

OBJECTIVES

► ABG Sampling

- ➤ Interpretation of ABG
 - Oxygenation status
 - Acid Base status

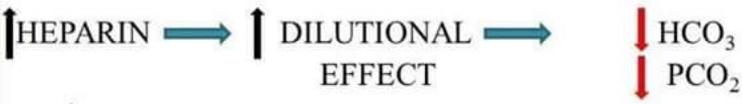
Case Scenarios

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 PCO₂

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

CHANGE IN VALUES 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→ more H+ → higher P CO₂ measured

C. P 0₂ (Clark Electrode)

 \Box 0₂ diffuses across membrane producing an electrical current measured as P 0₂.

Interpretation of ABG

- OXYGENATION
- □ ACID BASE

- G E N T
- ➤ Determination of PaO₂

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

As Age the expected PaO₂

• $PaO_2 = 109 - 0.4$ (Age)

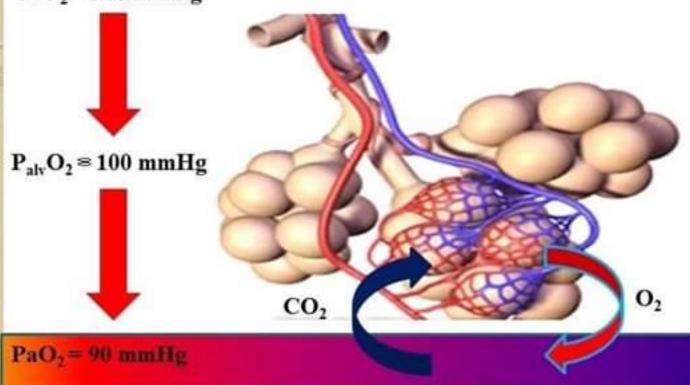
As FiO₂ the expected PaO₂

- · 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	1 285	180

A C I D

B

A

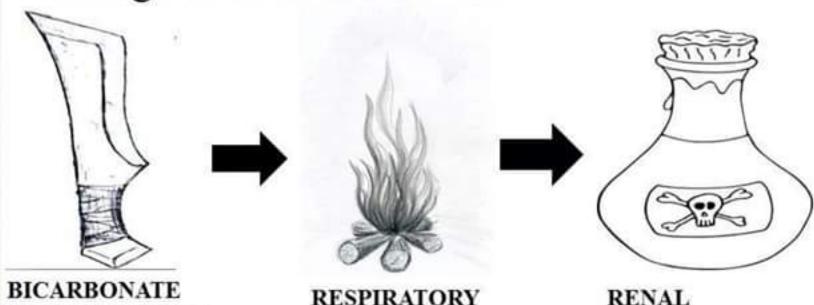
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BUFFER SYSTEM

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

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

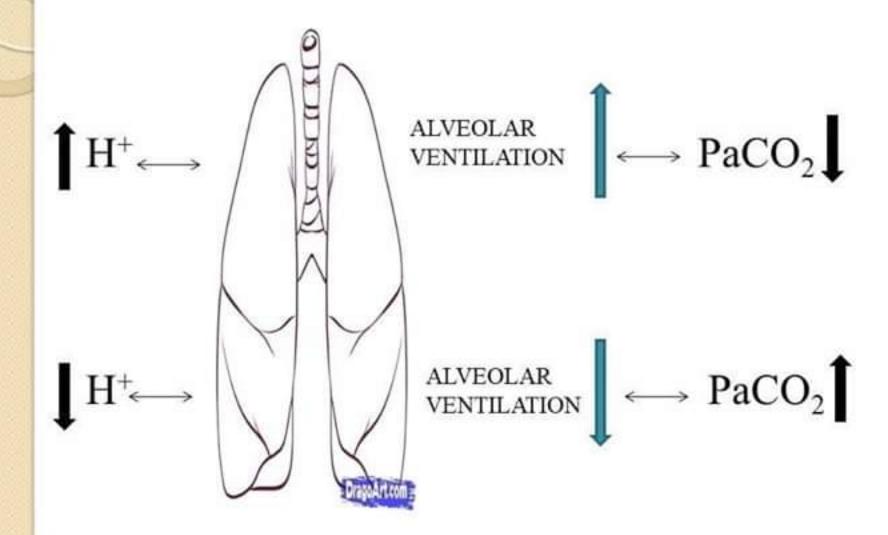
In Acidosis - Acid =
$$H^+$$

 $\uparrow H^+ + HCO_3 \longrightarrow H_2CO_3 \longrightarrow CO_2 + H_2O_3$

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

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

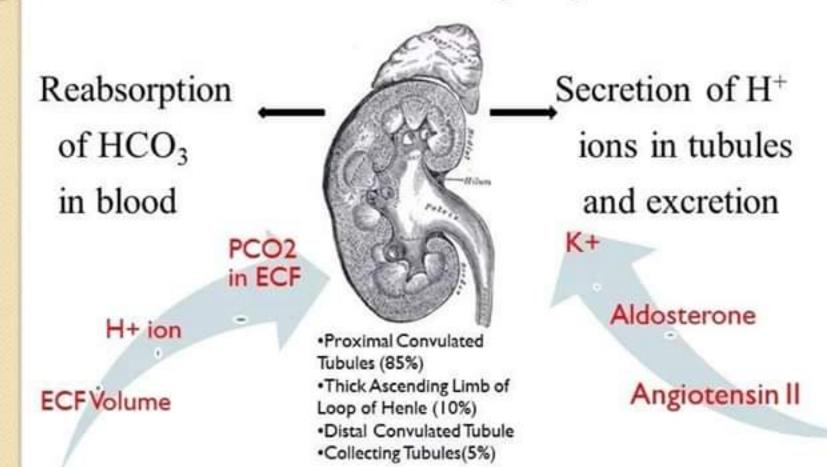
➤ Respiratory Regulation of Acid Base Balance-



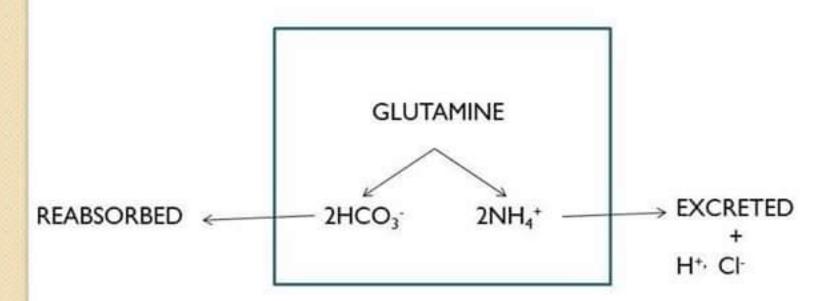
➤ Renal Regulation of Acid Base Balance

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

This is achieved in the following ways-



 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

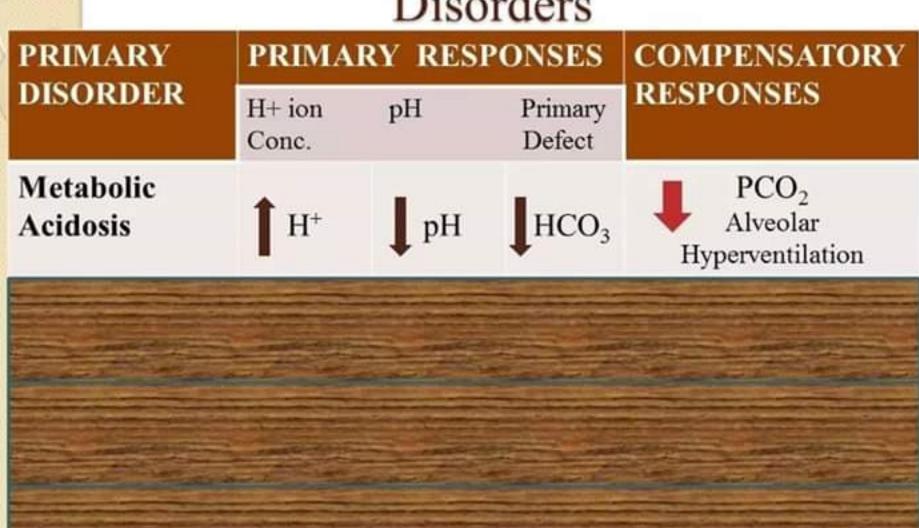
- □ACIDOSIS presence of a process which tends to ↓ pH by virtue of gain of H + or loss of HCO₃-
- □ALKALOSIS presence of a process which tends to ↑ pH by virtue of loss of H⁺ or gain of HCO₃⁻

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/l ↓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 – $1 \text{mmHg} \uparrow \text{ in PCO2} \rightarrow \text{HCO3} \uparrow \text{ by } 0.4 \text{meq/l}$ Resp. alkalosis – $1 \text{mmHg} \downarrow \text{ in PCO2} \rightarrow \text{HCO3} \downarrow \text{ by } 0.4 \text{meq/l}$ 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
pН	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 ± 4	meq/L
ΔНСО3	+2 to -2	meq/L

Is this ABG Authentic? STEPO ACIDEMIA or ALKALEMIA? STEP 1 RESPIRATORY or METABOLIC? STEP 2 If Respiratory – ACUTE or CHRONIC? STEP 3 Is COMPENSATION adequate? STEP 4 If METABOLIC – ANION GAP? STEP 5 If High gap Metabolic Acidosis— STEP 6 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/ $I = 24 \times (PCO_2 / HCO_3)$

$$\begin{array}{ccc} & & & & & & \\ & & & & \\ pH = -log \; [\; H^+] \\ \\ pH_{expected} = & pH_{measured} \; = \; ABG \; is \; \textbf{authentic} \end{array}$$

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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 \uparrow PCO₂ \uparrow or pH \downarrow PCO₂ \downarrow \longrightarrow METABOLIC

$$\triangleright$$
pH \uparrow PCO₂ \downarrow or pH \downarrow PCO₂ \uparrow \Longrightarrow 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 $\Delta pH_{(e-chronic)} = 0.003x \Delta pCO_2$

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	PARTIALLY	CHRONIC RESPIRATORY
DISORDER	COMPENSATED	DISORDER

STEP 4 ADEQUATE COMPENSATION?

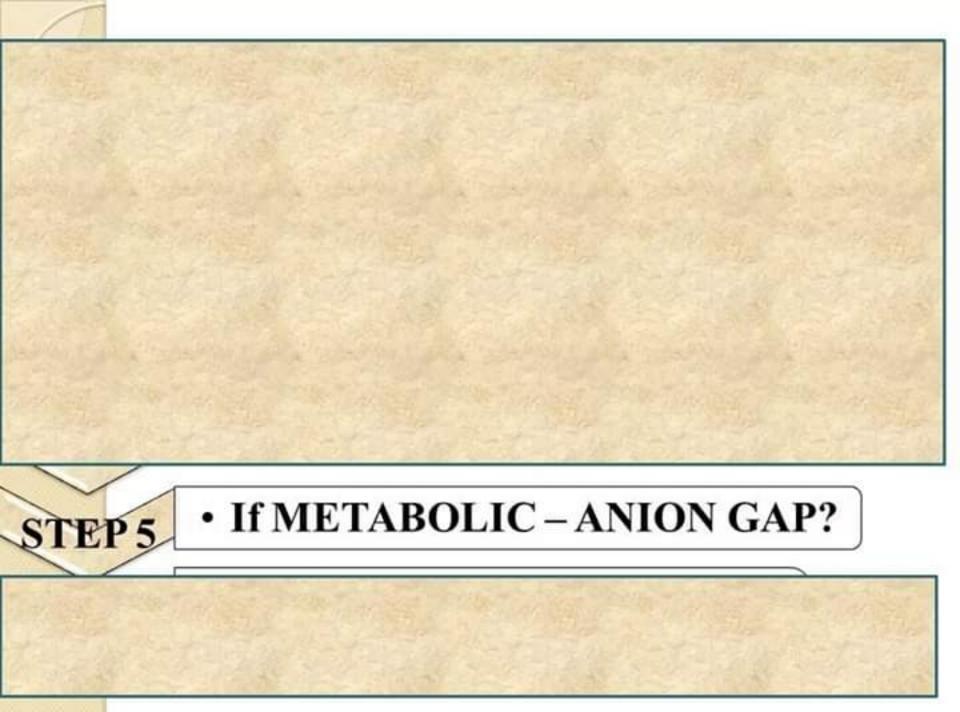
IS THE COMPENSATORY RESPONSE ADEQUATE OR NOT?

➤ METABOLIC DISORDER → PCO_{2expected}

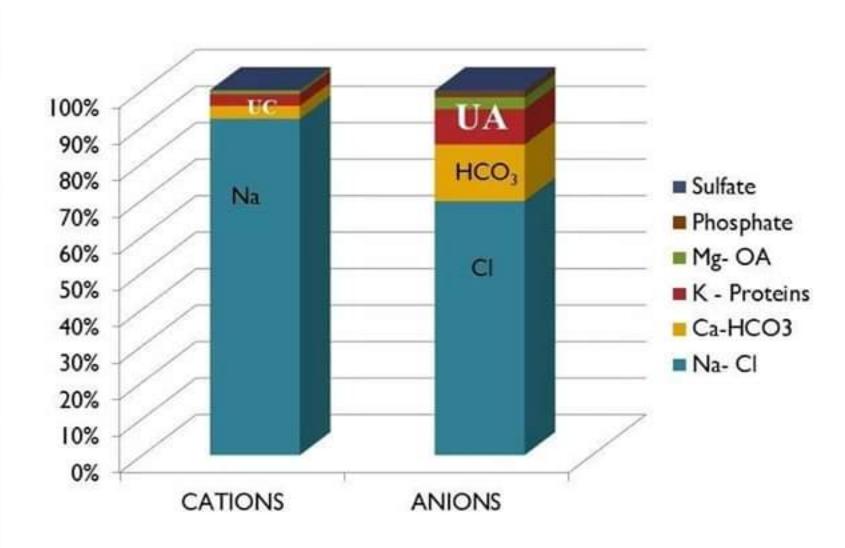
 PCO_2 measured $\neq PCO_2$ expected \implies MIXED DISORDER

➤ RESPIRATORY DISORDER → 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% I in S. Albumin -> 75% I in Anion Gap !!!

Metabolic Acidosis

Normal Anion Gap Metabolic Acidosis

Normal Anion Gap Acidosis

High Anion Gap Metabolic Acidosis

M	
	METHANOL
U	
	UREMIA - ARF/CRF
	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?

► △ Anion Gap = Measured AG – Normal AG

Measured AG – 12

 $\triangleright \Delta \text{ HCO}_3 = \text{Normal HCO}_3 - \text{Measured HCO}_3$

24 - Measured HCO₃

Ideally, Δ Anion Gap = Δ HCO₃ 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

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
pН	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

Δsis – Primary Metabolic Acidosis

= 1.48

High Anion Gap, compensated Cause- CRF

- Metabolic Alkalosis

Cause - ? Vomiting