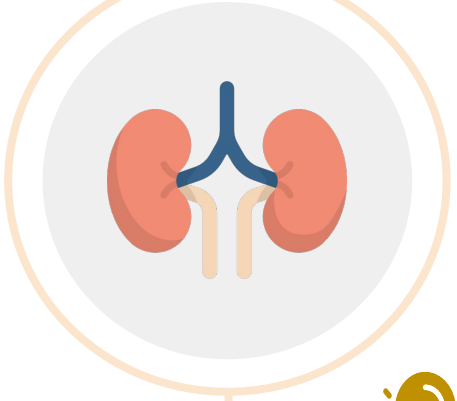
Acid-Base disorders



Online MedEds High-Yield Brilliant Summary!

Objectives :

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

Editing file

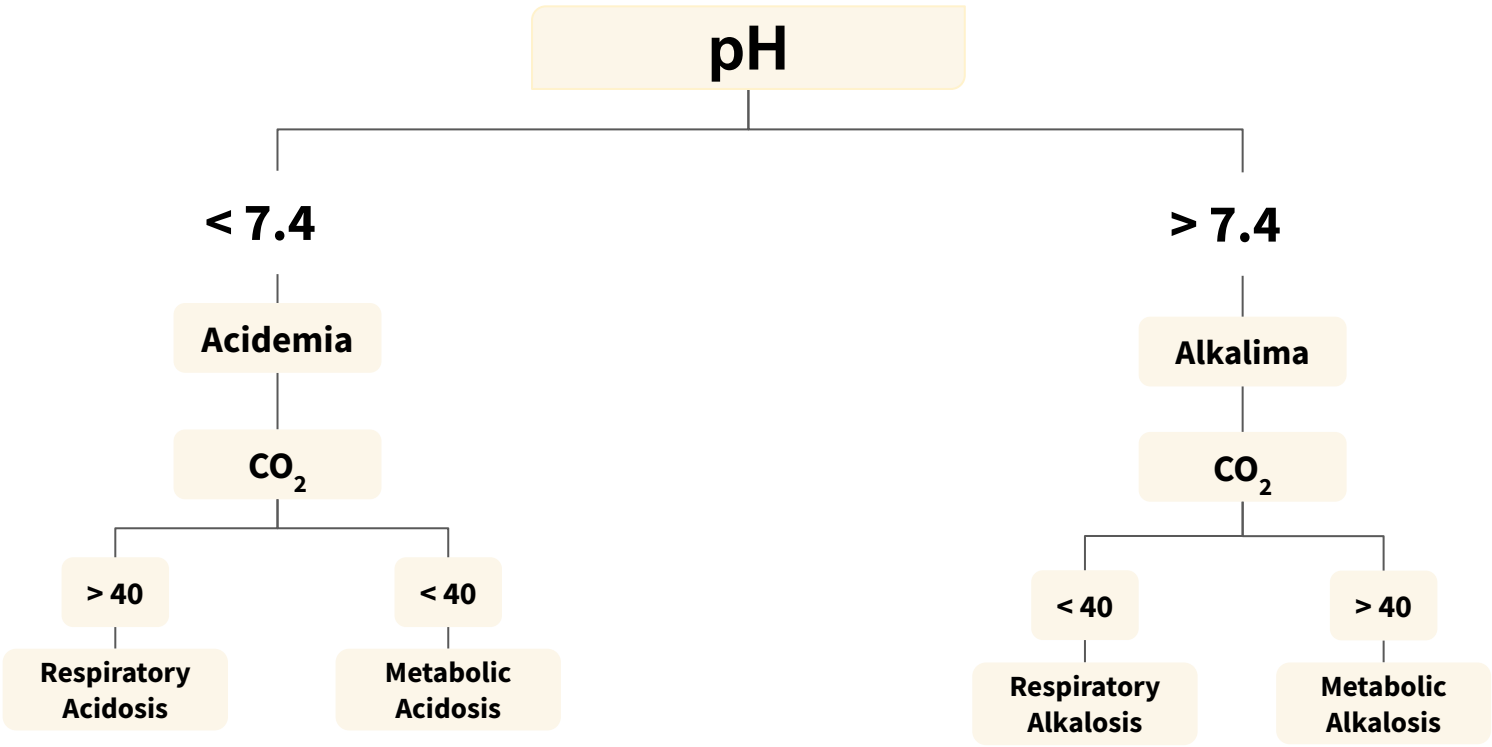
Color index

- Original text
- Females slides
- Males slides
- Doctor's notes 438
- Doctor's notes 439
- Text book
- Important
- Golden notes
- Extra

Lecture outline

Approach each Acid-Base Questions by determining the primary disturbance

1- First we look at the Ph ⟶ to determine Acidemia OR Alkalemia
2- Then we look at CO_2 ⟶ to determine the cause



Normal PH doesn't mean there's no disturbance, always look at bicarbonate and CO2 ⟶ mixed disturbance

Respiratory Acidosis

- Most probably caused by Hypoventilation;
Causes can be: (Opiate overdose) (Obstructive lung disease) (No muscular strength) (Obstructive sleep apnea)

Respiratory Alkalosis

- Most probably caused by Hyperventilation;
Causes can be: (Pain) (Anxiety) (Hypoxemia) (Basically anything that causes fast respiratory rate)

Metabolic Acidosis

- First look at the anion gap ; (> 12 = Anion Gap Acidosis) / (< 12 = Non Gap Acidosis)
A. Anion gap acidosis causes can be **(MUD PILES)** (M=methanol, U=uremia, D=DKA, P=propylene glycol, I=isopropyl alcohol, L=lactic Acidosis, E=ethylene glycol, S=salicylate)
B. Non gap acidosis ⟶ **check urine anion gap**, (if pos+ = RENAL TUBULAR ACIDOSIS) (if neg - = Diarrhea)

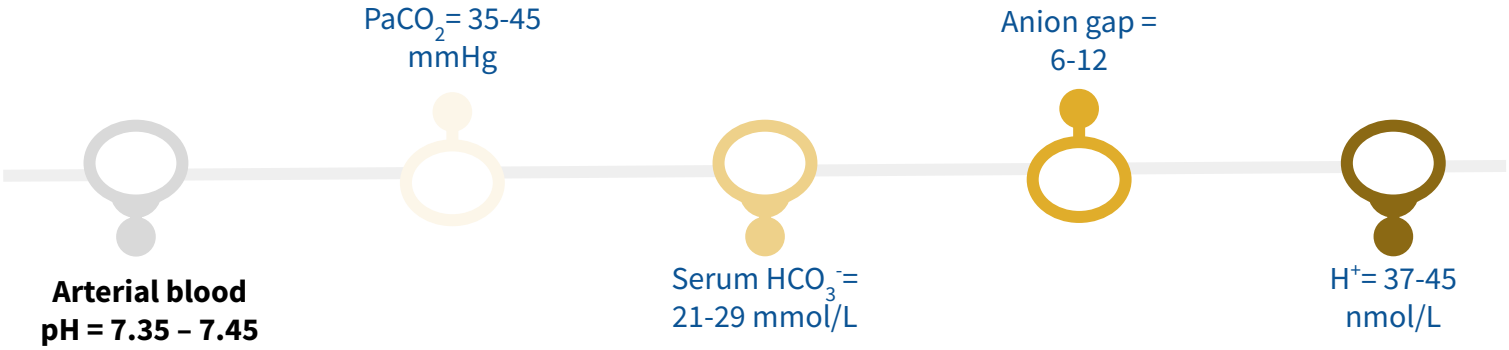
Metabolic Alkalosis

- First look at the Urine chlorine (to see if volume responsive or not)
Volume responsive ⟶ Urine chloride will be <10 ; causes can be (Volume depletion) (Emesis) (Diuretics)
NOT volume responsive ⟶ Check BP ; Hypertensive = Hyperaldosterone state / normal BP = Genetic Disease

Credits: ***Dustyn williams (Online meded)***

Introduction

◀ 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+ ions and maintained by several buffering systems.
  - A **decrease** in blood pH is called acidaemia 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_3^- or CO_2
- **Assessment of acid base abnormalities**: typically done using arterial blood gases (ABG)[1]
- 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
Problem	Hypoventilation	Hyperventilation	Gain of H^+ or loss of HCO_3^-	Gain of HCO_3^- or loss of H^+[3]
pH	↓	↑	↓	↑
HCO_3-	↑	↓	↓↓	↑↑
$PaCO_2$	↑↑[2]	↓↓	↓	↑

1: Mostly in ICU patients. Getting an ABG difficult and painful that's why we do VBG and CBG
2: PCO_2 does not rise above 55 mmHg because hypoxia then intervenes to drive respiration
3: [H+] can't be calculated in the blood

Respiratory Acidosis

Definition

Increased $PaCO_2$ and **decreased** pH

◄ Mechanism

- Process that primarily causes **elevation** in PaCO2.
- Reduce effective ventilation e.g. many chronic respiratory diseases (COPD) or drugs depressing the respiratory center.
- Alveolar Hypoventilation → **Accumulation** of CO_2 → Increases in $PaCO_2$ → Respiratory acidosis → pH decreases.
- HCO_3^- will increase (Compensation) but it needs time (12 -24 h) as the kidney need time to compensate

◄ Clinical features

- Signs of acute CO2 retention: headaches, confusion, and papilledema, flapping tremors

◄ Classification

	Acute Respiratory Acidosis	Chronic Respiratory Acidosis
Causes	1. Respiratory: airway obstruction, severe pneumonia, chest trauma/pneumothorax 2. Acute drug intoxication: narcotics, sedatives. 3. Residual neuromuscular blockade. 4. CNS disease (head trauma)	1. Chronic lung disease (COPD) 2. Neuromuscular disease 3. ★ Extreme obesity 4. Chest wall deformity 5. Muscular e.g. Duchenne dystrophy
pH	Low	Almost normal due compensatory mechanism.
Compensation	• Immediate renal compensatory↑of HCO3. • HCO3 ↑ by 1 mEq/l for every 10 mmHg ↑ in PaCO2.	HCO3 ↑ by 3-3.5 mEq/l for every 10 mmHg ↑ in PaCO2 (Due to renal adaptation)

➢ Treatment:

- Verify patency of airways.
- Give supplemental oxygen: If PaO_2 is low (<60 mmHg), Oxygen is contraindicated in COPD patients (CO2 retention) as it can exacerbate symptoms.
- Treat underlying cause.
- Intubation and mechanical ventilation might be required for:
  - Severe acidosis.
  - $PaCO_2$ > 60 or inability to increase PaO_2.
  - Mental deterioration.
  - Impending respiratory fatigue.

Respiratory Alkalosis

Definition

Decreased $PaCO_2$ and **increased** pH.

◀ Mechanism

- Process that primarily causes **reduction** in PaCO2
- Increase ventilation e.g.in response to hypoxia or secondary to metabolic acidosis.
- Alveolar hyperventilation → increased wash out CO2 → decrease in PaCO2 → increased pH.
- Compensation: HCO3- will decrease after (12 -24 h).

◀ Etiology

Hyperventilation of any Cause

- Overaggressive mechanical ventilation
- Pain, Sepsis, Pregnancy, Hepatic failure (cirrhosis)
- Medication (salicylate toxicity e.g. aspirin overdose)
- Anxiety Panic attack(most common), Fever (not severe) 2nd most common.
- Hypoxemia, Restrictive lung disease
- Pulmonary embolism, asthma, pneumonia.

Clinical Features:

lightheadedness, dizziness, anxiety, paresthesia, and perioral numbness

Tetany, Arrhythmias, Trousseau's sign[1] and Chvostek's sign[1] may be positive

◀ Classification

Acute Respiratory Alkalosis	Chronic Respiratory Alkalosis
HCO^{3-}↓ by 2 mEq/l for every 10 mmHg ↓ in $PaCO_2$	HCO^{3-} ↓ by 4-5 mEq/l for every 10 mmHg ↓ in $PaCO_2$.

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

Respiratory Alkalosis *(cont.)*

◀ Treatment

1. Treat underlying cause.
2. Sometimes: does not need to be treated (e.g., in the case of pregnancy).
3. Breathe into paper bag to recycle the exhaled CO2 (especially who have anxiety).

Metabolic Acidosis[1]

Definition

Loss of [HCO3] or **addition** of [H+] and decreased pH.

◀ Mechanism

- Process that primarily **reduced bicarbonate**
- Excessive H+ formation e.g. lactic acidosis, ketoacidosis.
- Reduce H+ excretion e.g. renal failure.
- Excessive HCO3- loss e.g. diarrhea.
- Compensation: Hyperventilation ⟶ decrease PCO2 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[2]:

- The difference between primary measured cations (Na+ and K+) and the primary measured anions (Cl- and HCO3-) in serum:
  - Anion gap = cations - anions ⟶ AG= [Na+] - ([Cl-] + [HCO3-])
  - Normal range is about 5-12 mmol/L
- It is helpful in determining the cause of a **metabolic acidosis**

1- Metabolic problems always shows compensation
2-This gap is normally made up of anions, such as phosphate and sulphate, as well as albumin.

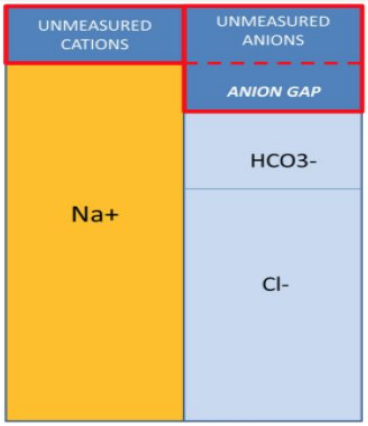
Metabolic Acidosis

◄ Classification & Etiology:

1-High Anion gap acidosis

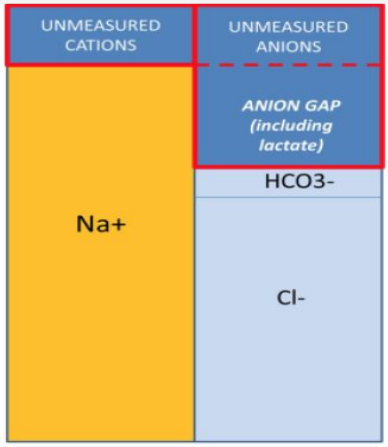
- **Causes of High Anion Gap Metabolic Acidosis (MUD PILES):**

Increased Endogenous production of anions distinct from Cl^- & HCO_3^-	
★ **L**actic acidosis Plasma lactate > 2 mmol/L	Type I: Tissue hypoxia & peripheral generation of lactate (circulatory failure & shock (**septic**, cardiogenic, hypovolemic)) Type II: Impaired metabolism of lactate (liver disease, metformin[1])
Diabetic Ketoacidosis[2]	★ **DKA**: caused by insulin deficiency & exacerbated by catecholamine & stress hormone excess → lipolysis → formation of acidic ketones (acetoacetate, 3-hydroxybutyrate, acetone) Other causes of ketoacidosis: • **Starvation ketoacidosis:** ↓ food intake in situations of high glucose demand e.g. neonates, pregnant & breastfeeding women • **Alcoholic ketoacidosis:** chronic malnutrition & recent alcohol binge
Uremia	Renal failure → ↓ NH_4^+ & H^+ excretion, decreased excretion of organic anions, sulfates, and phosphates.
INH	Impaired hepatic clearance of lactate
Increased Exogenous Intake	
Ethanol/Ethylene glycol poisoning	Accumulation of glycolate, calcium oxalate crystals
Methanol poisoning	Manifested as visual complaints
Propylene glycol (not paraldehyde)	is metabolized to lactic acid (lactate) and has the potential to cause a high anion gap metabolic acidosis
Aspirin poisoning[3]	Accumulation of **S**alicylates



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^+)

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

Metabolic Acidosis

◄ Classification & Etiology[1]:

2-Normal Anion Gap Acidosis

- HCO_3^- decreases and is replaced by Cl^- to maintain electroneutrality. Consequently, these disorders are sometimes referred to collectively as **hyperchloremic acidosis.**

↑ GI HCO_3^- loss	**Diarrhea**[2], small bowel fistula, pancreatic fistula, **urinary diversion procedure**, ileostomy, ureterosigmoidostomy
↑ Renal HCO_3^- loss	• Type II (proximal) RTA[3] • hyperparathyroidism • tubular damage e.g. drugs, heavy metals, paraproteins ★ **Treatment with carbonic anhydrase inhibitors**: Acetazolamide therapy
↓Renal H^+ excretion	• Type I (classical distal) RTA, Type IV RTA (aldosterone deficiency[4]) • CKD
↑ HCl production	• Ammonium chloride ingestion, ↑ catabolism of lysine, arginine • **Excessive administration of 0.9% saline**

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[5] 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- Just in case if you read it somewhere **Urinary anion gap** is used to determine what is the source of non anion gap acidosis Is it from the kidney or GI problems (you don't need to know about it)
2- Most common cause of normal AG metabolic acidosis
3- further discussed in **the next page.**
4- hypoaldosterone status could be due to: Addison's disease, spironolactone, amiloride, triamterene.
5- needed especially in normal AG metabolic acidosis

Metabolic Acidosis *(cont.)*

Renal tubular acidosis (RTA):

- Renal tubular acidosis (RTA) is a metabolic acidosis with a normal anion gap.
- RTA should be suspected when there is a hyperchloraemic acidosis with a normal anion gap in the absence of gastrointestinal disturbance.
- Plasma HCO_3^- < 21 mmol/L, urine pH > 5.3 = RTA
- Confirmed by acid load test

Type I (classical distal) RTA[1]

- The distal tubule is responsible for generating new bicarbonate under the influence of aldosterone.
- Drugs such as amphotericin and autoimmune diseases such as SLE or Sjögren syndrome can damage the distal tubule. If new bicarbonate cannot be generated at the distal tubule, then acid cannot be excreted into the tubule, raising the pH of the urine.
- **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.
- topiramate causes distal RTA.
- **Teats:** The best initial test is a UA looking for an abnormally high pH above 5.5. The most accurate test is to infuse acid into the blood with ammonium chloride. A healthy person will be able to excrete the acid and will decrease the urine pH. Those with distal RTA cannot excrete the acid and the urine pH will remain basic (over 5.5) despite an increasingly acidic serum.
- **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[2,3]

- **Impaired HCO3 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.
- In proximal RTA, tenofovir kills tubule
- **Treatment:** sodium bicarbonate: massive doses may be required to overcome the renal 'leak'.

Type IV RTA

- Also called **'hyporeninaemic hypoaldosteronism[4]'**
- type IV RTA occurs most often in diabetes.
- **impaired sodium reabsorption in the late distal tubule or cortical collecting duct, which is associated with reduced secretion of both K^+ and H+ ions**
- The cardinal features are hyperkalaemia and acidosis occurring in a patient with mild chronic kidney disease
- **Test:** by finding a persistently high urine sodium despite a sodium-depleted diet. In addition, hyperkalemia is a main clue to answering "What is the most likely diagnosis?"
- **Treatment:** fludrocortisone, sodium bicarbonate, diuretics, or ion exchange resins to remove potassium, or a combination of these.

1- Distal RTA calcifies the kidney parenchyma (nephrocalcinosis)
2- There is Type III: combination of type 2 and 1 but is extremely rare
3-Both proximal and distal RTA are hypokalemic. Potassium is lost in the urine
4- There is a decreased amount or effect of aldosterone at the kidney tubule.

Metabolic alkalosis

◀ Definition[1,2]:

Addition of [HCO3] or **loss** of [H+] and **increase** pH

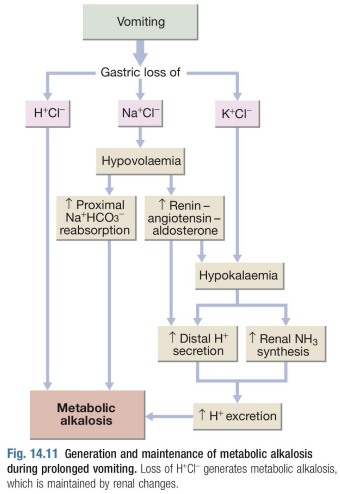


Fig. 14.11 **Generation and maintenance of metabolic alkalosis during prolonged vomiting.** Loss of H^+Cl^- generates metabolic alkalosis, which is maintained by renal changes.

◀ 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 HCO3- .
  - Loss of acid (H+) ex: from vomiting.
- Maintaining Metabolic alkalosis due to the kidney inability to excrete the excess HCO3
- Compensation: Hypoventilation ⟶ increased PCO2 (respiratory Acidosis) immediately (PaCO2 ↑ by 0.6 mmHg for every 1 mEq/l ↑ in HCO3).

Clinical Features:

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

◀ Classification & causes[3]:

	Hypovolemic (Saline sensitive, urine Cl^- <10 mEq/L)	**Normovolemic (Saline resistant, urine Cl^- >20 mEq/L)**
Definition	Metabolic alkalosis with ECF contraction (due to ⟶ fluid loss).	Metabolic alkalosis with ECF volume expansion (no fluid loss)

Causes	• Gastric loss of H+ (sustained vomiting)
	• Diuretic use: loop or thiazide.
	• Volume depletion
	• Post-hypercapnia
	• Villous adenoma of colon, diarrhea with high chloride content
		• Hypertensive:
		  ○ Primary Hyperaldosteronism
		  ○ Cushing Syndrome
		  ○ Glucocorticoid therapy
		• Hypo/normo tensive:
		  ○ Bicarbonate ingestion: massive or with kidney disease
		  ○ Bartter's syndrome & Gitelman's syndrome, Severe hypokalemia

Treatment	intravenous infusions of 0.9% saline with potassium supplements	management of the underlying cause

1- You cannot determine the etiology of metabolic alkalosis from the ABG.
2- The ABG in metabolic alkalosis will always have:
- Increased pH >7.40
- Increased pCO2 indicating respiratory acidosis as compensation
- Increased bicarbonate

3- Hypokalemia is one of the causes : H+ will move into the cytoplasm of the cells in order to get the K+ to compensate for Hyperkalemia

Steps in Acid-Base Analysis

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

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)

Step 3a — Determine the primary abnormality that is causing the abnormal pH

- If the **pH** is acidemic (<7.35), then look for **Low** HCO_3 (**Metabolic**) or **High** PCO_2 (**Respiratory**)
- If the **pH** is alkalemic (>7.45), then look for **High** HCO_3 (**Metabolic**) or **Low** PCO_2 (**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.

Step 3b — If pH is normal, that doesn't rule out mixed acidosis and alkalosis (Determine what is being mixed[1])

- Look for **high** or **low** PCO2= **Low** PCO2 suggests **respiratory alkalosis**/**High** PCO2 suggests **respiratory acidosis**
- Look for **high** or **low** HCO3= **Low** HCO3 suggests **metabolic acidosis/High** HCO3 suggests **metabolic alkalosis**

How to determine Is the respiratory disturbance acute or chronic?

- **Acute respiratory acidosis**: HCO3 **increase** by 1 mEq/l for every 10 mmHg **increase** in PaCO2.
- **Chronic respiratory acidosis**: HCO3 **increase** by 3-3.5 mEq/l for every 10 mmHg **increase** in PaCO2.
- **Acute respiratory alkalosis**: HCO3 **decrease** by 2 mEq/l for every 10 mmHg **decrease** in PaCO2.
- **Chronic respiratory alkalosis**: HCO3 **decrease** by 4-5 mEq/l for every 10 mmHg **decrease** in PaCO2.

1: Sometimes you may have mixed diseases (Metabolic Alkalosis AND Respiratory Acidosis)
2: there are 3 Clinical Scenarios that Produce a Mixed Disorder with Near Normal pH (e.g. increased AG metabolic acidosis + respiratory alkalosis)
• Cirrhosis • ASA overdose • Sepsis

Steps in Acid-Base Analysis

Step 4 ★ check for compensation[1]

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, HCO3 is 15** (hence, it is metabolic acidosis)
- Use the metabolic acidosis formula: **Expected PCO2** by using **Winter's formula PCO2 = 1.5 x HCO3 + 8 (±2[1])** = 1.5 x 15 + 8 = 30.5
- **So:** you expect the **PCO2** in this patient to be in the range of **28.5– 32.5**[4]
- Now, determine whether there is a compensation or an additional disorder:
  - If the PCO2 in this patient is **higher than 32.5** → consider additional[3] respiratory acidosis
  - If the PCO2 in the patient is **lower than 28.5** → consider additional respiratory alkalosis

Primary disorder		Expected compensation
Metabolic acidosis		• **$PaCO_2 = 1.5 \times HCO_3 + 8 \pm 2$** • **$\downarrow PaCO_2 = 1.2 \times \Delta HCO_3$** • **$PaCO_2$ ~ last two digits of pH**
Metabolic alkalosis		• **$\uparrow PaCO_2 = 0.7 \times \Delta HCO_3$**
Respiratory acidosis	**Acute**	• **$\uparrow HCO_3 = 0.1 \times \Delta PaCO_2$**
	Chronic (COPD)	• **$\uparrow HCO_3 = 0.35 \times \Delta PaCO_2$** • **$\downarrow pH = 0.003 \times \Delta PaCO_2$**
Respiratory alkalosis	**Acute**	• **$\downarrow HCO_3 = 0.2 \times \Delta PaCO_2$**
	Chronic	• **$\downarrow HCO_3 = 0.4 \times \Delta PaCO_2$**

Step 5 Calculate the anion gap

anion gap (AG): AG = Na – (Cl + HCO3)

- Normal anion gap = 6-12[5]
- 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.
- If there is a reduction of albumin the bicarb and Cl^- will increase
- **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: we have Compensatory mechanism to keep the pH from lifting too high changing in the PH will cause enzyme unfold and abnormal changing in the proteins shape
2: gives you a range
3: Please make sure that you differentiate between additional and compensated.
4: Memorize one compensation equation for each acid base abnormality. Example:
-If the PCO_2 of this patient was 35, then the patient's acid-base status will be : Metabolic Acidosis AND Respiratory Acidosis.
-If the PCO_2 of this patient was 30, then the patient's acid-base status will be : Metabolic Acidosis Compensated by Respiratory Alkalosis.
Someone With Metabolic Acidosis what happen? He will breathe fast but this will not bring the PH to normal range but the PCO2 will get down so it's not fully corrected and if the pH is normal that mean there is additional abnormality
5: The normal range is up to 14. It is Especially important in Metabolic Acidosis, crucial for the differential diagnosis.

Cases (Doctor's Slides)

◀ 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	**7.18**	7.35-7.45
PO_2	150 mmHg	82-105 mmHg
$PaCO_2$	**16 mmHg**	35-45 mmHg
HCO_3	**7 mmol/L**	22-26 mmol/L
Na^+	138 mmol/L	136-145 mmol/L
K^+	3.9 mmol/L	3.5-5 mmol/L
Cl^-	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**

$PaCO_2 = 1.5 \times HCO_3 + 8 \pm 2$

  - **Substitute the values**

$PaCO_2 = 1.5 \times 7 + 8 \pm 2$

$PaCO_2 = 18.5 \pm 2$

$PaCO_2 = (16.5 - 20.5)$

  - **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**

AG = Na - (Cl +HCO3)

AG = 138 - (95+7) = 36 (**high**)

- **Indicate what is causing the acid base disturbance?**
  - Lactic acidosis (associated with shock) shock > shifting to anaerobic metabolism > high lactate > low PH (metabolic acidosis)

◀ 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	**7.49**	7.35-7.45
PO_2	86 mmHg	82-105 mmHg
$PaCO_2$	**48.5 mmHg**	35-45 mmHg
HCO_3	**39 mmol/L**	22-26 mmol/L
Na^+	142 mmol/L	136-145 mmol/L
K^+	3 mmol/L	3.5-5 mmol/L
Cl^-	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**[1]

  $\uparrow PaCO_2 = 0.7 \times (39-24)$

  $\uparrow PaCO_2 = 10.5$
  - **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)

1- 39 is the value from Lab results | 24 is the normal value of bicarbonate

Cases (Doctor's Slides)

◀ 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	**7.21**	7.35-7.45
PO_2	61.5 mmHg	82-105 mmHg
$PaCO_2$	**83 mmHg**	35-45 mmHg
HCO_3	**34 mmol/L**	22-26 mmol/L
Na^+	140 mmol/L	136-145 mmol/L
K^+	4.7 mmol/L	3.5-5 mmol/L
Cl^-	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 acidosis)
- **Check whether the patient has compensation/additional disturbance.**
  - **Choose the formula**

Ask yourself, is it Acute or chronic? COPD = Chronic

$\uparrow HCO_3 = 0.35 \times \Delta PaCO_2$

  - **Substitute the values**

$\uparrow HCO_3 = 0.35 \times (83-40)$

$\uparrow HCO_3 = 15$

  - **Add to the normal range**

$\uparrow HCO_3 = \mathbf{24 + 15} = 39 \pm 2$

$\uparrow HCO_3 = (37-41)$

  - **Interpret the result**

there is an additional metabolic acidosis on top of the respiratory acidosis[1,2]

- **Calculate the anion gap**

$AG = Na - (Cl + HCO_3)$

$AG = 140 - (94+34) = 12$ (**normal**)

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

CO2 retention caused by COPD (CO2 accumulation may itself lead to drowsiness That further depresses respiratory drive)

1: because there is loss of bicarb, is less than what is expected

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	**7.25**	7.35-7.45
PO_2	101 mmHg	82-105 mmHg
$PaCO_2$	**31.5 mmHg**	35-45 mmHg
HCO_3	**17 mmol/L**	22-26 mmol/L
Na^+	134 mmol/L	136-145 mmol/L
K^+	3.4 mmol/L	3.5-5 mmol/L
Cl^-	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**

  $PaCO_2 = 1.5 \times 17 + 8 \pm 2$

  $PaCO_2 = 33.5 \pm 2$

  $PaCO_2 = (31.5\text{-}35.5)$
  - **Interpret the result**

  the metabolic acidosis is compensated properly by respiratory alkalosis.
- **Calculate the anion gap**

  AG = Na - (Cl +HCO3)

  AG = 134 - (104+17) = 13 (**normal**)
- **Indicate what is causing the acid base disturbance?**

  diarrhea

Helpful Figures

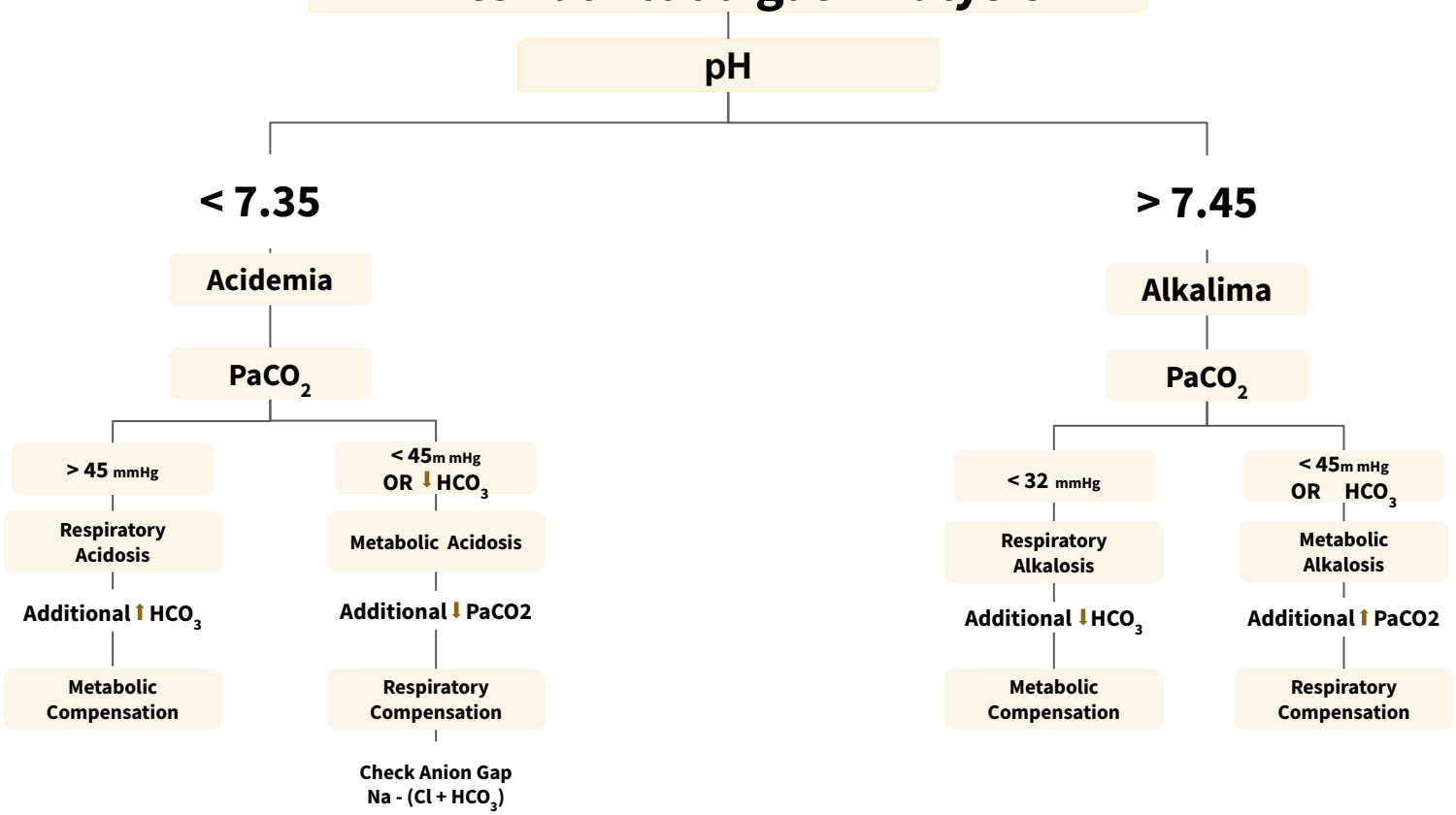
Causes of Respiratory Acidosis and Alkalosis

Respiratory alkalosis	Respiratory acidosis
Decreased pCO_2	Increased pCO_2
Increased minute ventilation	Decreased minute ventilation
Metabolic acidosis as compensation	Metabolic alkalosis as compensation
• Anemia • Anxiety • Pain • Fever • Interstitial lung disease • Pulmonary emboli	• COPD/emphysema • Drowning • Opiate overdose • Alpha 1-antitrypsin deficiency • Kyphoscoliosis • Sleep apnea/morbid obesity

Causes of Metabolic Acidosis with an Increased Anion Gap

	Cause	Test	Treatment
Lactate	Hypotension or hypoperfusion	Blood lactate level	Correct hypoperfusion
Ketoacids	DKA, starvation	Acetone level	Insulin and fluids
Oxalic acid	Ethylene glycol overdose	Crystals on UA	Fomepizole, dialysis
Formic acid	Methanol overdose	Inflamed retina	Fomepizole, dialysis
Uremia	Renal failure	BUN, creatinine	dialysis
Salicylates	Aspirin overdose	Aspirin level	Alkalinize urine

Arterial Blood gas Analysis



Summary

Arterial: 7.35-7.45 — **Normal pH** — Venous: 7.31-7.41

Metabolic Acidosis	Process that primarily **reduces bicarbonate**	1. Excessive H^+ formation: e.g. lactic acidosis, ketoacidosis 2. Reduced H^+ excretion: e.g. renal failure 3. Excessive HCO_3^- loss: e.g. diarrhea
Metabolic Alkalosis	Process that primarily **raises bicarbonate**	1. Extracellular fluid volume loss: e.g. vomiting or diuretics 2. Excessive potassium loss with subsequent hyperaldosteronism
Respiratory Acidosis	Process that primarily causes **elevation of $PaCO_2$** (Hypoventilation)	Reduced effective ventilation: e.g. many chronic respiratory diseases or drugs depressing the respiratory system
Respiratory Alkalosis	Process that primarily causes **reduction in $PaCO_2$** (Hyperventilation)	Increased ventilation: e.g. in response to hypoxia or secondary to a metabolic acidosis

Approaching Acid-base Abnormalities

Step 1: History and Physical Examination	Vomiting • Diarrhea • Hypoventilation • Respiratory disease • Medications (laxatives, diuretics, etc) • Diabetes
Step 2: Look at the pH	- **Normal** 7.35 – 7.45 (No abnormality or **mixed** acidosis and alkalosis) - **Low** <7.35 (acidemic) - **High** >7.45 (alkalemic)
Step 3: **a.** Determine the primary abnormality that is causing the abnormal pH	- If the pH is **acidemic**, look for: Low HCO_3^- (Metabolic) or High PCO_2 (Respiratory) - If the pH is **alkalemic**, look for High HCO_3^- Metabolic) or Low PCO_2 (Respiratory)
b. if pH is normal	- Rule out mixed acidosis and alkalosis - Look for high or low PCO_2 and for high or low HCO_3^-
Step 4: Check for compensation **(imp)**	Metabolic Acidosis: $PaCO_2 = 1.5 \times HCO_3 + 8\ (\pm 2)$ Or $\downarrow PaCO_2 = 1.2 \times \Delta HCO_3$ 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: $\uparrow HCO_3 = 0.35 \times \Delta PaCO_2$ 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)	$AG = Na - (Cl + HCO_3)$

Lecture Quiz

Q1: A 70 year old woman is brought to the emergency department by her daughter because of a 2 day history of non-bloody diarrhea. The patient has had 8-10 bowel movement daily and has not vomited. She has a history of Hypertension, coronary artery disease, hyperlipidemia, and type 2 DM. Current medication include furosemide, hydrochlorothiazide, linsopril, atrovistatin, metoprolol, metformin, and dapagliflozin. She appears ill and sleepy. She is 157 cm tall and weight 109 kg ; BMI is 44 kg/m2. Her temperature is 37.9oC, pulse is 115/min, respiration are 26/min, and blood pressure is 110/60 mmHg. Pulse oximetry shows an oxygen saturation of 97% on room air. Physical examination shoes dry mucous membranes. A fingerstick glucose test shows no abnormalities. Arterial blood gas analysis on room air shows: pH (7.24) / pO2 (85 mmHg) / pCO2 (39 mmHg) / HCO3 (16 mEq/L). Which of the following is the most likely acid-base abnormality in this patient?

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

Q2: 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; PCO2 = 3.3; PO2 = 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

Q3: 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 PCO2 3.0 kPa PO2 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

Q4: 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, PO2 14.1, PCO2 3.1 kPa, HCO3 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

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, HCO3- 13 meq/liter, PCO2 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

Answers: Q1:E | Q2:E | Q3:D | Q4:B | Q5:C | Q6:C

Answers Explanation File!

GOOD LUCK !

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