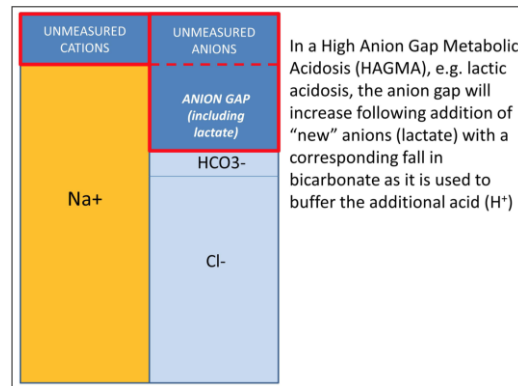
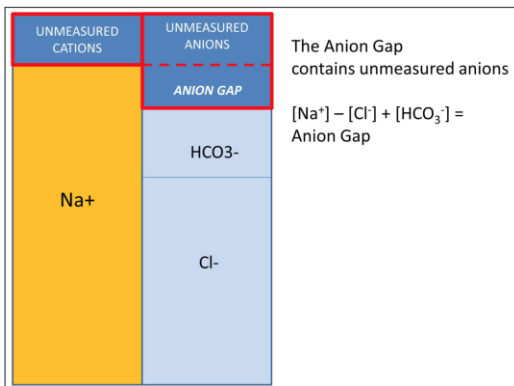
### ★ Step 5:

**Calculate the anion gap (AG):**  
 $AG = Na - (Cl + HCO_3)$

Albumin is the main unmeasured anion.

To overcome the effects of the hypoalbuminemia on the AG, the corrected AG can be used which is  $AG + (0.25 \times (40 - \text{albumin}))$  expressed in g/L.

when we see increase in anion gap that's mean there's additional acids like lactic acid and keto acid.



**Metabolic acidosis with normal anion gap suggests:**

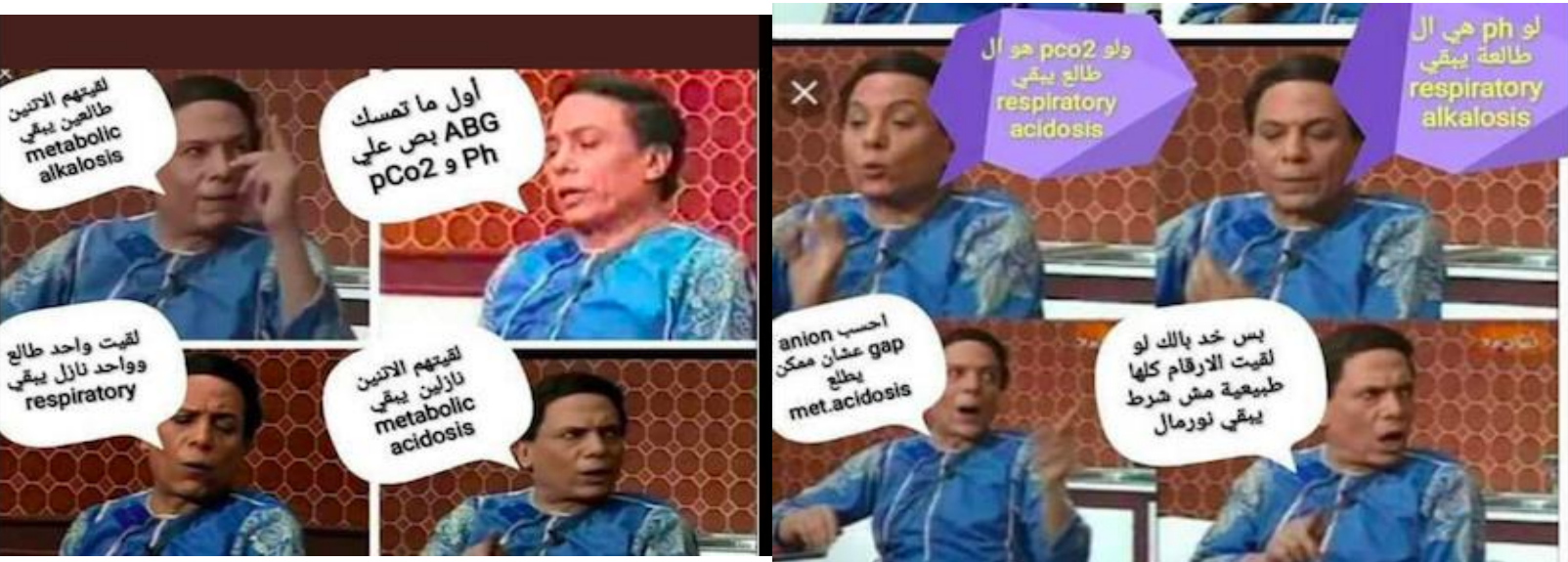
Normal anion gap
Gastrointestinal losses of bicarbonate
Renal tubular acidosis
Treatment with carbonic anhydrase inhibitors
Urinary diversion procedures
Excessive administration of 0.9% saline

**For a metabolic acidosis, is there an increased anion gap?**

- Anion gap = [Sodium] - ([Chloride] + [Bicarbonate]) (normal AG 8-12)
- Serum Osmolality = (2 x (Na + K)) + (BUN) + (glucose)

**Are there other metabolic processes present in a patient with an increased anion gap metabolic acidosis?**

to simplify things:





## Important Cases from the Doctor's Slides

### ★ Normal reference range

- pH (7.35-7.45), PCO<sub>2</sub> (35-45 mmHg), PO<sub>2</sub> (82-105 mmHg), HCO<sub>3</sub> (22-26 mmol/L), AG (8-12)
- Creatinine (40-110 umol/L), Urea (2.5-7.8 mmol/L), Na (136-145 mmol/L), K (3.5-5 mmol/L)

### ★ Case 1

**A 75-year-old man is admitted with septic shock. Shortly after admission, blood tests reveal the following:**

- pH 7.18, PO<sub>2</sub>= 150 mmHg, PaCO<sub>2</sub>= 16 mmHg, HCO<sub>3</sub> 7 mmol/L
- Na 138 mmol/L, K 3.9 mmol/L, Cl 95 mmol/L, Urea 8.2 mmol/L, Creatinine 102 μmol/L
- Please identify the acid base disturbance :

**1. The disturbance is metabolic acidosis.** Why? Because the pH is less than the normal range (7.35-7.45) and the bicarbonate is less than 22 mmol/L.

**2. Is the body properly compensating?**

$1.5 \times 7 + 8 = 18.5 \pm 2$ . The patient's value is 16 Which almost falls within the range, that means that the metabolic acidosis is being compensated properly with respiratory alkalosis.

**3. Anion gap:**

$$\text{Na} - (\text{Cl} + \text{HCO}_3) = 138 - (95 + 7) = 36$$

The anion gap is higher than normal. which indicates **metabolic acidosis**.

- Please indicate what is causing the acid base disturbance  
The cause is Lactic acidosis because which associated with shock.

### ★ Case 2

**A 68-year-old woman is being treated for congestive heart failure in the coronary care unit. After several days of treatment, the following results are returned:**

- pH 7.49, PO<sub>2</sub>= 86 mmHg, PaCO<sub>2</sub>= 48.5 mmHg, HCO<sub>3</sub> 39 mmol/L
- Na 142 mmol/L, K 3.0 mmol/L, Cl 85 mmol/L, Urea 9.3 mmol/L, Creatinine 84 μmol/L
- Please identify the acid base disturbance

**1. The disturbance is metabolic alkalosis.** Why? Because the pH is higher than the normal range (7.35-7.45) and the bicarbonate is also higher than 26 mmol/L.

**2. Is the body properly compensating?**

Here the calculation is different:

$0.7 \times (39 - 24) = 10.5 \rightarrow (10.5 + 40) = 50.5 \pm 2$  (this is the estimated increase in PaCO<sub>2</sub>). So it should be between 48.5-52.2 mmHg which means that the metabolic alkalosis is compensating properly with respiratory acidosis.

**3. Anion gap:**

$$\text{Na} - (\text{Cl} + \text{HCO}_3) = 142 - (85 + 39) = 18$$

**The anion gap is higher than normal here so the patient might have metabolic acidosis as well. but the doctor said to neglect it in this case for simplicity\***

- Please indicate what is causing the acid base disturbance.  
The cause of alkalosis here is the use of Diuretics (diuretics decrease blood volume so as a response to that, the kidneys increase reabsorption of sodium bicarbonate)

### ★ Case 3

**A 70-year-old man with chronic obstructive pulmonary disease (COPD) is admitted with increasing confusion. Shortly after admission, blood tests reveal the following:**

- pH 7.21, PO 61.5 mmHg, PaCO<sub>2</sub> 83 mmHg, HCO<sub>3</sub> 34 mmol/L
- Na 140 mmol/L, K 4.7 mmol/L, Cl 94 mmol/L Urea 8.2 mmol/L, Creatinine 66 μmol/L
- Please identify the acid base disturbance

**1. The disturbance is respiratory acidosis.** Why? Because the pH is less than the normal range (7.35-7.45) and the PaCO<sub>2</sub> is also significantly higher than 40 mmHg.

2. Is the body properly compensating?

First we have to determine whether the cause is Acute or Chronic, this mainly depends on the clinical scenario. Which means that if a patient presents with a stroke 2 or 3 hours ago and he cannot breathe so he developed Respiratory acidosis, this is Acute. A chronic scenario (like the case mentioned here) where the patient has COPD and he chronically retains CO<sub>2</sub> which eventually lead to respiratory acidosis.

$0.35 \times (83-40) = 15 \rightarrow (15+24=39 \pm 2)$  which means that 37-41mmol/L is the expected HCO<sub>3</sub>. The doctor said this was a tough call because it's close to the expected range but if you want to go by the book it means that the patient's body isn't compensating properly. So, the patient has **respiratory acidosis** with **metabolic acidosis**.

3. Anion gap:

$Na - (Cl+HCO_3) = 140 - (94+34) = 12$  (normal anion gap)

- Please indicate what is causing the acid base disturbance.  
The cause here is CO<sub>2</sub> retention caused by COPD  
(CO<sub>2</sub> accumulation may itself lead to drowsiness That further depresses respiratory drive.)

### ★ Case 4

**A 40-year-old man developed profuse diarrhea following antibiotic treatment of a chest infection. He is thirsty, and light headed. Shortly after admission, blood tests reveal the following:**

- pH 7.25, PO 101 mmHg, PaCO<sub>2</sub> 31.5 mmHg, HCO<sub>3</sub> 17 mmol/L
- Na 134 mmol/L, K 3.4 mmol/L, Cl 104 mmol/L, Urea 9.3 mmol/L, Creatinine 102 μmol/L
- Please identify the acid base disturbance

**1. The disturbance is metabolic acidosis.** Why? Because the pH is less than the normal range (7.35-7.45) and the bicarbonate is less than 22 mmol/L.

2. Is the body properly compensating?

The calculation in this case is similar to the first case

$1.5 \times 17 + 8 = 33.5 \pm 2$  mmHg ( so, expected PaCO<sub>2</sub> should be between 31.5-35.5) so the body here is compensating properly with respiratory alkalosis.

3. Anion gap:

$Na - (Cl+HCO_3) = 134 - (104+17) = 13$  (normal anion gap metabolic acidosis)

- Please indicate what is causing the acid base disturbance.  
The cause here is **diarrhea**, which is one of the causes of metabolic acidosis with a normal anion gap. Because the body retains chloride in response to the loss of bicarbonate through the GIT.



# Summary

	Metabolic Acidosis	Metabolic Alkalosis
Underlying cause	<b>How to determine the underlying cause?</b> By the <b>anion gap</b> .	<b>How to determine the underlying cause?</b> By saline responsiveness.
	<b>Normal AG Acidosis:</b> <b>Diarrhea</b> , Renal tubular acidosis.	<b>Saline Responsive Urine (cl-) &lt;20</b> with ECF contraction (due to fluid loss). Vomiting & diarrhea of high Cl (HCl loss), Diuretic (decreased ECF volume)
	<b>High AG Acidosis</b> <b>MUD PILES:</b> Methanol, Uremia, <b>DKA</b> , Propylene glycol, INH, Lactic acidosis (or diabetic ketoacidosis), Ethanol/Ethylene Glycol, Salicylates	<b>Saline resistant Urine (cl-) &gt;20</b> with ECF volume expansion (no fluid loss) <b>Hypertensive:</b> Hyperaldosteronism (potassium loss) Cushing Syndrome <b>Hypo/normo tensive:</b> Exogenous alkali load
Signs and symptoms	<b>Hyperventilation</b> Decreased in Cardiac output and tissue perfusion.	There are no characteristic signs and symptoms (most imp. Hx)
Treatment	Treat the underlying cause. Sodium bicarbonate for severe acidosis (esp. in <b>normal AG</b> acidosis). Mechanical ventilation if the patient is fatigued (esp. in <b>DKA</b> )	Treat the underlying cause. Give normal saline + potassium in <b>saline responsive</b> . Spironolactone (K+ sparing diuretic) in <b>saline resistant</b> .
	Respiratory Acidosis	Respiratory Alkalosis
Underlying cause	<b>Acute Respiratory Acidosis:</b> airway obstruction, pneumonia, pneumothorax, chest trauma, head trauma, <b>stroke</b> , drug toxicity	<b>Hyperventilation</b>  <b>most common Causes of hyperventilation:</b> <b>Anxiety, Fever</b>
	<b>Chronic Respiratory Acidosis:</b> <b>COPD</b> , morbid obesity, muscular and neuromuscular diseases	
Signs and symptoms	Drowsiness, confusion, headaches, flapping tremors.	Dizziness, anxiety, paresthesia, and perioral numbness. Tetany, Arrhythmias, Trousseau's sign & Chvostek's sign
Treatment	Treat the underlying cause. Give supplemental oxygen except in patients with COPD. Some cases require intubation and mechanical ventilation.	Treat the underlying cause. Breathing into a paper bag can help patients with anxiety.

## Questions

**1- In a man undergoing surgery, it was necessary to aspirate the contents of the upper gastrointestinal tract. After surgery, the following values were obtained from an arterial blood sample: pH 7.55, PCO<sub>2</sub> 52 mm Hg and HCO<sub>3</sub> - 40 mmol/l. What is the underlying disorder?**

- A. Metabolic Acidosis
- B. Metabolic Alkalosis
- C. Respiratory Acidosis
- D. Respiratory Alkalosis

**2- A student is nervous for a big exam and is breathing rapidly, what do you expect out of the followings**

- A. Metabolic Acidosis
- B. Metabolic Alkalosis
- C. Respiratory Acidosis
- D. Respiratory Alkalosis

**3- Which of the following laboratory results below indicates compensated metabolic alkalosis?**

- A. Low pCO<sub>2</sub> normal bicarbonate and, high pH
- B. Low pCO<sub>2</sub>, low bicarbonate, low pH
- C. High pCO<sub>2</sub>, normal bicarbonate and, low pH
- D. High pCO<sub>2</sub>, high bicarbonate and High pH

**4- A young woman is found comatose, having taken an unknown number of sleeping pills an unknown time before. An arterial blood sample yields the following values: pH – 6.90, HCO<sub>3</sub> - 13 meq/liter, PaCO<sub>2</sub> 68 mmHg. This patient's acid-base status is most accurately described as which of the following?**

- A. Uncompensated metabolic acidosis
- B. Uncompensated respiratory acidosis
- C. simultaneous respiratory and metabolic acidosis
- D. Respiratory acidosis with partial renal compensation

1.B

2.D

3.D

4.C