# ARTERIAL BLOOD GAS ANALYSIS

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# What is the ABG?

- Arterial blood gas analysis is an essential part for diagnosing and managing the patient's **oxygenation** status, **ventilation** status and **acid base balance**.
- Drawn from **arteries**( radial, brachial and femoral)

# What is an Arterial Blood Gas (ABG)?

- The components
  - pH / $PaCO_2$ / $PaO_2$ / $HCO_3$ / $O_2$sat / BE
- Desired ranges
  - pH – 7.35 - 7.45
  - $PaCO_2$ – 35 - 45 mmHg
  - $PaO_2$ – 80 - 100 mmHg
  - $HCO_3$ – 21 - 27
  - $O_2$sat – 95 - 100%
  - Base excess – +/-2 mEq/L

# PURPOSE OF ABG'S

- 1-To determine the presence and type of acid – base balance.
- 2-To check for severe breathing problem and lungs diseases by PO2 and PCo2 levels.
- 3-Assessment of the response to the therapeutic intervention such as mechanical ventilator.

# INDICATION OF ABG

- Respiratory failure
- Ventilated patient
- Cardiac failure
- Renal failure
- Sepsis and Burn
- Poisoning

# ACID-BASE BALANCE

❖The primary aim of keeping this delicate balance is to preserve the **Homeostasis** i.e. the highly complex interactions that maintain all body systems to functioning within a normal range.

❖Any extreme change in this balance (PH **< 6.8** or **> 7.8**) may result in disastrous changes e.g. **denaturation of proteins** & shut down of **all enzymatic and metabolic processes**. Such disturbed environment would be *incompatible with life*.

- **Acidosis ..physiological processes that lower the PH below 7.35**
- **Alkalosis ..physiological processes that raise the P above 7.45**

1. LUNGS – MOST RAPID , WASHOUT CO2
2. KIDNEYS – SLOWER ONSET – HCO3-

3. BUFFERING BY EXTRACELLULAR AND INTRACELLULAR PROTEINS AND MOLECULES(PHOSPHATES .. ETC)

**Buffering system of acid base**

# RESPIRATORY COMPENSATION

## Respiratory Mechanism

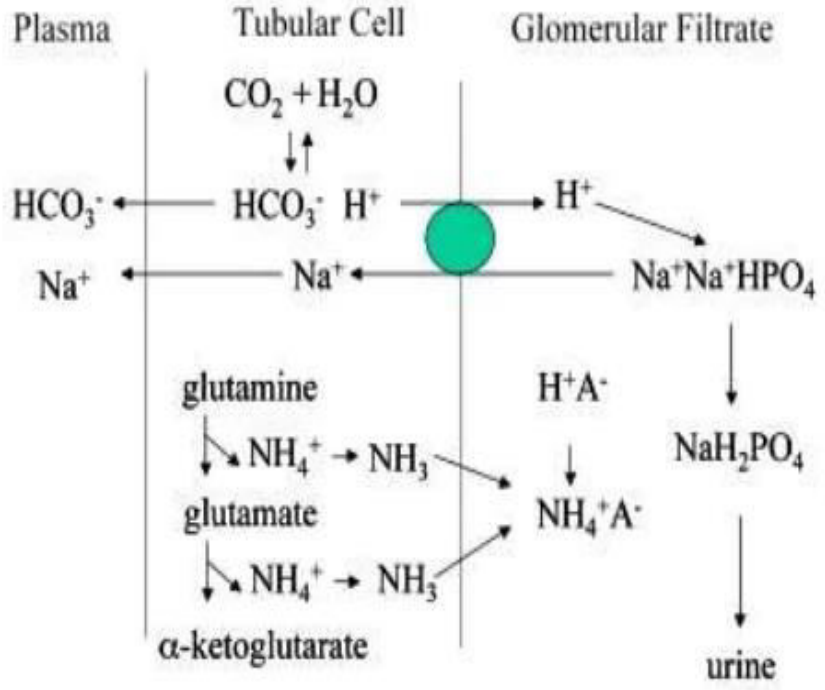
- Exhalation of carbon dioxide
- Powerful, but only works with **volatile acids**
- Doesn't affect **fixed acids** like lactic acid
- $CO_2 + H_20 \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^-$
- Body pH can be adjusted by changing rate and depth of breathing

## Respiratory Mechanisms

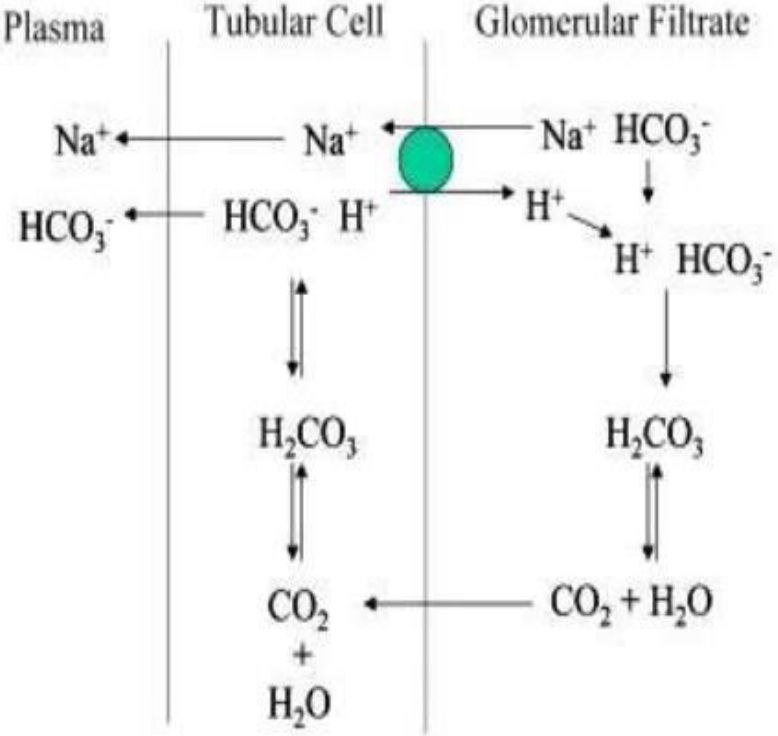
- Arterial PCO2 stimulates chemorecptors in the medulla oblongata
- An elevated arterial blood PCO2 is a stimulus to increase ventilation leading to increased expiration of CO2 hence increase blood pH
- Conversely, a drop in blood PCO2 inhibits ventilation; the consequent rise in blood [H2CO3] reduces the alkaline shift in blood pH

# RENAL BUFFER

## Renal Excretion of Acid, Sodium/Hydrogen Ion Exchange and Formation of Ammonia




## Renal Reclamation of Bicarbonate



# ANOTHER BUFFERING SYSTEMS

## Phosphate Buffer

- Major intracellular buffer
- $H^+ + HPO_4^{2-} \leftrightarrow H_2PO4^-$
- $OH^- + H_2PO_4^- \leftrightarrow H_2O + H_2PO_4^{2-}$

## Protein Buffers

- Includes hemoglobin, work in blood
- Carboxyl group gives up $H^+$
- Amino Group accepts $H^+$
- Side chains that can buffer $H^+$ are present on 27 amino acids.

# TYPES OF ACID-BASE IMBALANCE

- RespiratoryAcidosis
- RespiratoryAlkalosis
- MetabolicAcidosis
- MetabolicAlkalosis

# Acid Base Disorders

| Disorder | pH | $[H^+]$ | Primary disturbance | Secondary response |
|---|---|---|---|---|
| **Metabolic acidosis** | ↓ | ↑ | ↓ $[HCO_3^-]$ | ↓ $pCO_2$ |
| **Metabolic alkalosis** | ↑ | ↓ | ↑ $[HCO_3^-]$ | ↑ $pCO_2$ |
| **Respiratory acidosis** | ↓ | ↑ | ↑ $pCO_2$ | ↑ $[HCO_3^-]$ |
| **Respiratory alkalosis** | ↑ | ↓ | ↓ $pCO_2$ | ↓ $[HCO_3^-]$ |

# HOW TO SOLVE ACID-BASE PROBLEMS :

1-Check the pH

pH <7.35 = acidosis

pH >7.45 = alkalosis

2-Check the $HCO_3^-$ and $pCO_2$

3-Determine acid-base disorder

Acidosis + ↓ $HCO_3^-$ = metabolic acidosis

Acidosis + ↑$pCO_2$ = respiratory acidosis

Alkalosis + ↑$HCO_3^-$ = metabolic alkalosis

Alkalosis+ ↓ $pCO_2$ = respiratory alkalosis

4-For metabolic acidosis only: Calculate anion gap

# (I) Respiratory Acidosis

- It is defined as a pH less than 7.35 with a $PaCO_2$ greater than 45 mmHg.
- Acidosis is the accumulation of $co_2$ which combines with water in the body to produce **carbonic acid**, thus lowering the pH of the blood.

| ABG | pH | $PaCO_2$ | $HCO_3$ |
|---|---|---|---|
| Respiratory Acidosis |  |  | normal |

**Renal compensation (increased reabsorption of HCO3)** begins within **12 to 24 hours and takes 5 days or so to complete.**

# A. Acute respiratory acidosis.

There is an **immediate** compensatory elevation of HCO3

There is an **increase of 1 mmol/L for every 10** mm Hg increase in PaCO2.

# b. Chronic respiratory acidosis.

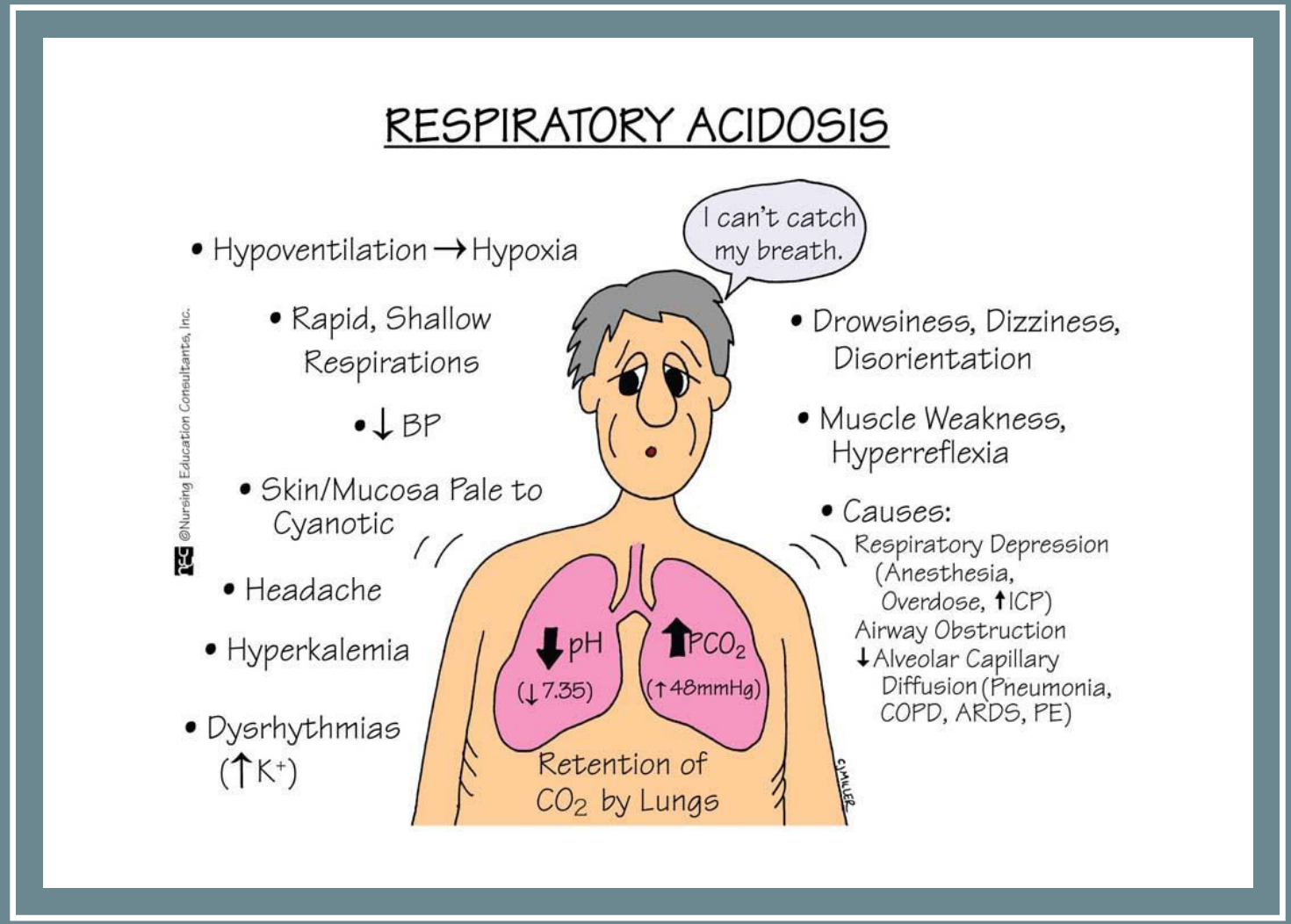
Renal adaptation occurs, and HCO3 − increases **by 4 mmol/L for every 10** mm Hg increase in PaCO2. This is generally seen in patients with underlying lung disease, such as chronic obstructive pulmonary disease **(COPD).**

# CAUSES OF RESPIRATORY ACIDOSIS: (ANYTHING CAUSE HYPOVENTILATION))

1. *Primary pulmonary diseases—for example,* ***COPD, airway obstruction***
2. *Neuromuscular diseases—for example,* ***myasthenia gravis***
3. *CNS malfunction—****injury to brainstem***
4. *Drug-induced hypoventilation (e.g., from* ***morphine, anesthetics, or sedatives****)*
5. ***Respiratory muscle fatigue***

# CLINICAL MANIFESTATION

- Muscular Weakness
- Tachypnea
- Blurred Vision
- Confusion
- Memory loss
- Restlessness



# MANAGEMENT

➢ ***Pharmacological:-***

- ***Opoid Antagonist:- Nalaxone***
- ***β2 Antagonist:- Formoterol, Albuterol***
- ***Anticholinergic:- Ipratropium bromide***
- ***Corticosteroids:Prednisone,Prednisolone***

## *NON PHARMACOLOGICAL :-*

*1) Dialysis:- To clear the toxin from blood.*

*2) Oxygen Therapy:- To minimize the chance of hypoxemia. If paO2 <60 mm Hg*

*3)Ventilator Support:- To subside the respiratory problem.*

- Patient is a 60-year-old male with pneumonia, admitted with dyspnea, fever and chills.
  - pHa = 7.28 (normal range 7.35 - 7.45)
  - $PaCO_2$ = 56 (35 - 45 mm Hg)
  - $PaO_2$ = 70 (68 - 92 mm Hg)
  - $[HCO_3]$ = 25 (22 - 26 mmol/L)
  - $SaO_2$ = 89% (95-100%)

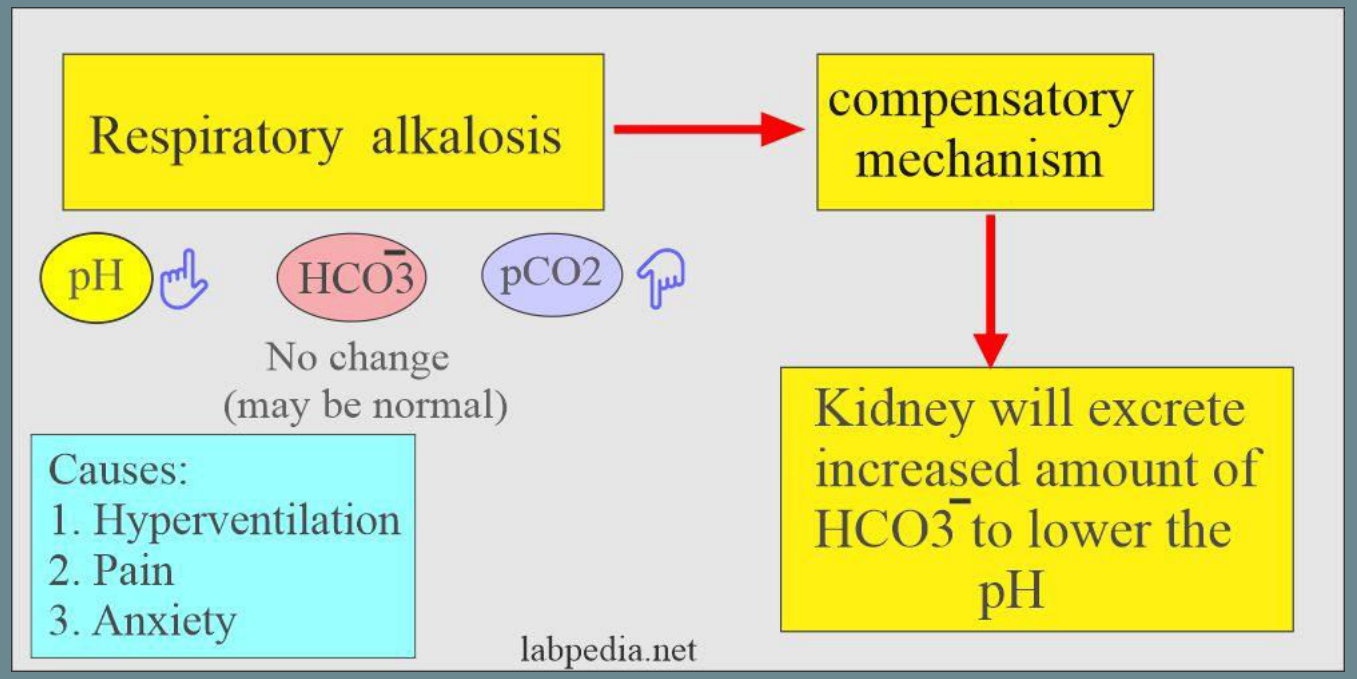
# #ANS

- uncompensated Respiratory acidosis Because pH is <7.4, with PaCO2>40 mmHg ,also we can tell that the disturbance is not compensated, because HCO3- should be higher than it is

# RESPIRATORY ALKALOSIS

Increased **pH** ( alkalosis)

Reduced **$pCO_2$** (respiratory)



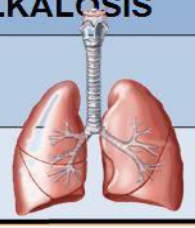
Respiratory alkalosis is usually caused by over-breathing (called hyperventilation) that occurs when you breathe very deeply or rapidly.

- Anxiety
- Pulmonary embolism , Pneumonia , Asthma
- Hypoxia
- Pregnancy
- Sepsis
- Mechanical ventilation
- High altitude
- Aspirin overdose

*In order to maintain blood pH within the normal range, **$HCO_3$ must decrease**, so **renal compensation occurs** (i.e., **$HCO_3$ excretion increases**). However, this does not occur acutely, but rather over the course of **several hours.***

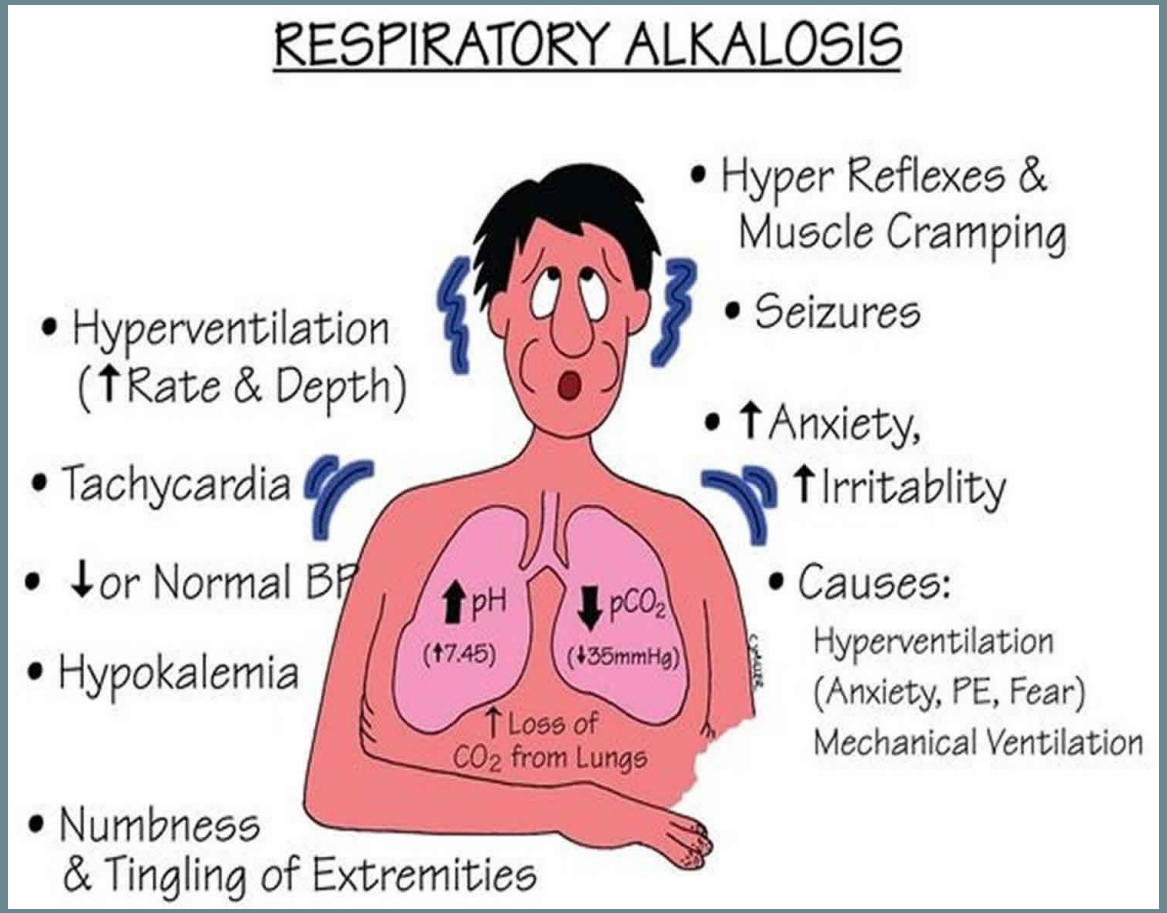
If there is reduction in bicarbonate this means renal compensation is happening which could be partial or complete

| **RESPIRATORY ALKALOSIS** | | |
|---|---|---|
| **Acute** | ↓ [$HCO_3^-$] | 2 mmol/L/10mmHg decrease in $PaCO_2$ |
| **Chronic** | ↓ [$HCO_3^-$] | 4 mmol/L/10mmHg decrease in $PaCO_2$ |



# CLINICAL MANIFESTATION

- Palpitation
- Tetany (muscle spasm)
- Convulsion
- Paralysis
- Chest pain



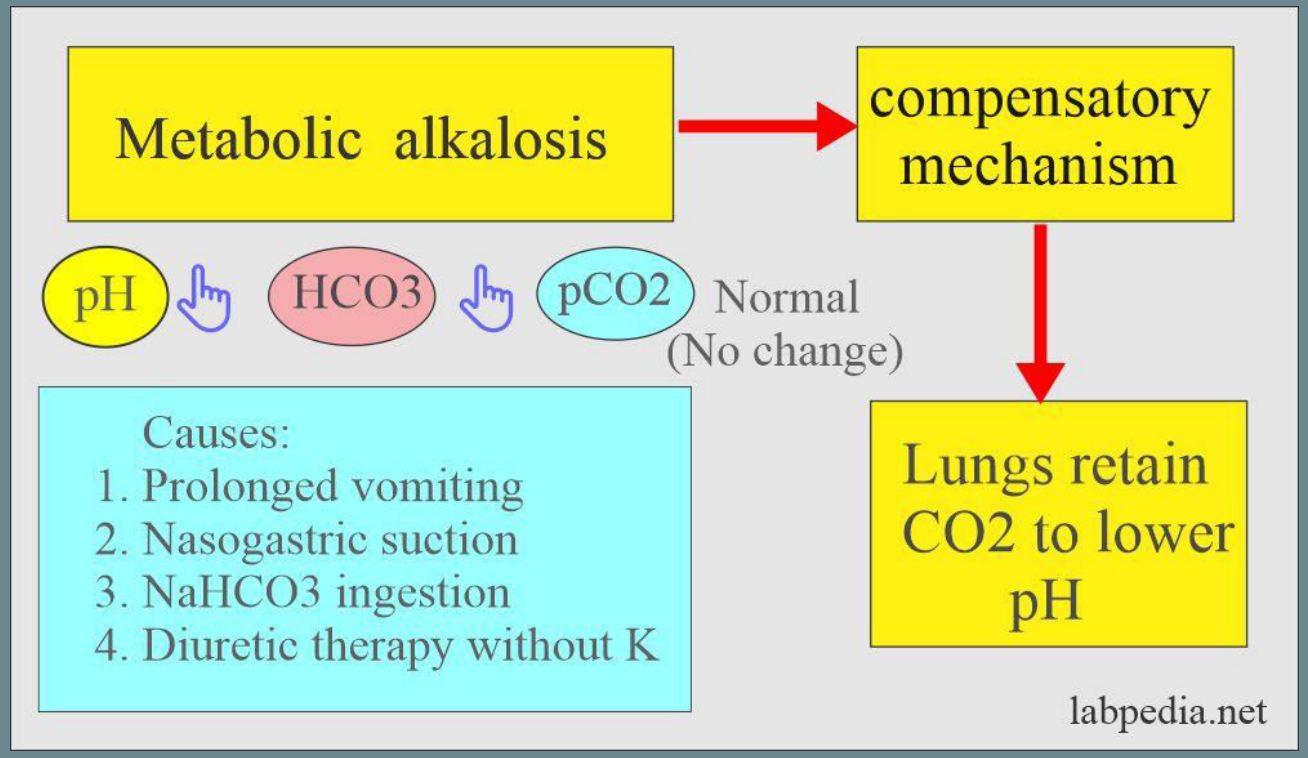
# MANAGEMENT

- ***pharmacological*:-**
  - *Antibiotic: Levofloxin*
  - *Antipyretic: Acetaminophen*
- ***Non-pharmacological*:-**
  - *Breath into a paper bag.*
  - *Restrict oxygen intake into the lungs.*

# METABOLIC ALKALOSIS

Increased **Ph** (alkalosis)

Increased **HCO3-** (metabolic)



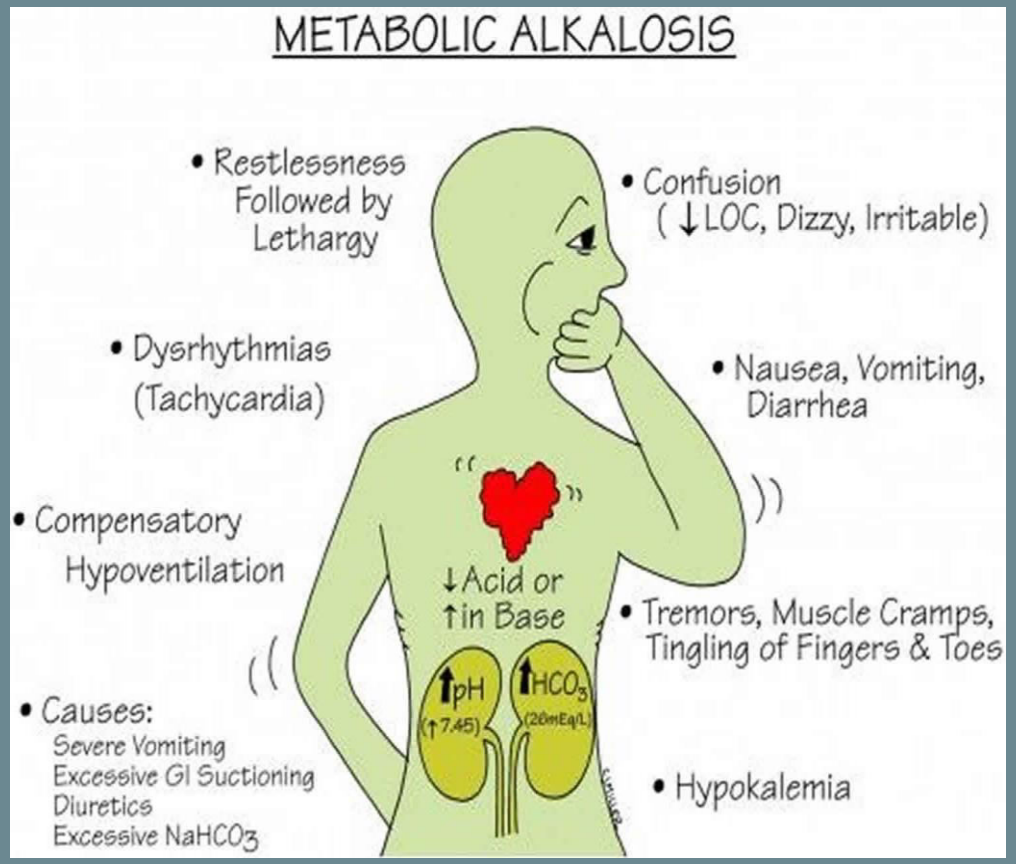

**To compensate the metabolic alkalosis respiration will be reduced in terms of rate and depth ( hypoventilation) which leads to hypercapnia ( increased pCO2 )**

# CAUSES

- Alcohol abuse
- Hyperaldosteronism
- Vomiting
- High fever
- Diuretic therapy
- Cystic fibrosis
- Hypokalemia

# CLINICAL MANIFESTATION

- **Headache lethargy & increase excitability.**
- **Delirium, Tetany and Seizure.**
- **Anginal symptoms**
- **Arrhythmia due to *hypokalemia***

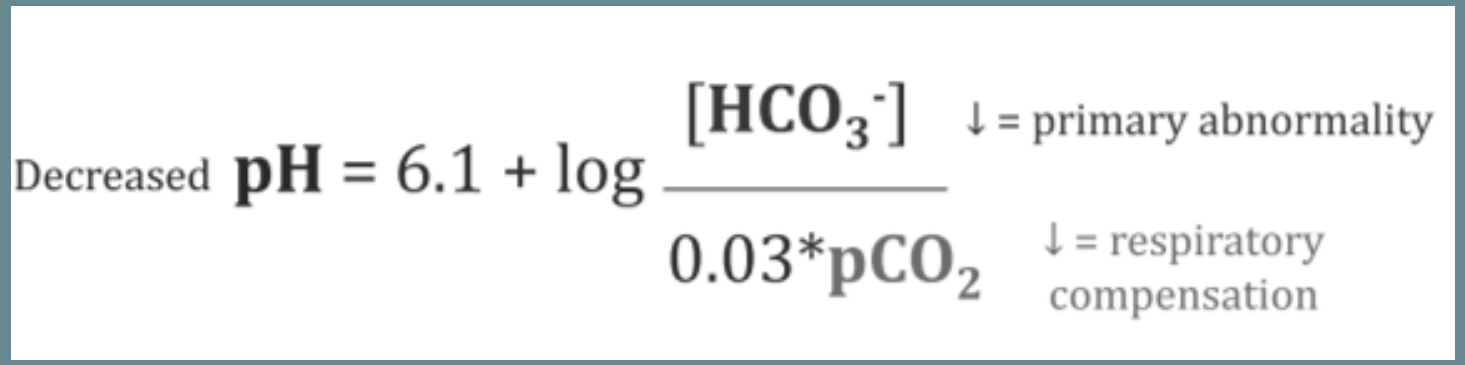


# MANAGEMENT

- Potassium sparing diuretics: Amiloride
- Acetazolamide
- NaCl fluid administration

# METABOLIC ACIDOSIS

- **Reduced pH (acidosis)**
- **Reduced HCO3- (metabolic acidosis)**
- **pCO2 ↓ = respiratory compensation**



# COMPENSATION

- Respiratory compensation to metabolic acidosis is Hyperventilation (respiratory alkalosis) which :

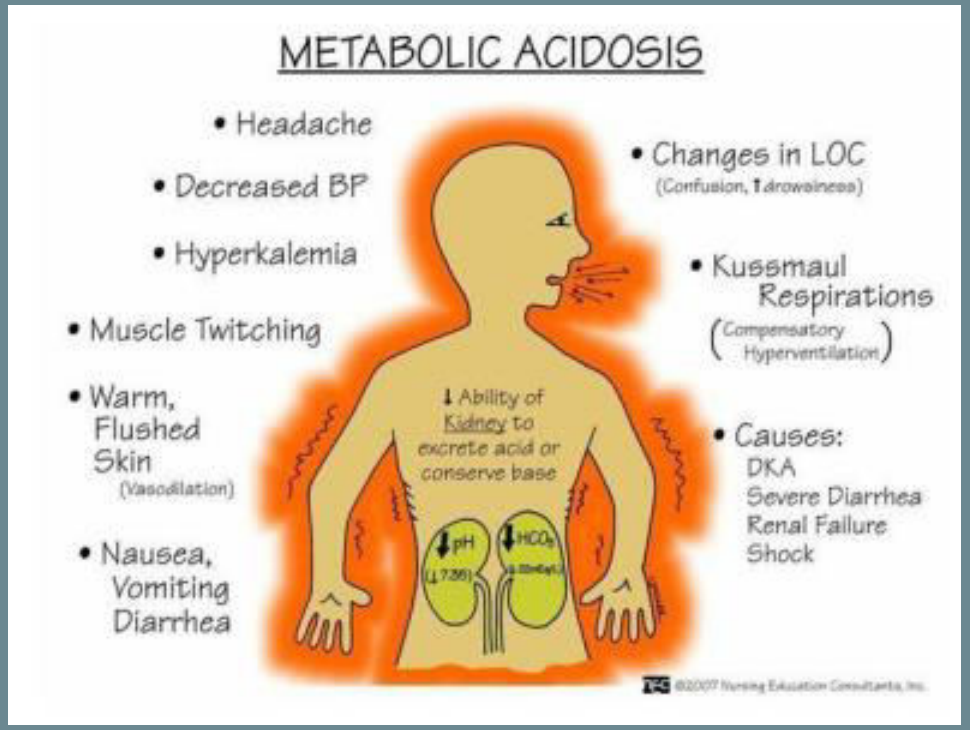
1. Lowers pCO2
2. Increases pH

- Winter's Formula gives expected pCO2
- If actual CO2 ≠ expected →mixed disorder

$$pCO2 = 1.5\ (HCO3\ -) + 8 +/- 2$$

# CLINICAL MANIFESTATION

- Rapid deep breathing (Kussmaul respirations)
- Hypotension
- Diarrhea
- Chest pain
- Arrhythmia
- Joint pain



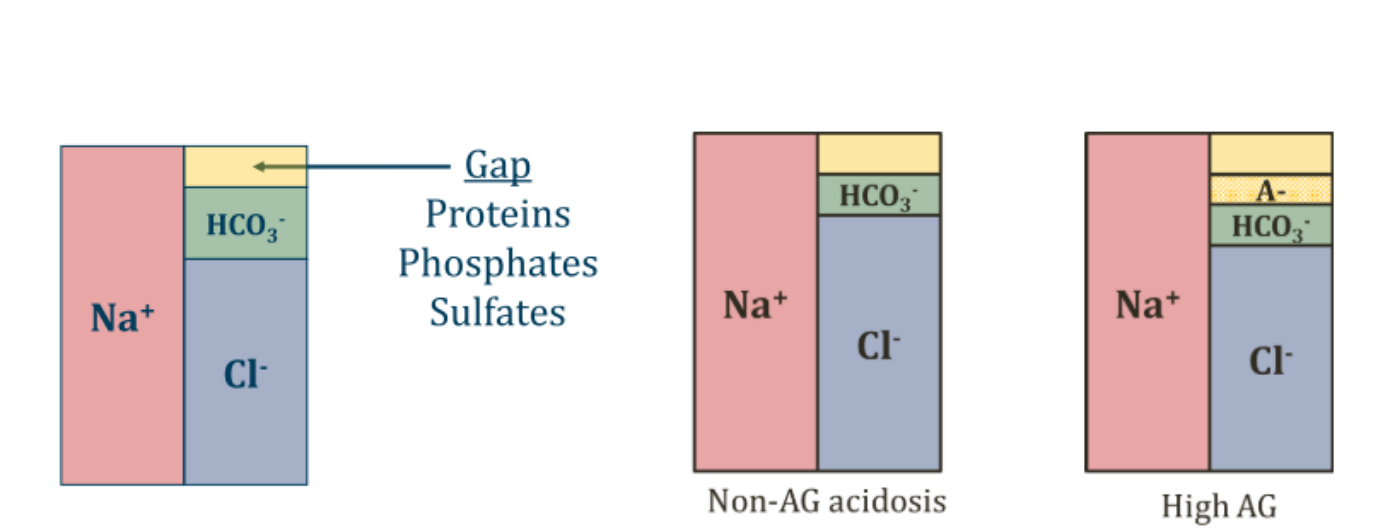
# THE ANION GAP

- Anion Gap = Na – (Cl- + HCO3-)

For example, if (Na+) 140 mEq/L , (Cl-) 103 mEq/L and (HCO3 -) 17

Anion Gap = 140 – (103 + 17) = 20 ( wide AG)

- Normal: 8 to 12




- Causes of metabolic acidosis categorized by anion gap:

1. Normal anion gap (Hyperchloremic metabolic acidosis )
2. Increased anion gap

due to kidney response to increase Cl reabsorption to maintain electroneutrality .

# NON-ANION GAP METABOLIC ACIDOSIS

- **Diarrhea**
- Loss of $HCO_3^-$ in stool
- Occurs with any fluid loss from bowel (fistula)
- **NaCl (saline) infusion**
- Influx of chloride ions ($Cl^-$)
- Shift of bicarbonate ions ($HCO_3^-$) into cells

- **Acetazolamide**
- **Loss of aldosterone effects**
- **Renal tubular acidosis**

→ Diagnosis by history, exam, labs
→ Treat underlying cause

# ANION GAP METABOLIC ACIDOSIS

- **M**ethanol
- **U**remia
- **D**iabetic ketoacidosis
- **P**ropylene glycol
- **I**ron tablets or INH
- **L**actic acidosis
- **E**thylene glycol
- **S**alicylates

MUD PILES

# 1.METHANOL, 2.ETHYLENE GLYCOL AND 3.PROPYLENE GLYCOL

- All are Solvents found in many products
- Methanol Metabolized to formic acid

  → Diagnosis: serum methanol level
  → Treatment: by Inhibition of alcohol dehydrogenase : Fomepizole, Ethanol or dialysis

- Ethylene Glycol Metabolized to glycolate and oxalate (Kidney toxins)

  → Diagnosis: serum ethylene glycol
  → Treatment: by Inhibition of alcohol dehydrogenase: Fomepizole Ethanol or Dialysis

- Propylene Glycol is Metabolized to pyruvic acid, acetic acid, lactic acid
  - Solvent for IV benzodiazepines

  → Treatment: dialysis

Methanol

**Classic scenario:**
- **Suspected ingestion**
- **Confusion**
- **Visual symptoms**

Ethylene Glycol

**Classic scenario:**
- **Suspected ingestion**
- **Flank pain, oliguria, anorexia (acute renal failure)**

Propylene Glycol

- Main clinical feature of overdose is CNS depression
- -No visual symptoms or nephrotoxicity

# OSMOLAR GAP

- Serum osmolarity mostly determined by sodium, glucose and urea
- Calculated osmolarity = (2 x [Na+]) + [glucose]/18 + [urea]/2.8)
- Osmolar Gap = measured osmolarity – calculated osmolarity
- Normal =< 10
- Causes of elevated osmolar gap:
- Mannitol
- **Methanol**
- **Ethylene glycol**

**4. Uremia:** in Advanced kidney disease as kidneys cannot excrete organic acids

Ttt : dialysis

**5. Diabetic Ketoacidosis:** in type I diabetics → when Insulin requirements cannot be met → Fatty acid metabolism instead occur producing ketone bodies

**6. Lactic Acidosis :**Low tissue oxygen delivery → production of lactate (lactic acidosis )

Clinical scenarios: TTT

- Shock (↓ tissue perfusion) → fluids, vasopressors, inotropes
- Ischemic bowel → surgery
- Metformin therapy (especially with renal failure) → stop drug
- Seizures → Post-ictal acidosis: no treatment required

Ttt by treating the underlying condition

**7. Iron :** hypoperfusion+ ferric irons (Fe3+) → Anion-gap metabolic acidosis

- Dx: serum iron level
- Ttt: GI decontamination or Defuroxamine

**8. Isoniazid**

**9. Aspirin Overdose :** Two acid-base disorders

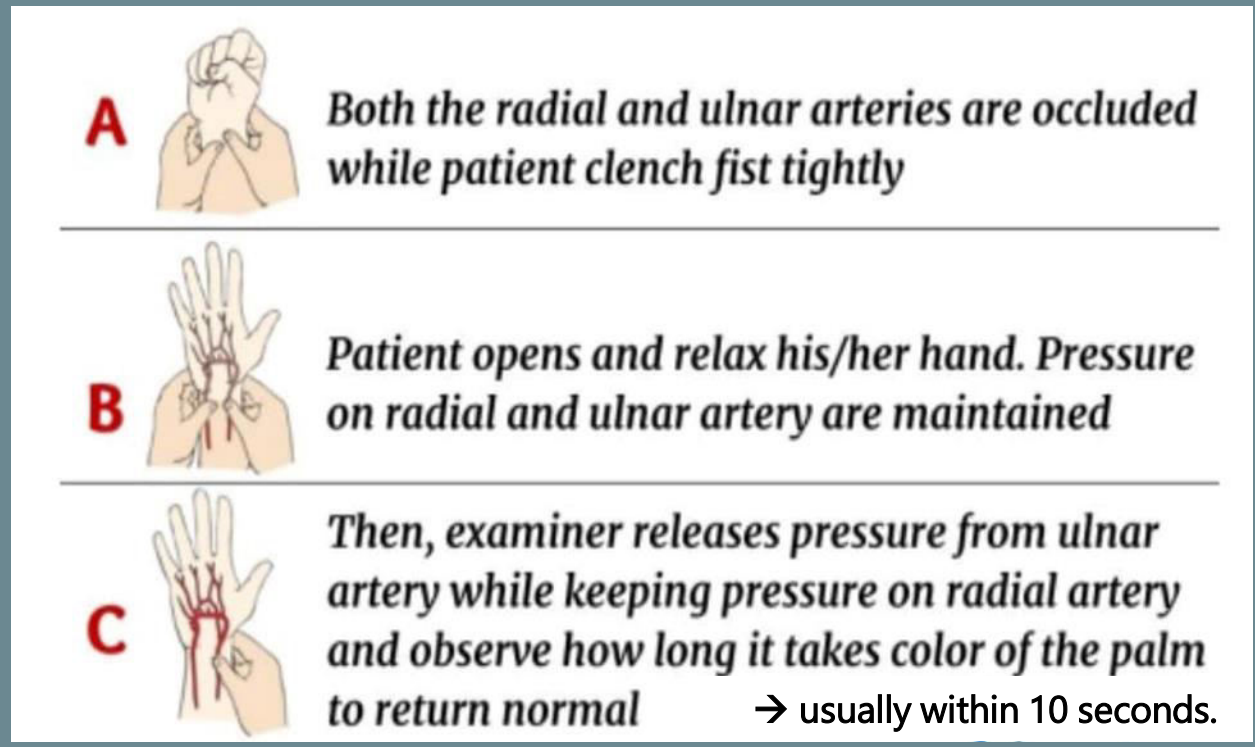
- Shortly after ingestion: respiratory alkalosis •
- Hours after ingestion: AG metabolic acidosis due to acid Accumulation
- Dx: serum salicylate level
- Ttt: urinary alkalinization by the administration of bicarbonate

# COMPLICATION OF ABG

- Bleeding
- Infection at puncture site
- Blood accumulating under skin
- Local pain
- Thrombus in artery
- Feeling faint
- Numbness of hand

# CONTRAINDICATION OF ABG

- Coagulopathy
- Artherosclerosis
- Infection at insertion site
- Abnormal modified Allen's test
- Use of thrombolytic agent




* The test is repeated on the same hand while releasing the radial artery first and continuing to compress the ulnar artery if evaluation of radial collateral blood flow is required

# SCENARIO 1

- A 63-year-old female who was admitted with shortness of breath. On your arrival, the patient appears drowsy and is on 10L of oxygen via a mask.
- You perform an ABG, which reveals the following results:
- **$PaO_2$:** 52.5 mmHg (82.5 – 97.5 mmHg)
- **pH:** 7.29 (7.35 – 7.45)
- **$PaCO_2$:** 68.2 mmHg (35.2 – 45 mmHg)
- **$HCO_3^-$:** 26 (22 – 26 mEq/L)

# ANS : UNCOMPENSATED RESPIRATORY ACIDOSIS

- pH reveals an acidosis
- the $PaCO_2$ is raised significantly and this is likely to be the cause of the acidosis.
- The **$HCO_3^-$** is normal, so the metabolic system is not contributing to the acidosis and also isn't compensating for the respiratory acidosis

# SCENARIO 2

- A 22-year-old female is brought into A&E by ambulance with a 5-day history of vomiting and lethargy. When you begin to talk with the patient you note that she appears disorientated and looks clinically dehydrated. At present, you are unable to gain any further details, but the patient looks very unwell from the end of the bed. You gain IV access, send off a routine panel of bloods and commence some fluids. You ask the nurse to check the patient's observations and she notes an increased respiratory rate, low blood pressure and tachycardia. You perform an ABG on the advice of your registrar. The results of the ABG are shown below (the patient was not on oxygen when this was taken).

- PaO2: 97.5 mmHg (82.5 – 97.5 mmHg)
- pH: 7.3 (7.35 – 7.45)
- PaCO2: 30.7 mmHg (35.2 – 45 mmHg)
- HCO3-: 13 (22 – 26 mEq/L)

# METABOLIC ACIDOSIS WITH PARTIAL RESPIRATORY COMPENSATION

- pH acidotic
- $HCO_3^-$ is low, which is in keeping with an acidosis, so the metabolic system is the cause of this patient's acidosis.
- The $CO_2$ is actually low and therefore the respiratory system doesn't appear to be contributing to the acidosis
- Is there a compensation ?

$pCO_2 = 1.5 (HCO_3^-) + 8 +/- 2$

1.5 (13 ) + 8 = 27.5 (+/- 2) ( 25.5-29.5) so partial compensation