



# **Arterial Blood Gas Interpretation**

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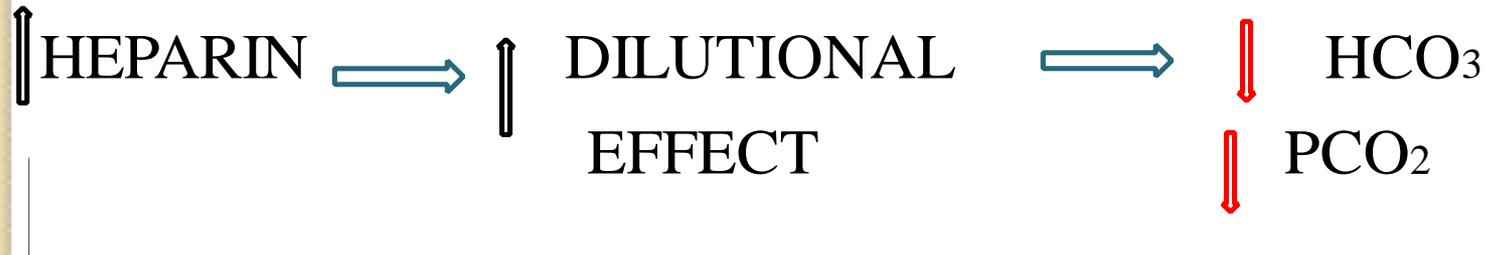
# OBJECTIVES

- ABG Sampling
- Interpretation of ABG
  - Oxygenation status
  - Acid Base status
- Case Scenarios

# ABG – Procedure and Precautions

- Ideally - Pre-heparinised ABG syringes
  - Syringe should be **FLUSHED** with 0.5ml of Heparin solution and emptied.

**Do not leave excessive heparin in the Syringe**



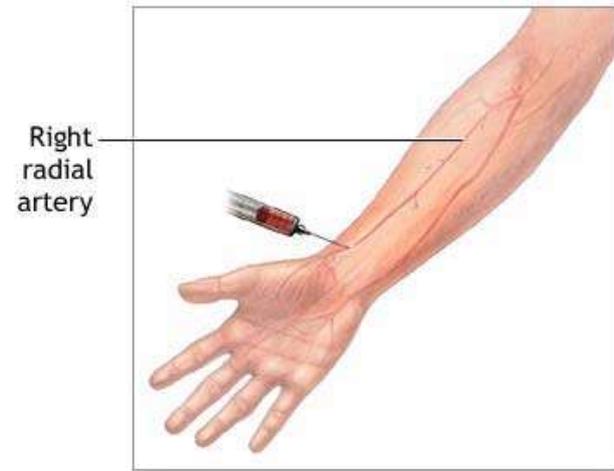
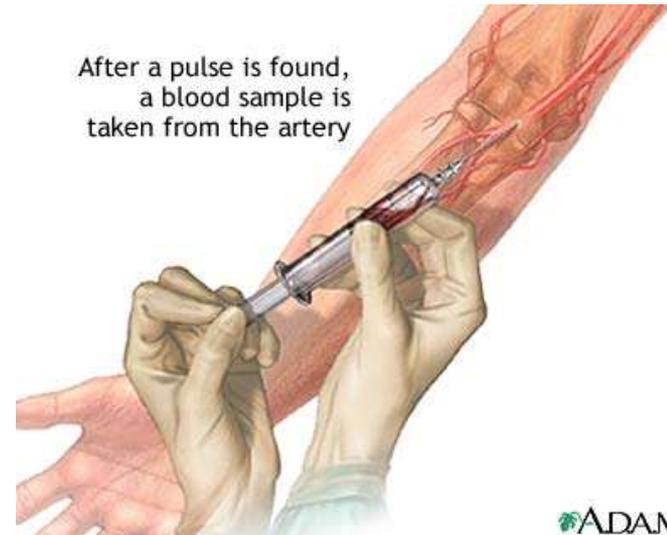
- ✓ Only small 0.5ml Heparin for flushing and discard it
- ✓ Syringes must have > 50% blood. Use only 2ml or less syringe

# Sites for obtaining ABG

- Radial artery ( most common )
- Brachial artery
- Femoral artery

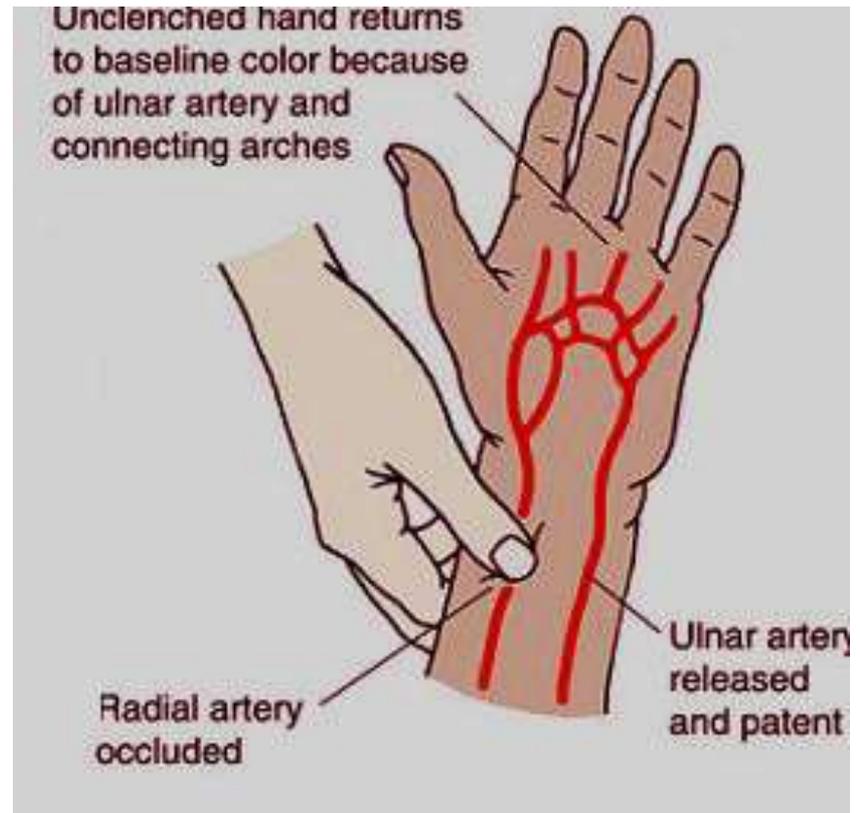
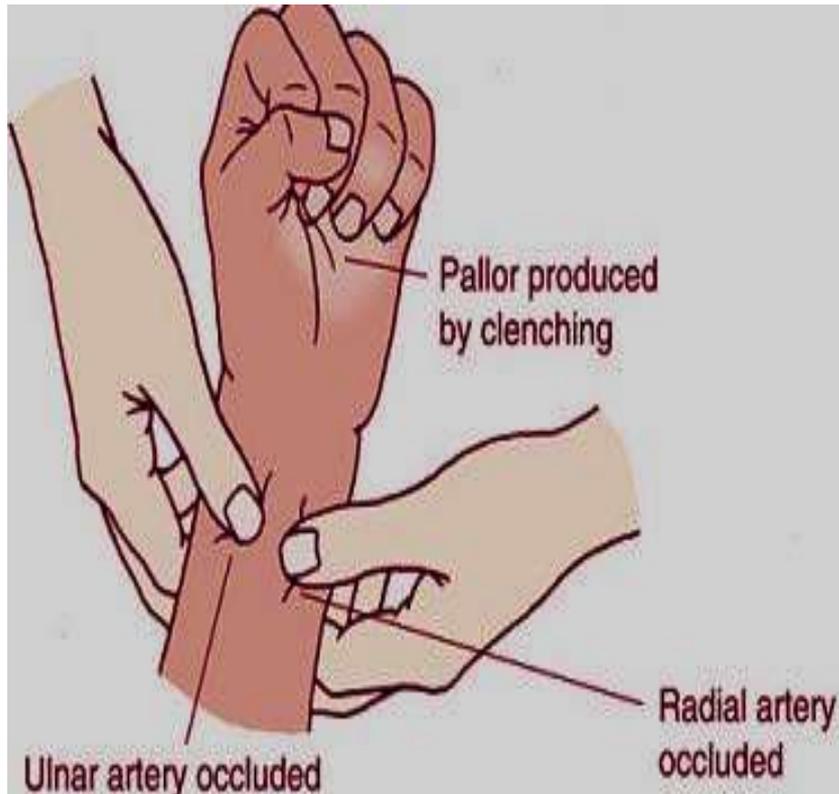
Radial is the most preferable site used because:

- It is easy to access
- It is not a deep artery which facilitate palpation, stabilization and puncturing
- The artery has a collateral blood circulation



# ALLEN'S TEST

It is a test done to determine that collateral circulation is present from the ulnar artery



- 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 IN VALUES EVERY 10 MINUTES	UNICED SAMPLE 37°C	ICED SAMPLE 4°C
pH	0.01	0.001
$PCO_2$	1 mm Hg	0.1 mm Hg
$PO_2$	0.1 %	0.01 %

# **Interpretation of ABG**

**❑ OXYGENATION**

**❑ ACID BASE**

# Blood Gas Report

- **Oxygenation Information**

- PaO<sub>2</sub> [oxygen tension]
- SaO<sub>2</sub> [oxygen saturation]

- **Acid-Base Information**

- PH
- PaCO<sub>2</sub>
- HCO<sub>3</sub> [measured]



## ► Determination of PaO<sub>2</sub>

PaO<sub>2</sub> is dependant upon → Age, FiO<sub>2</sub>, P<sub>atm</sub>

As Age ↑ the expected PaO<sub>2</sub> ↓

- $PaO_2 = 109 - 0.4 (Age)$

As FiO<sub>2</sub> ↑ the expected PaO<sub>2</sub> ↑

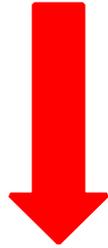
- Alveolar Gas Equation:
  - $P_{AO_2} = (P_B - P_{H_2O}) \times FiO_2 - PaCO_2/R$

**P<sub>AO<sub>2</sub></sub>** = partial pressure of oxygen in Alveolar gas, **P<sub>B</sub>** = Barometric Pressure (760mmHg), **P<sub>H<sub>2</sub>O</sub>** = water vapor pressure (47 mm Hg), **FiO<sub>2</sub>** = fraction of inspired oxygen, **R** = respiratory quotient (0.8)

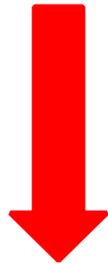
## ► Determination of the PaO<sub>2</sub> / FiO<sub>2</sub> ratio

Inspired Air FiO<sub>2</sub> = 21%

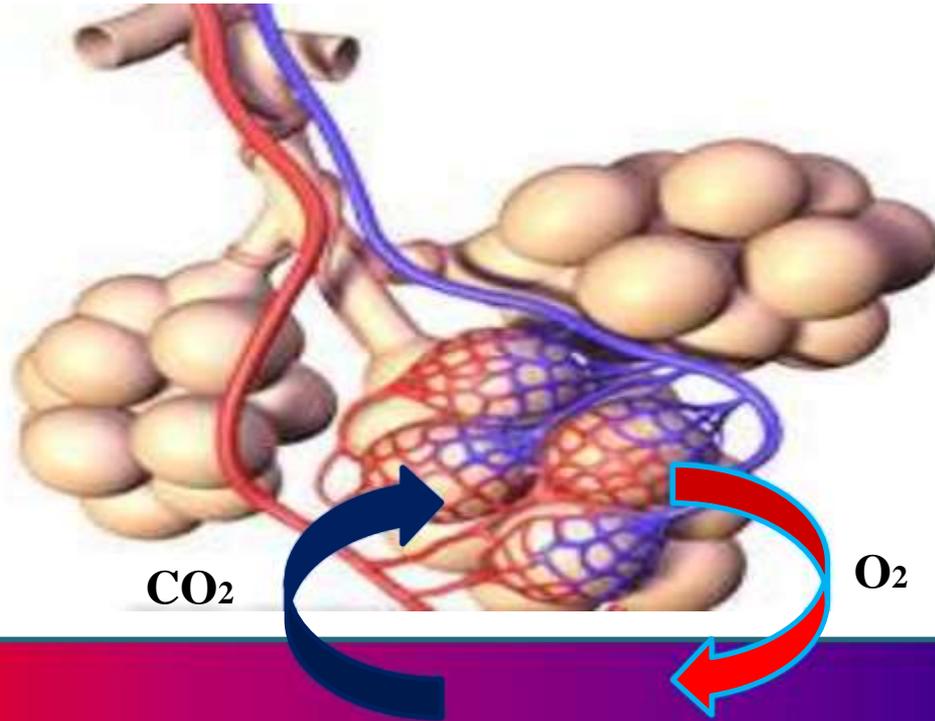
PiO<sub>2</sub> = 150 mmHg



PalvO<sub>2</sub> = 100 mmHg



PaO<sub>2</sub> = 95 mmHg



## PaO<sub>2</sub>/ FiO<sub>2</sub> ratio

- Gives understanding Patient Oxygenation with Respect to Oxygen delivered, more important than simply the PaO<sub>2</sub> value.

### *Example,*

	<b>Patient 1 On Room Air</b>	<b>Patient 2 On MV</b>
PaO <sub>2</sub>	68	90
FiO <sub>2</sub>	21% (0.21)	50% (0.50)
P:F Ratio	324 	180 

# **CLASSIFICATION OF HYPOXEMIA**

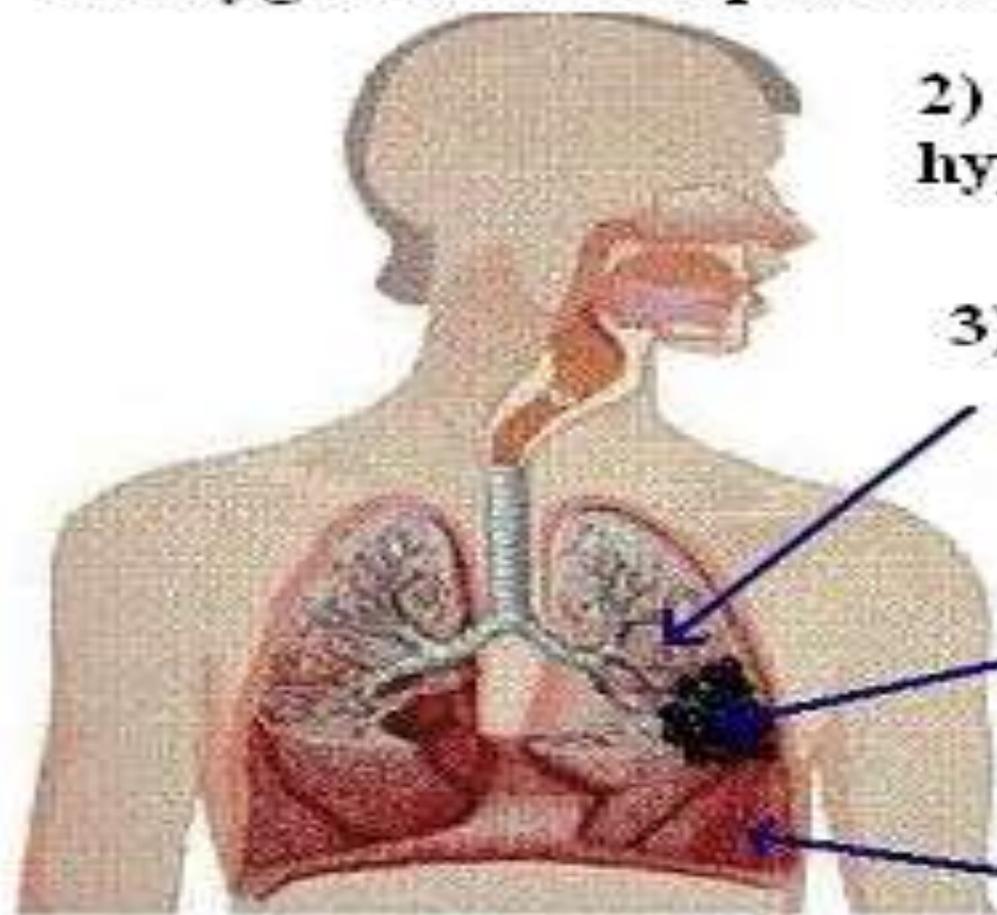
<b>Classifications</b>	<b>PaO<sub>2</sub> (rule of thumb)</b>
Normal	80-100 mm Hg
Mild hypoxemia	60-80 mm Hg
Moderate hypoxemia	40-60 mm Hg
Severe hypoxemia	<40 mm Hg

This classification is based on predicted **normal values for a patient who is less than 60 years old and breathing room air.** For older patients, **subtract 1 mm Hg for every year over 60 years of age** from the limits of mild and moderate hypoxemia.

A PaO<sub>2</sub> of less than 40 mm Hg represents severe hypoxemia at any age.

# Causes of hypoxemia

1) Reduced partial pressure of oxygen in the inspired air

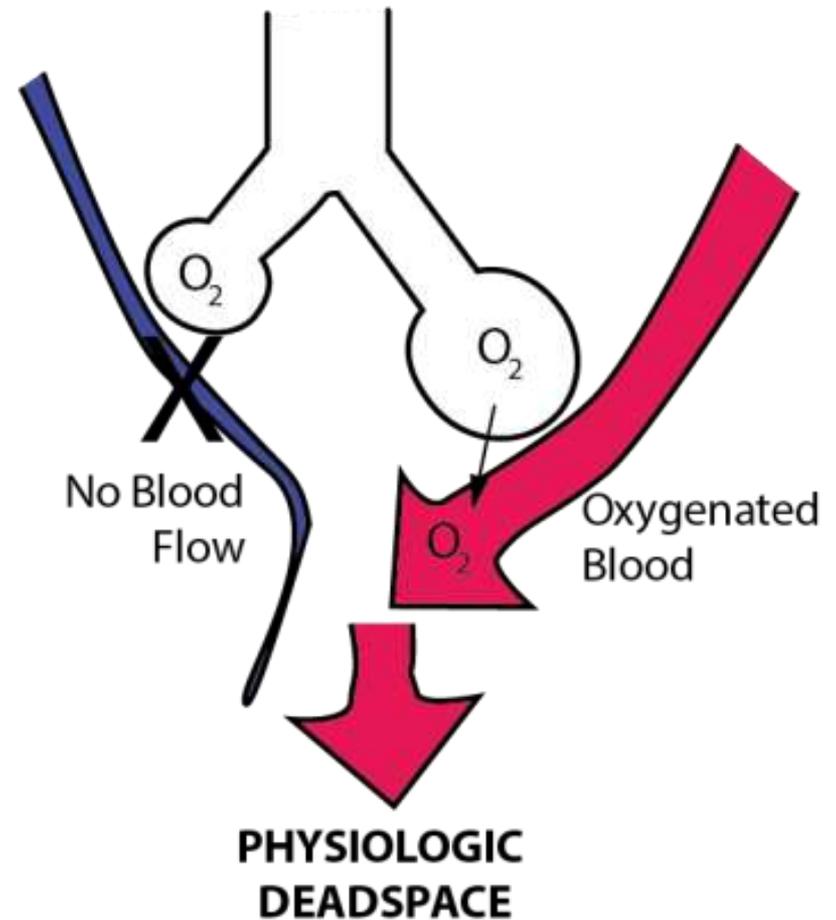
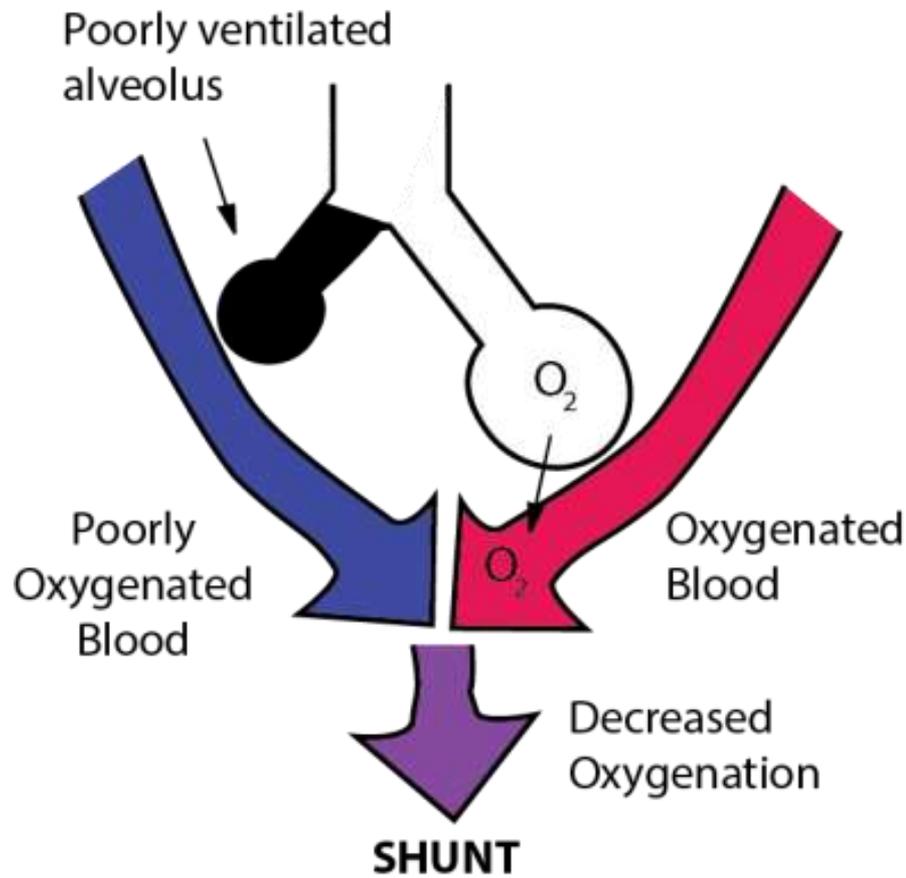


2) Alveolar hypoventilation

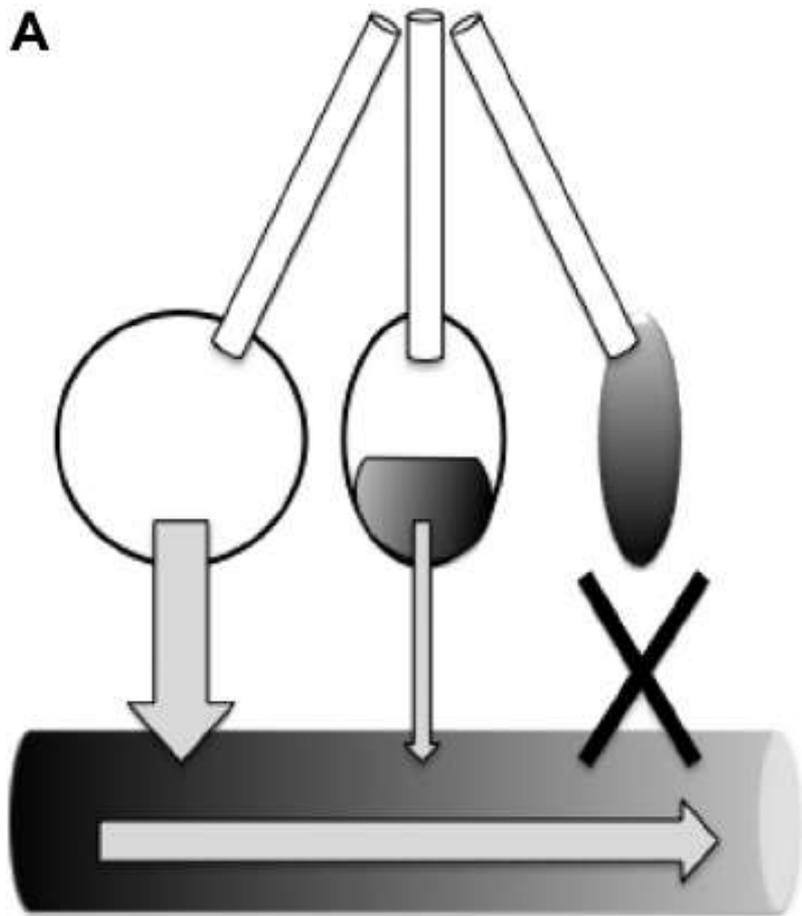
3) Ventilation-perfusion mismatch

4) Shunt (intracardiac or intrapulmonary)

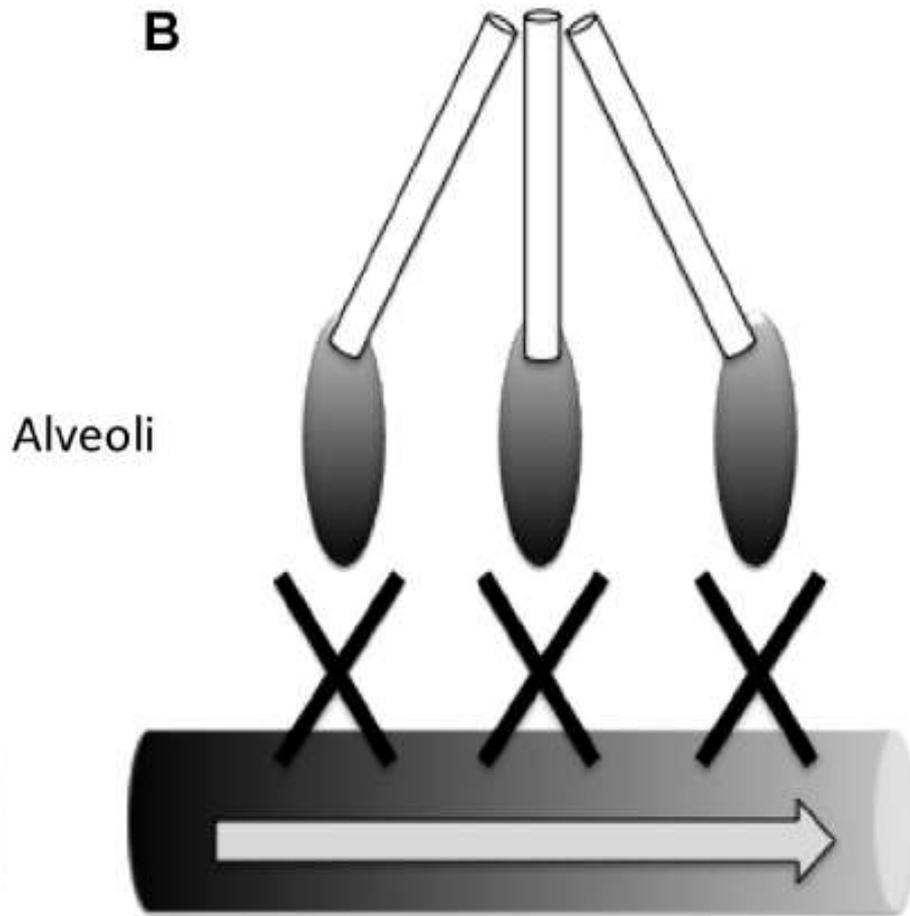
5) Impaired alveolar-capillary diffusion



Shunt is perfusion of poorly ventilated alveoli.  
Physiologic dead space is ventilation of poorly perfused alveoli.



VQ mismatch



Capillary

Shunt Physiology



## The A-a gradient

$$\text{A-a gradient} = P_A\text{O}_2 - P_a\text{O}_2$$

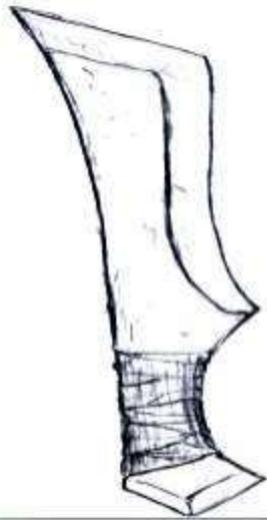
Normal = < 15mmHg

Normal rises 1mmHg per decade

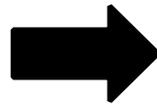
CAUSE	$P_{aO_2}$	A – a GRADIENT	EFFECT OF SUPPLEMENTAL O <sub>2</sub>
High Altitude	Decreased	Normal	Improves
Hypoventilation	Decreased	Normal	Improves
Diffusion Defect	Decreased	Increased	Improves
V <sup>o</sup> /Q Defect	Decreased	Increased	Improves
R → L Shunt	Decreased	Increased	Does not

# Acid Base Balance

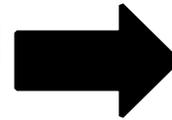
- $H^+$  ion concentration in the body is precisely regulated
- The body understands the importance of  $H^+$  and hence devised DEFENCES against any change in its concentration-



**BICARBONATE  
BUFFER SYSTEM**  
Acts in few seconds



**RESPIRATORY  
REGULATION**  
Acts in few minutes



**RENAL  
REGULATION**  
Acts in hours to days

A  
C  
I  
D  
  
B  
A  
S  
E

# 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  $HCO_3^-$
- **ALKALOSIS** – presence of a process which tends to ↑ pH by virtue of loss of  $H^+$  or gain of  $HCO_3^-$

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)

# Causes of Acid-Base Balance

## Metabolic Acidosis

- Diabetic ketoacidosis
- Diarrhea
- Renal failure
- Shock
- Aspirin overdose
- Sepsis

## Metabolic Alkalosis

- Loss of gastric secretions
- Overuse of antacids
- K<sup>+</sup> wasting diuretics

## Respiratory Acidosis

- Hypoventilation
- COPD
- Airway obstruction
- Drug overdose
- Chest trauma
- Pulmonary edema
- Neuromuscular disease

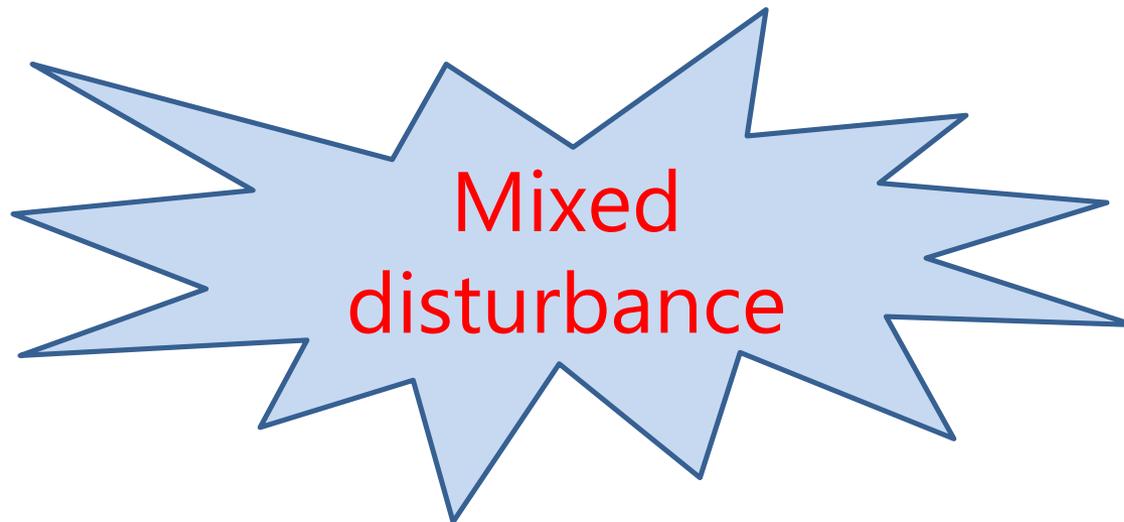
## Respiratory Alkalosis

- Hyperventilation
- Hypoxia
- Anxiety
- High altitude
- Pregnancy
- Fever

# Compensatory responses and their mechanisms.

<b>Primary disorder</b>	<b>Primary Chemical change</b>	<b>Compensatory response</b>	<b>Compensatory Mechanism</b>
Metabolic Acidosis	↓ HCO <sub>3</sub> <sup>-</sup>	↓ PCO <sub>2</sub>	Hyperventilation
Metabolic Alkalosis	↑ HCO <sub>3</sub> <sup>-</sup>	↑ PCO <sub>2</sub>	Hypoventilation
Respiratory Acidosis	↑ PCO <sub>2</sub>	↑ HCO <sub>3</sub> <sup>-</sup>	
Acute			Intracellular Buffering
Chronic			Renal Generation of HCO <sub>3</sub> <sup>-</sup>
Respiratory Alkalosis	↓ PCO <sub>2</sub>	↓ HCO <sub>3</sub> <sup>-</sup>	
Acute			Intracellular Buffering
Chronic			Renal secretion of HCO <sub>3</sub> <sup>-</sup>

If  $\text{PCO}_2$  &  $[\text{HCO}_3^-]$  move in opposite directions



# Normal Values

<b>ANALYTE</b>	<b>Normal Value</b>	<b>Units</b>
pH	<b>7.35 - 7.45</b>	
PCO <sub>2</sub>	<b>35 - 45</b>	mm Hg
PO <sub>2</sub>	<b>80 – 100</b>	mm Hg`
[HCO <sub>3</sub> ]	<b>22 – 26</b>	meq/L
SaO <sub>2</sub>	<b>95-100</b>	%
Anion Gap	<b>12±4</b>	meq/L

**Step Wise  
Approach to  
Interpretation  
of ABG  
Reports**



**STEP 1**

- Acidosis or Alkalosis?

**STEP 2**

- Respiratory or Metabolic?

**STEP 3**

- If Resp. Acute or Chronic and Compensation

**STEP 4**

- If Metabolic, Compensation and Anion gap?

# Step 1 Acidosis, Alkalosis, or normal?

- PH is  $< 7.35$ ,  $\Rightarrow$  Primary process is acidosis.
- PH is  $> 7.45$ ,  $\Rightarrow$  Primary process is alkalosis.

## Step 2: Is the primary disturbance Respiratory or Metabolic?

Look at the  $\text{paCO}_2$  and pH

If both go with the same direction the primary disturbance is  $\Rightarrow$  Metabolic

If both go with different direction the primary disturbance is  $\Rightarrow$  Respiratory

# Step 3: For Primary Respiratory disturbance, is it acute or chronic? then Compensation

## Acute or chronic

### PaCO<sub>2</sub> and pH

Acute condition.

for each 1 mm Hg PaCO<sub>2</sub>  $\Rightarrow$  pH changes 0.008 .

$$\text{pH changes } (\Delta \text{pH}) = 0.008 \times \Delta \text{PaCO}_2$$

Chronic condition.

for each 1 mm Hg PaCO<sub>2</sub>  $\Rightarrow$  pH changes 0.003

$$\text{pH changes } (\Delta \text{pH}) = 0.003 \times \Delta \text{PaCO}_2$$

IF RESPIRATORY, IS IT ACUTE OR CHRONIC?

➤ Acute respiratory disorder -  $\Delta \text{pH}_{(e\text{-acute})} = 0.008 \times \Delta \text{Pco}_2$

➤ Chronic respiratory disorder -  $\Delta \text{pH}_{(e\text{-chronic})} = 0.003 \times \Delta \text{pCO}_2$

➤ Compare,  $\text{pH}_{\text{measured}} (\text{pH}_m)$  v/s  $\text{pH}_{\text{expected}} (\text{pH}_e)$

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

# Step 3: For Primary Respiratory disturbance, is it acute or chronic? then Compensation

## Compensation

### PaCO<sub>2</sub> and HCO<sub>3</sub>

#### Respiratory acidosis:

➤ Acute condition.

for each 10mm Hg PaCO<sub>2</sub> ↑ ⇒ HCO<sub>3</sub> ↑ by 1 meq.

➤ Chronic condition.

for each 10mm Hg PaCO<sub>2</sub> ↑ ⇒ HCO<sub>3</sub> ↑ by 4 meq

#### Respiratory alkalosis:

➤ Acute condition.

for each 10mm Hg PaCO<sub>2</sub> ↓ ⇒ HCO<sub>3</sub> ↓ by 2 meq.

➤ Chronic condition.

for each 10mm Hg PaCO<sub>2</sub> ↓ ⇒ HCO<sub>3</sub> ↓ by 5 meq.

	acidosis	alkalosis
acute	1	2
chronic	4	5

# Step 4: For a metabolic disturbance, is the respiratory system compensating OK?

## Metabolic acidosis

$$\text{Expected PCO}_2 = (1.5 \times \text{HCO}_3^-) + 8 \pm 2$$

Winter's Equation

## Metabolic alkalosis

$$\text{Expected PCO}_2 = 40 + (0.6 \times \Delta\text{HCO}_3^-)$$

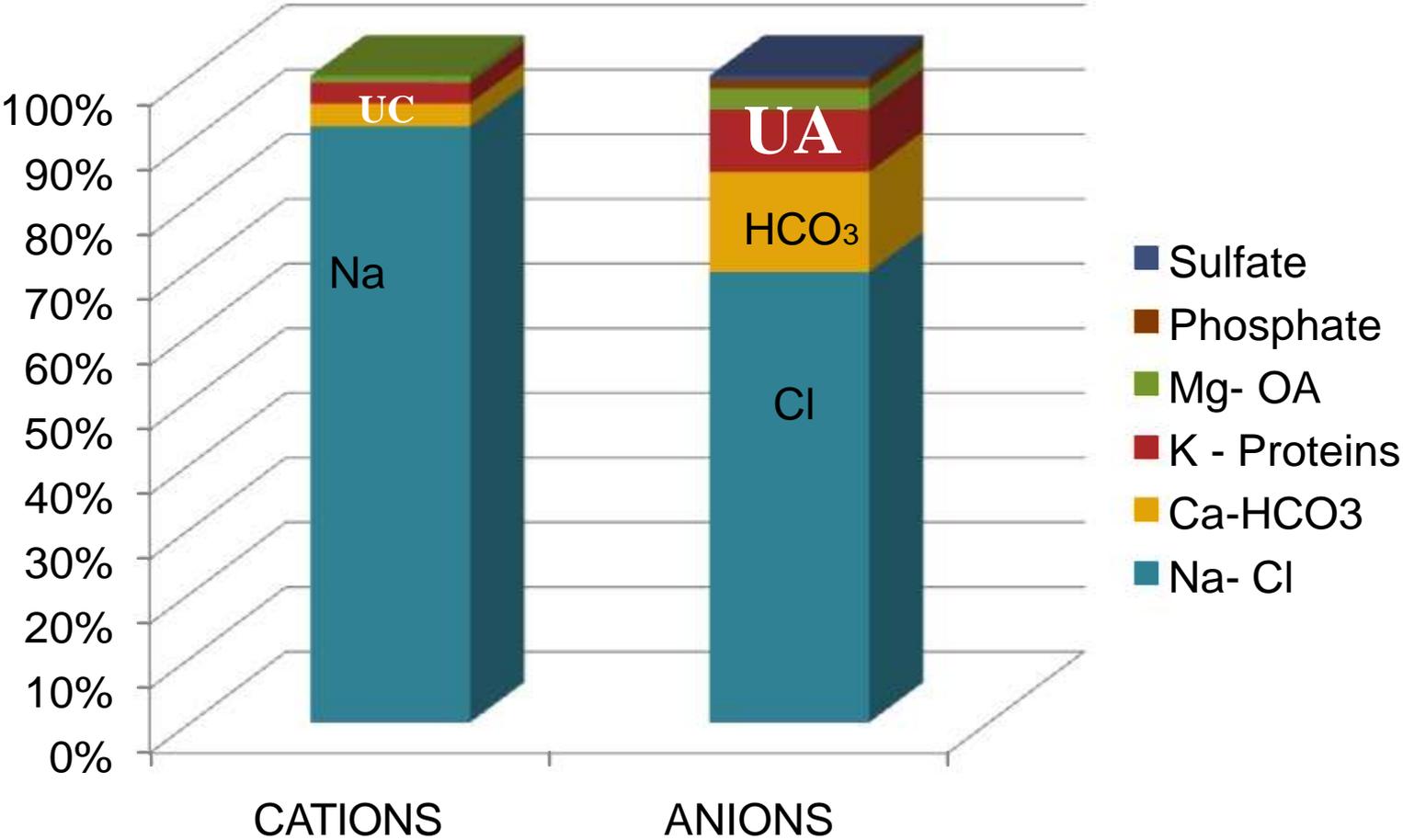
Quick rule of thumb :  $\text{PCO}_2 = \text{last 2 digits of pH}$



**For any metabolic disorder**

**Step4: For a metabolic acidosis, Anion gap?**

# Electrochemical Balance in Blood

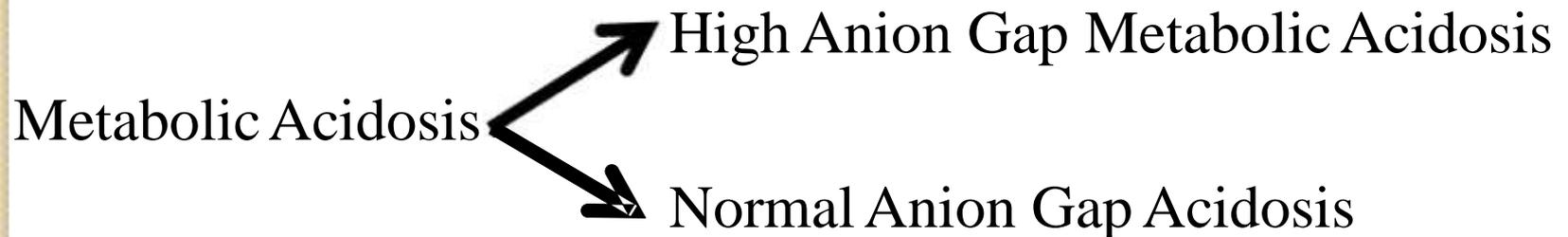


# METABOLIC ACIDOSIS- ANION GAP?

IN METABOLIC ACIDOSIS WHAT IS THE ANION GAP?

$$\square \text{ANION GAP (AG)} = (\text{Na} + \text{K}) - (\text{HCO}_3 + \text{Cl})$$

Normal Value =  $12 \pm 4$  ( 7- 16 Meq/l)



# CAUSES OF METABOLIC ACIDOSIS

(High anion gap) → (Normochloremic)

## ❖ LACTIC ACIDOSIS

## ❖ KETOACIDOSIS

- ✓ Diabetic
- ✓ Alcoholic
- ✓ Starvation

## ❖ RENAL FAILURE

(acute and chronic)

## ❖ TOXINS

- ✓ Ethylene glycol
- ✓ Methanol
- ✓ Salicylates
- ✓ Propylene glycol

# Causes of Normal Anion Gap (Hyperchloremic) Metabolic Acidosis

- H** Hyperalimentation
- A** Acetazolamide
- R** Renal tubular acidosis
- D** Diarrhea
- A** Adrenal insufficiency
- S** Spironolactone
- S** Saline infusion

# Anion Gap and Albumin

- The normal AG is affected by patients plasma albumin concentration.
- For every 1g/dl reduction in plasma albumin concentration the AG decreases by 2.5
- **Corrected AG = Calculated AG + [2.5 × (4 – albumin)]**

- A patient with poorly controlled IDDM missed his insulin for 3 days.

pH 7.1 HCO<sub>3</sub> 8 mEq/l PaCO<sub>2</sub> 20 mmhg Na 140  
mEq/l CL 106 mEq/l and urinary ketones +++

# Analysis

- pH is low so patient has **acidosis**. Low HCO<sub>3</sub> is suggestive of **metabolic acidosis**. PaCO<sub>2</sub> is also low suggestive of compensation.
- Expected compensation (fall in PaCO<sub>2</sub>) will be  
 $PaCO_2 = HCO_3 \times 1.5 + 8 = 8 \times 1.5 + 8 = 12 + 8 = 20$
- SO expected PaCO<sub>2</sub> will be 20 mmhg, which matches with actual PaCO<sub>2</sub>, suggestive of simple ABD.
- AG is 26 (AG=Na-(Cl+HCO<sub>3</sub>)=140-(106+8)=140-114=26, which is high, S/o high AG Metabolic Acidosis. Presence of urinary ketones suggests presence of diabetic ketoacidosis.
- So the patient has **high anion gap metabolic acidosis** due to DKA

- ABG of patient with stable CHF on furosemide is as follows

pH 7.48   HCO<sub>3</sub> 34 mEq/l   PaCO<sub>2</sub> 48 mmhg

- pH is high so patient has **alkalosis**.
- HCO<sub>3</sub> is high S/O **metabolic alkalosis**.
- PaCO<sub>2</sub> is high, S/O compensation (*follows same direction rule*)
- Expected compensation (rise in PaCO<sub>2</sub>) will be

- Expected PCO<sub>2</sub> = 40 + (0.6 X ΔHCO<sub>3</sub><sup>-</sup>)

- ΔHCO<sub>3</sub><sup>-</sup> = 34 - 24 = 10 mEq/L

So, Change in PaCO<sub>2</sub> = 40 + (10 x 0.6) = 46 mmHg,  
which almost matches with actual PaCO<sub>2</sub> which is 48  
mEq/L, Suggestive of simple ABD.

- So patient has **primary metabolic alkalosis due to diuretics**.

- Following sleeping pills ingestion, patient presented in drowsy state with sluggish respiration with respiratory rate 4/min.

pH 7.1 HCO<sub>3</sub> 28 mEq/L PaCO<sub>2</sub> 80 mmhg PaO<sub>2</sub> 42 mmhg

- pH is low so patient has acidosis.
- High PaCO<sub>2</sub> is S/O respiratory acidosis.
- Low PaO<sub>2</sub> –hypoxia, supports diagnosis of respiratory failure- acidosis. HCO<sub>3</sub> is also high suggestive of compensation (*same direction rule*).

### Is it Acute OR chronic respiratory disorder???

IN ACUTE ( $\Delta \text{pH}$ ) =  $0.008 \times \Delta \text{PaCO}_2 = 0.008 \times (80-40) = 0.32$

IN CHRONIC ( $\Delta \text{pH}$ ) =  $0.003 \times \Delta \text{PaCO}_2 = 0.003 \times (80-40) = 0.12$

$\Delta \text{pH} = 7.4 - 7.1 = 0.3 \dots \dots \dots$  So It is Acute Disorder

PH ↓    CO<sub>2</sub> ↑    HCO<sub>3</sub> ↑ .....HCO<sub>3</sub> increased for compensation but PH is still abnormal so there is partial compensation

- So, the patient has **Acute respiratory acidosis partially compensated due to respiratory failure, due to sleeping pills.**

## Clinical correlation: Example 1

- A 15 year old boy is brought from examination hall in apprehensive state with complain of tightness of chest.

pH 7.54   HCO<sub>3</sub> 21 mEq/L   PaCO<sub>2</sub> 21 mm of hg

## Example 1 : Analysis

- pH is high so patient has **alkalosis**.
- Low PaCO<sub>2</sub> is suggestive of **respiratory alkalosis**.

**Is it Acute OR chronic respiratory disorder???**

**IN ACUTE ( $\Delta$  pH) =  $0.008 \times \Delta$ PaCO<sub>2</sub> =  $0.008 \times (40-21) = 0.15$**

**IN CHRONIC ( $\Delta$  pH) =  $0.003 \times \Delta$ PaCO<sub>2</sub> =  $0.003 \times (40 -21) = 0.057$**

**$\Delta$  pH =  $7.54 - 7.4 = 0.14$ ..... So It is Acute Disorder**

HCO<sub>3</sub> decreased for compensation but PH is still abnormal so there is partial compensation

- **So the patient has acute respiratory alkalosis partially compensated due to anxiety.**

- A case of hepatic failure has persistent vomiting

**pH 7.54   HCO<sub>3</sub> 38 mEq/L   PaCO<sub>2</sub> 44 mmhg**

**pH is high so patient has alkalosis. HCO<sub>3</sub> is high S/O metabolic alkalosis** (due to vomiting). PaCO<sub>2</sub> is high suggestive of compensation (follows same direction rule)

- Expected compensation (rise in PaCO<sub>2</sub>) will be

$$\text{Rise in PaCO}_2 = 0.6 \times \text{rise in HCO}_3 = 0.6 \times (38 - 24) = 0.6 \times 14 = 8.4$$

- So expected PaCO<sub>2</sub> will be  $40 + 8.4 = 48.4$  mmhg. But actual value of PaCO<sub>2</sub> is lesser than expected PaCO<sub>2</sub> (44 vs 48.4 mmhg) which suggests presence of additional respiratory alkalosis (hepatic failure can cause respiratory alkalosis).

- So, patient has mixed disorder, **metabolic alkalosis with respiratory alkalosis.**

# CASE 1

62 years old Male patient

- COPD
- Breathlessness, progressively increased, aggravated on exertion, 2 days
- Chronic smoker
- expiratory rhonchi

22/7/2011	7:30 am
pH	7.20
PCO <sub>2</sub>	92 mmHg
PO <sub>2</sub>	76 mmHg
Actual HCO <sub>3</sub>	28.00 mmol/l
SO <sub>2</sub>	89
FiO <sub>2</sub>	37%

- STEP 1 – ACIDEMIA
- STEP 2 – pH ↓ PCO<sub>2</sub> ↑ Respiratory
- STEP 3 PH expected  
PH acute =  $7.40 - 0.008(92 - 40) = 6.984$   
PH chronic =  $7.40 - 0.003(92 - 40) = 7.244$   
PH (7.2) b/w 6.984 to 7.244

Primary Respiratory Acidosis,  
partially compensated

STEP 4

Mild hypoxemia

# CASE 2

63 years old ,Male patient

- CRF
- Breathlessness
- Decreased Urine Otpt. 2days
- Vomiting 10-15

31/7/2011	11:30pm
pH	7.18
PCO2	21.00
PO2	82
Actual HCO3	7.80
Base Excess	-18.80
SO2	95
Na	140.6
Chloride	102
K	4
Albumin	2.4

31/7/11	11:30pm
pH	7.18
PCO2	21.00
PO2	90
Actual HCO3	7.80
Base Excess	-18.80
SO2	95
Na	140.6
Chloride	102
K	4
Albumin	2.4

➤ STEP 1 – ACIDEMIA

➤ STEP 2 – pH ↓ PCO2 ↓  
METABOLIC

➤ STEP4 – PCO2 expected  
 $PCO2 = (1.5 \times HCO3) + 8 \pm 2$   
 $(1.5 \times 7.8) + 8 \pm 2 =$   
 $19.7 \pm 2 = 17.7 - 21.7$

➤ STEP5 ANION GAP  
 $= (Na + K) - (HCO3 + Cl)$   
 $= (140.6 + 4) - (7.80 + 102)$   
 $= 34.8$

HIGH AG Met. Acidosis

Thank  
you!