

Arterial Blood Gas Analysis

Dr Umar



Overview

ABG Sampling


Interpretation of ABG

- Gas Exchange

- Acid Base status



Applications of ABG

- To document respiratory failure and assess its severity
 - To monitor patients on ventilators and assist in weaning
 - To assess acid base imbalance in critical illness
 - To assess response to therapeutic interventions and mechanical ventilation
 - To assess pre-oppatients
- 

ABG – Procedure and Precautions

Where to place -- the options



Radial
Dorsalis Pedis
Femoral
Brachial

Technical Errors

Excessive Heparin

- Ideally : Pre-heparinised ABG syringes
- Syringe **FLUSHED** with 0.5ml 1:1000 Heparin & emptied
- **DO NOT LEAVE EXCESSIVE HEPARIN IN THE SYRINGE**

↑ HEPARIN

↑ DILUTIONAL
EFFECT

↓ HCO_3^-
↓ pCO_2

ABG Syringe



Technical Errors

- Risk of alteration of results ↑ with:
 - 1) ↑ **size** of syringe/needle
 - 2) ↓ **vol** of sample

- ✓ Syringes must have > 50% blood
- ✓ Use only 3ml or less syringe
- ✓ 25% lower values if 1 ml sample taken in 10 ml syringe (0.25 ml heparin in needle)

Technical Errors

Air Bubbles

➤ pO_2 150 mm Hg & pCO_2 0 mm Hg

□ **Contact with AIR BUBBLES**

↑ **pO_2** & ↓ **pCO_2**

➤ Seal syringe immediately after sampling

Body Temperature

➤ Affects values of **pCO_2** and **HCO_3^-** only

➤ ABG Analyser controlled for Normal Body temperatures

Technical Errors

WBC Counts

- 0.01 ml O₂ consumed/dL/min
- Marked increase in high TLC/plt counts : ↓ pO₂
- Chilling / immediate analysis

ABG Syringe must be transported earliest via COLD CHAIN

Change/10 min	Uniced 37°C	Iced 4°C
pH	0.01	0.001
pCO ₂	1 mm Hg	0.1 mm Hg
pO ₂	0.1%	0.01%



ABG Equipment

- ❑ **3 electrode system** that measures three fundamental variables - **pO₂**, **pCO₂** and **pH**
- ❑ All others parameters such as HCO₃⁻ computed by software using standard formulae



Interpretation of ABG

- Gas exchange
 - Acid Base Status
- 



Gas exchange



Assessment Of Gas exchange

- PaO₂ vs SpO₂
- Alveolar-arterial O₂ gradient
- PaO₂/FiO₂ ratio
- PaCO₂

Determinants of PaO₂

PaO₂ is dependant upon \longrightarrow Age, FiO₂, P_{atm}

As Age \uparrow the expected PaO₂ \downarrow

- $\text{PaO}_2 = 109 - 0.4 (\text{Age})$

As FiO₂ \uparrow the expected PaO₂ \uparrow

- Alveolar Gas Equation:

- $\text{P}_A\text{O}_2 = (\text{P}_B - \text{P}_{\text{H}_2\text{O}}) \times \text{FiO}_2 - \text{pCO}_2/\text{R}$



Hypoxemia

- Normal PaO₂ : 95 – 100 mm Hg
- Mild Hypoxemia : PaO₂ 60 – 80 mm Hg
- Moderate Hypoxemia : PaO₂ 40 – 60 mm Hg
– tachycardia, hypertension, cool extremities
- Severe Hypoxemia : PaO₂ < 40 mm Hg –
severe arrhythmias, brain injury, death

Alveolar-arterial O₂ gradient

- P(A-a)O₂ is the alveolar-arterial difference in partial pressure of oxygen
- $PAO_2 = 150 - PaCO_2/RQ$
- Normal range : 5 - 25 mm Hg (increases with age)
- Increase P(A-a)O₂ : lung parenchymal disease

PaO_2 / FiO_2 ratio

Inspired Air $FiO_2 = 21\%$

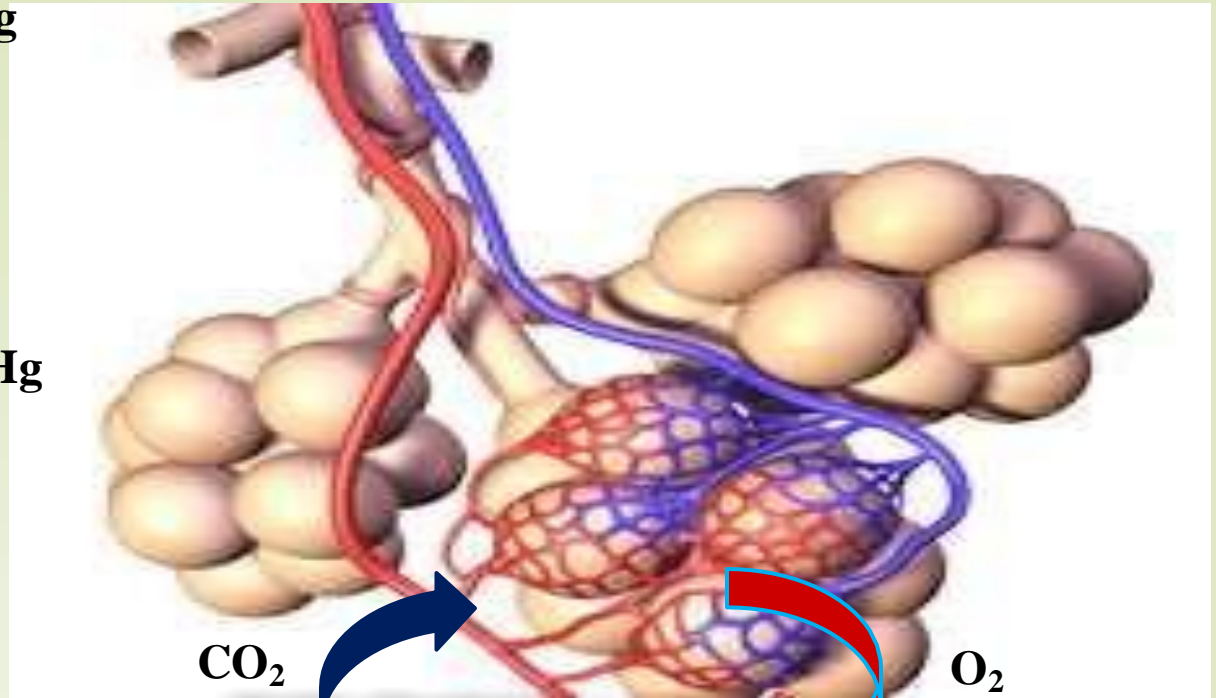
$PiO_2 = 150$ mmHg



$P_{alv}O_2 = 100$ mmHg



$PaO_2 = 90$ mmHg




Berlin criteria for ARDS severity

<i>PaO₂/ FiO₂ ratio</i>	<i>Inference</i>
200 - 300 mm Hg	Mild ARDS
100 - 200 mm Hg	Moderate ARDS
< 100 mm Hg	Severe ARDS

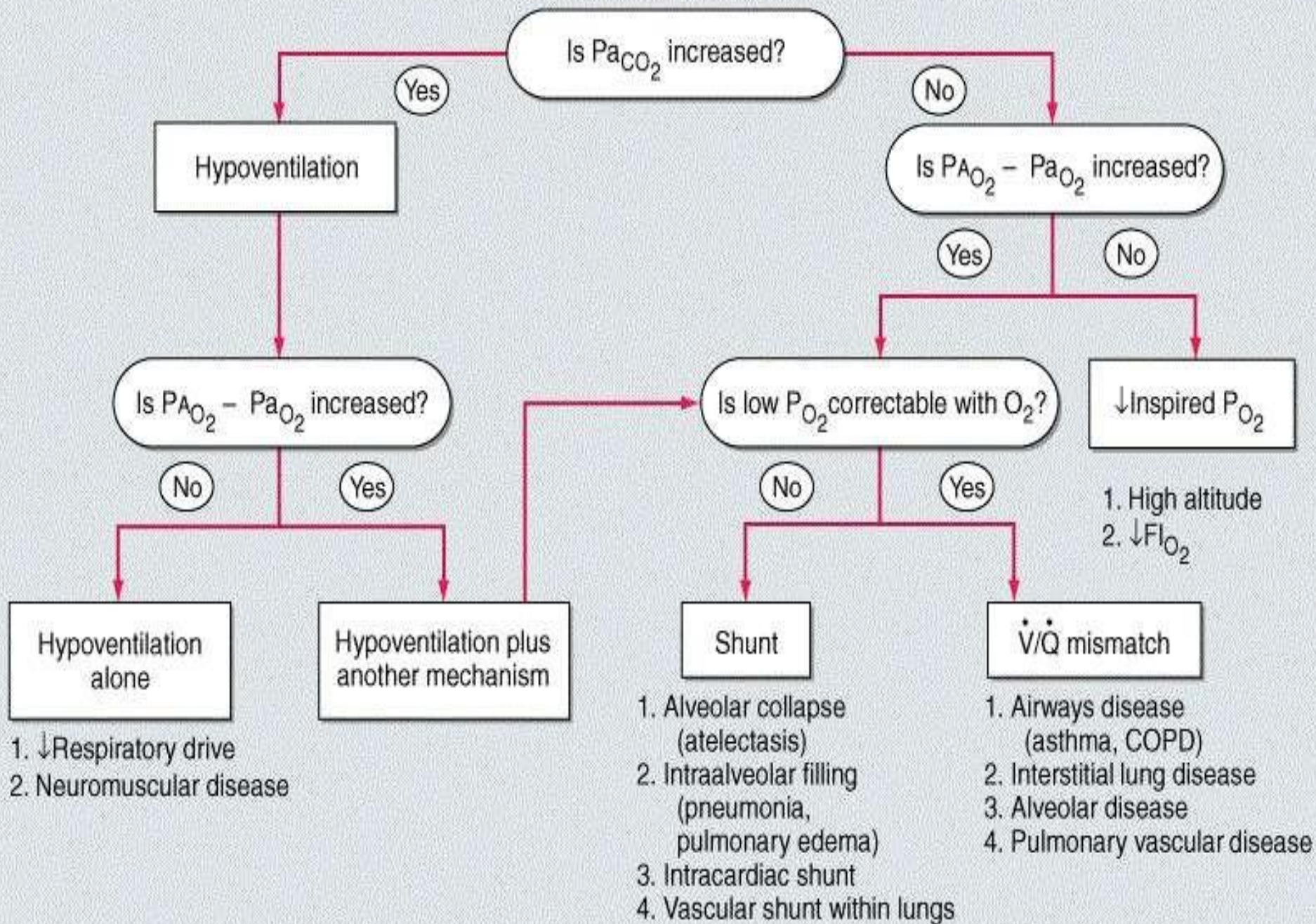
ARDS is characterized by an acute onset within 1 week, bilateral radiographic pulmonary infiltrates, respiratory failure not fully explained by heart failure or volume overload, and a PaO₂/FiO₂ ratio < 300 mm Hg



Hypercapnia

- PaCO₂ is directly proportional to CO₂ production and inversely proportional to alveolar ventilation
 - Normal PaCO₂ is 35 – 45 mm Hg
- 

APPROACH TO PATIENT WITH HYPOXEMIA





Acid Base Status

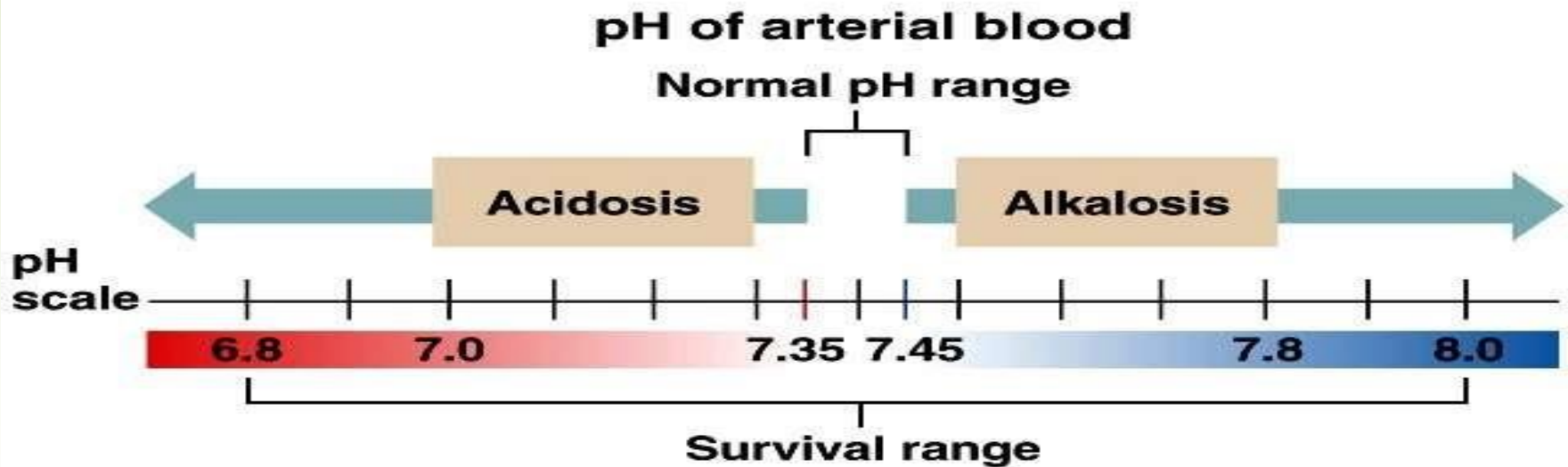
Basics

- Nano equivalent = 1×10^{-9}

$pH = -\log [H^+]$: Sorensen formula

- $[H^+] = 40 \text{ nEq/L}$ (16 to 160 nEq/L) at pH-7.4

Copyright © The McGraw-Hill Companies, Inc. Permission required for reproduction or display.



Henderson-Hasselbalch Equation

- Correlates metabolic & respiratory regulations

$$\text{pH} = \text{pK} + \log \frac{\text{HCO}_3^-}{.03 \times [\text{PaCO}_2]}$$

- *Simplified*

$$\text{pH} \sim \frac{\text{HCO}_3^-}{\text{PaCO}_2}$$

First line of defense against pH shift

Chemical buffer system

Bicarbonate buffer system

Phosphate buffer system

Protein buffer system

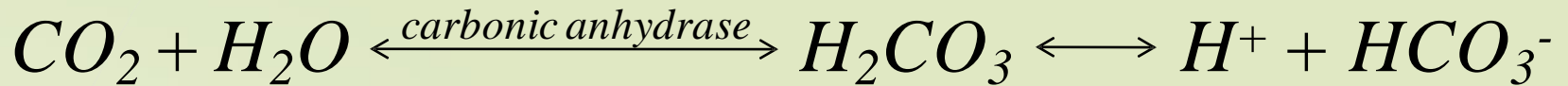
Second line of defense against pH shift

Physiological buffers

Respiratory mechanism (CO₂ excretion)

Renal mechanism (H⁺ excretion)

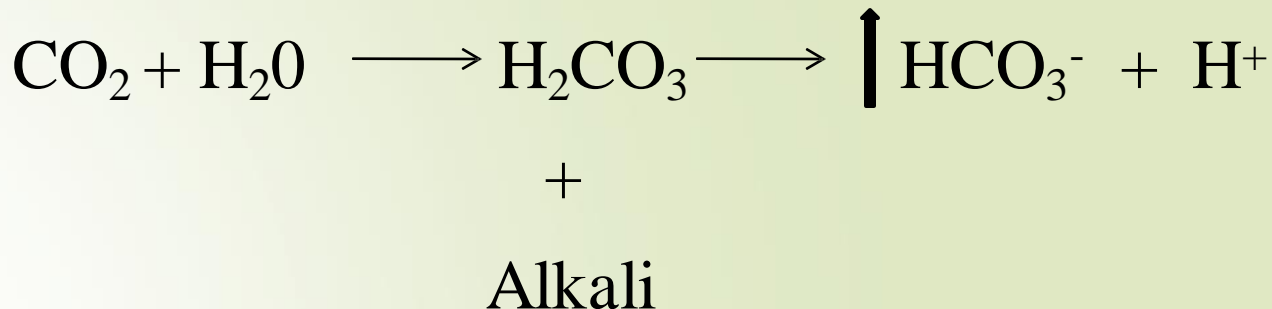
Bicarbonate Buffer System



Acidosis : Acid = H⁺

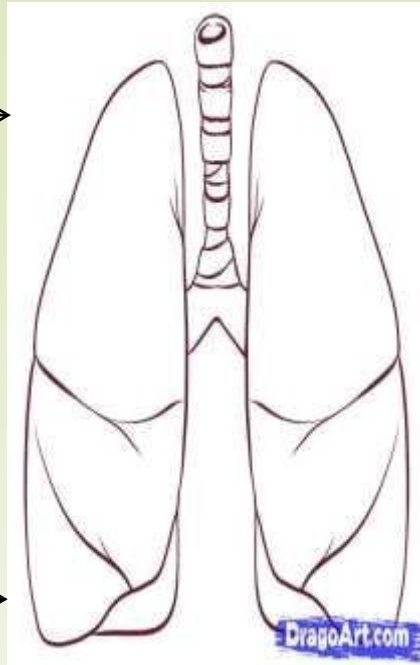


Alkalosis : Alkali + Weak Acid = H₂CO₃

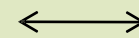


Respiratory Regulation

↑ H^+

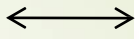


ALVEOLAR
VENTILATION

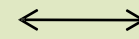


$PaCO_2$ ↓

↓ H^+



ALVEOLAR
VENTILATION

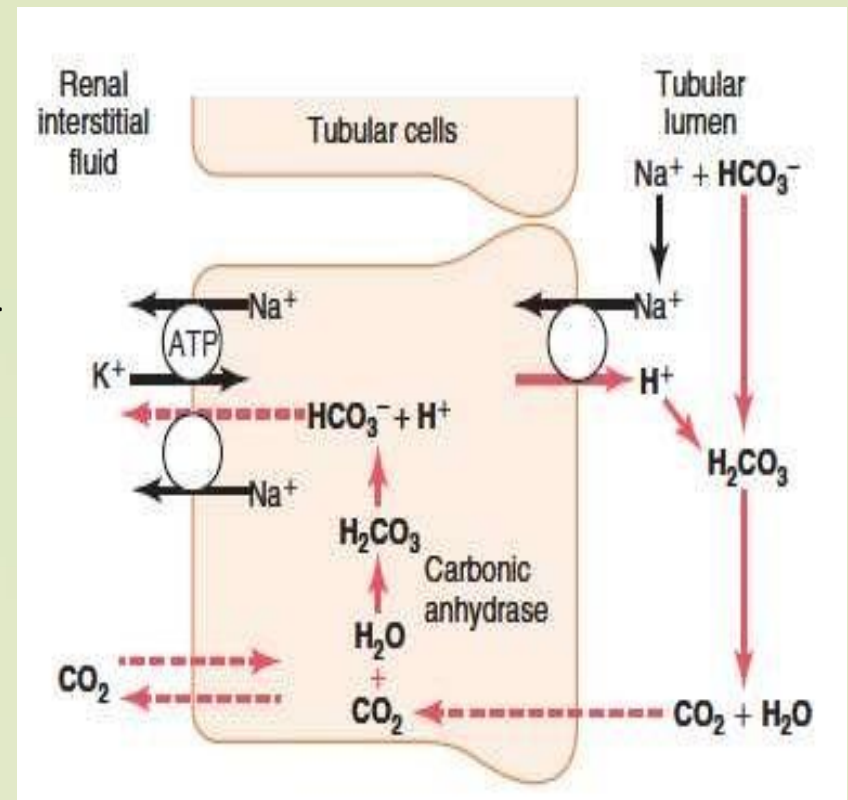


$PaCO_2$ ↑

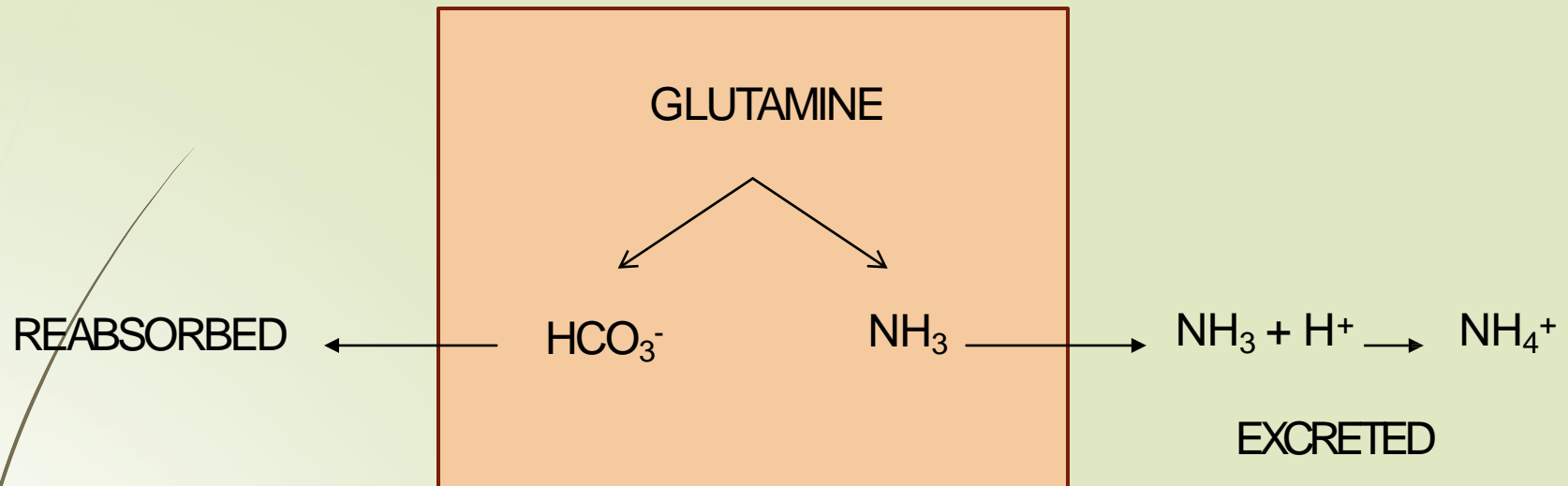
Renal Regulation

Kidneys control the acid-base balance by excreting either a basic or an acidic urine

- Excretion of HCO_3^-
- Regeneration of HCO_3^- with excretion of H^+



Excretion of excess H^+ & generation of new HCO_3^- : The Ammonia Buffer System



- In **chronic acidosis**, the dominant mechanism of acid eliminated is **excretion of NH_4^+**



Response...

Bicarbonate Buffer System

- Acts in few seconds

Respiratory Regulation

- Starts within minutes good response by 2hrs, complete by 12-24 hrs

Renal Regulation

- Starts after few hrs, complete by 5-7 days

Abnormal Values

pH < 7.35

- Acidosis (metabolic and/or respiratory)

pH > 7.45

- Alkalosis (metabolic and/or respiratory)

paCO₂ > 45 mm Hg

- Respiratory acidosis (alveolar hypoventilation)

paCO₂ < 35 mm Hg

- Respiratory alkalosis (alveolar hyperventilation)

HCO₃⁻ < 22 meq/L


- Metabolic acidosis

HCO₃⁻ > 26 meq/L

- Metabolic alkalosis



Simple Acid-Base Disorders



Simple acid-base disorder – a single primary process of acidosis or alkalosis with or without compensation

Compensation...

The body always tries to normalize the pH so...

- $p\text{CO}_2$ and HCO_3^- rise & fall together in simple disorders
- Compensation never overcorrects the pH
- Lack of compensation in an appropriate time defines a 2nd disorder
- Require normally functioning lungs and kidneys

Characteristics of 1° acid-base disorders

DISORDER	PRIMARY RESPONSE			COMPENSATORY RESPONSE
Metabolic acidosis	↑ [H ⁺]	↓ PH	↓ HCO ₃ ⁻	↓ pCO ₂
Metabolic alkalosis	↓ [H ⁺]	↑ PH	↑ HCO ₃ ⁻	↑ pCO ₂
Respiratory acidosis	↑ [H ⁺]	↓ PH	↑ pCO ₂	↑ HCO ₃ ⁻
Respiratory alkalosis	↓ [H ⁺]	↑ PH	↓ pCO ₂	↓ HCO ₃ ⁻

Disorder	Compensatory response
<i>Respiratory acidosis</i>	
Acute	↑ HCO ₃ ⁻ 1 mEq/L per 10 mm Hg ↑ pCO ₂
Chronic	↑ HCO ₃ ⁻ 3.5 mEq/L per 10 mm Hg ↑ pCO ₂
<i>Respiratory alkalosis</i>	
Acute	↓ HCO ₃ ⁻ 2 mEq/L per 10 mm Hg ↓ pCO ₂
Chronic	↓ HCO ₃ ⁻ 5 mEq/L per 10 mm Hg ↓ pCO ₂
<i>Metabolic acidosis</i>	↓ pCO ₂ 1.3 mm Hg per 1 mEq/L ↓ HCO ₃ ⁻ (Limit of CO ₂ is 10 mm Hg)
<i>Metabolic alkalosis</i>	↑ pCO ₂ 0.7 mm Hg per 1 mEq/L ↑ HCO ₃ ⁻ (Limit of CO ₂ is 55 mm Hg)

Mixed Acid-base Disorders

Presence of more than one acid base disorder simultaneously

Clues to a mixed disorder:

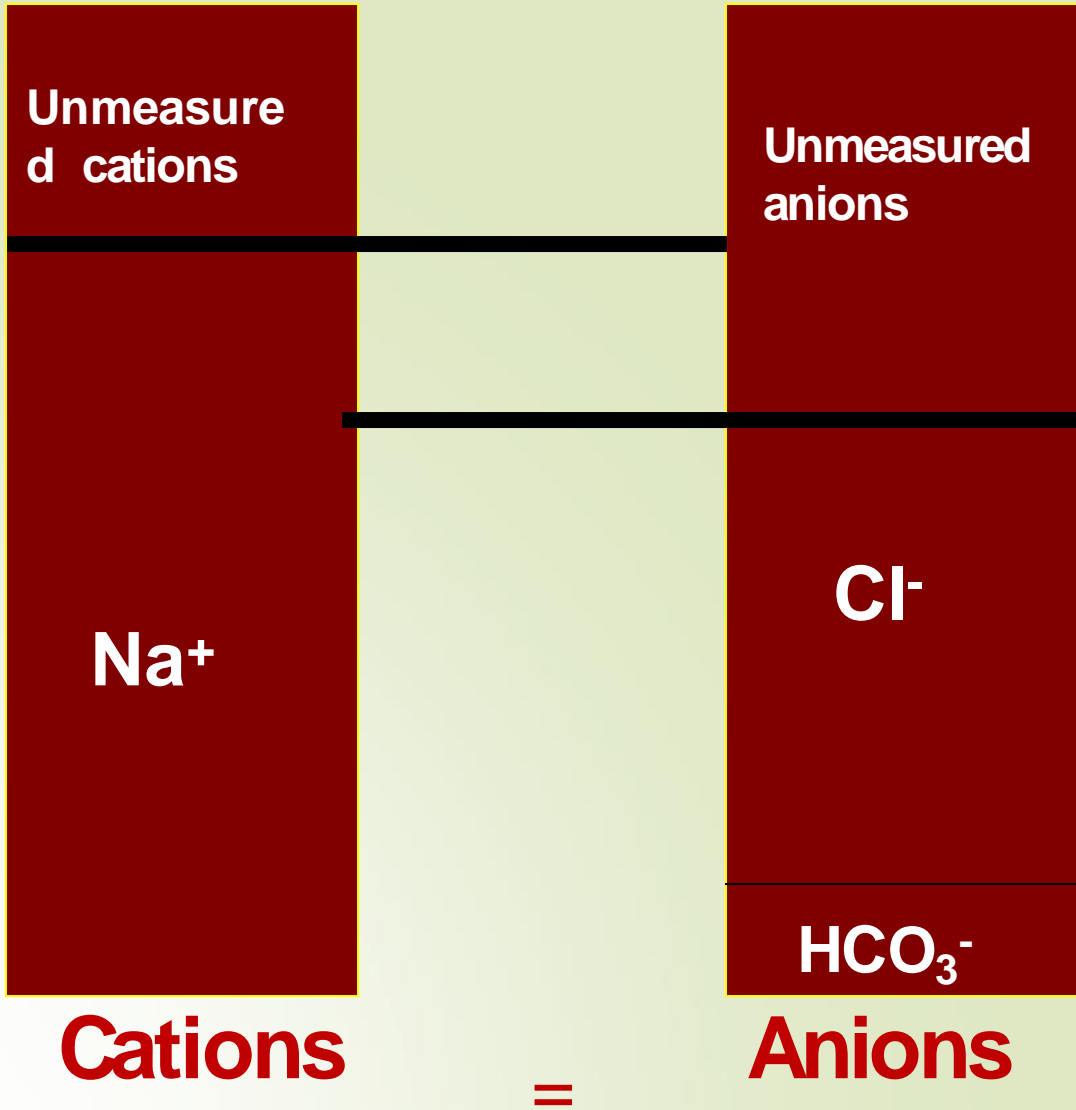
- Normal pH with abnormal HCO_3^- or pCO_2
- pCO_2 and HCO_3^- move in opposite directions
- pH changes in an opposite direction for a known primary disorder

Anion Gap

$$AG = [Na^+] - [Cl^- + HCO_3^-]$$

- Elevated anion gap represents metabolic acidosis
- Normal value: 12 ± 4 mEq/L
- Major unmeasured anions
 - albumin
 - phosphates
 - sulfates
 - organic anions

**Anion Gap =
Metabolic
Acidosis**



Cations

=

Anions



Increased Anion Gap

- Diabetic Ketoacidosis
- Chronic Kidney Disease
- Lactic Acidosis
- Alcoholic Ketoacidosis
- Aspirin Poisoning
- Methanol Poisoning
- Ethylene Glycol Poisoning
- Starvation

Normal Anion Gap

- Diarrhea
- Renal Tubular Acidosis
- Addisons Disease
- Carbonic Anhydrase Inhibitors


Delta Gap

- The difference between patient's AG & normal AG
- The coexistence of 2 metabolic acid-base disorders may be apparent


$$\text{Delta gap} = \text{Anion gap} - 12$$



$$\text{Delta Gap} + \text{HCO}_3^- = 22-26 \text{ mEq/l}$$

- If >26, consider additional metabolic alkalosis
- If <22, consider additional non AG metabolic acidosis




**STEP-BY-STEP ANALYSIS
OF
ACID-BASE STATUS**



- 
- 
1. Look at the ***pO₂*** (<80 mm Hg) and ***O₂ saturation*** (<90%) for ***hypoxemia***




2. Look at the *pH*

- ❑ < 7.35 : ACIDOSIS
 - ❑ > 7.45 : ALKALOSIS
 - ❑ $7.35 - 7.45$: normal/mixed disorder
- 




3. Look at $p\text{CO}_2$

- ❑ > 45 mm Hg : Increased (Acidic)
- ❑ < 35 mm Hg : Decreased (Alkalotic)





4. Look at the HCO_3^-

- ❑ > 26 mEq/L : Increased (Alkalotic)
- ❑ < 22 mEq/L : Decreased (Acidic)




5. Determine the acid-base disorder, match either the $p\text{CO}_2$ or the HCO_3^- with the pH





6. *Compensation...* are the CO_2 or HCO_3^- of opposite type ?





Is the compensation adequate??

METABOLIC DISORDER \longrightarrow $\text{PCO}_{2\text{expected}}$

- $\text{PCO}_{2\text{measured}} \neq \text{PCO}_{2\text{expected}}$ \longrightarrow MIXED DISORDER


RESPIRATORY DISORDER \longrightarrow $\text{pH}_{\text{expected}}$

- $\text{pH}_{\text{m}} \neq \text{pH}_{\text{e range}}$ \longrightarrow MIXED DISORDER



7. Calculate the *anion gap* if it is more there is Metabolic acidosis



$$AG = [Na^+] - [Cl^- + HCO_3^-]$$



8. Does the anion gap explain the change in HCO_3^- ?

Calculate *Delta gap*

(rule out co-existence of 2 acid-base disorders)



9. Examine the patient to determine whether the clinical signs are compatible with the acid-base analysis...

Arterial blood sample

pH < 7.40

pH > 7.40

Acidosis

Alkalosis

[HCO₃⁻] < 24 mEq/L

PCO₂ > 40 mm Hg

[HCO₃⁻] > 24 mEq/L

PCO₂ < 40 mm Hg

Metabolic acidosis

Respiratory acidosis

Metabolic alkalosis

Respiratory alkalosis

PCO₂ < 40 mm Hg

[HCO₃⁻] > 24 mEq/L

PCO₂ > 40 mm Hg

[HCO₃⁻] < 24 mEq/L

Respiratory compensation

Renal compensation

Respiratory compensation

Renal compensation

* 1.2 mm Hg ↓ PCO₂ per 1 mEq/L ↓ in [HCO₃⁻]

* 3.5 mEq/L ↑ [HCO₃⁻] per 10 mm Hg ↑ in PCO₂

* 0.7 mm Hg ↑ PCO₂ per 1 mEq/L ↑ in [HCO₃⁻]

* 5 mEq/L ↓ [HCO₃⁻] per 10 mm Hg ↓ in PCO₂



***Treat the patient
not the ABG!!!***

Thank you...