

Fb/Nurse-Info

OBJECTIVES

ABG Sampling

- Interpretation of ABG
 - Oxygenation status
 - Acid Base status

Case Scenarios

ABG – Procedure and Precautions

Site- (Ideally) Radial Artery Brachial Artery Femoral Artery

Ideally - Pre-heparinised ABG syringes

- Syringe should be **FLUSHED** with 0.5ml of 1:1000 Heparin solution and emptied.

DO NOT LEAVE EXCESSIVE HEPARIN IN THE SYRINGE



- ✓ Only small 0.5ml Heparin for flushing and discard it
- ✓ Syringes must have > 50% blood. Use only 2ml or less syringe.

- Ensure No Air Bubbles. Syringe must be sealed immediately after withdrawing sample.
 - Contact with AIR BUBBLES

Air bubble =
$$PO_2$$
 150 mm Hg, PCO_2 0 mm Hg
Air Bubble + Blood = PO_2 PCO_2

➤ ABG Syringe must be transported at the earliest to the laboratory for EARLY analysis via COLD CHAIN

CHANGE INVALUES EVERY 10 MINUTES	UNICED SAMPLE 37°C	ICED SAMPLE 4°C
pН	0.01	0.001
PCO ₂	I mm Hg	0.1 mm Hg
PO ₂	0.1 %	0.01 %

- ➤ Patients **Body Temperature** affects the values of PCO₂ and HCO₃.
 - ■ABG Analyser is controlled for Normal Body temperatures
 - Any change in body temp at the time of sampling leads to alteration in values detected by the electrodes
- Cell count in PO₂
- ➤ ABG Sample should always be sent with relevant information regarding O₂, FiO₂ status and Temp.

ABG ELECTRODES

- A. pH (Sanz Electrode)
- Measures H+ ion concentration of sample against a known pH in a reference electrode, hence potential difference. Calibration with solutions of known pH (6.384 to 7.384)
- B. PCO₂ (Severinghaus Electrode)
- □ CO₂ reacts with solution to produce H+ higher CO₂- \rightarrow more H+ \rightarrow higher P CO₂ measured
- C. P 0₂ (Clark Electrode)
- O₂ diffuses across membrane producing an electrical current measured as P O₂.

Interpretation of ABG □ OXYGENATION □ ACID BASE

G E N A T

➤ Determination of PaO₂

 PaO_2 is dependant upon \implies Age, FiO_2 , P_{atm}

• $PaO_2 = 109 - 0.4$ (Age)

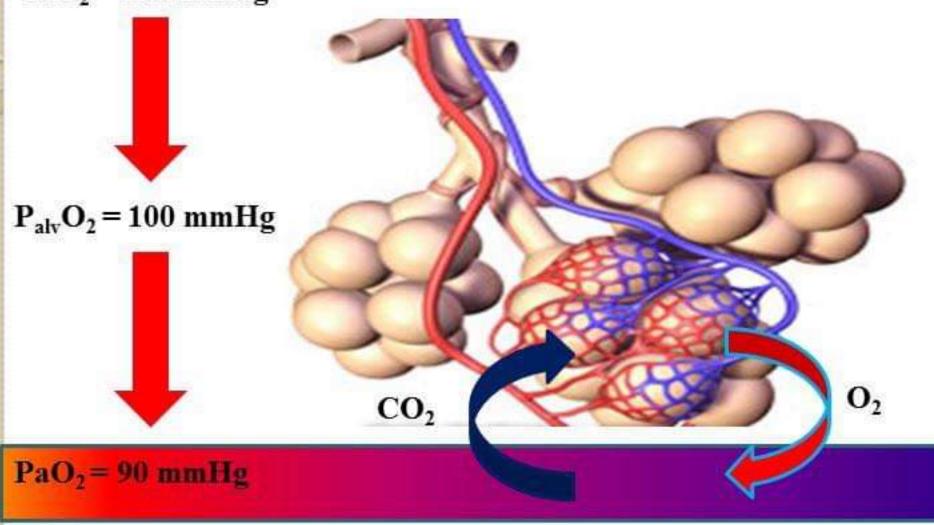
As FiO₂ the expected PaO₂

- Alveolar Gas Equation:
 - $P_AO_2 = (P_B-P_{h2o}) \times FiO_2 pCO_2/R$

 P_AO_2 = partial pressure of oxygen in alveolar gas, P_B = barometric pressure (760mmHg), P_{h2o} = water vapor pressure (47 mm Hg), FiO_2 = fraction of inspired oxygen, PCO_2 = partial pressure of CO_2 in the ABG, R = respiratory quotient (0.8)

➤ Determination of the PaO2 / FiO2 ratio

Inspired Air $FiO_2 = 21\%$ $PiO_2 = 150 \text{ mmHg}$



PiO ₂ / FiO ₂ Ratio	Inference
476	Normal
< 300	Acute Lung Injury
< 200	ARDS (along with other criteria)

PO₂/FiO₂ ratio (P:F Ratio)

➤ Gives understanding that the patients
OXYGENATION with respect to OXYGEN delivered is more important than simply the PO₂ value.

Example,

	Patient 1 On Room Air	Patient 2 On MV
PO2	60	90
FiO2	21% (0.21)	50% (0.50)
P:F Ratio	285	180

A C D

BA

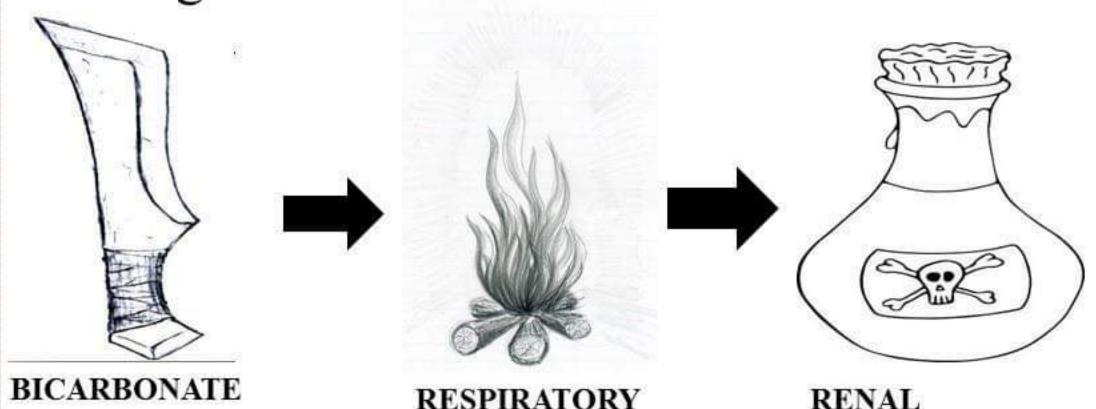
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BUFFER SYSTEM

Acts in few seconds

Acid Base Balance

- ➤ H⁺ ion concentration in the body is precisely regulated
- The body understands the importance of H⁺ and hence devised DEFENCES against any change in its concentration-



REGULATION

Acts in few minutes

REGULATION

Acts in hours to days

Regulation of Acid Base

> Bicarbonate Buffer System

$$CO_2 + H_2O \stackrel{carbonic anhydrase}{\longleftarrow} H_2CO_3 \stackrel{\longleftarrow}{\longleftarrow} H^+ + HCO_3^-$$

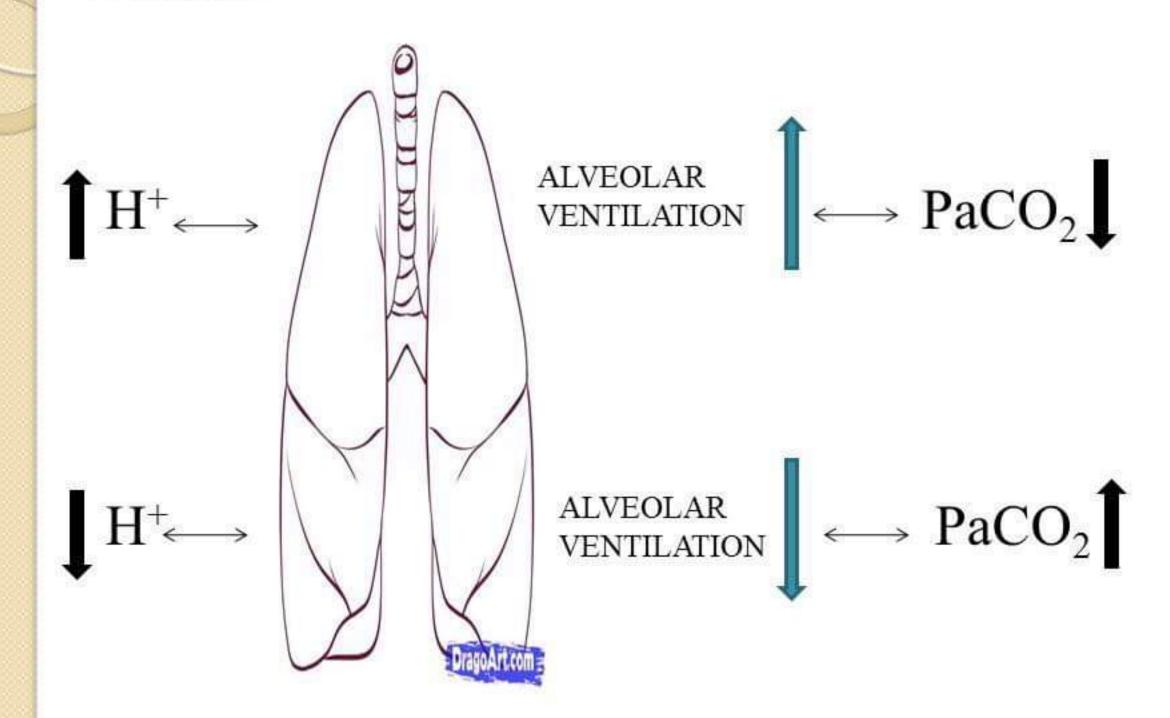
In Acidosis - Acid =
$$H^+$$

 \uparrow $H^+ + HCO_3 \longrightarrow H_2CO_3 \longrightarrow CO_2 + H_2O$

In Alkalosis - Alkali + Weak Acid =
$$H_2CO_3$$

 $CO_2 + H_2O \longrightarrow H_2CO_3 \longrightarrow \uparrow HCO_3^- + H^+$
ALKALI

➤ Respiratory Regulation of Acid Base Balance-



> Renal Regulation of Acid Base Balance

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

This is achieved in the following ways-

Reabsorption of HCO₃ in blood

> PCO2 in ECF

H+ ion

ECF Volume



Proximal Convulated
 Tubules (85%)

- •Thick Ascending Limb of Loop of Henle (10%)
- Distal Convulated Tubule
- Collecting Tubules (5%)

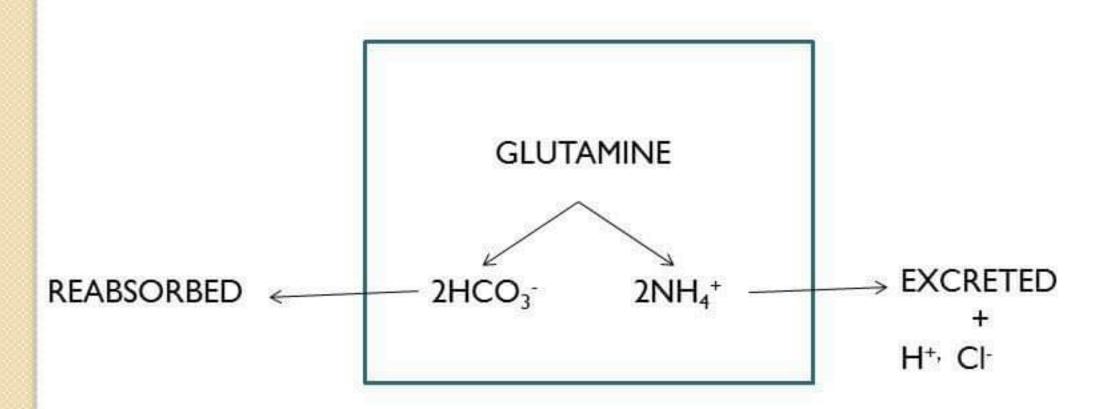
Secretion of H⁺ ions in tubules and excretion

K+

Aldosterone

Angiotensin II

 Another mechanism by which the kidney controls the acid base balance is by the Combination of excess H+ ions in urine with AMMONIA and other buffers- A mechanism for generating NEW Bicarbonate ions



 In CKD, the dominant mechanism by which acid is eliminated by the Kidneys is excretion of NH4+

Assessment of ACID BASE Balance

Definitions and Terminology

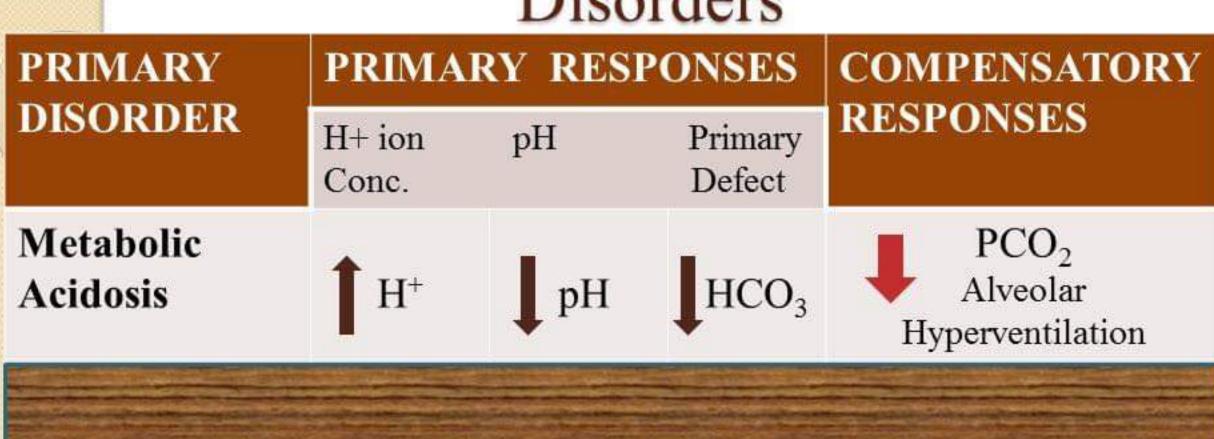
- □ACIDOSIS presence of a process which tends to ↓pH by virtue of gain of H + or loss of HCO₃-
- □ALKALOSIS presence of a process which tends to ↑ pH by virtue of loss of H⁺ or gain of HCO₃⁻

If these changes, change pH, suffix 'emia' is added

- ACIDEMIA reduction in arterial pH (pH<7.35)
- ALKALEMIA increase in arterial pH (pH>7.45)

- Simple Acid Base Disorder/ Primary Acid Base disorder a single primary process of acidosis or alkalosis due to an initial change in PCO₂ and HCO₃.
- □ Compensation The normal response of the respiratory system or kidneys to change in pH induced by a primary acid-base disorder
- The Compensatory responses to a primary Acid Base disturbance are never enough to correct the change in pH, they only act to reduce the severity.
- ■Mixed Acid Base Disorder Presence of more than one acid base disorder simultaneously .

Characteristics of Primary ACID BASE Disorders





Compensation

Metabolic Disorders – Compensation in these disorders leads to a change in PCO₂

METABOLIC ACIDOSIS

- $PCO_2 = (1.5 \times [HCO_3^-]) + 8 + 2$
- $PCO_2 = [HCO_3 -] + 15$
- For every 1mmol/1 ↓in HCO₃ the PCO₂ ↓falls by 1.25 mm Hg

METABOLIC ALKALOSIS

- $PCO_2 = (0.7 \text{ X } [HCO_3^-]) + 21 \pm 2$
- $PCO_2 = [HCO_3^-] + 15$
- For every 1mol/1 †in HCO₃ the PCO₂ † by 0.75 mm Hg

In Respiratory Disorders

PCO2 Kidney HCO3 Reabsorption

Compensation begins to appear in 6 - 12 hrs and is fully developed only after a few days.

1.ACUTE

Before the onset of compensation

Resp. acidosis — 1mmHg ↑ in PCO2—HCO3 ↑ by 0.1meq/l Resp. alkalosis — 1mmHg ↓ in PCO2 —HCO3↓ by 0.2 meq/l

2.CHRONIC (>24 hrs)

After compensation is fully developed

Resp. acidosis – 1mmHg ↑ in PCO2 → HCO3 ↑ by 0.4meq/l

Resp. alkalosis – 1mmHg↓ in PCO2 → HCO3↓ by 0.4meq/l

Respiratory Disorders – Compensation in these disorders leads to a change in HCO_{3.}

RESPIRATORY ACIDOSIS

• ACUTE pH=7.40-0.008(PCO₂-40)

CHRONIC pH=7.40-0.003(PCO₂-40)

RESPIRATORY ALKALOSIS

ACUTE pH=7.40+0.008(40-PCO₂)

• CHRONIC pH=7.40+0.003(40-PCO₂)

to Interpretation Of ABG reports

Six steps logical approach originally proposed by Narins and Emmett (1980) and modified by Morganroth in 1991

Normal Values

ANALYTE	Normal Value	Units
pH	7.35 - 7.45	
PCO2	35 - 45	mm Hg
PO2	72 – 104	mm Hg`
[HCO3]	22 - 30	meq/L
SaO2	95-100	%
Anion Gap	12 <u>+</u> 4	meq/L
ΔHCO3	+2 to -2	meq/L

STEP 0 STEP 1

Is this ABG Authentic?

ACIDEMIA or ALKALEMIA?

STEP 2

RESPIRATORY or METABOLIC?

STEP 3

If Respiratory – ACUTE or CHRONIC?

STEP 4

Is COMPENSATION adequate?

STEP 5

• If METABOLIC – ANION GAP?

STEP 6

 If High gap Metabolic Acidosis— GAP GAP?

Is this ABG authentic?

• $pH = - log [H^+]$

Henderson-Hasselbalch equation

$$pH = 6.1 + log \underline{HCO_3}$$

$$0.03 \times PCO_2$$

The [HCO3-] mentioned on the ABG is actually calculated using this equation from measured values of PCO₂ nd pH

•
$$[H+] neq/l = 24 \times (PCO_2 / HCO_3)$$

$$pH = -log [H^+]$$

 $pH_{expected} = pH_{measured} = ABG is authentic$

Reference table for pH v/s [H⁺]

H ⁺ ion	PII
100	7.00
79	7.10
63	7.20
50	7.30
45	7.35
40	7.40
35	7.45
32	7.50
25	7.60

$$[H+] neq/l = 24 X (PCO_2 / HCO_3)$$

STEP 1 ACIDEMIA OR ALKALEMIA?

Look at pH

<7.35 - acidemia

>7.45 – alkalemia

❖ RULE – An acid base abnormality is present even if either the pH or PCO2 are Normal.

STEP 2 RESPIRATORY or METABOLIC?

IS PRIMARY DISTURBANCE RESPIRATORY OR METABOLIC?

$$\triangleright$$
pH \triangleq PCO₂ \triangleq or pH \downarrow PCO₂ \downarrow \Longrightarrow METABOLIC

$$\triangleright$$
pH \triangleq PCO₂ \blacksquare or pH \blacksquare PCO₂ \triangleq RESPIRATORY

❖RULE- If either the pH or PCO₂ is Normal, there is a mixed metabolic and respiratory acid base disorder.

STEP 3

RESPIRATORY-ACUTE/CHRONIC?

IF RESPIRATORY, IS IT ACUTE OR CHRONIC?

- ► Acute respiratory disorder $\Delta pH_{(e-acute)} = 0.008x \Delta Pco_2$
- >Chronic respiratory disorder $\Delta pH_{(e-chronic)} = 0.003x \Delta pCO_2$
- ightharpoonup Compare, $pH_{measured}(pH_m)$ v/s $pH_{expected}(pH_e)$

$\mathbf{pH}_{(m)} = \mathbf{pH}_{(e-acute)}$	pH _(m) = between pH _(e- acute) & pH _(e- chronic)	$pH_{(m)} = pH_{(e-chronic)}$
ACUTE RESPIRATORY DISORDER	PARTIALLY COMPENSATED	CHRONIC RESPIRATORY DISORDER

STEP 4 ADEQUATE COMPENSATION?

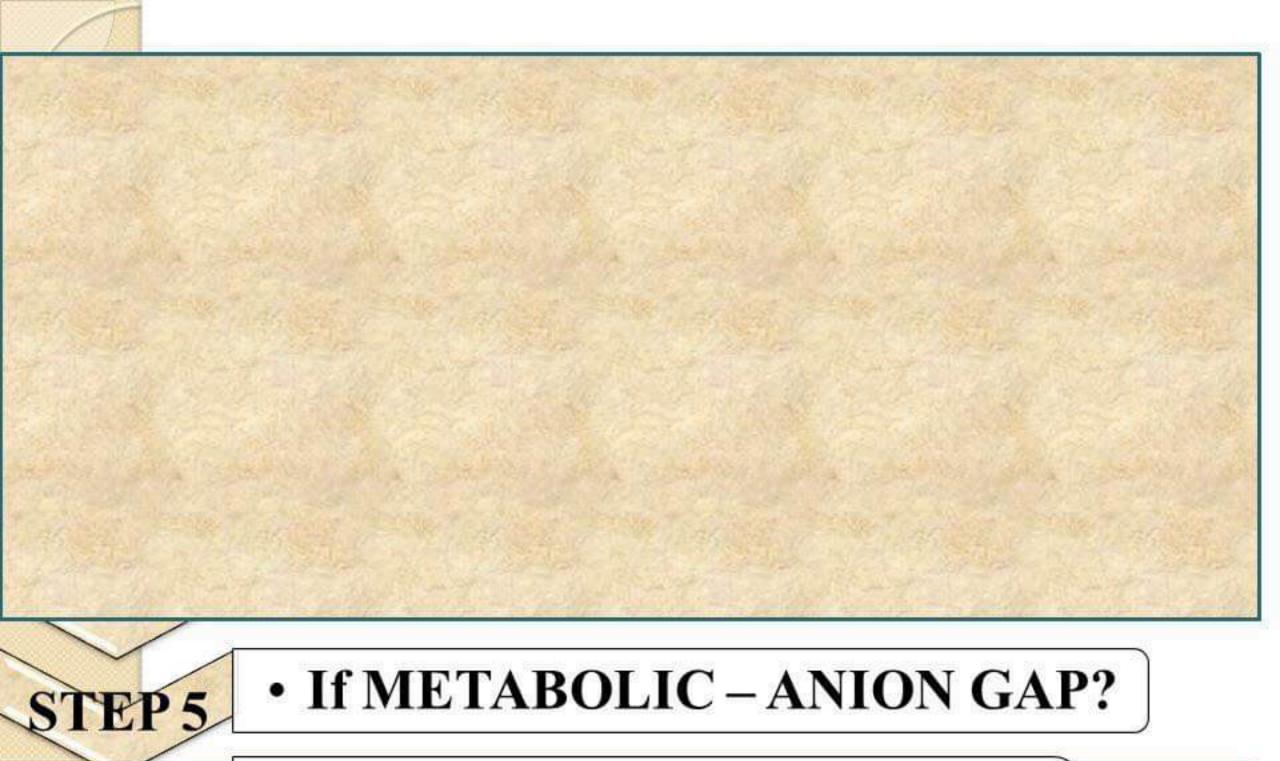
IS THE COMPENSATORY RESPONSE ADEQUATE OR NOT?

➤ METABOLIC DISORDER ——— PCO_{2expected}

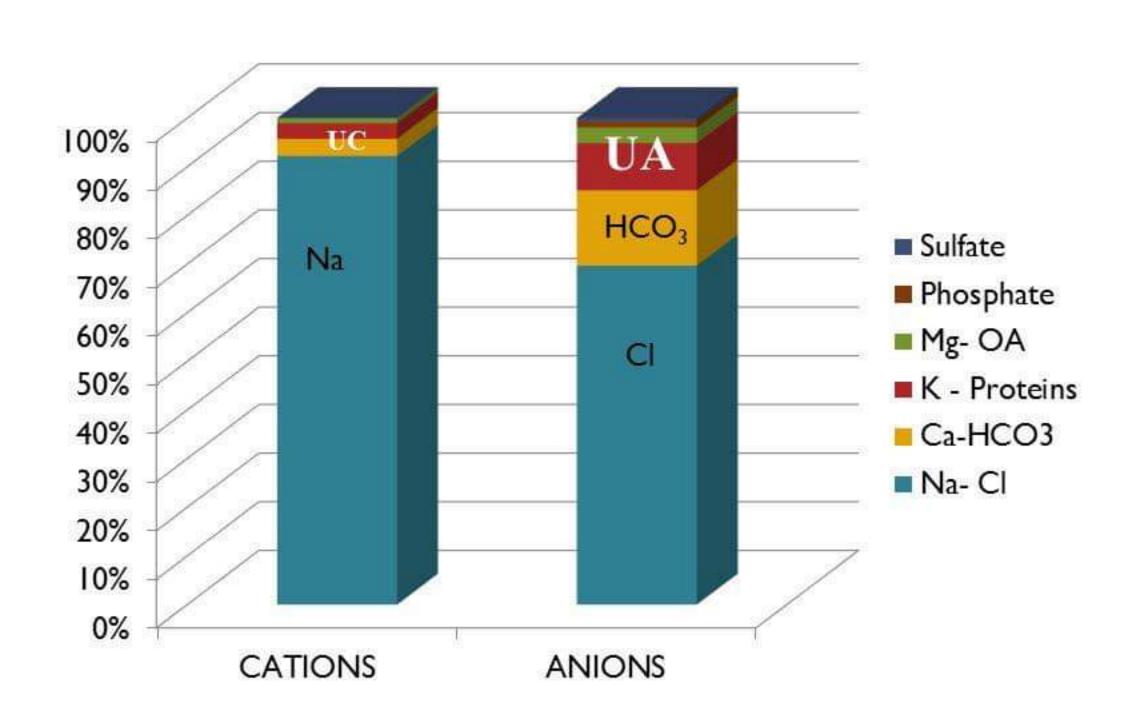
 PCO_2 measured $\neq PCO_2$ expected \implies MIXED DISORDER

ightharpoonup RESPIRATORY DISORDER \longrightarrow pH_{expected acute-chronic}

 $pH_m \neq pH_e range \implies MIXED DISORDER$



Electrochemical Balance in Blood



Anion Gap

AG based on principle of electroneutrality:

- > Total Serum Cations = Total Serum Anions
- > M cations + U cations = M anions + U anions
- > Na + (K + Ca + Mg) = HCO₃ + Cl + (PO4 + SO4 + Protein + Organic Acids)
- $> Na + UC = HCO_3 + Cl + UA$
- > But in Blood there is a relative abundance of Anions, hence

- $> Na (HCO_3 + Cl) = UA UC$
- $> Na (HCO_3 + Cl) = Anion Gap$

STEP 5

METABOLIC ACIDOSIS-ANION GAP?

IN METABOLIC ACIDOSIS WHAT IS THE ANION GAP?

 \square ANION GAP(AG) = Na – (HCO₃ + Cl)

Normal Value = 12 ± 4 (7- 16 Meq/l)

Adjusted Anion Gap = Observed AG +2.5(4.5- S.Albumin) 50% in S. Albumin $\Longrightarrow 75\%$ in Anion Gap !!!

Metabolic Acidosis

High Anion Gap Metabolic Acidosis

Normal Anion Gap Acidosis

High Anion Gap Metabolic Acidosis

M	METHANOL
U	UREMIA - ARF/CRF
D	DIABETIC KETOACIDOSIS & other KETOSIS
P	PARALDEHYDE, PROPYLENE GLYCOL
	ISONIAZIDE, IRON
	LACTIC ACIDOSIS
E	ETHANOL, ETHYLENE GLYCOL
s	SALICYLATE

STEP 6

CO EXISTANT METABOLIC DISORDER – "Gap Gap"?

C/O HGAG METABOLIC ACIDOSIS, ANOTHER DISORDER?

 $\triangleright \Delta$ Anion Gap = Measured AG – Normal AG

Measured AG - 12

 Δ HCO₃ = Normal HCO₃ – Measured HCO₃

24 – Measured HCO₃

Ideally, $\Delta Anion Gap = \Delta HCO_3$

For each 1 meq/L increase in AG, HCO3 will fall by 1 meq/L

 $\triangle AG/\triangle HCO_3^- = 1 \rightarrow Pure High AG Met Acidosis$ $<math>\triangle AG/\triangle HCO_3^- > 1 \rightarrow Assoc Metabolic Alkalosis$ $<math>\triangle AG/\triangle HCO_3^- < 1 \rightarrow Assoc N AG Met Acidosis$

Clinical CASE SCENARIOS

Fb/Nurse-Info

CASE 1

Mr. Shamshuddin, 62/M, Nagina

- > k/c/o COPD
- Breathlessness, progressively increased, aggravated on exertion, 2 days
- Chronic smoker
- O/E
 RS- B/L expiratory rhonchi

22/7/11	7:30 am
рН	7.20
PCO2	92 mmHg
PO2	76 mmHg
Actual HCO3	21.00 mmol/l
SO2	89
FiO2	37%

22/7/11	7:30 am
pН	7.20
PCO2	92 mmHg
PO2	76 mmHg
Actual HCO3	21.00 mmol/l
SO2	89
FiO2	37%

- ➤ STEP 1 ACIDEMIA
- ➤ STEP 2 pH PCO₂ Respiratory
- ➤ STEP 3 pH expected
 - \square pH acute = 7.40 0.008(92-40) 7.40 - 0.008(52)

6.984

 \square pH chronic = 7.40 - 0.003(92-40)

7.244

- □pH b/w 6.98 to 7.244
- Primary Respiratory Acidosis, partially compensated

CASE 2

Mr.Dharam Dutt, 63/M, Bijnor

- k/c/o CRF(conservativeRx)
- Breathlessness
- Decreased Urine Otpt. 2days
- > Vomiting 10-15
- > O/E

No pedal edema, dehydration+

RS - B/L A/E Normal

31/7/11	11:30pm
pН	7.18
PCO2	21.00
PO2	90
Actual HCO3	7.80
Base Excess	-18.80
SO2	95
Na	140.6
Chloride	102
T.Protein	6
Albumin	2.4

31/7/11	11:30pm
pН	7.18
PCO2	21.00
PO2	90
Actual HCO3	7.80
Base Excess	-18.80
SO2	95
Na	140.6
Chloride	102
T.Protein	6
Albumin	2.4

- ➤ STEP 1 ACIDEMIA
- > STEP 2 pH PCO2 METABOLIC
- STEP 4 PCO2expected
 PCO2exp = (1.5 x HCO3)+8±2
 (1.5X7.80)+8±2
 19.7+2= 17.7 21.7
- > STEP5 ANION GAP = Na – (HCO3 +Cl) = 140.6-(7.80+102) = 30.8
- ✓ AG corrected for albumin = 30.8+5.25
 AG = 36.05
 HIGH AG Met. Acidosis

31/7/11	11:30pm
pН	7.18
PCO2	21.00
PO2	90
Actual HCO3	7.80
Base Excess	-18.80
SO2	95
Na	140.6
Chloride	102
T.Protein	6
Albumin	2.4

> STEP 6 – GAP GAP = (AG-12)/(24-HCO3) = 36.05-12/24-7.80 = 24.05/16.2

Gap/gap > 1 = add. Metabolic alkalosis

Asis – Primary Metabolic Acidosis

= 1.48

High Anion Gap, compensated Cause- CRF

Metabolic Alkalosis

Cause - ? Vomiting