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Acid Base Balance

★ Objectives:

State the normal value for PH,PCO2,HCO3

Understand the basic mechanism of acid base disturbance

Interpret basic acid base disturbance

List common differential diagnosis for different acid base disorder

★ Resources Used in This lecture:

Doctor`s notes , davidson's , team 433 , step up to medicine.

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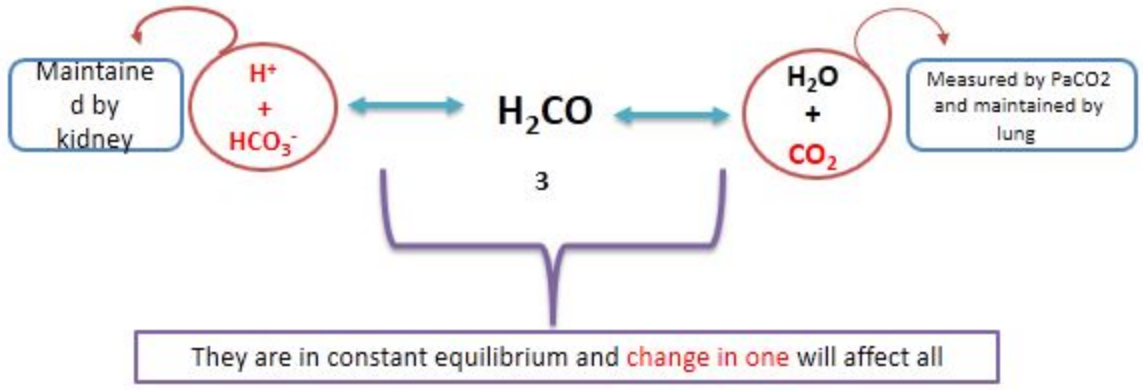
Physiology overview

- **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.**
- **Most importantly the bicarbonate-carbonic acid buffer pair that depends on the balance between bicarbonate ions and carbonic acid:**



Anion Gap (AG) :

- Usually blood serum level is neutralized electronically which means it's charged→ zero. → cations = the anions.
- Most prominent cations are Na and K.
- Most prominent anions are Cl and HCo3 + A gap.
- To know this gap we subtract the anions from the cations normally it's between 8-12.
- Anion in the gap is usually (phosphate,sulphate, albumin and organic acids).

Calculations:

- **Anion gap = cations - anions**
- **Anion gap = [Sodium] - (Chloride + Bicarbonate)**
- **Anion gap = ($Na^+ + K^+$) - ($Cl^- + HCO_3^-$)**
- **AG= (Na+)-(Cl- + HCO3 -)**

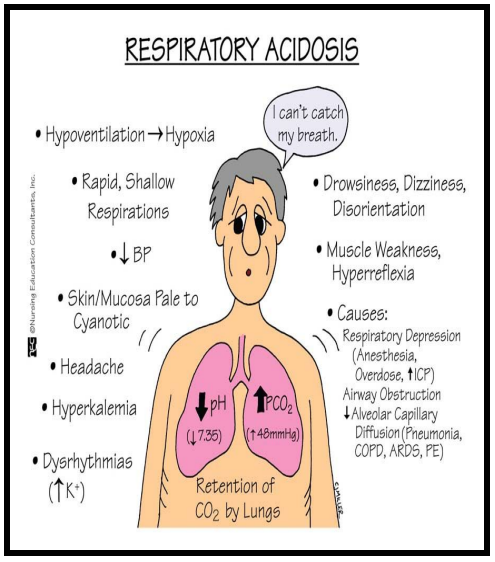
Normal Values You need to memorise it :

★ **arterial blood pH = 7.35 – 7.45 → 7.4**
★ **P_aCO_2 = 35-45 → 40**
★ **Serum HCO_3^-.= 22-26 → 24**
★ **Anion gap = 8-12 → 6**

Respiratory acidosis

Definition

Increased PaCO2 mmHg and decreased pH.



Mechanism:

- ❑ Hypoventilation → accumulation of CO_2 → increases in PCO2 → Respiratory acidosis → PH decreases.
- ❑ HCO_3^- will increase (Metabolic alkalosis) but it need time (12 -24 h) as the kidney need time to compensate.

Etiology based on system:

- ❑ **CNS** : damage of the respiratory center in the brainstem

Caused by:

- Stroke
- Hemorrhage
- Trauma ,
- Tumor
- Medication such as (Commonly sleeping pills, other: morphine, anesthetics and narcotics)

- ❑ **PNS :** Demyelinating disease Of PNS ex." **guillain-barré syndrome**[1] usually follow diarrhea or flu like illness → followed by ascending paralysis from legs going up.
- ❑ **Neuromuscular junction**: Myasthenia gravis
- ❑ **Muscular disease**: intercostal muscle atrophy, such as:

- Duchenne dystrophy
- Congenital muscle atrophy

- ❑ **Chest wall** : such as severe scoliosis

- **Bronchial tree such as:** COPD or Emphysema (irreversible bronchoconstriction not responding to bronchodilators → retain CO2 → exchange gases lung defect→ leading to acute/chronic Respiratory alkalosis.)

- ❑ **Other causes**: Drowning, Sleep apnea and Morbid obesity.

Note : any disease that reduces CO_2 clearance (inhibit adequate ventilation) can lead to respiratory acidosis.

[1] It`s a acute or subacute characterised by ascending paralysis (start at leg and go up) , so when it severe enough it can cause respiratory compromised . so we have to do vital capacity to see whether the respiratory muscle or respiration are affected , in severe cases we will see respiratory acidosis

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**
PH	**Low**	**Normal due compensatory mechanism.**
Compensation	-Immediate compensatory elevation of HCO_3. $-HCO_3^-$ increase by 1 mEq/l for every 10 mmHg increase in $PaCO_2$.	$-HCO_3^-$ increase by 3-3.5 mEq/l for every 10 mmHg increase in $PaCO_2$. (Due to renal adaptation)

Clinical features :

- **Symptoms** : Somnolence, confusion, myoclonus[2] with asterixis (Flapping tremors).
- **Signs of acute CO_2 retention:** headaches, confusion, and papilledema.

Pathophysiology of papilledema :

- Increased $PaCO_2$ causes increased cerebral blood flow which increases CSF pressure⟶ Resulting in generalized CNS depression.
- Increased free calcium due to displacement from albumin by H^+

Treatment

1. Verify patency of airways.
2. Give supplemental oxygen : If PaO_2 is low (<60 mmHg)
3. Treat underlying cause.

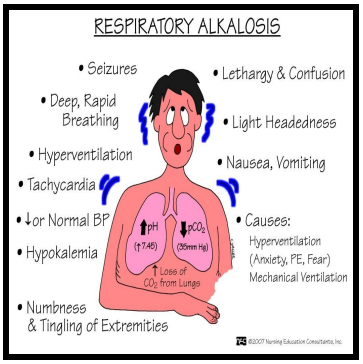
[2] Involuntary jerk of muscle

Oxygen is contraindicated in COPD patients as it can exacerbate symptoms.

4. Intubation and mechanical ventilation might be required for:

- Severe acidosis (PH <7)
- PaCO2 > 60 or inability to increase PaO2
- Mental deterioration
- Impending respiratory fatigue

Respiratory alkalosis



Definition

Decreased PaCO2 mmHg and increased pH.

Mechanism:

- **Alveolar hyperventilation → increased wash out CO_2 → decrease in $PaCO_2$ → increased PH**
- **Compensation : HCO_3^- will decrease (Metabolic Acidosis) after (12 -24 h).**

Classification

- **Acute respiratory alkalosis :** HCO_3^- decrease by 2 mEq/l for every 10 mmHg decrease in PaCO2.
- **Chronic respiratory alkalosis :** HCO_3^- decrease by 4-5 mEq/l for every 10 mmHg decrease in PaCO2.

Etiology

–Anxiety (most common)	–Pain
–Sepsis	–Fever (not severe) (2nd most common)
–Thyrotoxicosis	–Pregnancy
–Hepatic failure (cirrhosis)	–Drugs (aspirin overdose)
–Hypoxemia	–Restrictive lung disease
–Severe congestive heart failure	- Pulmonary embolism , asthma , pneumonia

- Any disease that increase the respiratory rate inappropriately can lead to respiratory alkalosis.
- Pregnancy → increase serum prostaglandin → Hyperventilation → respiratory Alkalosis.
- Aspirin overdose → overstimulation of respiratory centre → Hyperventilation → respiratory Alkalosis.

Clinical features :

- Lightheadedness, dizziness, anxiety, paresthesias, and perioral numbness.
- Tetany
- Arrhythmias in severe cases
- In severe cases, Trousseau's sign and Chvostek's sign may be positive.

Pathophysiology:

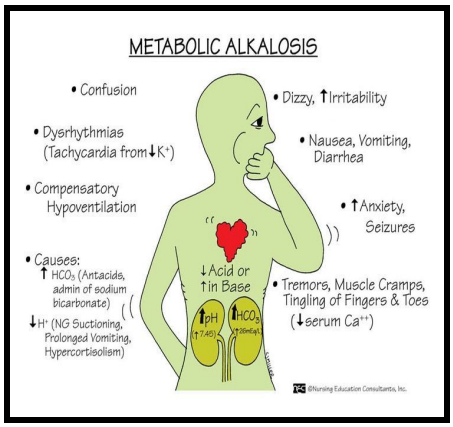
- ❑ Lightheadedness, dizziness, anxiety, paresthesias, and perioral numbness :
  - Vasoconstriction ⟶ decreased cerebral blood flow
  - alkalosis ⟶ increased binding of calcium to albumin ⟶ reduction in ionised calcium
- ❑ Tetany & Arrhythmias
  - alkalosis ⟶ increased binding of calcium to albumin ⟶ reduction in ionised calcium

Treatment

- Treat the underlying cause.
- Breathe into paper bag to recycle the exhaled CO_2 (especially who have anxiety).

You don't treat with Respiratory alkalosis in pregnancy

Metabolic acidosis



Definition

Decreased HCO_3 and decreased pH.

Mechanism

Either:

- Increase acid gaining either Exogenous Intake or Endogenous production.
- Decrease acid excretion.
- Loss or decrease production of bicarbonate.

❑ Compensation: Hyperventilation → decrease P_{CO_2} (respiratory Alkalosis) **immediately.** $PaCO_2$ decreases by 1 mmHg for every 1 mEq/l decrease in HCO_3..

Classification and Etiology

1. High Anion Gap Causes:

		Causes	Test	Treatment
Increased Endogenous production	**Lactate**	Hypotension or hypoperfusion or Excessive expenditure (seizure)	Blood lactate level	Correct hypoperfusion
	Ketoacidosis	DKA, Starvation , prolonged alcohol consumption	Acetone Level	Insulin and fluids
	Uremia	Renal failure	BUN , CR	Dialysis
Increased Exogenous Intake	**Oxalic acid**	Ethylene glycol overdose	Crystals on UA	Fomepizole[3], Dialysis
	Formic acid	Methanol overdose	Inflamed retina	Fomepizole, Dialysis
	Salicylates	Aspirin overdose	Aspirin level	Alkalinize urine.

2.Normal Anion Gap → Cl^- will be added so AG will not be changed.

GI	Loss of HCO_3	Diarrhea , fistula in bowel or pancreas . ureterosigmoid.
Renal	Decrease HCO_3 reabsorption.	Proximal Tubular Acidosis (RTA Type 2): caused by amyloidosis, myeloma , fanconi syndrome , or heavy metals (Wilson disease)
	Decreased production of HCO_3	Distal Tubular Acidosis (RTA Type 1) :caused by Autoimmune diseases (sjogren's syndrome and SLE) or medication such as amphotericin.
	Carbonic anhydrase inhibition	Due to diuretics as acetazolamide.
	Early renal failure	Impaired generation of ammonia
Others	Post- hypocapnia	Respiratory alkalosis→ renal wasting of HCO_3 →rapid correction of resp. Alkalosis → transient acidosis until HCO_3 is regenerated.
	Dilutional	Due to rapid infusion of bicarbonate - free IV fluids.

[3] Fomepizole is used as an antidote in confirmed or suspected methanol or ethylene glycol poisoning. Fomepizole is a competitive inhibitor of alcohol dehydrogenase, the enzyme that catalyzes the initial steps in the metabolism of ethylene glycol and methanol to their toxic metabolites.

To distinguish between RTA & Diarrhea we perform Urine Anion Gap.

- UAG= Sodium - Chloride.
- In RTA there is a defect in acid secretion ⟶ so less Cl into urine⟶ result of UAG positive number.
- In Diarrhea Excretion acid is intact⟶ H+ is excreted with Cl- in urine ⟶ UAG negative number.

Pathophysiology :

- RTA Type 1 : damage Distal Tubular Acidosis ⟶ decrese generation of new HCO3 ⟶ decreased excretion H ions ⟶ PH of urine become High ⟶ (increase risk of Renal stones (calcium oxalate
- RTA Type 2 : damage of Proximal tubules ⟶ decrease the absorption of HCO3 ⟶ High HCO3 in urine ⟶ depleted body of bicarbonate ⟶ distal tubules start reabsorb the HCO3

Note : Hypokalemia present in both RTA 1 & 2 due high level of aldosterone

- Decrease acid excretion such as Hypoaldosteronism (RTA Type 4) :common in DM

Clinical features :

1. Hyperventilation (deep rhythmic breathing) also called Kussmaul respiration.
2. Decreased in Cardiac output and 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 problems always show compensation.

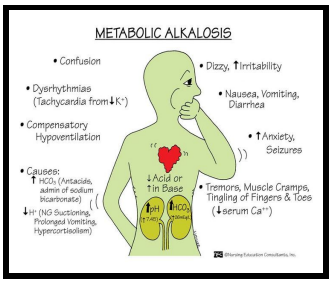
Metabolic alkalosis

Definition

Increased pH and increased HCO_3^-

Uncomplicated metabolic alkalosis is typically transient , because kidney can normally excrete the excess HCO_3^-

Mechanism:



- ❏ 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 HCO3.
- ❏ Compensation : Hypoventilation → increased Pco2 (respiratory Acidosis) immediately (PaCO2 increases by 0.6 mmHg for every 1 mEq/l increases in HCO3)

Classification and Causes

	Saline –Responsive U(cl^-) <20	Saline –resistant U(cl^-) >20
Definition	Metabolic alkalosis with ECF contraction (due to → fluid loss)	Metabolic alkalosis with ECF volume expansion (no fluid loss)
Causes	• Gastric loss of H^+ and generation of HCO3 such as vomiting (HCl loss) , NGT drainage, • Diuretic use → These decrease the ECF volume, body HCO3 content is normal, but plasma HCO3 increases due to ECF contraction. ( ) • Volume depletion • Post-hypercapnia • Villous adenoma of colon,diarrhea with high chloride content	**Hypertensive:** • Hyperaldosteronism either primary or secondary. • Non-mineralocorticoid • Cushing Syndrome **Hypo/normo tensive:** • Exogenous alkali load either IV or oral sodium bicarbonate • Bartter's syndrome &, Gitelman's syndrome • Severe hypokalemia

- Metabolic alkalosis → hypokalemia because decreased serum H+ will keep K inside intracellular , as well as the increase in kidney K + excretion seen in hyperaldosteronism, and diuretics intake.
- Metabolic acidosis → hyperkalemia because High serum H+ will shift K from intracellular to Extracellular.
- The urine chloride level is very important to differentiate between saline-sensitive and saline-resistance.

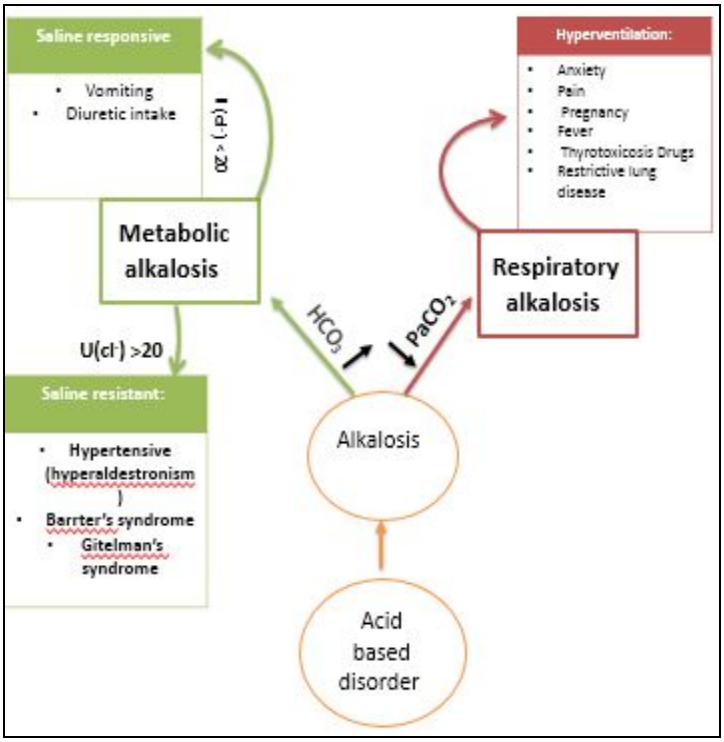
Clinical Feature

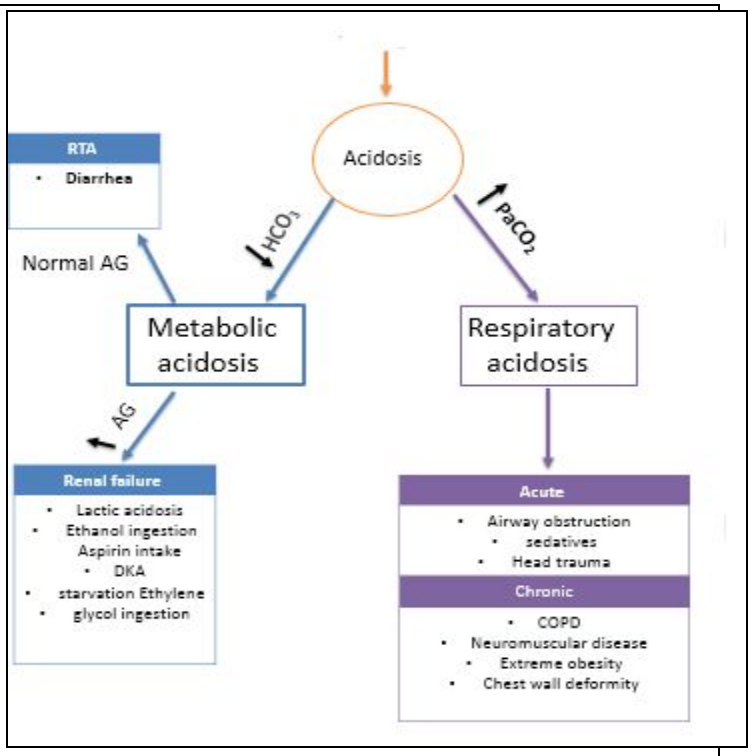
- There is no characteristic signs and symptoms (most imp. Hx)

Treatment

1. Treat the underlying cause.
2. Give normal saline plus potassium in saline responsive.
3. Spironolactone (K+ sparing diuretic) might be considered in saline resistant.

Summary





From step up : arterial blood gas interpretation :

CO_2 level	• If elevated , think of either respiratory acidosis or compensation for metabolic alkalosis • If low , think of either respiratory alkalosis or compensation for metabolic acidosis
HCO_3^- level	• If elevated , think of either metabolic alkalosis or compensation for respiratory acidosis • If low , think of either metabolic acidosis or compensation for respiratory alkalosis

Effects of acidosis and alkalosis

acidosis	• Right shift in oxygen-hemoglobin dissociation curve diminishes the affinity of hemoglobin for oxygen (increasing tissue oxygen delivery) • CNS depression • Decreased pulmonary blood flow • Arrhythmias and Impaired myocardial • Hyperkalemia
alkalosis	• Decreased cerebral blood flow • Left shift in oxygen-hemoglobin dissociation curve increases the affinity of hemoglobin for oxygen (decreasing oxygen delivery to tissues) • Arrhythmias • Tetany and seizures

Primary disturbance					
Primary Disorder	Problem	pH	HCO_3^-	P_aCO_2	Doctor`s Notes
Metabolic acidosis	gain of H^+ or loss of HCO_3^-	↓	↓	↓	-Gain acid : 1- by generate endogenous acid (ketoacidosis like in diabetic ketoacidosis or in starvation ketoacidosis) or (lactic acidosis , hypotension or hypoxia or renal failure) 2- by exogenous acid by ingestion something from outside like (alcohol intoxication , ethanol , methanol , etc) -loss of HO_3: diarrhea or renal tubular acidosis
Metabolic alkalosis	gain of HCO_3^- or loss of H^+	↑	↑	↑	-gain of HCO_3^- either by receiving IV(HCO_3^-) or oral ($NAHCO_3$) - loss of acid (HCl) from vomiting
Respiratory acidosis	hypoventilation	↓	↑	↑	- Whenever the patient hypoventilate he will not wash out CO_2 then CO_2 will accumulate result in respiratory acidosis which increases in $PaCO_2$ So the PH will decrease as compensatory mechanism the HCO_3^- will increase but problem it need 24 hours so the kidney need time to compensate
Respiratory alkalosis	hyperventilation	↑	↓	↓	Whenever the patient hyperventilation he will wash out CO_2 so decrease in $PaCO_2$ result in increased PH as compensatory mechanism the HCO_3 will decrease but also the kidney need 24 hours to compensate