

# Acid-Base disorders

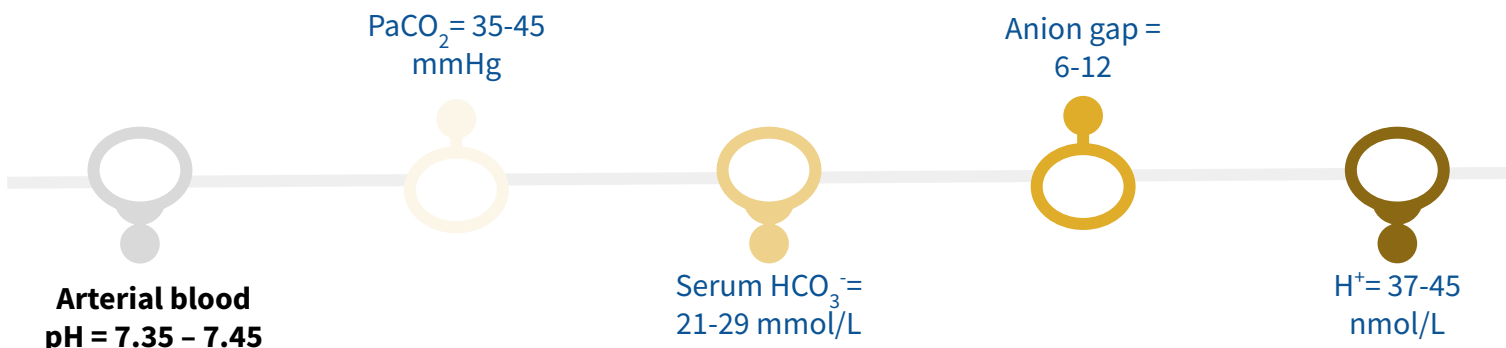
## Objectives:

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

## Color index:

Original text Females slides Males slides  
Doctor's notes Textbook Important Golden notes Extra

## Normal values



## Basic recall

### Definition

Acid-base balance is concerned with maintaining a normal hydrogen ion concentration in the body fluids. This balance is achieved by utilization of buffers 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>
- **Assessment of acid base abnormalities:** 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**
- The clinical picture is often dominated by the underlying cause rather than the acid-base abnormality itself
- Always check the reference range in your local laboratory.

## ➤ Primary disturbance:

Primary disorder	Respiratory acidosis	Respiratory alkalosis	Metabolic acidosis	Metabolic alkalosis
<b>Problem</b>	Hypoventilation	Hyperventilation	Gain of H <sup>+</sup> or loss of HCO <sub>3</sub> <sup>-</sup>	Gain of HCO <sub>3</sub> <sup>-</sup> or loss of H <sup>+</sup>
<b>pH</b>	↓	↑	↓	↑
<b>HCO<sub>3</sub><sup>-</sup></b>	↑	↓	↓↓	↑↑
<b>PaCO<sub>2</sub><sup>1</sup></b>	↑↑	↓↓	↓	↑

1: PCO<sub>2</sub> does not rise above 55 mmHg because hypoxia then intervenes to drive respiration

## Definition

Increased  $\text{PaCO}_2$  and decreased pH

## Mechanism

- Process that primarily causes **elevation** in  $\text{PaCO}_2$ .
- Reduce effective ventilation e.g. many **chronic respiratory diseases (COPD)** or drugs depressing the respiratory center.
- Alveolar Hypoventilation → **Accumulation** of  $\text{CO}_2$  → Increases in  $\text{PaCO}_2$  → Respiratory acidosis → pH decreases.
- $\text{HCO}_3^-$  will increase (Compensation) but it needs time (12 -24 h) as the kidney need time to compensate

## Clinical features

- Signs of acute  $\text{CO}_2$  retention: headaches, confusion, and papilledema, flapping tremors

## Classification

	Acute Respiratory Acidosis	Chronic Respiratory Acidosis
<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>	Low	Almost normal due compensatory mechanism.
<b>Compensation</b>	<ul style="list-style-type: none"> <li>• Immediate renal compensatory ↑ of <math>\text{HCO}_3^-</math>.</li> <li>• <math>\text{HCO}_3^-</math> ↑ by 1 mEq/l for every 10 mmHg ↑ in <math>\text{PaCO}_2</math>.</li> </ul>	$\text{HCO}_3^-$ ↑ by 3-3.5 mEq/l for every 10 mmHg ↑ in $\text{PaCO}_2$ (Due to renal adaptation)

## Treatment:

- Verify patency of airways.
- Give supplemental oxygen: If  $\text{PaO}_2$  is low (<60 mmHg), Oxygen is contraindicated in COPD patients ( $\text{CO}_2$  retention) as it can exacerbate symptoms.
- Treat underlying cause.
- Intubation and mechanical ventilation might be required for:
  - Severe acidosis.
  - $\text{PaCO}_2 > 60$  or inability to increase  $\text{PaO}_2$ .
  - Mental deterioration.
  - Impending respiratory fatigue.

# Respiratory Alkalosis

## Definition

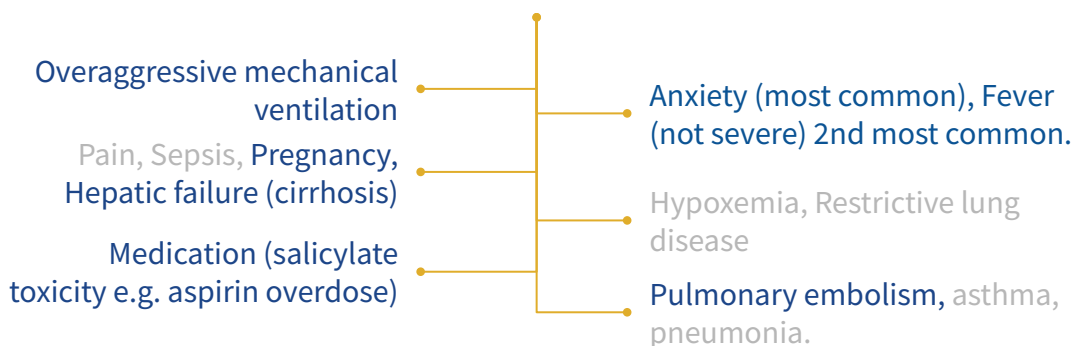
Decreased  $\text{PaCO}_2$  and increased pH.

## Mechanism

- Process that primarily causes **reduction** in  $\text{PaCO}_2$
- Increase ventilation e.g. in response to hypoxia or secondary to metabolic acidosis.
- Alveolar hyperventilation → increased wash out  $\text{CO}_2$  → decrease in  $\text{PaCO}_2$  → increased pH.
- Compensation:  $\text{HCO}_3^-$  will decrease after (12 -24 h).

## Etiology

### Hyperventilation of any Cause



## Clinical Features:

lightheadedness, dizziness, anxiety, paresthesia, and perioral numbness

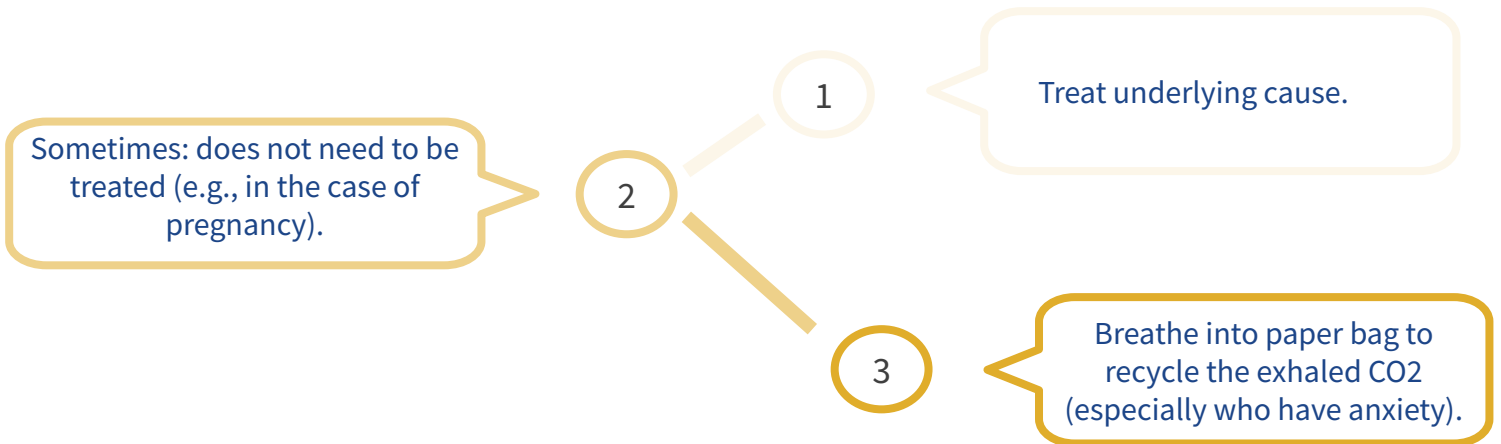
Tetany, Arrhythmias, Trousseau's sign<sup>1</sup> and Chvostek's sign<sup>1</sup> may be positive

## Classification

Acute Respiratory Alkalosis	Chronic Respiratory Alkalosis
$\text{HCO}_3^- \downarrow$ by 2 mEq/l for every 10 mmHg $\downarrow$ in $\text{PaCO}_2$	$\text{HCO}_3^- \downarrow$ by 4-5 mEq/l for every 10 mmHg $\downarrow$ in $\text{PaCO}_2$ .

1: alkalosis promotes the binding of calcium to albumin, resulting in a reduction in ionised calcium concentrations

## ◀ Treatment



# Metabolic Acidosis

## Definition

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

## ◀ Mechanism

- Process that primarily **reduced 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.
- **Compensation: 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

## ◀ The Anion gap<sup>1</sup>:

- 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 →  $AG = ([Na^+] + [K^+]) - ([Cl^-] + [HCO_3^-])$
  - Normal range is about 5-11 mmol/L
- It is helpful in determining the cause of a **metabolic acidosis**

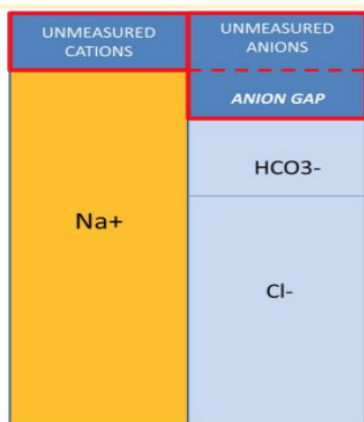
1: This gap is normally made up of anions, such as phosphate and sulphate, as well as albumin.

## Classification & Etiology:

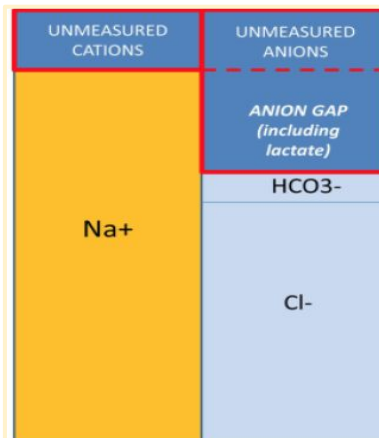
### 1-High Anion gap acidosis

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

Increased Endogenous production of anions distinct from Cl <sup>-</sup> & HCO <sub>3</sub> <sup>-</sup>	
<p>★ <b>Lactic acidosis</b> Plasma lactate &gt; 2 mmol/L</p>	<p>Type I: Tissue hypoxia &amp; peripheral generation of lactate (circulatory failure &amp; shock (<b>septic</b>, cardiogenic, hypovolemic)) Type II: Impaired metabolism of lactate (liver disease, metformin<sup>1</sup>)</p>
<p><b>Diabetic Ketoacidosis</b><sup>2</sup></p>	<p>★ <b>DKA</b>: caused by insulin deficiency &amp; exacerbated by catecholamine &amp; stress hormone excess → lipolysis → formation of acidic ketones (acetoacetate, 3-hydroxybutyrate, acetone) Other causes of ketoacidosis:</p> <ul style="list-style-type: none"> <li>★ <b>Starvation ketoacidosis</b>: ↓ food intake in situations of high glucose demand e.g. neonates, pregnant &amp; breastfeeding women</li> <li>★ <b>Alcoholic ketoacidosis</b>: chronic malnutrition &amp; recent alcohol binge</li> </ul>
<p><b>Uremia</b></p>	<p>Renal failure → ↓ NH<sub>4</sub><sup>+</sup> &amp; H<sup>+</sup> excretion, decreased excretion of organic anions, sulfates, and phosphates.</p>
<p><b>INH</b></p>	<p>Impaired hepatic clearance of lactate</p>
Increased Exogenous Intake	
<p><b>Ethanol/Ethylene glycol poisoning</b></p>	<p>Accumulation of glycolate, calcium oxalate crystals</p>
<p><b>Methanol poisoning</b></p>	<p>Manifested as visual complaints</p>
<p><b>Propylene glycol</b> (not paraldehyde)</p>	<p>is metabolized to lactic acid (lactate) and has the potential to cause a high anion gap metabolic acidosis</p>
<p><b>Aspirin poisoning</b><sup>3</sup></p>	<p>Accumulation of <b>Salicylates</b></p>



The Anion Gap contains unmeasured anions  
 $[Na^+] - [Cl^-] + [HCO_3^-] =$   
 Anion Gap



In a High Anion Gap Metabolic Acidosis (HAGMA), e.g. lactic acidosis, the anion gap will increase following addition of “new” anions (lactate) with a corresponding fall in bicarbonate as it is used to buffer the additional acid (H<sup>+</sup>)

1: Inhibit lactate metabolism

2: Treat with insulin

3: salicylate overdose may cause both primary metabolic acidosis and primary respiratory alkalosis. Treat by removal of salicylate by dialysis

## Classification & Etiology:

### 2-Normal Anion Gap Acidosis

- $\text{HCO}_3^-$  decreases and is replaced by  $\text{Cl}^-$  to maintain electroneutrality. Consequently, these disorders are sometimes referred to collectively as **hyperchloraemic acidoses**.

↑ GI $\text{HCO}_3^-$ loss	<b>Diarrhea</b> <sup>1</sup> , small bowel fistula, pancreatic fistula, <b>urinary diversion procedure</b> , ileostomy, ureterosigmoidostomy
↑ Renal $\text{HCO}_3^-$ loss	<ul style="list-style-type: none"> <li>• Type II (proximal) RTA<sup>2</sup></li> <li>• hyperparathyroidism</li> <li>• tubular damage e.g. drugs, heavy metals, paraproteins</li> <li>• <b>Treatment with carbonic anhydrase inhibitors: Acetazolamide therapy</b></li> </ul>
↓ Renal $\text{H}^+$ excretion	<ul style="list-style-type: none"> <li>• Type I (classical distal) RTA, Type IV RTA (aldosterone deficiency<sup>3</sup>)</li> <li>• CKD</li> </ul>
↑ HCl production	<ul style="list-style-type: none"> <li>• Ammonium chloride ingestion, ↑ catabolism of lysine, arginine</li> <li>• <b>Excessive administration of 0.9% saline</b></li> </ul>

## Clinical Features:

1- **Hyperventilation** (deep rhythmic breathing) also called **Kussmaul respiration**.

2- Tissue malfunction such as altered cardiac & central nervous system

## Treatment:

**1** Identify & correct the underlying cause

**2** IV bicarbonate<sup>4</sup> is best reserved for severe acidosis or evidence of tissue dysfunction

**3** Mechanical ventilation might be needed if the patient is fatigued (esp. in DKA)

1: Most common cause of normal AG metabolic acidosis

2- further discussed in [the next page](#).

3- hypoaldosterone status could be due to: Addison's disease, spironolactone, amiloride, triamterene.

4- needed especially in normal AG metabolic acidosis

## Renal tubular acidosis (RTA):



- RTA should be suspected when there is a hyperchloraemic acidosis with a normal anion gap in the absence of gastrointestinal disturbance.
- Plasma  $\text{HCO}_3^- < 21$  mmol/L, urine pH  $> 5.3$  = RTA
- Confirmed by acid load test

### Type I (classical distal) RTA

- **Impaired acid secretion in late distal tubule of cortical collecting duct intercalated cells**
- Consists of: acidosis, hypokalemia, Inability to lower the urine pH below 5.3 despite systemic acidosis, Low urinary ammonium production, Low urinary citrate (owing to increased citrate absorption in the proximal tubule where it can be converted to bicarbonate), Hypercalciuria.
- **Treatment:** sodium bicarbonate, potassium supplements and citrate. Thiazide diuretics are useful by causing volume contraction and increased proximal sodium bicarbonate reabsorption.

### Type II (proximal) RTA

- **Impaired  $\text{HCO}_3^-$  reabsorption in proximal tubule**
- The cardinal features are acidosis, hypokalaemia, an inability to lower the urine pH below 5.5 despite systemic acidosis, and the appearance of bicarbonate in the urine despite a subnormal plasma bicarbonate.
- **Treatment:** sodium bicarbonate: massive doses may be required to overcome the renal 'leak'.

### Type IV RTA

- Also called '**hyporeninaemic hypoaldosteronism**'
- **impaired sodium reabsorption in the late distal tubule or cortical collecting duct, which is associated with reduced secretion of both  $\text{K}^+$  and  $\text{H}^+$  ions**
- The cardinal features are hyperkalaemia and acidosis occurring in a patient with mild chronic kidney disease
- **Treatment:** fludrocortisone, sodium bicarbonate, diuretics, or ion exchange resins to remove potassium, or a combination of these.



## Definition:

Addition of  $[HCO_3^-]$  or loss of  $[H^+]$  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  $HCO_3^-$ .
  - Loss of acid ( $H^+$ ) ex: from vomiting.
- Maintaining Metabolic alkalosis due to the kidney inability to excrete the excess  $HCO_3^-$
- Compensation: Hypoventilation  $\rightarrow$  increased  $PCO_2$  (respiratory Acidosis) immediately ( $PaCO_2 \uparrow$  by 0.6 mmHg for every 1 mEq/l  $\uparrow$  in  $HCO_3^-$ ).

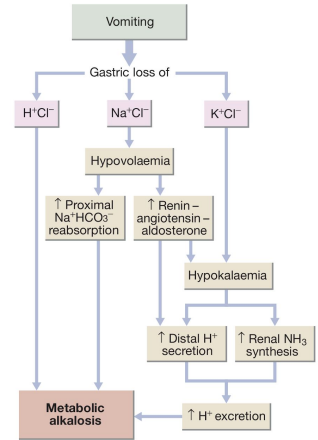


Fig. 14.11 Generation and maintenance of metabolic alkalosis during prolonged vomiting. Loss of  $H^+Cl^-$  generates metabolic alkalosis, which is maintained by renal changes.

## Clinical Features:

Tetany, apathy, confusion, drowsiness, cardiac arrhythmias & neuromuscular irritability are common when alkalosis is severe

## Classification & causes:

	Hypovolemic (Saline sensitive, urine $Cl^- < 10$ mEq/L)	Normovolemic (Saline resistant, urine $Cl^- > 20$ mEq/L)
<b>Definition</b>	Metabolic alkalosis with ECF contraction (due to $\rightarrow$ fluid loss).	Metabolic alkalosis with ECF volume expansion (no fluid loss)
<b>Causes</b>	<ul style="list-style-type: none"> <li>• Gastric loss of <math>H^+</math> (sustained vomiting)</li> <li>• Diuretic use: loop or thiazide.</li> <li>• Volume depletion</li> <li>• Post-hypercapnia</li> <li>• Villous adenoma of colon, diarrhea with high chloride content</li> </ul>	<ul style="list-style-type: none"> <li>• Hypertensive:                             <ul style="list-style-type: none"> <li>○ Primary Hyperaldosteronism</li> <li>○ Cushing Syndrome</li> <li>○ Glucocorticoid therapy</li> </ul> </li> <li>• Hypo/normo tenseive:                             <ul style="list-style-type: none"> <li>○ Bicarbonate ingestion: massive or with kidney disease</li> <li>○ Bartter's syndrome &amp; Gitelman's syndrome, Severe hypokalemia</li> </ul> </li> </ul>
<b>Treatment</b>	intravenous infusions of 0.9% saline with potassium supplements	management of the underlying cause

## 1 Step 1 History & physical examination

### look for clues that may lead to the abnormalities in pH

- Vomiting: causes loss of acid and gastric contents, which suggests development of alkalosis
- Diarrhea
- Hypoventilation
- Respiratory disease
- Medications (laxatives, diuretics, etc)
- Diabetes

## 2 Step 2 Look at the pH

### Determine if it is

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

## 3 Step 3a 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**)

**Note:** Compensation will not return the pH to the normal range, it's just a mechanism which the body trying to reduce the impact.

## 3 Step 3b If pH is normal, that doesn't rule out mixed acidosis and alkalosis (Determine what is being mixed<sup>1</sup>)

- Look for **high** or **low** PCO<sub>2</sub>= **Low** PCO<sub>2</sub> suggests **respiratory alkalosis**/**High** PCO<sub>2</sub> suggests **respiratory acidosis**
- Look for **high** or **low** HCO<sub>3</sub>= **Low** HCO<sub>3</sub> suggests **metabolic acidosis**/**High** HCO<sub>3</sub> suggests **metabolic alkalosis**

### How to determine Is the respiratory disturbance acute or chronic?

- **Acute respiratory acidosis:** HCO<sub>3</sub> **increase** by 1 mEq/l for every 10 mmHg **increase** in PaCO<sub>2</sub>.
- **Chronic respiratory acidosis:** HCO<sub>3</sub> **increase** by 3-3.5 mEq/l for every 10 mmHg **increase** in PaCO<sub>2</sub>.
- **Acute respiratory alkalosis:** HCO<sub>3</sub> **decrease** by 2 mEq/l for every 10 mmHg **decrease** in PaCO<sub>2</sub>.
- **Chronic respiratory alkalosis:** HCO<sub>3</sub> **decrease** by 4-5 mEq/l for every 10 mmHg **decrease** in PaCO<sub>2</sub>.

1: Sometimes you may have mixed diseases ( Metabolic Alkalosis AND Respiratory Acidosis )

## 4 Step 4

### check for compensation

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

example:

- A patient has **diabetic ketoacidosis**, pH is 7.29, HCO<sub>3</sub> is 15 (hence, it is metabolic acidosis)
- Use the metabolic acidosis formula: Expected PCO<sub>2</sub> by using Winter's formula  $PCO_2 = 1.5 \times HCO_3 + 8 (\pm 2^1) = 1.5 \times 15 + 8 = 30.5$
- So: you expect the PCO<sub>2</sub> in this patient to be in the range of **28.5– 32.5<sup>3</sup>**
- Now, determine whether there is a compensation or an additional disorder:
  - If the PCO<sub>2</sub> in this patient is **higher than 32.5** → consider additional<sup>2</sup> respiratory acidosis
  - If the PCO<sub>2</sub> in the patient is **lower than 28.5** → consider additional respiratory alkalosis

### Compensation calculation

Primary disorder		Expected compensation
Metabolic acidosis		<ul style="list-style-type: none"> <li>• <math>PaCO_2 = 1.5 \times HCO_3 + 8 \pm 2</math></li> <li>• <math>\downarrow PaCO_2 = 1.2 \times \Delta HCO_3</math></li> <li>• <math>PaCO_2 \sim</math> last two digits of pH</li> </ul>
Metabolic alkalosis		<ul style="list-style-type: none"> <li>• <math>\uparrow PaCO_2 = 0.7 \times \Delta HCO_3</math></li> </ul>
Respiratory acidosis	Acute	<ul style="list-style-type: none"> <li>• <math>\uparrow HCO_3 = 0.1 \times \Delta PaCO_2</math></li> </ul>
	Chronic	<ul style="list-style-type: none"> <li>• <math>\uparrow HCO_3 = 0.35 \times \Delta PaCO_2</math></li> <li>• <math>\downarrow pH = 0.003 \times \Delta PaCO_2</math></li> </ul>
Respiratory alkalosis	Acute	<ul style="list-style-type: none"> <li>• <math>\downarrow HCO_3 = 0.2 \times \Delta PaCO_2</math></li> </ul>
	Chronic	<ul style="list-style-type: none"> <li>• <math>\downarrow HCO_3 = 0.4 \times \Delta PaCO_2</math></li> </ul>

## 5 Step 5

### Calculate the anion gap

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

- Normal anion gap = 6-12<sup>4</sup>
- Albumin is the main unmeasured anion. To overcome the effects of hypoalbuminemia on the AG, the corrected AG can be used which is **AG + (0.25 X (40-albumin))** expressed in g/L.
- An increase in anion gap that means there's **additional acids** like lactic acid and keto acid.
- Get back to pages to check for high AG metabolic acidosis vs normal AG metabolic acidosis

1: gives you a range

2: Please make sure that you differentiate between additional and compensated.

3: Memorize one compensation equation for each acid base abnormality. Example:

-If the PCO<sub>2</sub> of this patient was 35, then the patient's acid-base status will be : Metabolic Acidosis **AND** Respiratory Acidosis.

-If the PCO<sub>2</sub> of this patient was 30, then the patient's acid-base status will be : Metabolic Acidosis **Compensated by** Respiratory Alkalosis. .

4: The normal range is up to 14. It is Especially important in Metabolic Acidosis, crucial for the differential diagnosis.

## Case study 1:

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

	Case	Normal range
pH	<b>7.18</b>	7.35-7.45
PO <sub>2</sub>	150 mmHg	82-105 mmHg
PaCO <sub>2</sub>	<b>16 mmHg</b>	35-45 mmHg
HCO <sub>3</sub>	<b>7 mmol/L</b>	22-26 mmol/L
Na <sup>+</sup>	138 mmol/L	136-145 mmol/L
K <sup>+</sup>	3.9 mmol/L	3.5-5 mmol/L
Cl <sup>-</sup>	95 mmol/L	
Urea	8.2 mmol/L	2.5-7.8 mmol/L
Creatinine	102 μmol/L	40-110 umol/L

- **Identify the acid-base disturbance.**

- **Metabolic acidosis**

- **Check whether the patient has compensation/additional disturbance.**

- **Choose the formula**

$$\text{PaCO}_2 = 1.5 \times \text{HCO}_3 + 8 \pm 2$$

- **Substitute the values**

$$\text{PaCO}_2 = 1.5 \times 7 + 8 \pm 2$$

$$\text{PaCO}_2 = 18.5 \pm 2$$

$$\text{PaCO}_2 = (16.5 - 20.5)$$

- **Interpret the result**

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.

- **Calculate the anion gap**

$$\text{AG} = \text{Na} - (\text{Cl} + \text{HCO}_3)$$

$$\text{AG} = 138 - (95 + 7) = 36 \text{ (high)}$$

- **Indicate what is causing the acid base disturbance?**

- Lactic acidosis (associated with shock)

## Case study 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:

	Case	Normal range
pH	<b>7.49</b>	7.35-7.45
PO <sub>2</sub>	86 mmHg	82-105 mmHg
PaCO <sub>2</sub>	<b>48.5 mmHg</b>	35-45 mmHg
HCO <sub>3</sub>	<b>39 mmol/L</b>	22-26 mmol/L
Na <sup>+</sup>	142 mmol/L	136-145 mmol/L
K <sup>+</sup>	3 mmol/L	3.5-5 mmol/L
Cl <sup>-</sup>	85 mmol/L	
Urea	9.3 mmol/L	2.5-7.8 mmol/L
Creatinine	84 μmol/L	40-110 umol/L

- **Identify the acid-base disturbance.**  
**Metabolic alkalosis**
- **Check whether the patient has compensation/additional disturbance.**
  - **Choose the formula**  
 $\uparrow PaCO_2 = 0.7 \times \Delta HCO_3$
  - **Substitute the values**  
 $\uparrow PaCO_2 = 0.7 \times (39-24)$   
 $\uparrow PaCO_2 = 10.5$
  - **Add to the normal range**  
 $\uparrow PaCO_2 = 40 + 10.5 = 50.5 \pm 2$   
 $\uparrow PaCO_2 = (48.5-52.5)$
  - **Interpret the result**  
the metabolic alkalosis is compensated properly by respiratory acidosis.
- **Indicate what is causing the acid base disturbance?**  
use of **Diuretics** (diuretics decrease blood volume so as a response to that, the kidneys increase reabsorption of sodium bicarbonate)

## Case study 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:

	Case	Normal range
pH	<b>7.21</b>	7.35-7.45
PO <sub>2</sub>	61.5 mmHg	82-105 mmHg
PaCO <sub>2</sub>	<b>83 mmHg</b>	35-45 mmHg
HCO <sub>3</sub>	<b>34 mmol/L</b>	22-26 mmol/L
Na <sup>+</sup>	140 mmol/L	136-145 mmol/L
K <sup>+</sup>	4.7 mmol/L	3.5-5 mmol/L
Cl <sup>-</sup>	94 mmol/L	
Urea	8.2 mmol/L	2.5-7.8 mmol/L
Creatinine	66 μmol/L	40-110 umol/L

- **Identify the acid-base disturbance.**

**Respiratory acidosis** (+ metabolic alkalosis)

- **Check whether the patient has compensation/additional disturbance.**

- **Choose the formula**

Ask yourself, is it Acute or chronic?<sup>1</sup>

$$\uparrow\text{HCO}_3 = 0.1 \times \Delta\text{PaCO}_2$$

- **Substitute the values**

$$\uparrow\text{HCO}_3 = 0.1 \times (83-40)$$

$$\uparrow\text{HCO}_3 = 4.3$$

- **Add to the normal range**

$$\uparrow\text{HCO}_3 = \underline{24 + 4.3} = 28.3 \pm 2$$

$$\uparrow\text{HCO}_3 = (26.3-30.3)$$

- **Interpret the result**

there is an additional metabolic alkalosis on top of the respiratory acidosis<sup>2</sup>

- **Indicate what is causing the acid base disturbance?**

CO<sub>2</sub> retention caused by COPD (CO<sub>2</sub> accumulation may itself lead to drowsiness That further depresses respiratory drive)

1: 437 doctor solved it as chronic, making the additional disturbance a metabolic acidosis. his explanation: 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 leads to respiratory acidosis.

2: When the clinically obtained acid-base parameters do not accord with the predicted compensation shown, a mixed acid-base disturbance should be suspected. For example, a respiratory acidosis due to narcotic overdose with metabolic alkalosis due to vomiting.

## Case study 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:

	Case	Normal range
pH	<b>7.25</b>	7.35-7.45
PO <sub>2</sub>	101 mmHg	82-105 mmHg
PaCO <sub>2</sub>	<b>31.5 mmHg</b>	35-45 mmHg
HCO <sub>3</sub>	<b>17 mmol/L</b>	22-26 mmol/L
Na <sup>+</sup>	134 mmol/L	136-145 mmol/L
K <sup>+</sup>	3.4 mmol/L	3.5-5 mmol/L
Cl <sup>-</sup>	104 mmol/L	
Urea	9.3 mmol/L	2.5-7.8 mmol/L
Creatinine	102 μmol/L	40-110 umol/L

- **Identify the acid-base disturbance.**  
**Metabolic acidosis**
- **Check whether the patient has compensation/additional disturbance.**
  - **Choose the formula**  
 $PaCO_2 = 1.5 \times HCO_3 + 8 \pm 2$
  - **Substitute the values**  
 $PaCO_2 = 1.5 \times 17 + 8 \pm 2$   
 $PaCO_2 = 33.5 \pm 2$   
 $PaCO_2 = (31.5-35.5)$
  - **Interpret the result**  
the metabolic acidosis is compensated properly by respiratory alkalosis.
- **Calculate the anion gap**  
 $AG = Na - (Cl + HCO_3)$   
 $AG = 134 - (104 + 17) = 13$  (normal)
- **Indicate what is causing the acid base disturbance?**  
diarrhea

# Summary

Arterial: 7.35-7.45

Normal pH

Venous: 7.31-7.41

Metabolic Acidosis	Process that primarily <b>reduces bicarbonate</b>	<ol style="list-style-type: none"> <li>1. Excessive H<sup>+</sup> formation: e.g. lactic acidosis, ketoacidosis</li> <li>2. Reduced H<sup>+</sup> excretion: e.g. renal failure</li> <li>3. Excessive HCO<sub>3</sub><sup>-</sup> loss: e.g. diarrhea</li> </ol>
Metabolic Alkalosis	Process that primarily <b>raises bicarbonate</b>	<ol style="list-style-type: none"> <li>1. Extracellular fluid volume loss: e.g. vomiting or diuretics</li> <li>2. Excessive potassium loss with subsequent hyperaldosteronism</li> </ol>
Respiratory Acidosis	Process that primarily causes <b>elevation of PaCO<sub>2</sub></b> (Hypoventilation)	Reduced effective ventilation: e.g. many chronic respiratory diseases or drugs depressing the respiratory system
Respiratory Alkalosis	Process that primarily causes <b>reduction in PaCO<sub>2</sub></b> (Hyperventilation)	Increased ventilation: e.g. in response to hypoxia or secondary to a metabolic acidosis

## Approaching Acid-base Abnormalities

<b>Step 1:</b> History and Physical Examination	Vomiting • Diarrhea • Hypoventilation • Respiratory disease • Medications (laxatives, diuretics, etc) • Diabetes
<b>Step 2:</b> Look at the pH	<ul style="list-style-type: none"> <li>- <b>Normal</b> 7.35 – 7.45 (No abnormality or <b>mixed</b> acidosis and alkalosis)</li> <li>- <b>Low</b> &lt;7.35 (acidemic)</li> <li>- <b>High</b> &gt;7.45 (alkalemic)</li> </ul>
<b>Step 3:</b> a. Determine the primary abnormality that is causing the abnormal pH	<ul style="list-style-type: none"> <li>- If the pH is <b>acidemic</b>, look for: Low HCO<sub>3</sub><sup>-</sup> (Metabolic) or High PCO<sub>2</sub> (Respiratory)</li> <li>- If the pH is <b>alkalemic</b>, look for High HCO<sub>3</sub><sup>-</sup> (Metabolic) or Low PCO<sub>2</sub> (Respiratory)</li> </ul>
b. if pH is normal	<ul style="list-style-type: none"> <li>- Rule out mixed acidosis and alkalosis</li> <li>- Look for high or low PCO<sub>2</sub> and for high or low HCO<sub>3</sub><sup>-</sup></li> </ul>
<b>Step 4:</b> Check for compensation ( <b>imp</b> )	<p>Metabolic Acidosis: PaCO<sub>2</sub> = 1.5 x HCO<sub>3</sub><sup>-</sup> + 8 (±2) Or ↓ PaCO<sub>2</sub> = 1.2 x ΔHCO<sub>3</sub><sup>-</sup></p> <p>Metabolic Alkalosis: ↑PaCO<sub>2</sub> = 0.7x ΔHCO<sub>3</sub><sup>-</sup></p> <p>Acute Respiratory Acidosis: ↑HCO<sub>3</sub><sup>-</sup> = 0.1 x ΔPaCO<sub>2</sub></p> <p>Chronic Respiratory Acidosis: ↑HCO<sub>3</sub><sup>-</sup> = 0.35 x ΔPaCO<sub>2</sub></p> <p>Acute Respiratory Alkalosis: ↓HCO<sub>3</sub><sup>-</sup> = 0.2 x ΔPaCO<sub>2</sub></p> <p>Chronic Respiratory Alkalosis: ↓HCO<sub>3</sub><sup>-</sup> = 0.4 x ΔPaCO<sub>2</sub></p>
<b>Step 5:</b> Calculate the anion gap (AG)	AG = Na - (Cl + HCO <sub>3</sub> <sup>-</sup> )



# Lecture Quiz

**Q1: A 32-year-old builder presents in accident and emergency in a distressed state. He reports suffering from chest pain for the last 2 weeks, the pain is sharp and only occurs when he moves heavy objects. He has a family history of cardiovascular disease and is worried about a heart attack. His blood gas findings are as follows: pH = 7.47; PCO<sub>2</sub> = 3.3; PO<sub>2</sub> = 15.3; bicarbonate = 17.53. The most likely diagnosis is:**

- A. Respiratory acidosis with metabolic compensation**
- B. Acute metabolic acidosis**
- C. Respiratory alkalosis with metabolic compensation**
- D. Metabolic acidosis with respiratory compensation**
- E. Acute respiratory alkalosis**

**Q2: A 22-year-old woman is found unconscious in her room and brought into accident and emergency. A urine dipstick is positive for glucose and ketones and blood analysis shows the following results:**

**pH 6.9 PCO<sub>2</sub> 3.0 kPa PO<sub>2</sub> 13 kPa Sodium 144 mmol/L Potassium 5.0 mmol/L Urea 11 Glucose 20 Chloride 100 Bicarbonate 2.9**

**The most likely anion gap is:**

- A. 180**
- B. 118**
- C. 139.2**
- D. 46.1**
- E. 28**

**Q3: You are informed that one of your ward patients has been breathless over the last hour and has been quite anxious since her relatives left after visiting. The patient is a 67-year-old woman who was admitted 6 days ago for a left basal pneumonia which has responded well with intravenous antibiotics. Her past medical history includes dementia and hypertension. You are asked by your registrar to interpret the patient's arterial blood gas (ABG) measurements taken during her tachypnoea: pH 7.49 kPa, PO<sub>2</sub> 14.1, PCO<sub>2</sub> 3.1 kPa, HCO<sub>3</sub> 24. From the list of answers below, choose the most appropriate ABG interpretation:**

- A. Metabolic alkalosis**
- B. Respiratory alkalosis**
- C. Type 1 respiratory failure**
- D. Respiratory acidosis**
- E. None of the above**

**Q4: The most common cause of hyperventilation:**

- A. Asthma**
- B. Hypoxemia**
- C. Anxiety**
- D. Fever**

**Q5: Which of the following assessments is preferred for obtaining blood gases in children (less painful):**

- A. Arterial blood gases (ABG)**
- B. Venous blood gases (VBG)**
- C. Capillary blood gases (CBG)**
- D. None of the above**

**Q6: 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><sup>-</sup> 13 meq/liter, PCO<sub>2</sub> 68 mmHg. This patient's acid-base status is most accurately described as (From 437 team work):**

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

# THANKS!!

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*Send us your feedback:  
We are all ears!*

