ARTERIAL BLOOD GAS ANALYSIS AND ITS IMPORTANCE IN NEUROSURGERY

Presented By : Dr.Manish Kasliwal

Why ABG…..??

- Knowledge of ABG analysis is important for every physician involved in treating critically ill patients.
- Underlying acid-base disturbances are inevitable in these patients.
- Arterial blood gas analysis reveals oxygenation status, adequacy of ventilation and acid-base balance.
- It plays a significant role in documenting and monitoring respiratory failure.

- The aims of doing a blood gas analysis are to detect

  * the presence and severity of hypoxemia and hyper(hypo)carbia.

  * changes in acid-base homeostasis, which might need further investigation and intervention.

Techniques….Arterial puncture

- **Site selection is crucial :**
  - **Radial,**
  - **Dorsalis pedis,**
  - **Brachial, or**
  - **Femoral artery (Linked to higher rates of hematoma and infection and should be used only as a last resort).**

Radial : Most common

- **The collaterality of blood flow to the hand must be checked by the Allen's test.**
- **PREPARATION OF SITE: antiseptic**
- **hand hyperextended,fixed.**
- **Firm pressure must be applied at the site of arterial puncture for at least two minutes preferably five minutes. (VERY IMPORTANT)**

Techniques….Arterial puncture

- **Air bubbles should be removed .**
- **Seal the needle with a rubber stopper to prevent the influx of air.**
- **Gently roll the syringe between your fingers to mix the blood with the heparin.**
- **ABG – as early as possible . Ideally within 30 minutes.**
- **Blood is a living medium and continues to consume oxygen and produce carbon dioxide. Blood gas results may be inaccurate if the specimen is not processed promptly.**

Techniques….. Indwelling arterial lines

- **Arterial lines provide access for frequent blood sampling and quantitative trends in blood pressure .**
  - **Umbilical In neonates**
  - **Peripheral**
    - ***radial , posterior tibial, dorsalis pedis***

COMPLICATIONS:

- **Vascular:** thrombus formation, limb ischaemia
- **Perforation:** haemorrhage
- **Miscellaneous:** extravasation of cannula, difficulties with sampling
- **Infectious**

Techniques….. Indwelling arterial lines

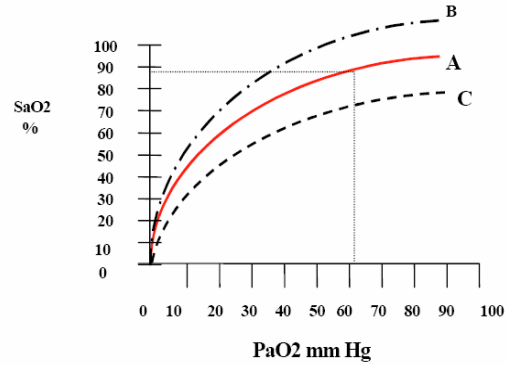
- **Assemble equipment**
- **Using aseptic technique, aspirate 1ml of blood using a new sterile 2ml syringe and do not discard it.**
- **Place heparinised syringe, open tap to allow blood into syringe. To adequately mix the sample invert the syringe 4 X and roll the syringe . Do not shake the sample.**
- **Replace blood previously withdrawn**
- **The third syringe of heparinised saline solution (0.2-0.3ml) is used to flush the line clear.**
- **Determine patency of arterial line by recommencing infusion.**

Why an ABG instead of Pulse oximetry?

When …..

- Hb saturation
- Immediate
- Continuous data
- Non-invasive

BECAUSE……



- Pulse oximetry becomes unreliable when saturations fall below 70-80%.

- Technical sources of error (ambient or fluorescent light, hypo perfusion, nail polish, skin pigmentation)

- Pulse oximetry cannot interpret met hemoglobin or carboxyhemoglobin.

- Pulse oximetry does not assess ventilation ($PaCO_2$) or acid base status.

Normal metabolism and its dysfunction

Cellular function :dependent on regular supply of glucose, oxygen and water.

Volatile acids like carbonic acid from tissue oxidation

Respiratory system :eliminates volatile acids in the form of CO_2

Fixed acids like sulphuric acid, phosphoric acid, lactic acid, keto acid (products of intermediary metabolism) constantly produced.

Renal mechanisms eliminate fixed acids in the form of hydrogen ions.

In pathological states: accumulation of the above acids and resulting in acid-base disturbance.

Renal and respiratory system take the brunt to mitigate the acid-base disturbance. The buffer base system which includes intra cellular and extra cellular buffers helps to maintain homeostasis in the immediate period.

What is Acid-base balance

- Acid-base balance is defined by the concentration of hydrogen ions.

- In order to achieve homeostasis, there must be a balance between the intake or production of hydrogen ions and the net removal of hydrogen ions from the body.

Acid & Base

- Molecules containing hydrogen atoms that can **release** hydrogen ions in solutions are referred to as an acid.
- An example of an acid is hydrochloric acid (HCL)
- A base is an ion that can **accept** a hydrogen ion.
- An example of a base is the bicarbonate ion. (HCO3)

How is Acid-Base balance measured

- Hydrogen ion concentration is expressed on a logarithm scale using pH units (part/percentage hydrogen).
- 7.0 being neutral
- Body systems carefully control pH of the body within the range of 7.35 - 7.45

Henderson - Hasselbalch Equation

pH = pKa + log [HCO3-]/[H2CO3]

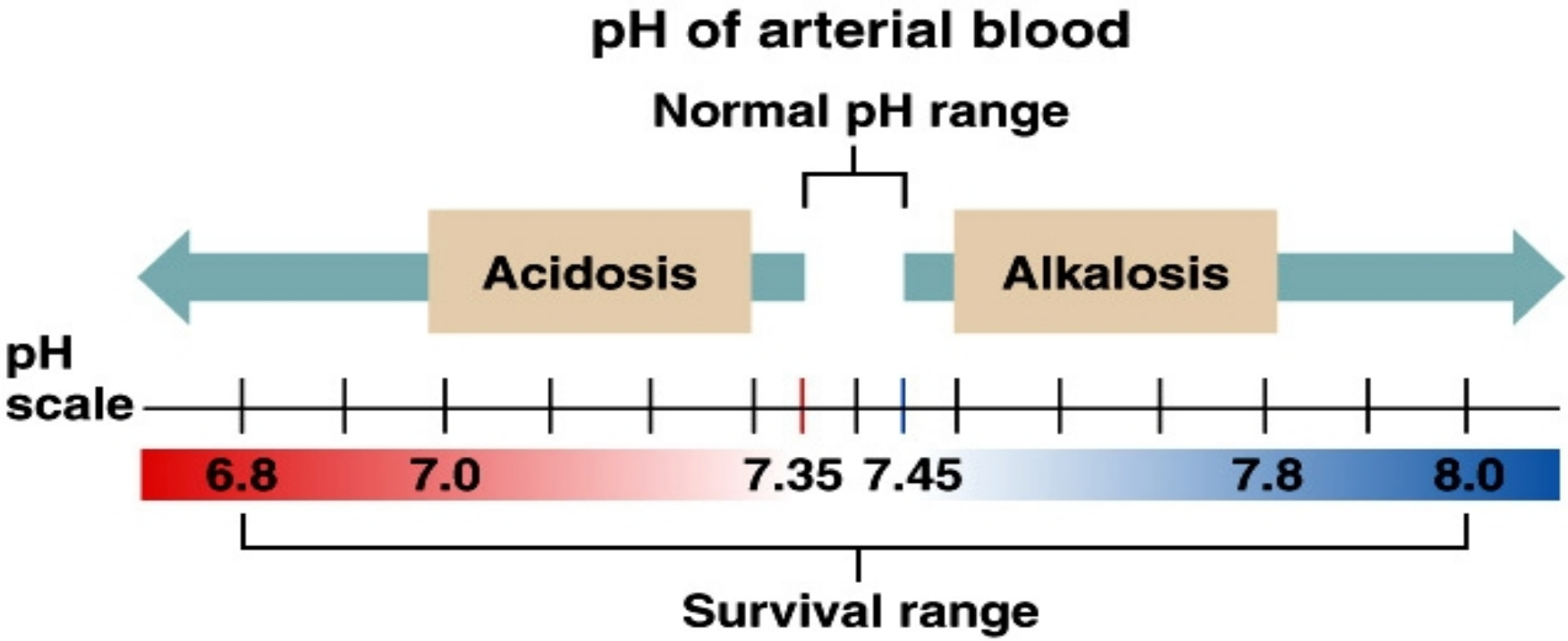
pH = pKa + log [HCO3-]/**0.03** x PCO2

pH = **6.1** + log **[HCO3-]**/0.03 x **PCO2**

7.4 = 6.1 + log **20/1**

7.4 = 6.1 + 1.3

- The solubility constant of CO_2 is 0.03
- The pK_a of carbonic acid is 6.1
- Plasma pH equals 7.4 when buffer ratio is 20/1
- Plasma pH may be affected by a change in either the bicarbonate concentration or the PCO_2
- The $[HCO_3^-]$ and PCO_2 values determine plasma pH



How the Body defends against fluctuations in pH

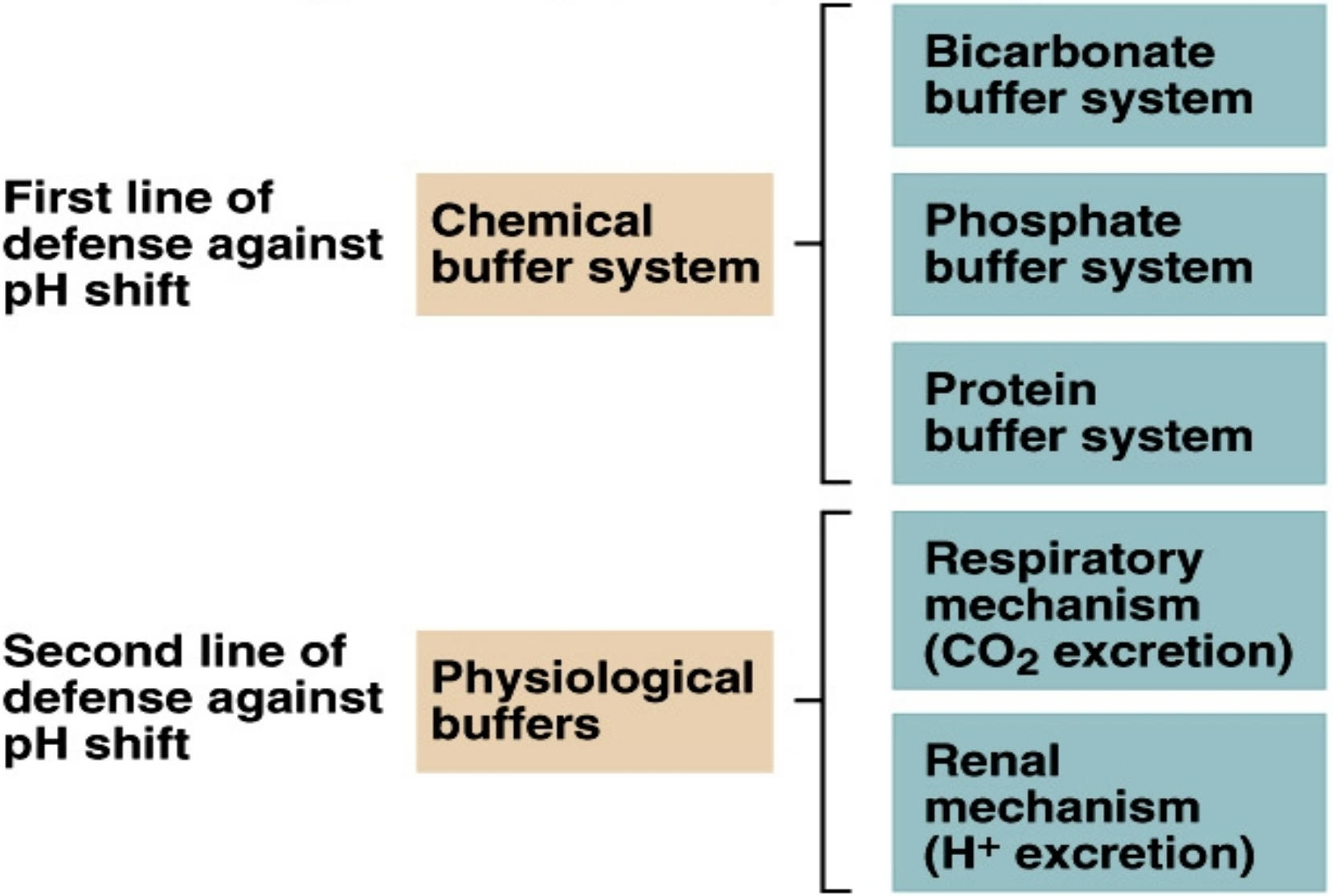
- A buffer is a solution that contain a weak acid and its conjugate base or a weak base and its conjugate acid

- Buffers are substances that neutralize acids or bases in effect, limiting the change in hydrogen ion concentration (and so pH) when hydrogen ions are added or removed from the solution. (Like a Sponge !!!!!)

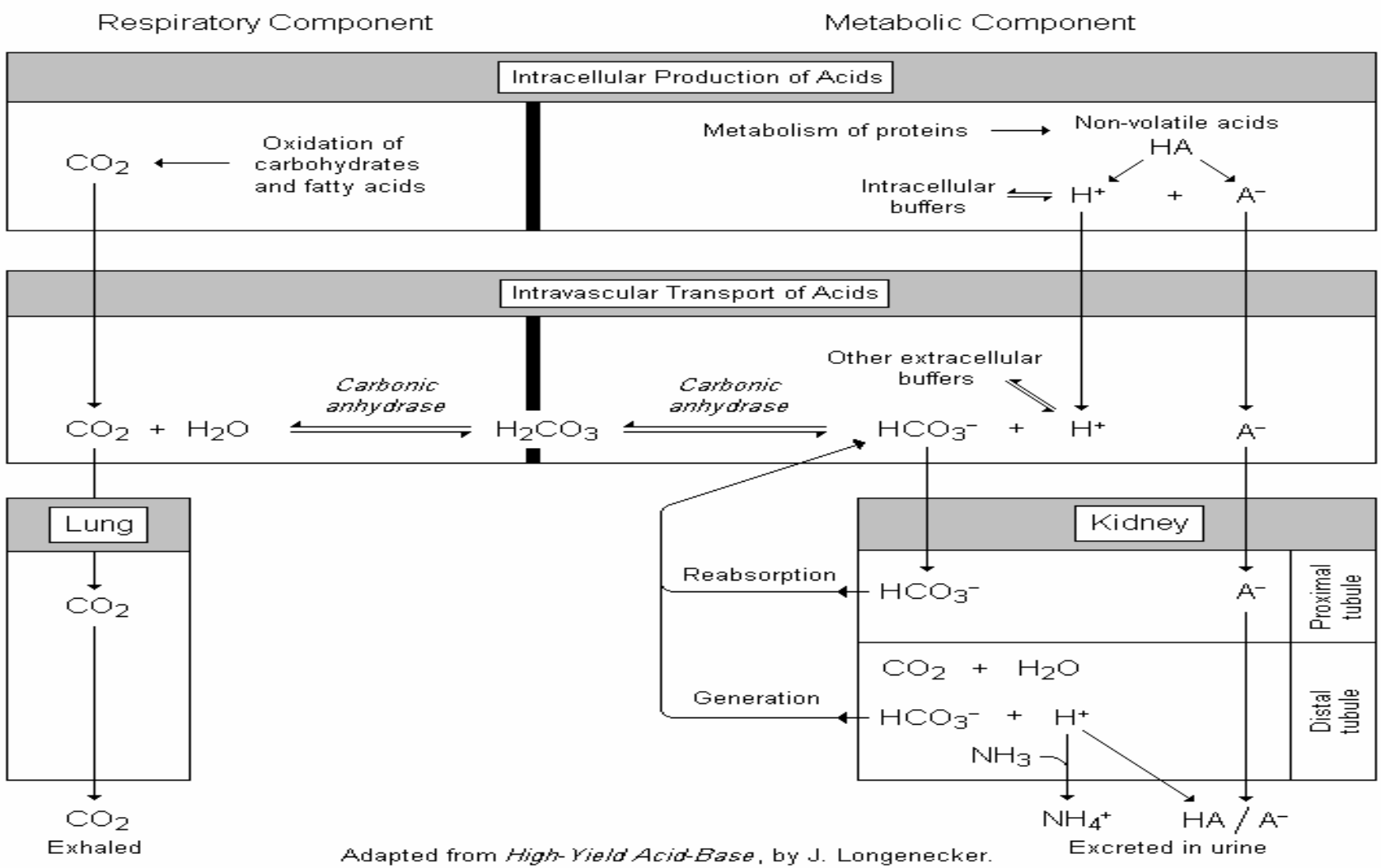
How the Body defends against fluctuations in pH

Three Systems in the body:

- **Buffers in the blood – Immediately. Serve as a first line of defense against changes in the acid-base balance**
- **Respiration through the lungs – Intermediate**
- **Excretion by the kidneys – More Slowly**



Overview of Acid-Base Physiology



Adapted from *High-Yield Acid-Base*, by J. Longenecker.

- Physiological buffers

1. bicarbonate/carbonic acid(ECF)
2. hemoglobin(BLOOD)
3. plasma proteins(ICF)
4. phosphate (URINARY)
5. ammonia (URINARY)
6. bone(ECF)

Buffer base (BB)

- **Sum of the bicarbonate and the non-volatile buffer ions (specially serum albumin, phosphate, hemoglobin)normally it is 48 mmol/l**

- **BB increases in met alkalosis & decreases in met acidosis**

Respiration through the Lungs

- CO_2 which is formed during cellular metabolism forms Carbonic acid in the blood decreasing the pH

- When the pH drops respiration rate increases this hyperventilation increases the amount of CO_2 exhaled thereby lowering the carbonic acid concentration and restoring homeostasis

Excretion by the Kidneys

- The kidneys play the primary role in maintaining long term control of Acid-Base balance
- The kidney does this by selecting which ions to retain and which to excrete

ACID-BASE DISORDERS

- Simple acid-base disorders have one primary abnormality.
- The four primary disorders are
  - respiratory acidosis,
  - respiratory alkalosis,
  - metabolic acidosis
  - metabolic alkalosis.
  - Mixed acid-base disorders have more than one abnormality. Two to three primary disorders can be combined together to result in a mixed disorder.

Acid-base Values and Acid-base Disturbances

Condition Normal	HCO3 22-26	pCO2 35-45	pH 7.35-7.45
Metabolic Acidosis	<22meq/l	35-45	<7.35
Metabolic Alkalosis	>26	35-45	>7.45
Respiratory Acidosis	>24	>45	<7.35
Respiratory Alkalosis	<24	<35	>7.45

Compensation

Primary Disorder	Compensatory Mechanism
Metabolic acidosis	Increased ventilation
Metabolic alkalosis	Decreased ventilation
Respiratory acidosis	Increased renal reabsorption of HCO_3^- in the proximal tubule Increased renal excretion of H^+ in the distal tubule
Respiratory alkalosis	Decreased renal reabsorption of HCO_3^- in the proximal tubule Decreased renal excretion of H^+ in the distal tubule

Compensation……

- It is the body response to acid-base imbalance.
- **Complete** if brought back within normal limits
- **Partial :** if range is still outside norms.
- The body NEVER overcompensates !!!
- Metabolic disturbance : **Respiratory compensation in the form of** hyperventilation or hypoventilation.
- If problem is respiratory : Renal mechanisms can bring about **metabolic compensation.**

Compensation……

- Compensation : attempt to return the pH to normal
- ABG's show that compensation is present when
  - the pH returns to normal or near normal
- If the nonprimary system is in the normal range (CO_2 35 to 45) (HCO_3 22-26), then that system is not compensating for the primary.
- For example:
  - In respiratory acidosis (pH<7.35, CO_2>45), if the HCO_3 is >26, then the kidneys are compensating by retaining bicarbonate.
  - If HCO_3 is normal, then not compensating.

Compensation in metabolic disorders

DISTURBANCES	RESPONCES	EXPECTED CHANGE
METABOLIC ACIDOSIS	↓ $PaCO_2$	[1.5 x HCO3] + (8 ± 2)
METABOLIC ALKALOSIS	↑ $PaCO_2$	[0.7 x HCO3] + (21 ± 2)

Compensation in respiratory acid-base disorder

Disturbance	Response	Expected change
Respiratory acidosis		
Acute	↑HCO_3	1meq/10mm ↑$PaCO_2$
Chronic	↑HCO_3	4meq/10mm ↑$PaCO_2$
Respiratory alkalosis		
Acute	↓ HCO_3	2 meq /10mm ↓ $PaCO_2$
Chronic	↓ HCO_3	4meq /10mm ↓ $PaCO_2$

Compensation in respiratory acid-base disorder

Acute respiratory acidosis	▲ pH=0.008 x ▲ Pco2
Chronic respiratory acidosis	▲ pH=0.003 x ▲ Pco2
Acute respiratory alkalosis	▲ pH=0.008 x ▲ Pco2
Chronic respiratory alkalosis	▲ pH=0.017 x ▲ Pco2

Respiratory Acidosis

- pH < 7.35, $PaCO_2$ > 45mm Hg
- Mechanism - Hypoventilation or Excess CO_2 Production
- Etiology - COPD, Neuromuscular Disease, Respiratory Center Depression, Late ARDS, Inadequate mechanical ventilation, Sepsis or Burns, Excess carbohydrate intake
- Compensation - Kidneys eliminate hydrogen ion and retain bicarbonate ion

Respiratory Acidosis - Contd

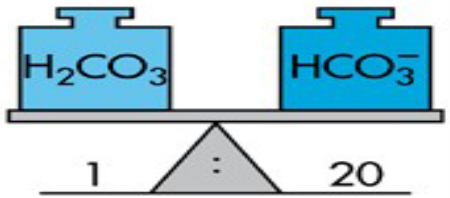
1. **Symptoms**

   - Dyspnea, Disorientation or coma
   - Dysrhythmias

2. **Treatment**

   - Treat underlying cause
   - Support ventilation
   - Correct electrolyte imbalance

a) Metabolic balance before onset of acidosis



H_2CO_3 : Carbonic acid

HCO_3^- : Bicarbonate ion

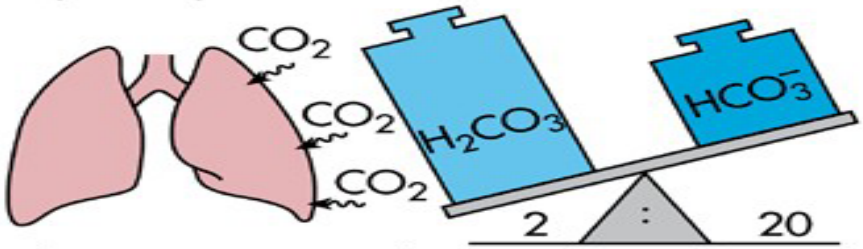
$(Na^+ \bullet HCO_3^-)$

$(K^+ \bullet HCO_3^-)$

$(Mg^{++} \bullet HCO_3^-)$

$(Ca^{++} \bullet HCO_3^-)$

b) Respiratory acidosis



Breathing is suppressed, holding CO_2 in body

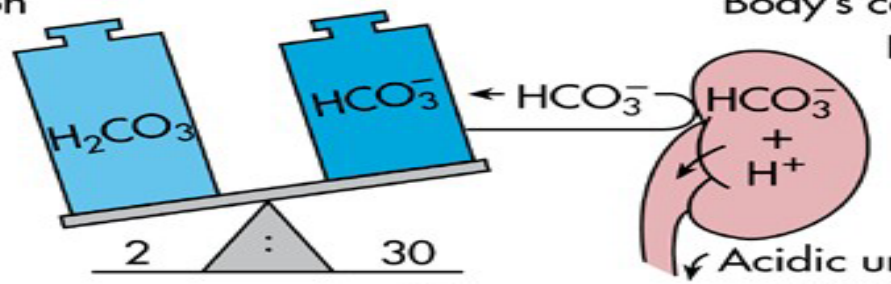
Primary change

pH — decreases

P_{CO_2} — increases

HCO_3^- — no change

c) Body's compensation



Kidneys conserve HCO_3^- ions and eliminate H^+ ions in acidic urine

Respiratory Alkalosis

– pH above 7.45 , CO_2 less than 35

- Etiology - Hyperventilation due to
  - » Extreme anxiety, stress, or pain
  - » Elevated body temperature
  - » Over ventilation with ventilator
  - » Hypoxia
  - » Drug overdose (e.g.. Salicylates)
  - » Hypoxemia (emphysema, asthma or pneumonia)
  - » CNS trauma or tumor

Respiratory Alkalosis (cont)

Symptoms

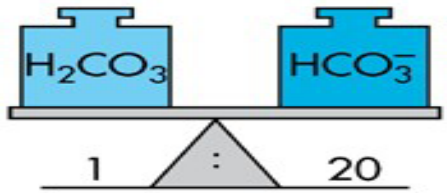
- Tachypnea or Hyperpnea
- Complaints of chest pain
- Light-headedness, syncope, coma, seizures
- Numbness and tingling of extremities
- Difficult concentrating, tremors, blurred vision
- Weakness, Paresthesia, Tetany

Respiratory Alkalosis (cont)

Compensation - Kidneys conserve hydrogen ion & Excrete bicarbonate ion

- Treatment
  - Monitor ABGs
  - Treat underlying disease
  - Assist patient to breathe more slowly
  - Help patient to breathe in a paper bag or apply rebreather mask
  - Sedation

a) Metabolic balance before onset of alkalosis



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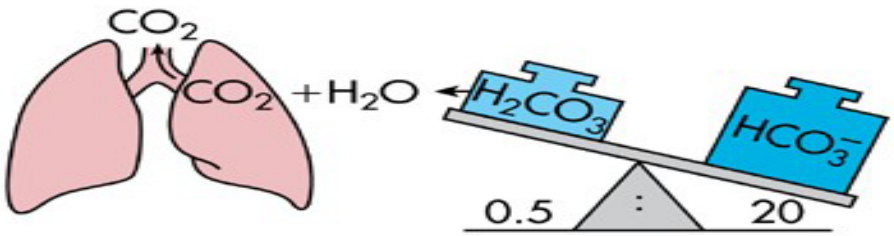


Hyperactive breathing "blows off" CO_2

Primary change

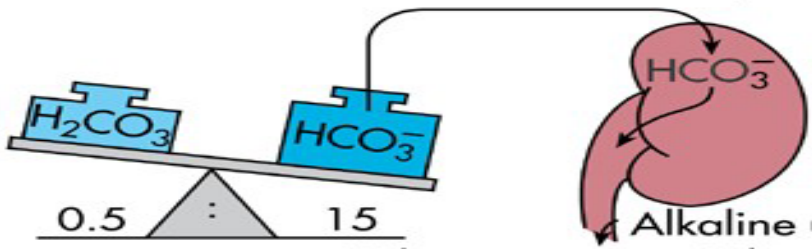
pH — increases

P_{CO_2} — decreases

HCO_3^- — no change

c) Body's compensation

Body's correction



Kidneys conserve H^+ ions and eliminate HCO_3^- in alkaline urine

Metabolic Alkalosis

1. Etiology
   a. Acid loss due to
      - Vomiting
      - Gastric suction
   b. Loss of potassium due to - Steroids, Diuresis
   c. Antacids (overuse of)

2. Symptoms - Hypoventilation (compensatory)
   - Dysrhythmias, Dizziness, Paresthesia, Numbness, Tingling of extremities
   - Hypertonic muscles, Tetany

Metabolic Alkalosis – Contd

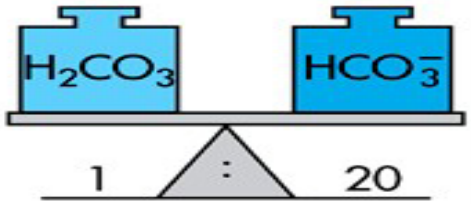
- Lab: pH > 7.45, Bicarbonate > 26
  - CO_2 normal or increased w/comp
  - Hypokalemia, Hypocalcaemia

3. Treatment
   - Treat underlying cause
   - Give potassium
   - Chloride replacement mainstay of therapy.
   - NaCl, HCl or KCl.

Metabolic Alkalosis – Contd

- Isotonic saline most common because the Cl responsive MA associated with volume depletion.
- Cl deficit : 0.3 x Wt. (kg) x (100- Plasma Cl)
- Vol : Cl Deficit/ 154 (L)
- KCl generally not an effective because cannot be corrected more than 40 meq/hr.
- HCl : corrosive.

a) Metabolic balance before onset of alkalosis



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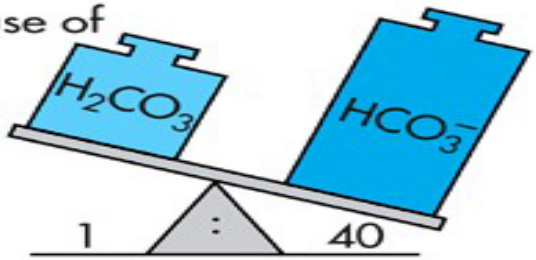
b) Metabolic alkalosis HCO_3^- increases because of loss of chloride ions or excess ingestion of sodium bicarbonate



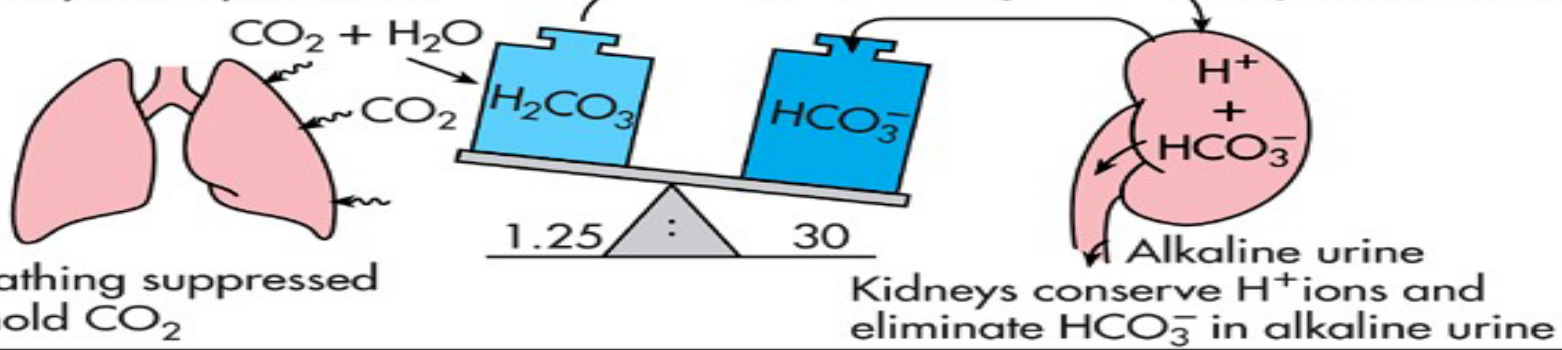
Primary change

pH — increases

P_{CO_2} — no change

HCO_3^- — increases

c) Body's compensation



Metabolic Acidosis

Etiology

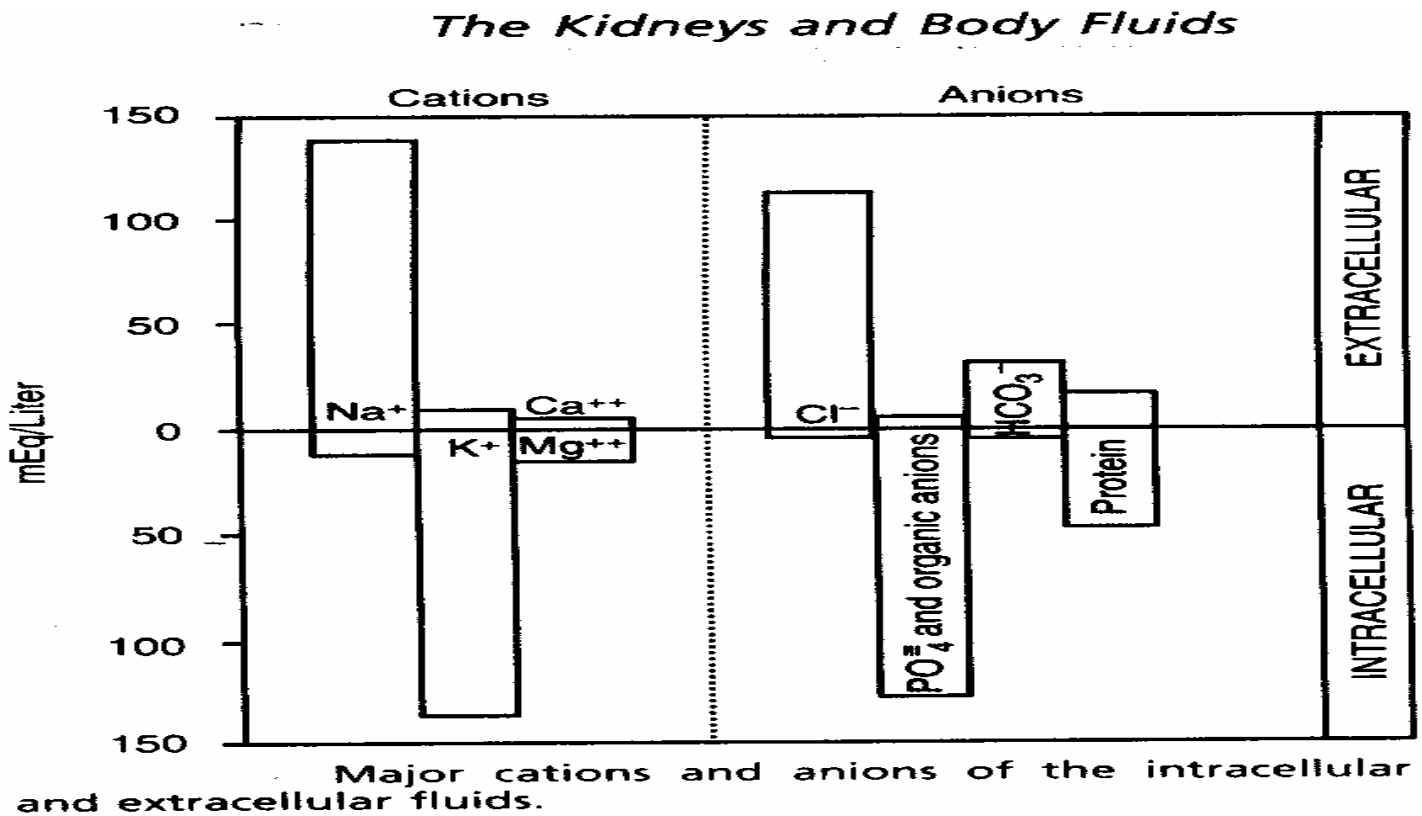
1. Conditions that increase acids in the blood
    - Renal Failure
    - DKA
    - Starvation or Malnutrition
    - Lactic acidosis

2. Prolonged diarrhea

3. Toxins

4. Carbonic anhydrase inhibitors - Diamox

Concept of Anion Gap

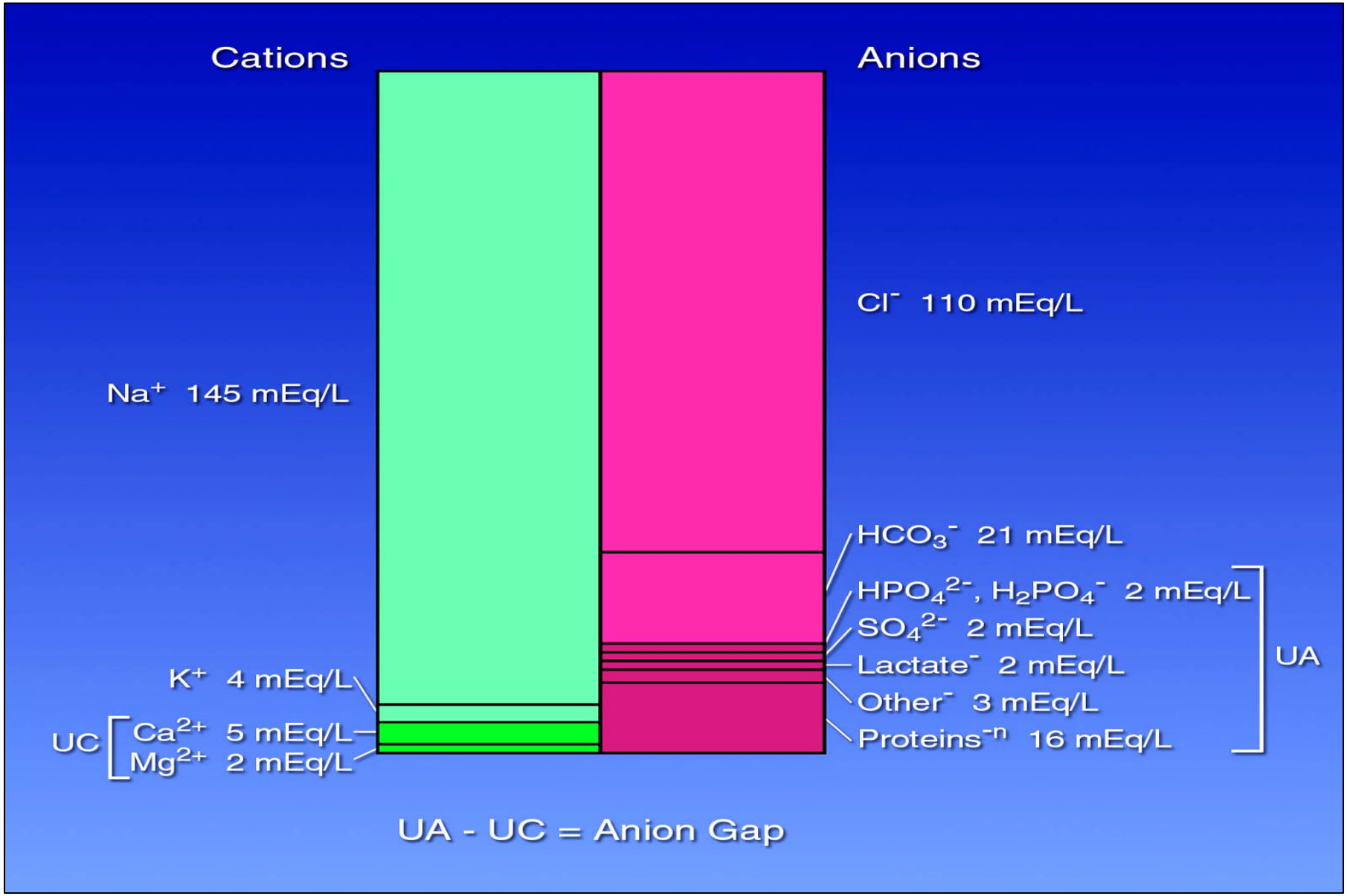


Major cations and anions of the intracellular and extracellular fluids.

Concept of Anion Gap…….

- Organisms exist in a state of electro neutrality with major and minor cations balanced by similar anions.
- There are anions and cations that are easily measured, i.e. Na^+, Cl^- and HCO_3^-.
- Normally $[Na^+]$ is in excess of the sum of $[Cl^-]$ and $[HCO_3^-]$.
- Unmeasured anions include inorganic anions (SO_4^{2-} and PO_4^{3-}), and organic anions (lactate, β-hydroxybutyrate and salicylate), and anionic proteins.
- $(AG) = [Na^+] - ([Cl^-] + [HCO_3^-]) = 10 \pm 2$ mEq /L

Concept of Anion Gap



Concept of Urinary Anion Gap

- The cations and anions normally present in urine are Na+, K+, NH4+, Ca++,Mg++ and Cl-, HCO3-, sulphate, phosphate and some organic anions.

- Only Na+, K+ and Cl- are commonly measured.
- Cl- + UA = Na+ + K+ + UC

  UAG = (UA - UC) = [Na+]+ [K+] - [Cl-]

- The Urinary Anion gap : differentiate between GIT and renal causes of a hyperchloraemic metabolic acidosis.

- Urinary Anion Gap (UAG) provides a rough index of Urinary ammonium excretion. Ammonium is positively charged so a rise in its Urinary concentration will cause a fall in UAG .

Concept of Urinary AG ....

- If the acidosis is due to loss of base via the bowel : the kidneys can response appropriately by increasing ammonium excretion : net loss of H+ from the body : decreased UAG.

- If the acidosis is due to loss of base via the kidney : not able to increase ammonium excretion : UAG will not be increased.

- In a patient with a hyperchloraemic metabolic acidosis:

  - A negative UAG suggests GIT loss of bicarbonate (eg diarrhoea)
  - A positive UAG suggests impaired renal distal acidification (i.e. renal tubular acidosis).

Anion-gap

HIGH

L or N

- Lactic acidosis
- Ketoacidosis
  - *diabetes
  - *alcohol
  - *starvation
- Toxins
  - *Salicylate,methanol ,ethyl glycol
- Renal failure

Urinary anion-gap

+VE

*Renal

Tubular

Acidosis

I,II,IV

-VE

*Diarrhea

*Fistulae

Clinical Manifestations Of Metabolic Acidosis

- Headache, Drowsiness, Nausea, Vomiting, Diarrhea
- Kussmaul's Respiration, Fruity smelling breath
- Hyperkalemia, Hypotension, Bradycardia
- G.I. Distension

CONSEQUENCES OF SEVERE ACIDEMIA (pH <7.2)

- Cardiovascular: impaired cardiac output and perfusion, cardiac arrhythmias.
- Cerebral: altered mental status.
- Respiratory: hyperventilation progressing to respiratory failure due to respiratory muscle fatigue.
- Metabolic: hyperkalemia can lead to lethal cardiac arrhythmia.

Compensation for Metabolic Acidosis

- **Increased ventilation**
- **Renal excretion of hydrogen ions if possible**
- **K^+ exchanges with excess H^+ in ECF**
- **H^+ into cells, K^+ out of cells**

Treatment….Principles

- 1. Accurate diagnosis of the cause.
- 2. Treat the underlying disorder as the primary therapeutic goal
  - Fluid, insulin and electrolyte replacement : DKA
  - Administration of Bicarbonate and/or dialysis may be required for acidosis associated with renal failure
  - Restoration of an adequate intravascular volume and peripheral perfusion : Lactic acidosis.
- 3. Supportive treatment (eg fluids, oxygen, treatment for hyperkalaemia) including all appropriate emergency management
- 4. In most of the cases IV Sodium bicarbonate NOT necessary, NOT helpful, & may even be harmful in the treatment of metabolic acidosis.

Indications for direct correction of acidosis by giving base:

- **The cause cannot be corrected. e.g. In non organic acidosis.**

- **Where the acidosis is depressing the circulation (i.e. to break the vicious circle of myocardial depression)**

TREATMENT

- NaHCO3 is not given intravenously until the blood pH is at least 7.2 and the plasma [HCO3-] 10 mmol / L.
- HCO3 deficit (meq) = 0.6 x BW x (Desired HCO3 – Measured HCO3)
- One half should be given over 30 minutes and the remaining over 4-6 hrs.
- The goal is to increase the pH to 7.25 and HCO3 level to 15 meq/ L and NOT to normal.
- ABG should be determined after 30 min.
- Maintain adequate ventilation.

Hazards of bicarbonate

- Hypernatremia
- Hyperosmolality
- Volume overload
- Rebound or ‘overshoot’ alkalosis
- Hypokalaemia
- Impaired oxygen unloading due to left shift of the oxyhaemoglobin dissociation curve
- Acceleration of lactate production by removal of acidotic inhibition of glycolysis
- Hypercapnia.

Bicarbonate containing buffer solutions

	7.5% $NaHCO_3$	Carbicarb
Sodium	0.9 mEq/ml	0.9 mEq/ml
Bicarbonate	0.9 mEq/ml	0.3 mEq/ml
Dicarbonate	-	0.3 mEq/ml
PCO_2	>200mmHg	3 mmHg
Osmolality	1461mOsm/kg	1667 mOsm/kg
pH (25 C)	8.0	9.6

- Carbicarb is a buffer solution that is 1:1 mixture of sodium bicarbonate and disodium carbonate, It has less bicarbonate and much lower PCO_2 than 7.5% $NaHCO_3$. solution.

- Carbicarb is more effective buffer than $NaHCO_3$

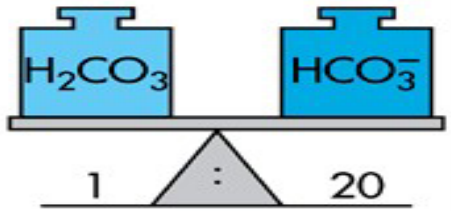
- Tromethamine (TRIS or THAM) provides intracellular and extra cellular buffering without generating CO_2

- THAM provides effective buffering over the pH range of 6.8 – 8.8

- Available in 0.3M solution (0.3 mEq/l)

- THAM (mEq/l) = 0.3 × Bodyweight (kg) × base deficit

a) Metabolic balance before onset of acidosis



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HCO_3^- : Bicarbonate ion

$(Na^+ \bullet HCO_3^-)$

$(K^+ \bullet HCO_3^-)$

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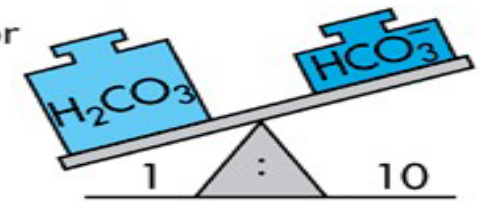
$(Ca^{++} \bullet HCO_3^-)$

b) Metabolic acidosis HCO_3^- decreases because of excess presence of ketones, chloride, or organic acid ions



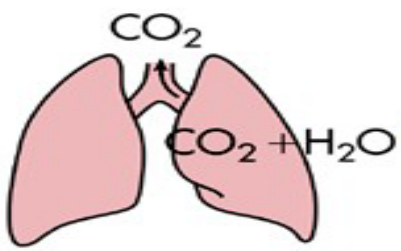
Primary change

pH — decreases

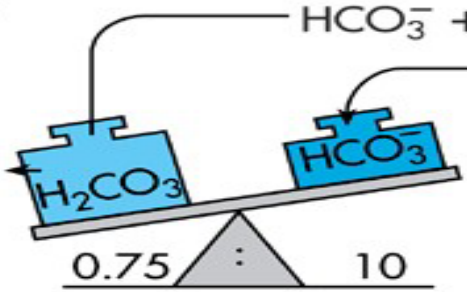
P_{CO_2} — no change

HCO_3^- — decreases

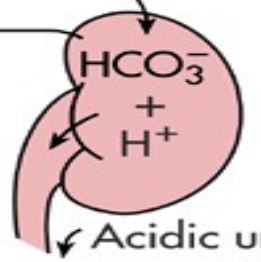
c) Body's compensation



Hyperactive breathing to "blow off" CO_2



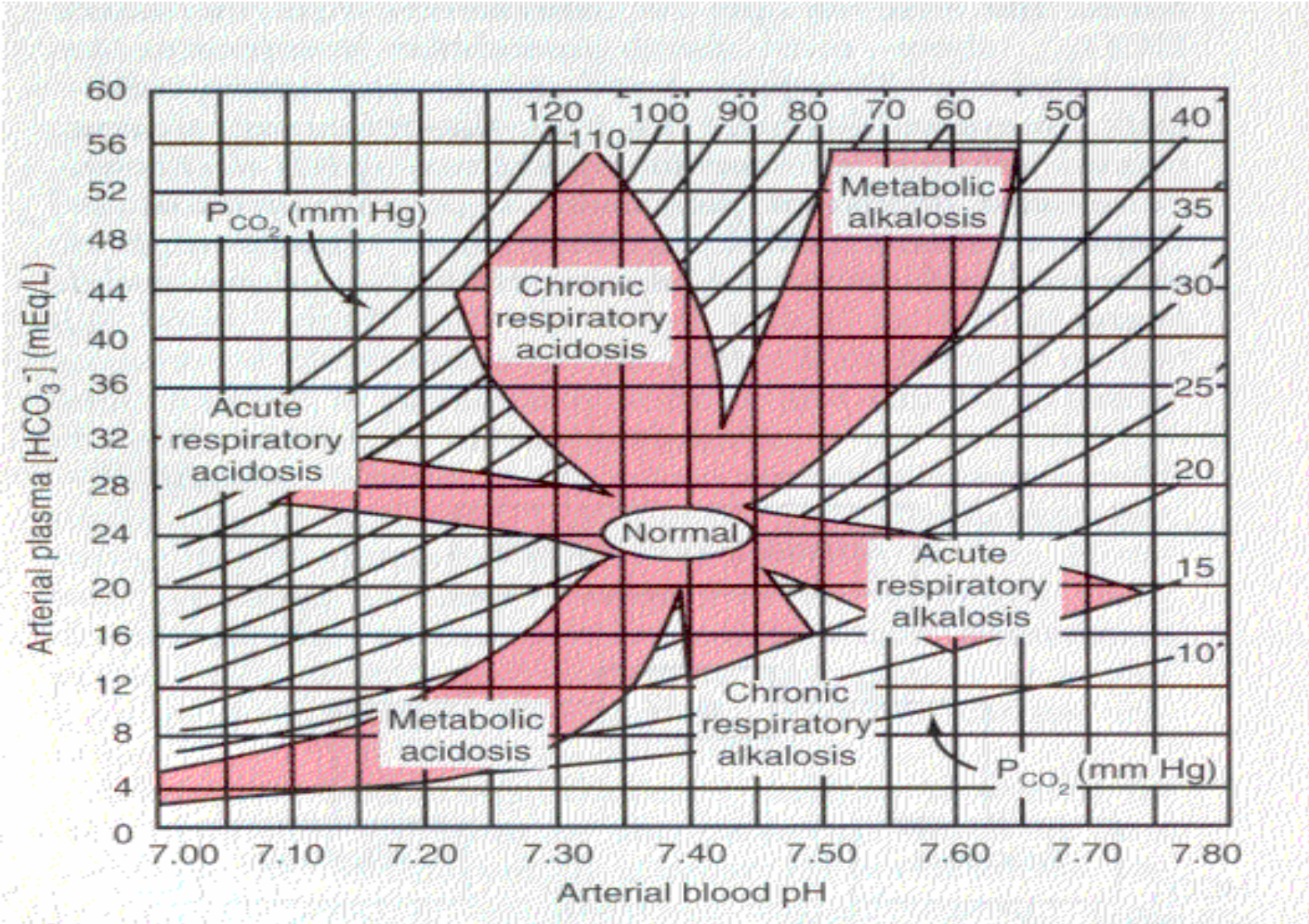
Body's correction



Acidic urine

Kidneys conserve HCO_3^- and eliminate H^+ ions in acidic urine

Acid-base Nomogram



Mixed Acid-base disorders are common

- In chronically ill respiratory patients, mixed disorders are probably more common than single disorders

  e.g., RAc + MAlk, RAc + Mac, Ralk + MAlk.

- In renal failure (and other patients) combined MAlk + MAc is also encountered.

- Always be on look out for mixed acid-base disorders. They can be missed easily !!

Tips to diagnosing mixed acid-base disorders

Don't interpret any blood gas data for acid-base diagnosis without closely examining the serum electrolytes: Na^+, K^+, Cl^- and CO_2.

- A serum CO_2 out of the normal range always represents some type of acid-base disorder.
- High serum HCO3 indicates MAlk &/or bicarbonate retention as compensation for resp.acid.
- Low serum HCO3 indicates MAci. &/or bicarbonate excretion as compensation for respiratory alkalosis

Tips to diagnosing mixed acid-base disorders - Contd

Single acid-base disorders do not lead to normal blood pH.

Although pH can be normal (7.35 - 7.45) with a mild single disorder, a truly normal pH with distinctly abnormal HCO_3^- and $PaCO_2$ invariably suggests two or more primary disorders.

Example: pH 7.40, $PaCO_2$ 20 mm Hg, HCO_3^- 12 mEq/L, in a patient with sepsis. Normal pH results from two co-existing and unstable acid-base disorders: acute respiratory alkalosis and metabolic acidosis.

ABG Interpretation

(I)

RADIOMETER ABL 800 FLEX

ABL835 E M TECHNOLOGIES — 10:09 AM — 9/30/2005
PATIENT REPORT — Syringe - S 195uL — Sample # 3665

Identifications

Patient ID	NSOT 3
Patient First Name	GUPTESHWAR
Sample type	Arterial
T	37.0 °C
$FO_2(I)$	21.0 %
Report Layout	ABG [FULL]

Blood Gas Values			
‡ pH	7.452		[7.350 - 7.450]
pCO_2	33.9	mmHg	[32.0 - 45.0]
‡ pO_2	163	mmHg	[83.0 - 108]
Temperature Corrected Values			
pH(*T*)	7.452		
$pCO_2(T)$	33.9	mmHg	
$pO_2(T)$	163	mmHg	
Electrolyte Values			
cNa^+	137	mmol/L	[136 - 146]
‡ cK^+	3.3	mmol/L	[3.4 - 4.5]
‡ cCa^{2+}	1.12 *	mmol/L	[1.15 - 1.29]
‡ cCl^-	107	mmol/L	[98 - 106]
Metabolite Values			
cGlu	104	mg/dL	[70 - 105]
‡ cLac	3.0	mmol/L	[0.5 - 1.6]
Oximetry Values			
ctHb	12.8	g/dL	[12.0 - 16.0]
‡ sO_2	100.8	%	[95.0 - 99.0]
Calculated Values			
$cHCO_3^-(P)_c$	23.3	mmol/L	
Anion Gap_c	6.3	mmol/L	
Hct_c	39.4	%	
ctO_{2e}	18.1	Vol%	
$pO_2(A\text{-}a)_e$	.....	mmHg	
$mOsm_c$	280.0	mmol/kg	
$cBase(B)_c$	0.3	mmol/L	
$cBase(Ecf)_c$	-0.2	mmol/L	
ctO_{2e}	18.1	Vol%	
$cH^+{}_c$	35.3	nmol/L	

Notes

‡ Value(s) above the critical limits
‡ Value(s) below the critical limits
c Calculated value(s)
e Estimated value(s)
* User correction applied to value(s)

Printed 10:10:17AM 05-09-30

E S C H W E I L E R
SYSTEM 3000 BGA PLUS E

NAME : Manju Devi

#		
DATE	10.02.06	
TIME	10:51	
BP	733	mmHg
TEMP.	37.0	C
HB	11.0	g/0.1
HCT	33.0	%
FIO2	40.0	%
RQ	0.85	
PO2	237.2	mmHg
PCO2	30.2	mmHg
PH	7.565	
K	3.6	mmol/l
NA	143	mmol/l
HCO3A	26.5	mmol/l
HCO3S	30.6	mmol/l
BE	6.6	mmol/l
SBE	5.5	mmol/l
TCO2	27.2	mmol/l
BB	52.9	mmol/l
O2SAT	99.7	%
O2-CT	15.4	%
P50	22.37	mmHg
AADO2	3.8	mmHg

ACID / BASE STATUS

COMB. RESP. ALKALOSIS
AND NON-RESP. ALKALOSIS

xxxxxxxxxxxxxxxxxxxxxx

pH

- pH indicates the acidity or alkalinity of the sample. pH is the negative logarithm of the hydrogen ion activity, pH = - log(H+).

  The measure of the overall acid-base status of the blood.

- Most metabolic processes depend on pH being kept within a relatively

  narrow range.

- Reference ranges

  pH reference range : 7.35 - 7.45

*p*O2 Arterial oxygen tension

*p*O2 is the oxygen partial pressure in a gas phase in equilibrium with the blood. Indicator of the oxygen uptake in the lungs.

N : 83-108 mmHg. Declines with age.

A normal *p*O2, while breathing room air, indicates an adequate pulmonary oxygen uptake.

- High *p*O2 leads to cellular hyperoxia. Toxic, if sustained. Unless a high level is specifically desired, FIO2 should be reduced to normalize *p*O2.

- If *p*O2 is too low, signify an inadequacy of the oxygen uptake from the lungs.

  Review Pulmonary and ventilatory status. Changes in *F*O2(I) and/or optimizing ventilator settings may be indicated along with, if possible, specific treatment of the pulmonary or cardiac changes causing the hypoxemia.

*p*CO2
Carbon dioxide tension

- *p*CO2 is the carbon dioxide partial pressure in a gas phase in equilibrium with the blood. 35 – 45 mm Hg

A. Low *p*CO2 Alveolar hyperventilation. Common causes:

Primary:

Excessive mechanical ventilation or Psychogenic hyperventilation

Decreasing *p*CO2(a) : pulmonary vasodilatation and vasoconstriction in several parts including the cerebral vasculature.

The net result of decreasing *p*CO2 may therefore be an impairment of oxygen supply to the tissues, especially in the central nervous system (CNS).

Increasing *p*CO2(a) may cause hypoxemia because the alveolar oxygen tension falls according to the alveolar gas equation. In addition, the right shift of the ODC, induced by acute respiratory acidosis, reduces arterial *c*tO2.

*p*CO2
Carbon dioxide tension

- Secondary:
  - Compensatory to metabolic acidosis
  - Secondary to central nervous system affection
  - Secondary to hypoxia
- B. High *p*CO2 Alveolar hypoventilation (hypercarbia):
  - Acute or chronic pulmonary disease
  - Upper airway obstruction (e.g., sleep apnea syndrome)
  - Diminished ventilatory drive due to central nervous system depression - either primary or secondary to sedation or analgesics - or compensatory to metabolic alkalosis
  - Insufficient, or intentionally low (“permissive hypercapnia”), mechanical ventilation

- **Alveolar-Arterial O2Gradient**

- Difference between the measured pressure of oxygen in the blood stream and the calculated oxygen in the alveolus. N < 15 mmHg

- Indicates whether hypoxia is a reflection of hypoventilation or due to deficiency in oxygenation

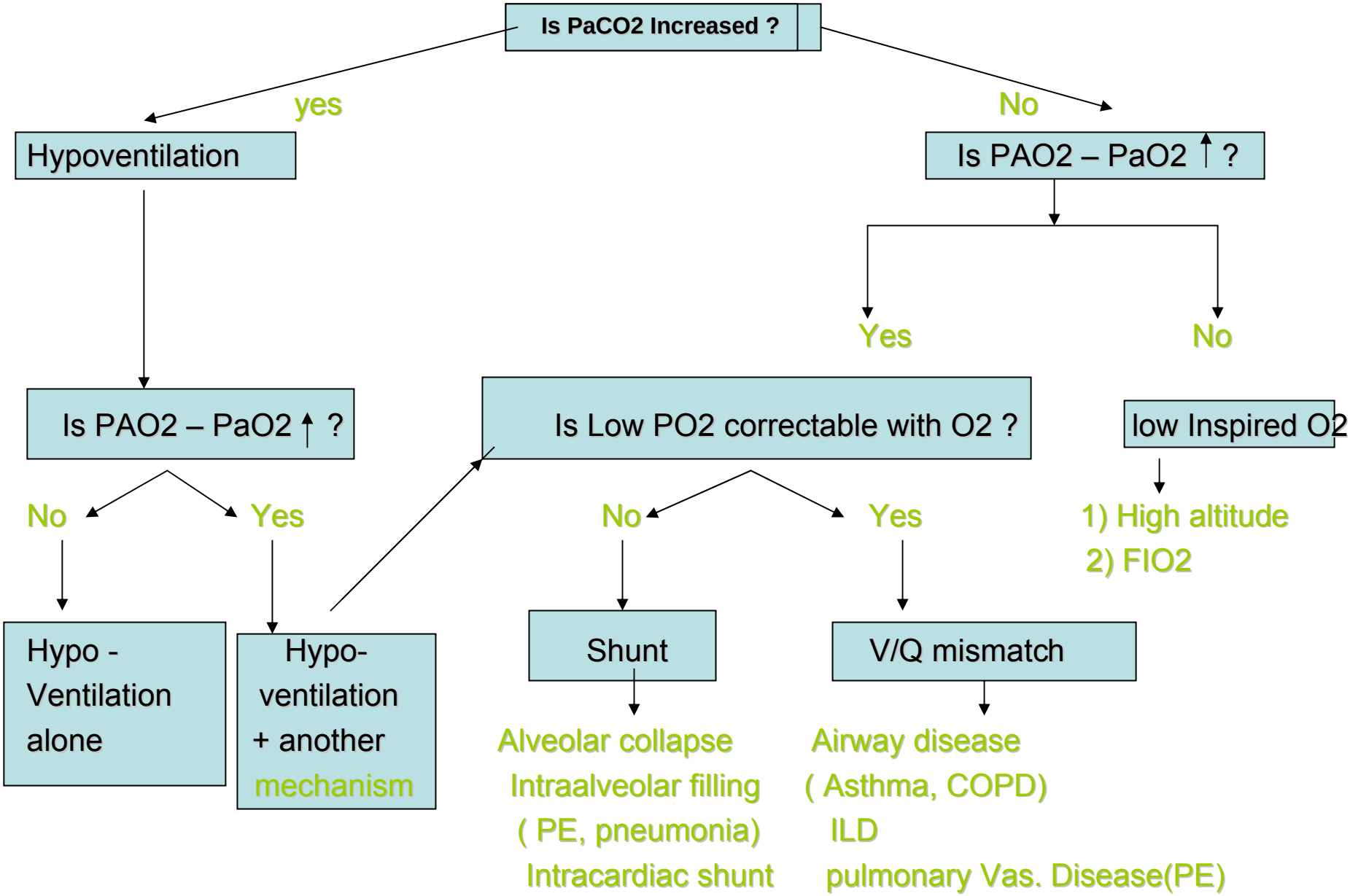
- P (A- a) O2 = (BP - pH2O) x FiO2 - (PaCO2 / R) - PaO2

  BP = 760 mmHg, pH2O = 47 mmHg, R = 0.8

- P (A- a) O2 = 150 - (1.25 x PaCO2) - PaO2 mm Hg

- A normal A-a gradient in the face of hypoxemia suggests the hypoxemia is due to hypoventilation and not due to underlying lung disorders.

- An increased A-a gradient identifies decreased oxygen in the arterial blood compared to the oxygen in the alveolus.


Is PaCO2 Increased ?
yes
No
Hypoventilation
Is PAO2 – PaO2 ↑ ?
Yes
No
Is PAO2 – PaO2 ↑ ?
Is Low PO2 correctable with O2 ?
low Inspired O2
No
Yes
No
Yes
1) High altitude
2) FIO2
Hypo - Ventilation alone
Hypo- ventilation + another mechanism
Shunt
V/Q mismatch
Alveolar collapse
Intraalveolar filling
(PE, pneumonia)
Intracardiac shunt
Airway disease
(Asthma, COPD)
ILD
pulmonary Vas. Disease(PE)

*s*O2
Arterial oxygen saturation

*s*O2 :ratio between the concentrations of O2Hb and HHb + O2Hb

*s*O2(a) is the percentage of oxygenated hemoglobin in relation to the amount of hemoglobin capable of carrying oxygen.

Reference ranges
Normal range: 95 – 99 %

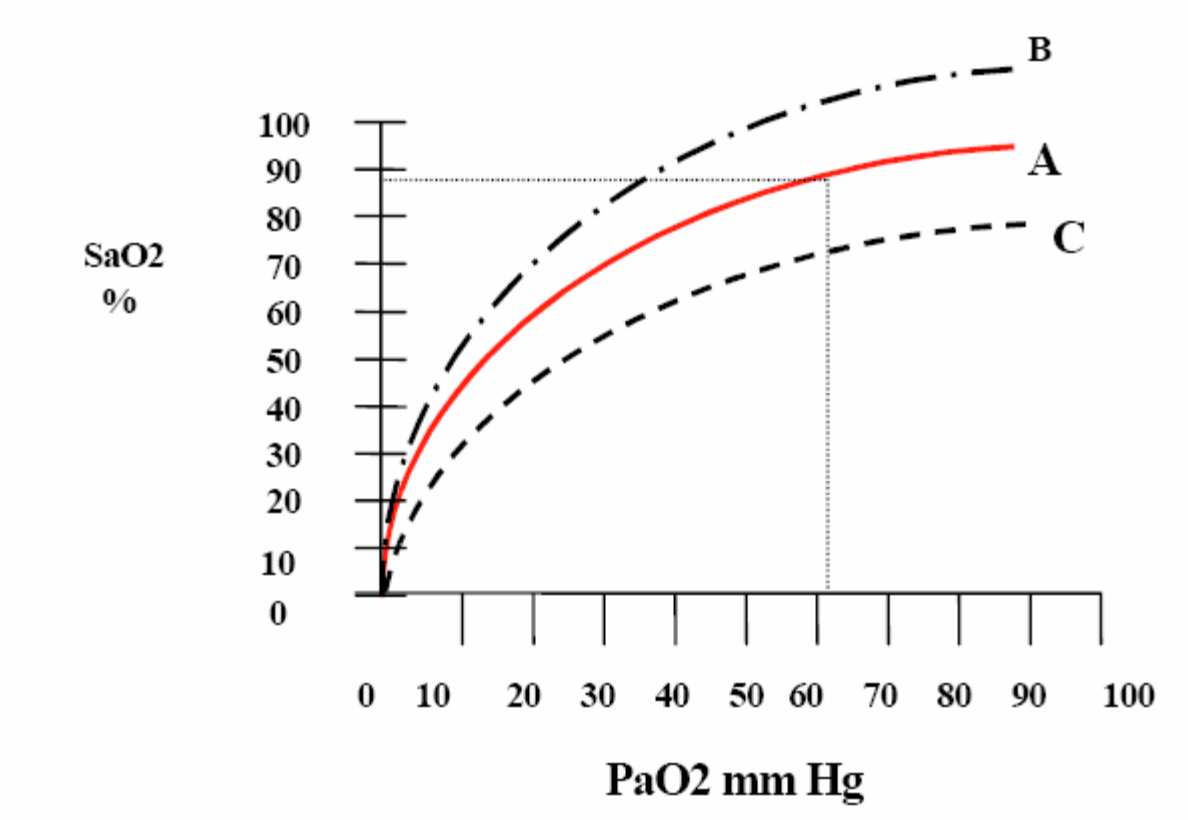
Clinical interpretation
Normal sO2:

Sufficient utilization of actual oxygen transport capacity.

Low *s*O2:

- Impaired oxygen uptake
- Right shift of ODC

Oxyhemoglobin Dissociation Curve



*c*tO2
Arterial concentration of total oxygen

- *c*tO2 is the concentration of the total oxygen in the blood.
- *c*tO2 = *s*O2 × 1.34 × *c*tHb + 0.0031 × *p*O2 ml / dl.
- Reference ranges : 8.8-22.3 mL / dL
- Normal *c*tO2 indicates an adequate oxygen content of the arterial blood.
- High *c*tO2:
  High *c*tO2, despite normal *p*O2, can only be caused by high ctHb (i.e., hemoconcentration, polycytemia, or excessive red-cell transfusion).
- Low *c*tO2:
  Low *c*tO2 may be caused by hypoxemia (low *p*O2) or if *p*O2 is normal, by a low ctHb and/or dyshemoglobinemia..

cBase(a)
Actual Base excess

"Base excess" is the absolute deviation (in mmol /L) of the buffer base amount from the normal level in blood.

The amount of acid (in mmol) required to restore 1 litre of blood to its normal pH, at a PCO2 of40mmHg.

The base excess reflects only the metabolic component of any disturbance of acid base balance. Reference ranges: ± 3 mmol / L

A low BE signifies metabolic acidosis, and a high BE signifies metabolic alkalosis.

BE is preferable to SBC in acid-base analysis, being a more exact indicator of "metabolic" buffer capacity (i.e., accounting for variations in buffer systems apart from the bicarbonate buffer).

*c*Base(Ecf)
Standard Base excess

- Standard base excess is an in vivo expression of base excess.

Base Excess is the in *vitro* value calculation for whole blood described by Siggaard-Andersen. To calculate Standard Base excess, also known as in vivo Base excess simply set the hemoglobin value to 5 g/100ml.

Standardized base excess (SBE) it is computed by blood gas analyzer by using Van Slyke equation

$$SBE = 0.9287 \times \blacktriangle pH \times \blacktriangle HCO_3^-$$

- What does *c*Base(Ecf) tell you

- *c*Base(Ecf) is the base excess in the total extracellular fluids, of which blood (the intravascular part) represents approx. one third. As buffering capacities differ in the extra cellular compartments (i.e., the intravascular vs. the extravascular compartment), *c*Base(Ecf) is an estimate more representative of in vivo base excess than is BE.

*c*HCO3-
Actual bicarbonate

The actual bicarbonate is the value calculated from the blood gas sample. It is calculated using the measured pH and *p*CO2 values.

What does *c*HCO3- tell you

An increased level of *c*HCO3- may be due to a primary metabolic alkalosis or a compensatory response to primary respiratory acidosis.
Decreased levels of *c*HCO3- are seen in metabolic acidosis and as a compensatory mechanism to primary respiratory alkalosis.

Reference ranges

22 – 26 mmol /L

*c*HCO3-(aP,st)
Standard bicarbonate

Standard bicarbonate (*c*HCO3-(P,st)) is the concentration of bicarbonate in plasma from blood which has been equilibrated with a gas mixture with *p*CO2 = 40 mmHg at 37 °C.

Thus, "standardizing" measurement conditions eliminates any respiratory influence on the bicarbonate concentration.

Hence, a low bicarbonate concentration signifies metabolic acidosis, a high bicarbonate concentration signifies metabolic alkalosis.

It gives a better estimate of the metabolic problem causing acid base imbalance.

Reference ranges

22 - 26 mmol/L

Normal values for arterial blood gases

Blood Gas Parameter	Parameter Reported & Symbol Used	Normal Value
Carbon dioxide tension	PCO_2	35 – 45 mm Hg (average, 40)
Oxygen tension	PO_2	80 – 100 mm Hg
Oxygen percent saturation	SO_2	97
Hydrogen ion concentration	pH	7.35 – 7.45
Bicarbonate	HCO_3^-	22 – 26 mmol/L

- STEP1 ⇨ LOOK FOR pH
  - <7.36 –ACIDOSIS
  - >7.44- ALKALOSIS
- STEP2 ⇨ LOOK FOR PCO_2

If PCO_2 CHANGES IN OPPOSITE DIRECTION OF pH ↓

PRIMARY RESPIRAT-ORY DISORDER ↓

If PCO_2 CHANGES IN SAME DIRECTION OF pH ↓

PRIMARY METABOLIC DISORDER ↓

STEP 3 ⇨ NOW LOOK FOR COMPENSATION WHETHER SIMPLE ACID-BASE DISORDER OR MIXED

Disturbance	Response	Expected change
Respiratory acidosis		
Acute	↑HCO3	1meq/10mm ↑PaCO2
Chronic	↑HCO3	4meq/10mm ↑PaCO2
Respiratory alkalosis		
Acute	↓ HCO3	2 meq /10mm ↓ PaCO2
Chronic	↓HCO3	4meq /10mm ↓ PaCO2

DISTURBANCES	RESPONCES	EXPECTED CHANGE
MET ACID	$PaCO_2$	[1.5 x HCO3] + (8 ± 2)
MET ALK	$PaCO_2$	[0.7 x HCO_3^-] + (21 ± 2)

- STEP 4 → IF COMPENSATION IS DIFFERENT THAN CALCULATED THEN DISORDER IS MIXED

- LOOK pH AND THEN DECIDE WHAT MAY BE THE MET CAUSE

LOOK PCO_2 AND THEN DECIDE WHAT MAY BE THE RESP DISORDER

CALCULATE ANION GAP IN METABOLIC ACIDOSIS
&
URINARY CHLORIDE IN METABOLIC ALKALOSIS

- STEP 5 → FINAL DIAGNOSIS

CASE 1

- A 58-year-old woman of ca cx came in the emergency department for acute dyspnea, sweating & disorientation.On neurological examination she was drowsy.Her ABG was done on room air and report was
- pH -7.20
- $PaCO_2$-65
- PaO_2-45
- HCO_3-28
- BE- 3

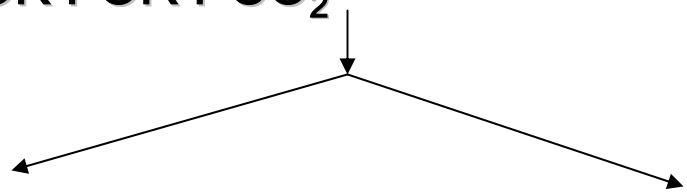
Na^+ -140

K^+ -4.3

Cl^- -103

INTERPRETATION

- STEP1 ⇨ LOOK FOR pH - 7.20 –ACIDOSIS
- STEP2 ⇨ LOOK FOR PCO_2



PCO_2 65 MEAN CHANGES IN

- IF PCO_2 CHANGES IN SAME DIRECTION OF pH

PRIMARY METABOLIC DISORDER

OPPOSITE DIRECTION OF pH

PRIMARY RESPIRATORY ACIDOSIS

STEP → NOW LOOK FOR COMPENSATION WHETHER SIMPLE RESP ACIDOSIS OR MIXED

Disturbance	Response	Expected change
Respiratory acidosis		
Acute	**↑HCO3= 2.5**	**1meq/10mm ↑PaCO2(25)**
Chronic	**↑HCO3**	**4meq/10mm ↑PaCO2**

$PaCO_2$=65 HCO_3=27

HCO_3 SHOULD BE 26.5

- STEP 4 → COMPENSATION IS SAME AS CALCULATED MEANS SIMPLE RESIRATORY ACIDOSIS

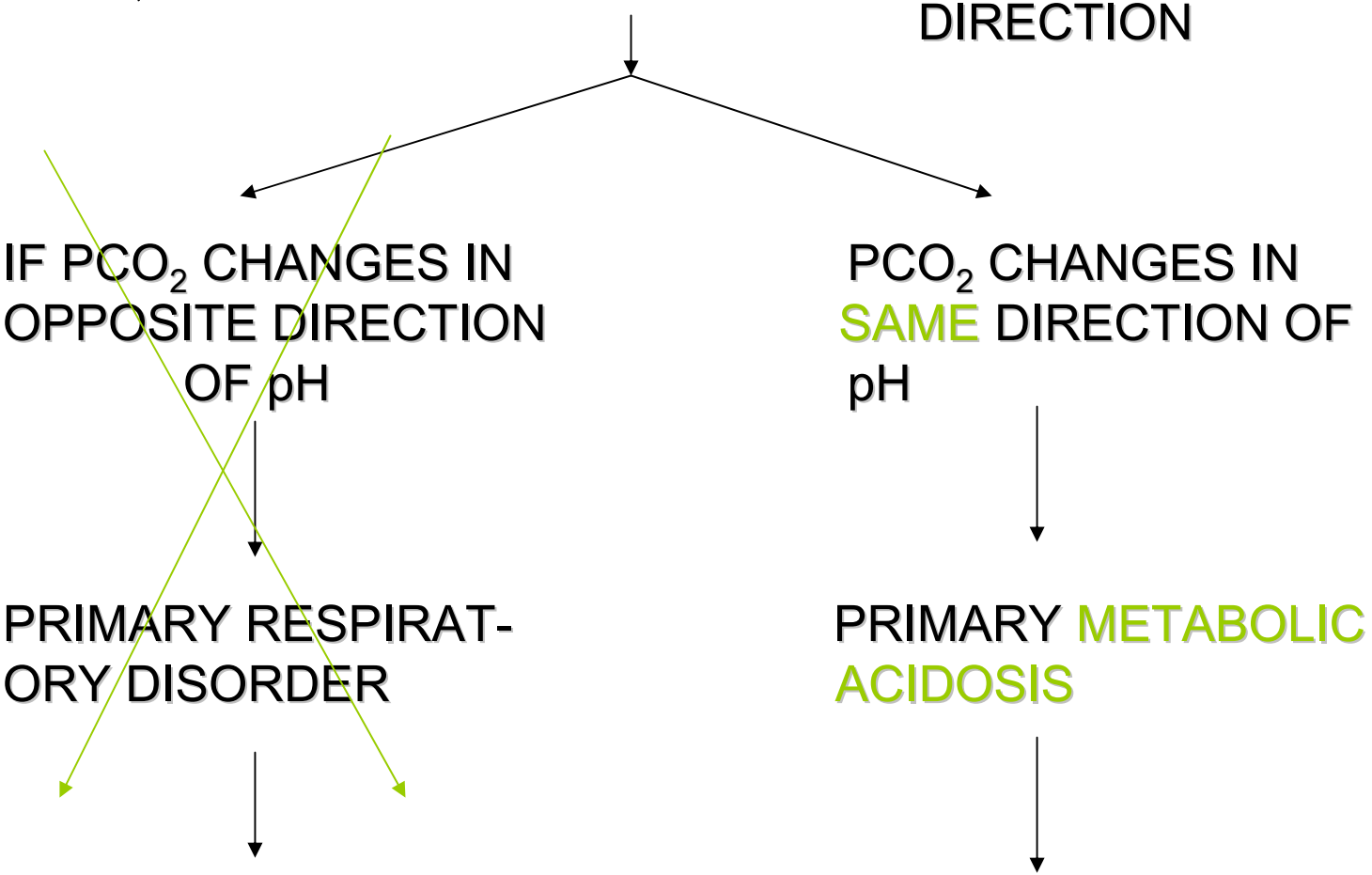
- STEP 5 → RESPIRATORY ACIDOSIS

CASE 2

- A 17-year old pt with Hodgkin's lymphoma with known IDDM entered the casualty with Kussmaul's breathing and an irregular pulse. His respiratory rate was 40/min & BP was 140/90.Room air ABG values and vital signs were;
- pH 7.05 Na^+-146mmol/l
- Po2 108 mm Hg K^+ -5.6mmol/l
- Pco2 12 mm Hg. Cl^- -100mmol/l
- BE - 30 mmol /L
- HCO3- 5 mmol /L

INTERPRETATION

- STEP1 ⇨ LOOK FOR pH - 7.05 –ACIDOSIS
- STEP2 ⇨ LOOK FOR PCO_2 - 12 – CHANGES IN SAME DIRECTION



STEP 3 ⇨ NOW LOOK FOR COMPENSATION
WHETHER SIMPLE METABOLIC ACIDOSIS OR MIXED

HCO_3=05
$PaCO_2$=12

DISTURBANCES	RESPOESE	EXPECTED CHANGE
MET ACID	↓ $PaCO_2$	1.5 x $[HCO_3^-]$ + 8 +/- 2 1.5 x 5 + 8 +/-2 = 15.5 +/- 2

- STEP 4 ⇨ COMPENSATION IS MORE THAN CALCULATED MEANS SOME RESP CAUSE TO DECREASE IN CO2

↓

RESP ALKALOSIS

- STEP 5 ⇨ **MET ACIDOSIS WITH RESP ALKALOSIS**

CASE 3

- 43-year-old man of ca lung comes in the emergency room for severe pneumonia. His respiratory rate is 38/min and he is using accessory breathing muscles,pulse is 130/min,BP is 80/56 and neurologically irritable.ABG done and report is
- pH -7.25
- $PaCO_2$ –55
- PaO_2 -45
- HCO_3 –15
- BE -(-8)

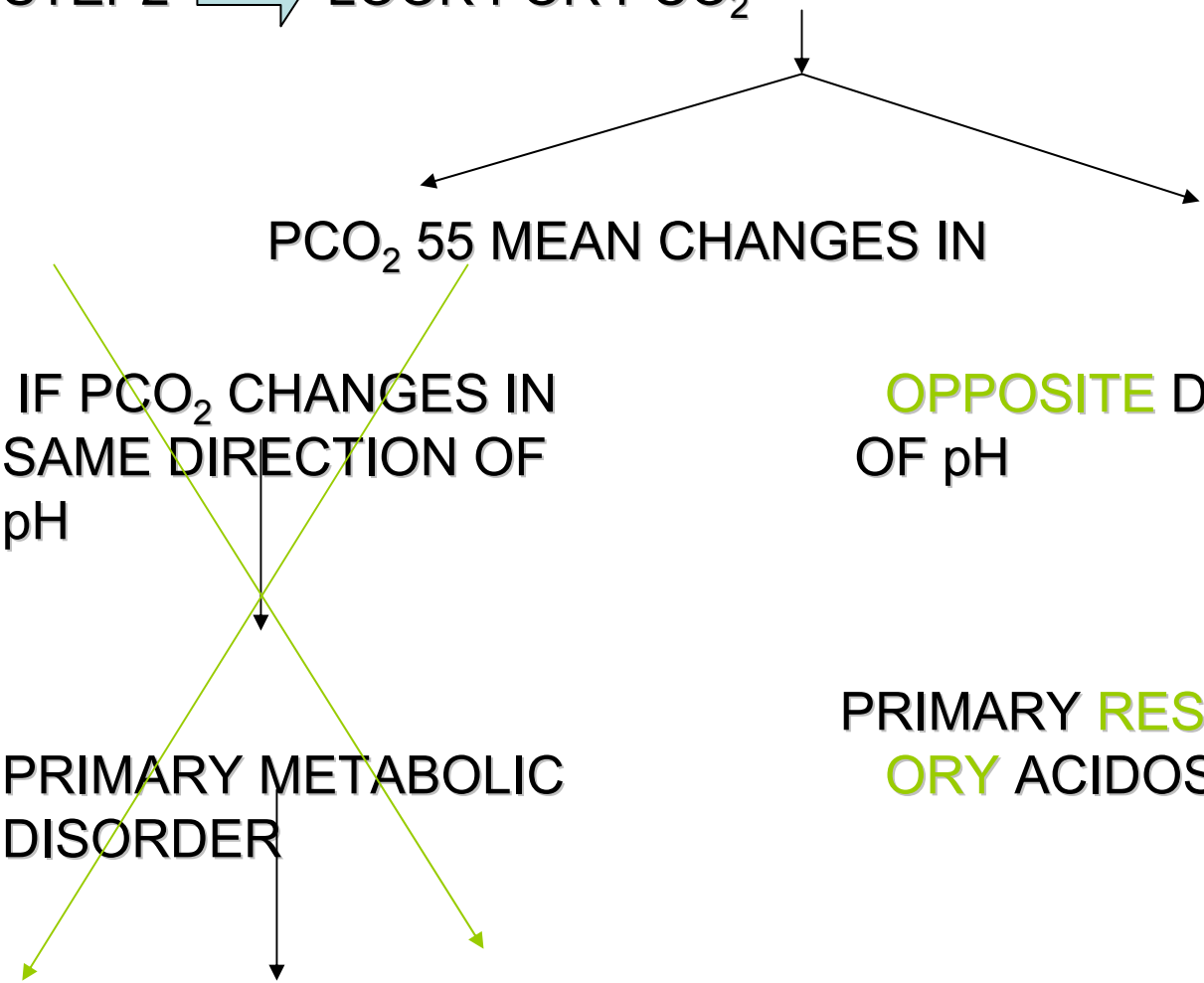
Na^+ -145

K^+ -4.8

Cl^- -98

INTERPRETATION

- STEP1 ⇨ LOOK FOR pH - 7.25 –ACIDOSIS
- STEP2 ⇨ LOOK FOR PCO_2



STEP 3 ⇨ NOW LOOK FOR COMPENSATION WHETHER SIMPLE ACID-BASE DISORDER OR MIXED

Disturbance	Response	Expected change
Respiratory acidosis		
Acute	**↑HCO3=1.5**	**1meq/10mm ↑PaCO2**
Chronic	**↑HCO3**	**4meq/10mm ↑PaCO2**

$PaCO_2$=55 HCO_3=15

HCO_3 SHOULD BE 25.5

- STEP 4 ⇨ **COMPENSATION IS OPPO THE CALCULATED MEANS SOME MET CAUSE TO DECREASE HCO3 MEANS**

MET ACIDOSIS

- STEP 5 ⇨ RESP ACIDOSIS WITH MET ACIDOSIS


Thank You