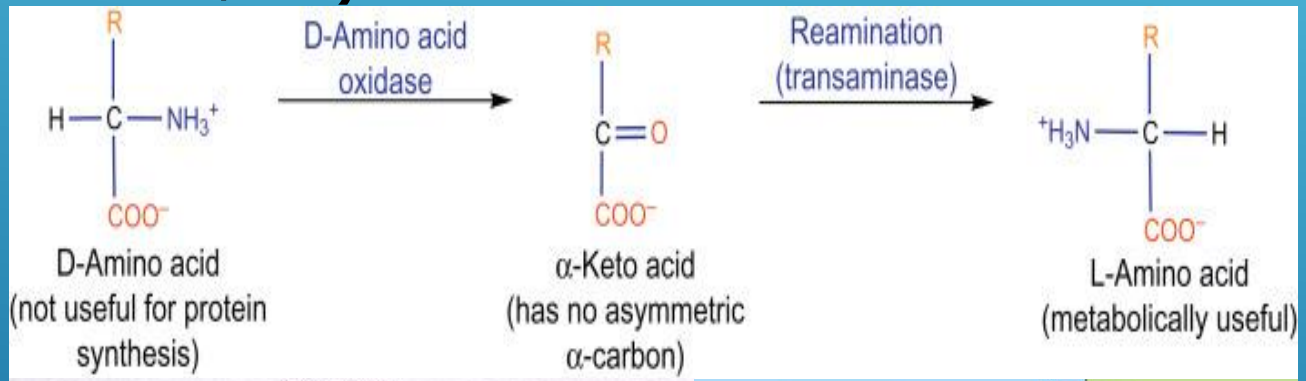
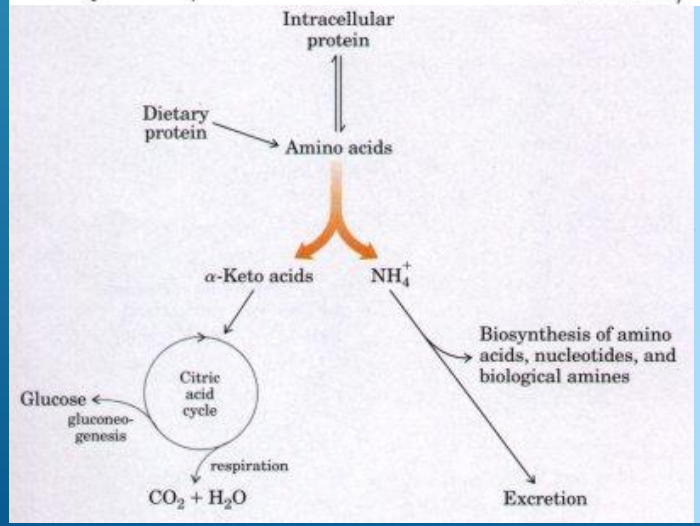
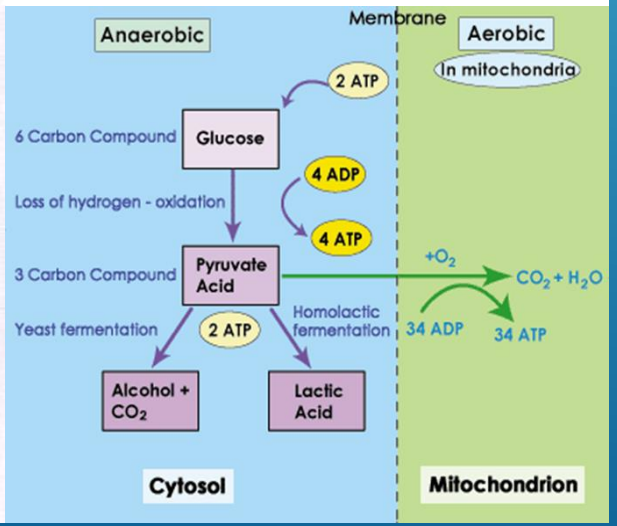
ACID-BASE BALANCE

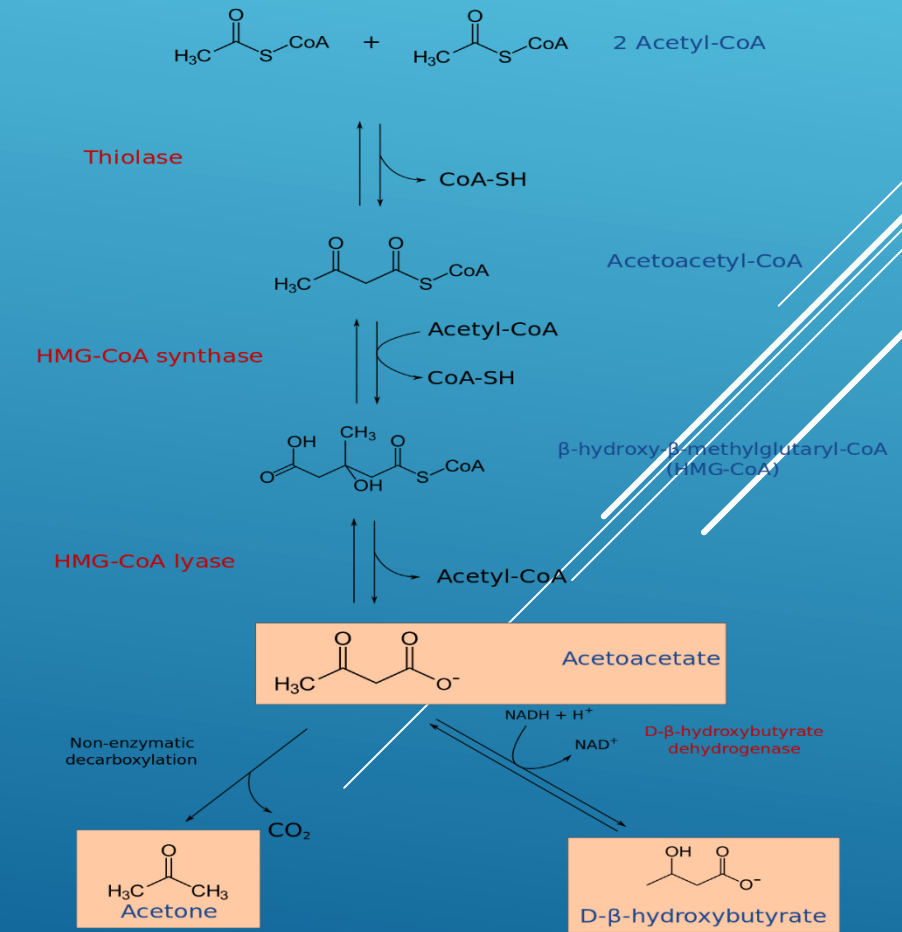
PRODUCTION OF HYDROGEN IONS

- The processes of metabolism generate hydrogen ions:
- **1) Oxidation amino acids, glucose, and fatty acids produce small amounts of acids (40-80 mmol/24h).**

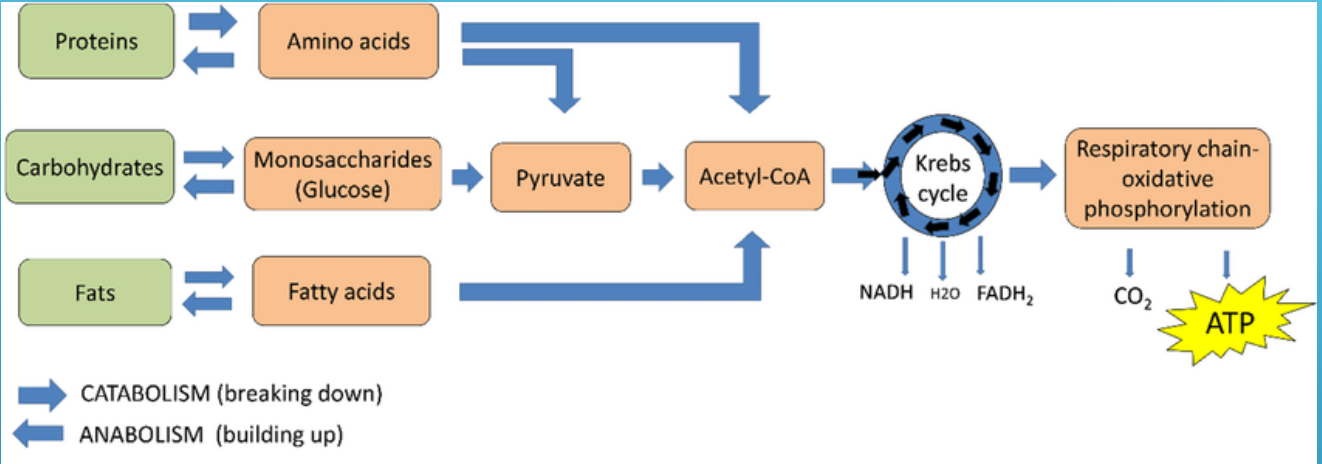








- **2) CO2 that is released from oxidative metabolism of carbohydrates, lipids, and proteins produce large amounts of acids (15,000 mmol/24h).**



- **Although CO2 does not contain H+ ions it rapidly reacts with H_2O to form carbonic acid (H_2CO_3), which further dissociates into H+ and HCO_3- ions.**

$$CO_2 + H_2O \Longrightarrow H_2CO_3 \Longrightarrow HCO_3^- + H^+$$

- The H+ ion concentration [H+] in the blood and ECF (body fluids) is approximately between 35 - 45 nmol/L **(pH 7.35 – 7.45)**.
- **-** A reduction by > 0.03 units leads to **acidosis**, and an increase leads to **alkalosis**.
- **- Acid base balance or homeostasis:** is the equilibrium between the acid production and the rate of its removal

- The body tries to keep this value constant within tight limits (as large shifts in pH are incompatible with life) **by three major mechanisms**:
- **a) Buffer systems:** Buffer systems react **quickly** to bind H+ or OH- ions to prevent drastic changes in the pH.
- **b) Exhalation of CO2:** Because of the relationship between CO2 and H+, alterations in respiratory rate affect changes in pH by changing the CO2 concentration of the body.
- **c) Kidney secretion of H+ and regeneration of HCO3-:** H+ secretion from distal tubules of the nephrons directly into filtrate acidifies urine and removes the H+ form the body. The kidneys not only secrete hydrogen ions but also they regenerate bicarbonate ions.

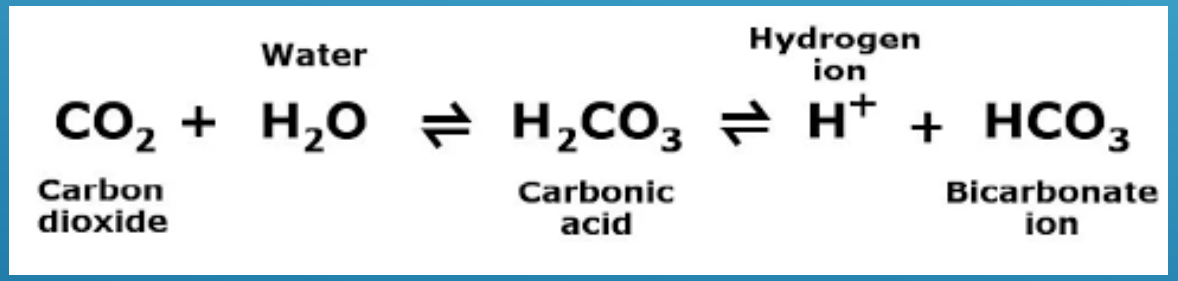
BUFFER SYSTEMS

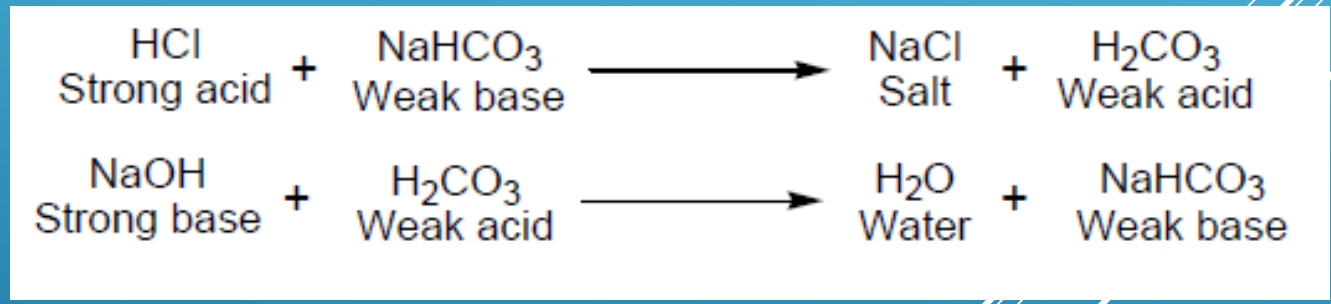
- **- Buffers: are solutions which resist the change in pH when an acid or alkali is added to it.**
- **- They do not remove H+ from the body, rather they temporarily mop up any excess H+ that are produced.**

- **- Buffers are usually a mixture of:**
- **- A weak acid and a strong base e.g: carbonic acid/sodium hydroxide mixture (H_2CO_3/NaOH).**
- **- A weak base and a strong acid e.g: sodium bicarbonate /hydrogenchlorid ($NaHCO_3$ /HCl).**

1. BICARBONATE BUFFER SYSTEM

- The bicarbonate buffering system is the most important buffer in the **ECF**. Bicarbonate (HCO3−) combines with H+ to form carbonic acid (H2CO3). This buffer system is unique in that the (H_2CO_3) can dissociate to water and carbon dioxide. Note that the effect of CO_2 in the aqueous solution is acidic.

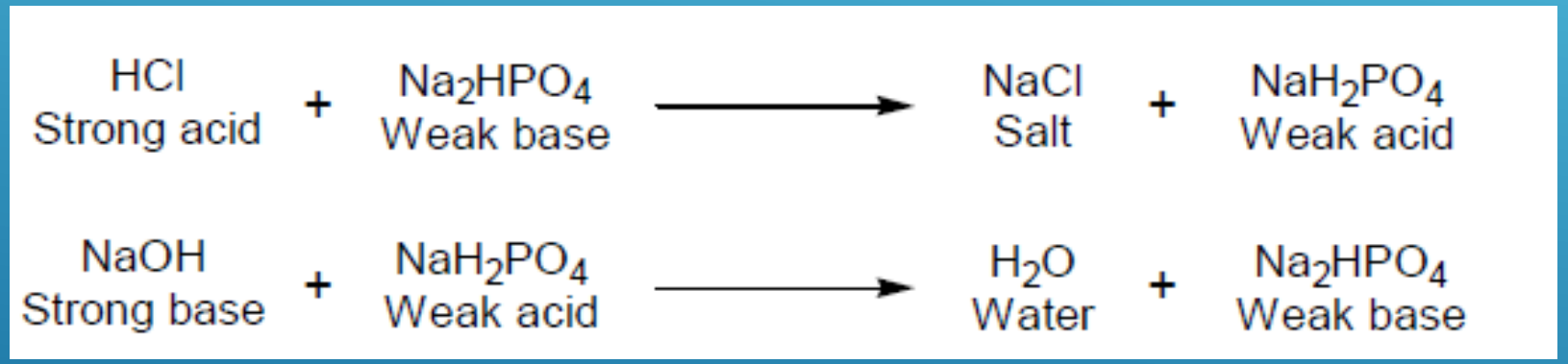




this buffer remove excess H^+ from the ECF at the expense of bicarbonate

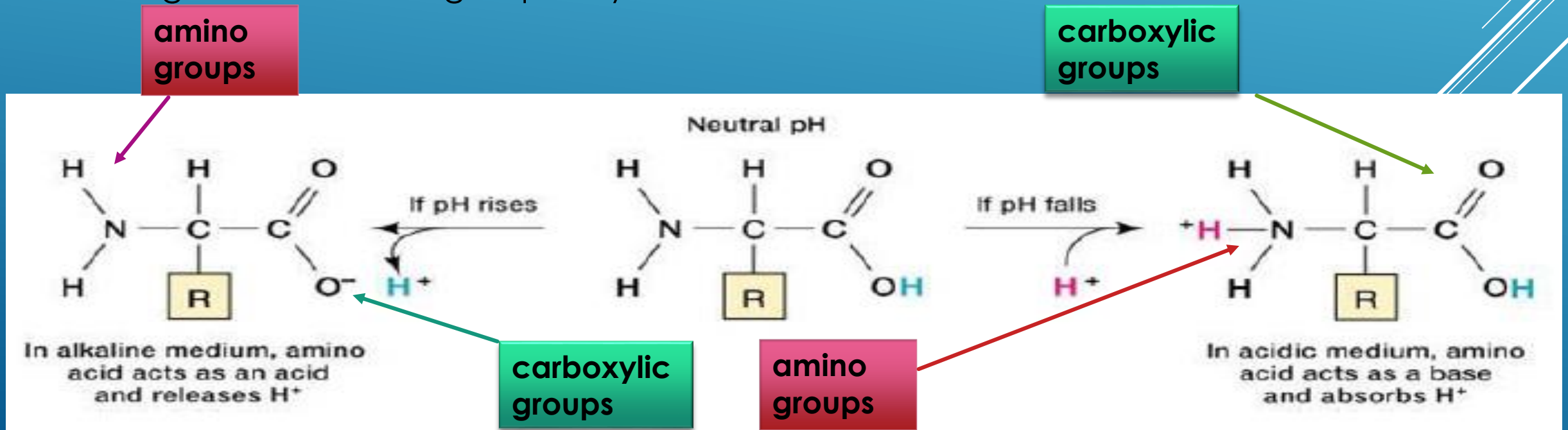
2. PHOSPHATE BUFFER SYSTEM

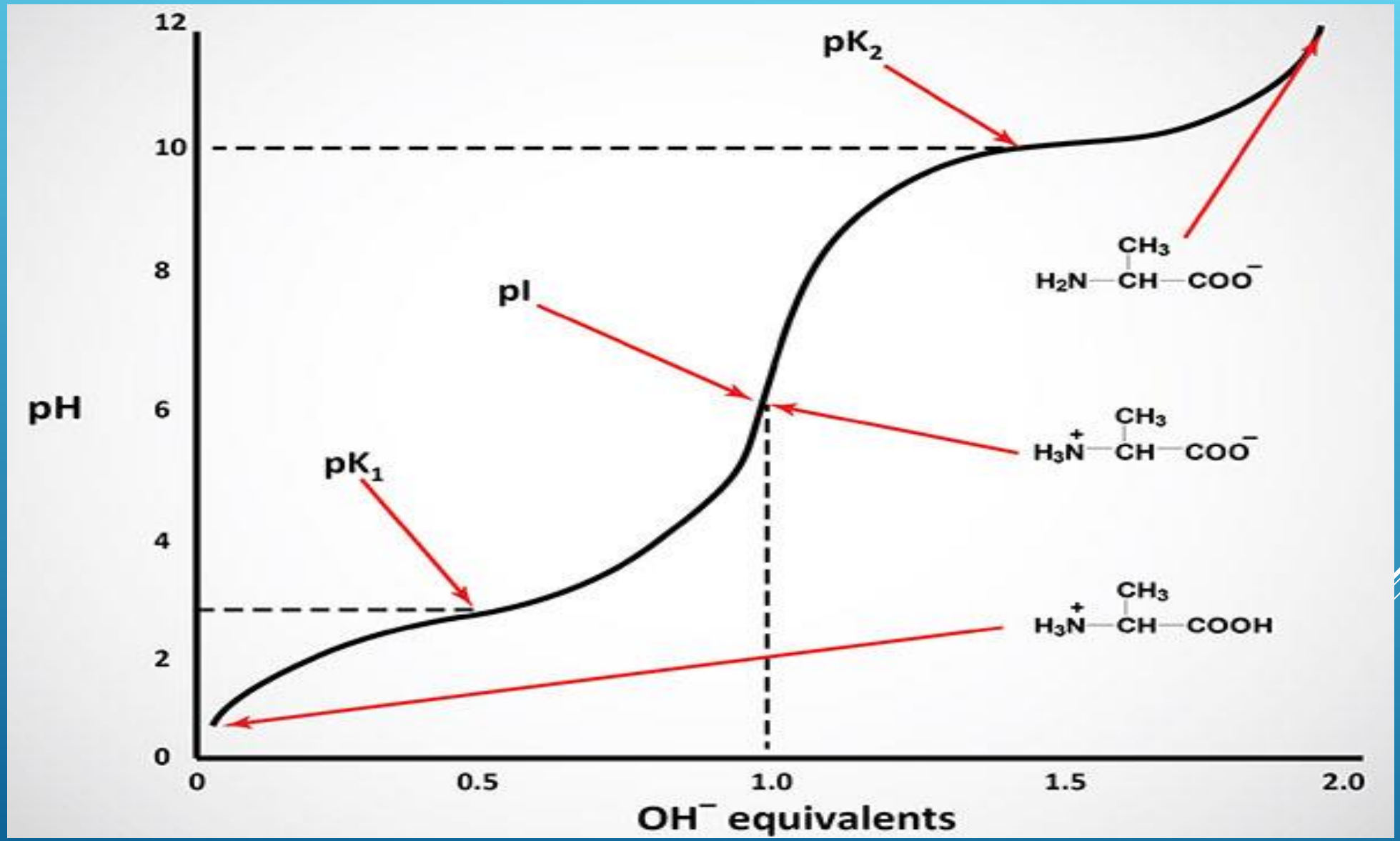
- Phosphate acts as **ICF, ECF and urine buffer**. Remember that phosphoric acid is a weak acid



3. PROTEIN BUFFER SYSTEM

- As composed of amino acids which have acidic and basic properties, proteins can act a buffering system either **intra- or extra-cellularly**. The protein buffer system is the most abundant buffer in cells and plasma **(ECF and ICF**)
- (remember that the amino groups of amino acids are weak bases whereas carboxylic groups are weak acids, thus amino acids have a buffering capacity, see figure below). In the blood, hemoglobin is the major buffering protein, whereas in plasma, plasma proteins such as albumin have a significant buffering capacity.

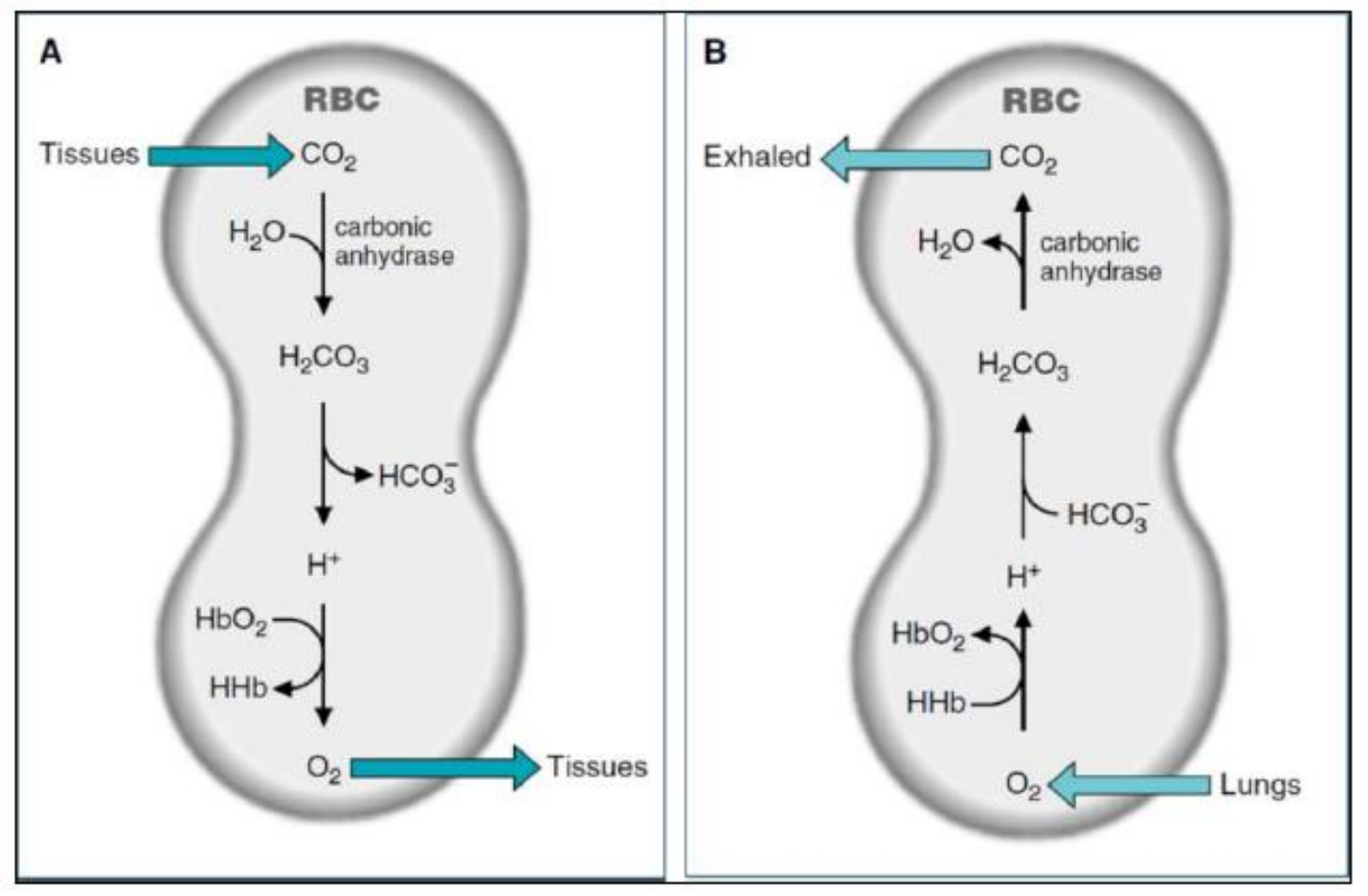



12
10
8
6
4
2
0
pH
0
0.5
1.0
1.5
2.0
OH⁻ equivalents
pK2
pI
pK1
CH3
H2N—CH—COO⁻
CH3
H3N+—CH—COO⁻
CH3
H3N+—CH—COOH

D) HAEMOGLOBIN AS BUFFER (BOHR EFFECT)

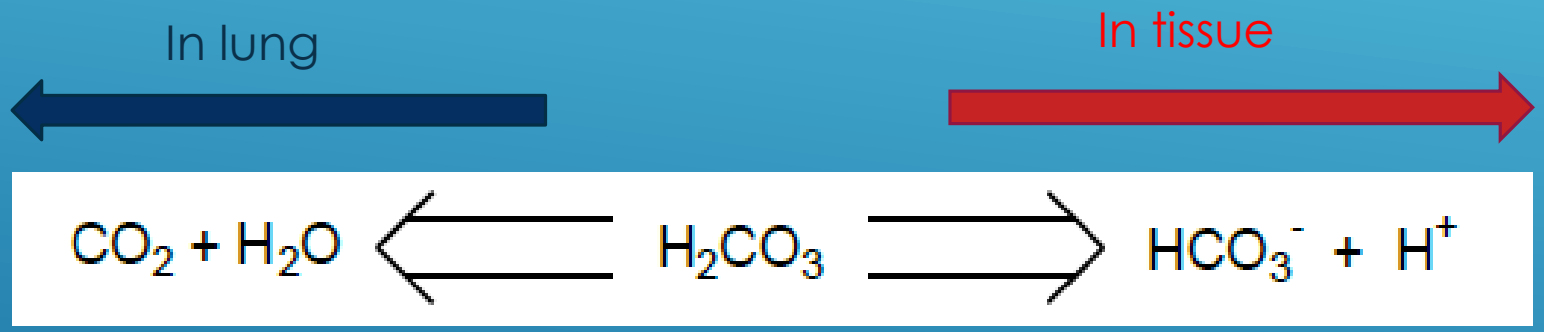
- Haemoglobin is not only important in the carriage of oxygen to the tissues but also in the transport of CO_2 and in buffering H^+ ions.
- **1-** Haemoglobin binds both CO_2 and H^+ , which consider as a powerful buffer.
- **2-** In the tissues, dissolved CO_2 passes into the RBC down its concentration gradient where it combines with water to form carbonic acid. This reaction is catalyzed by the enzyme carbonic anhydrase.
- **3- Carbonic acid then dissociates into bicarbonate and** H^+ ions.
- **4- The** H^+ bind to reduced haemoglobin to form HHb.
- **5-** HCO_3^- generated by this process pass back into the plasma in exchange for Cl^-. This ensures that there is no change of negative ions by RBC.
- **6-** In the lungs this process is reversed and H^+ bound to haemoglobin recombine with HCO_3^- to form CO_2 which passes into the alveoli.

BOHR EFFECT


A
RBC
Tissues
CO_2
H_2O
carbonic anhydrase
H_2CO_3
HCO_3^-
H^+
HbO_2
HHb
O_2
Tissues
B
RBC
Exhaled
CO_2
H_2O
carbonic anhydrase
H_2CO_3
HCO_3^-
H^+
HbO_2
HHb
O_2
Lungs

EXHALATION OF CO2

- - Breathing plays a most important role in the control of acid-base balance. Remember:



- **In the tissues where carbon dioxide is abundant, the reaction is shifted to the right**
- **In the lungs where H+ are liberated from hemoglobin, the reaction is shifted to the left**

ROLE OF KIDNEY

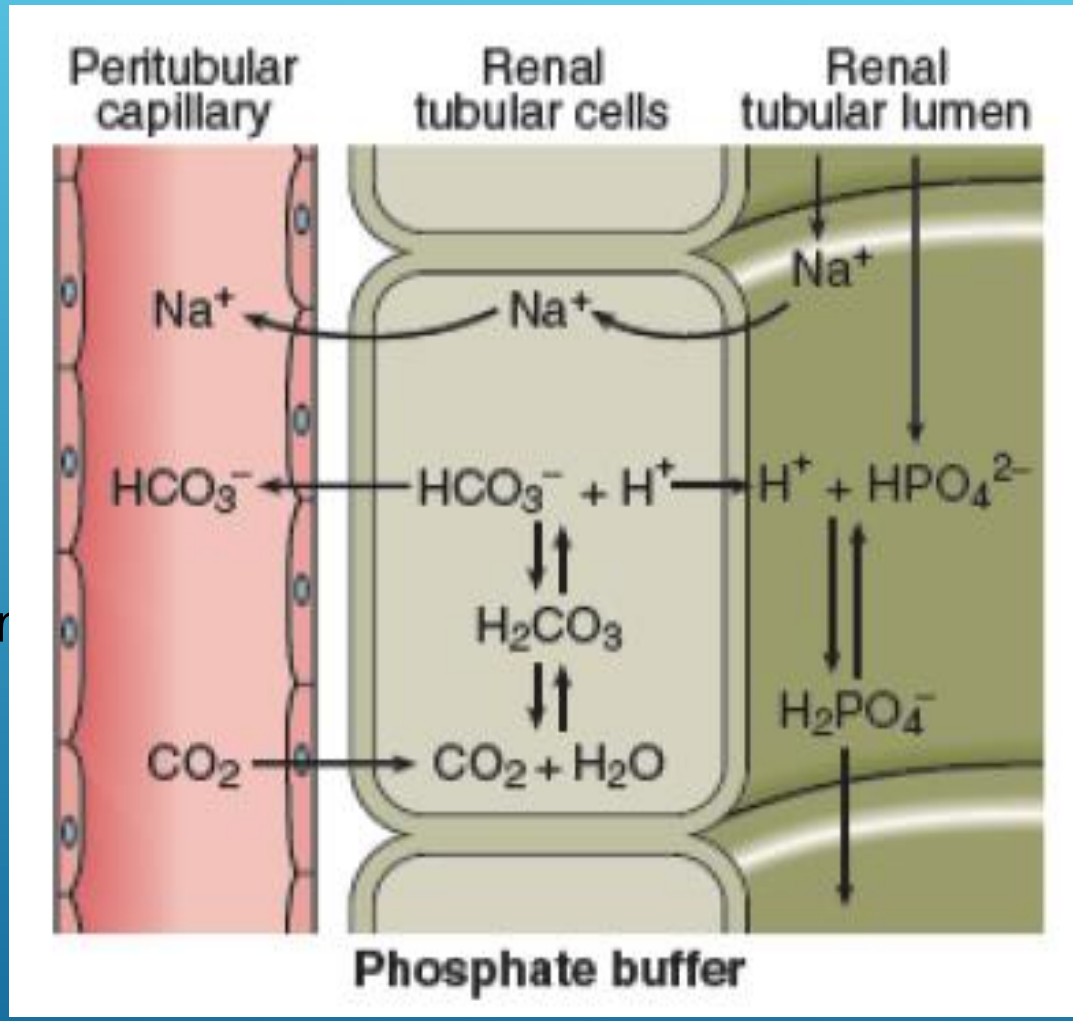
A) EXCRETION OF H+

- **-** The tubules of the kidneys secrete H+ directly into the filtrate so that urine is acidified and the H+ are lost from the body.
- **-** H+ ions are actively secreted in the proximal and distal tubules, but the maximum urinary [H+] is around 0.025 mmol/l (pH 4.6).

- **- Therefore, in order to excrete the 30-40** mmol of H+ required per day, a urine volume of 1200 L would have to be produced.
- **-** However, **buffering of H+ also occurs in the urine**. This allows the excretion of these large quantities of H+ without requiring such huge urine volumes.
- **-** The predominant buffers in the urine are **phosphate** and **ammonia**.
- **- H+ ion secretion is an active process and requires energy in the form of ATP.**

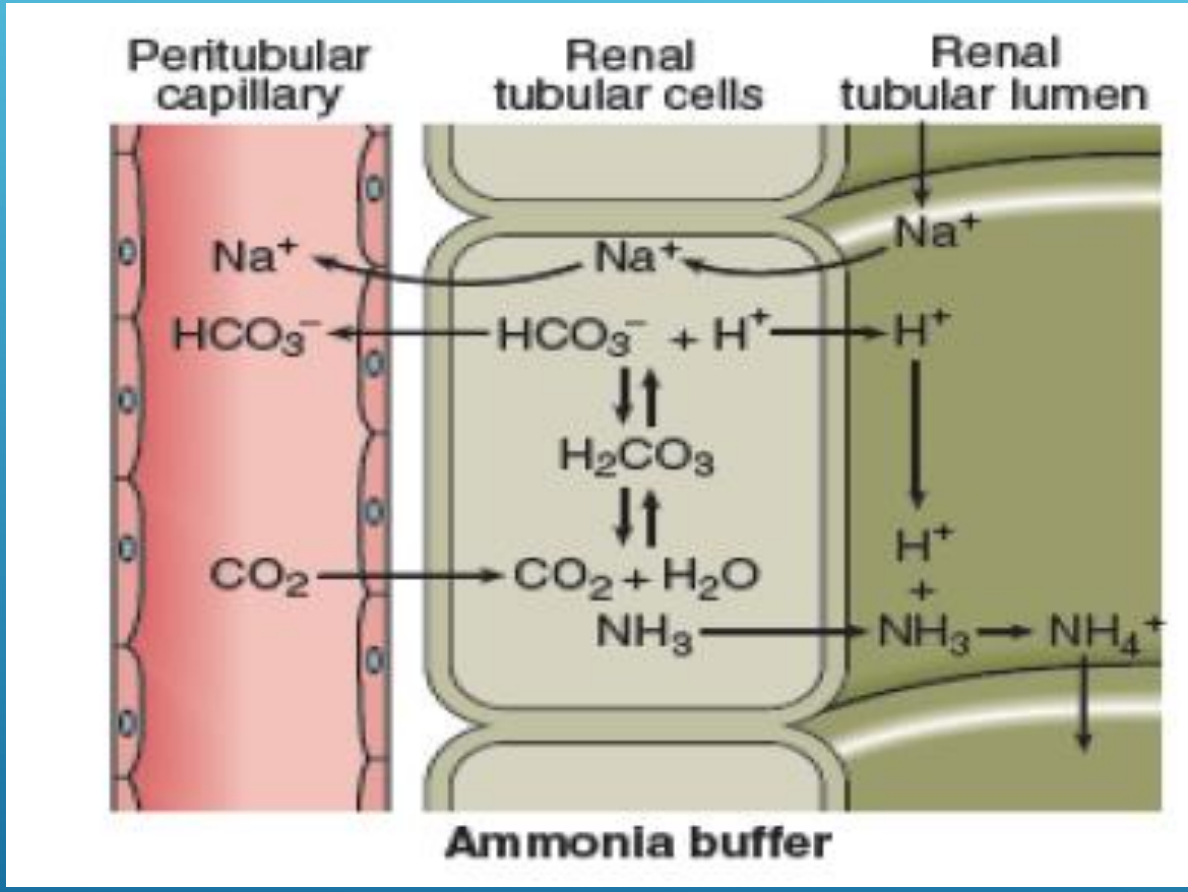
PHOSPHATE BUFFERING FOR URINE:

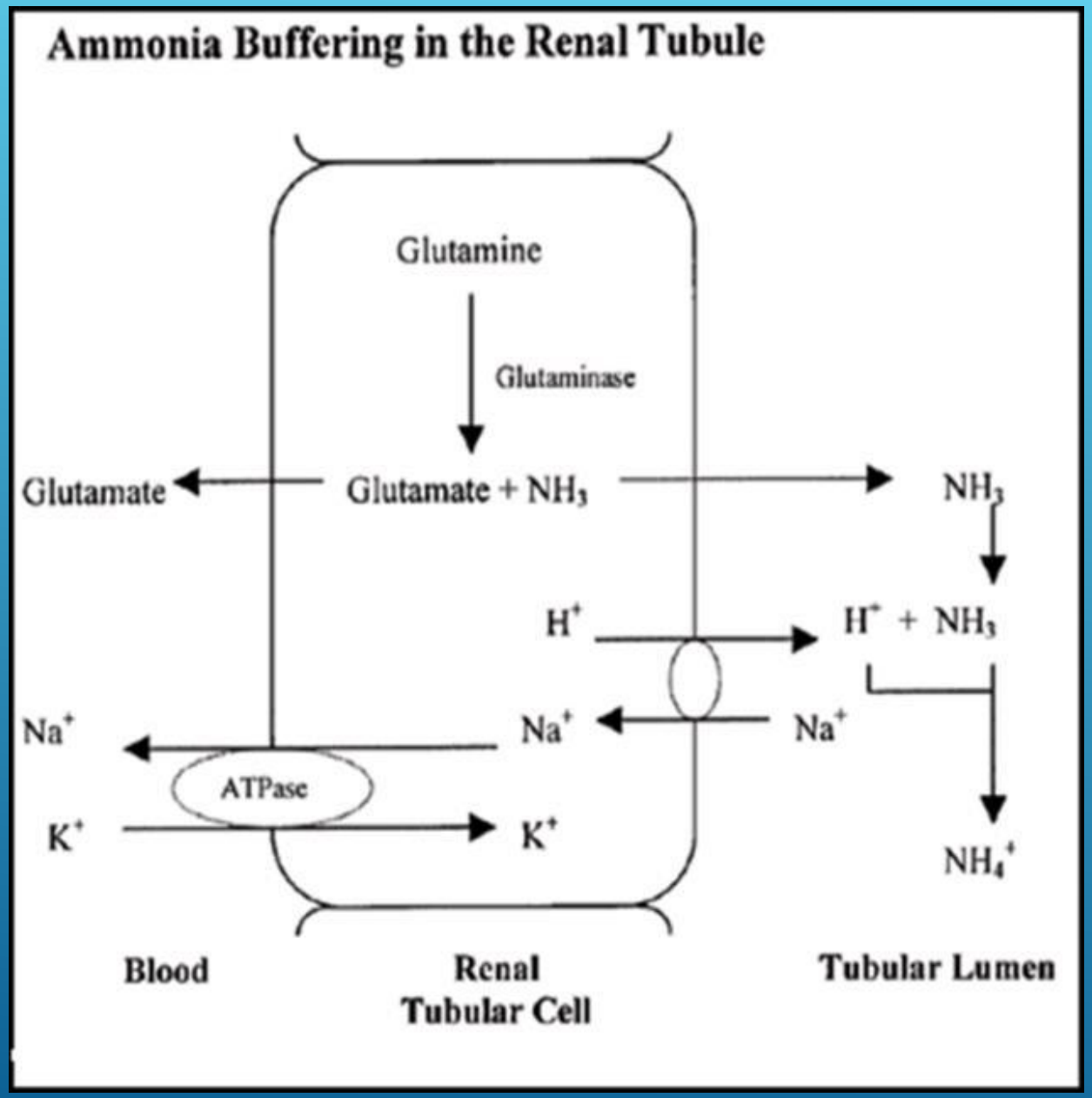
- - Phosphate is freely filtered by the glomerulus and passes down the tubule where it combines with H+ to form H_2PO_4-.
- - Hydrogen ions are secreted in exchange for sodium ions; the energy for this exchange comes from the Na-K-ATPase that maintains the concentration gradient for sodium



AMMONIA BUFFERING FOR URINE:

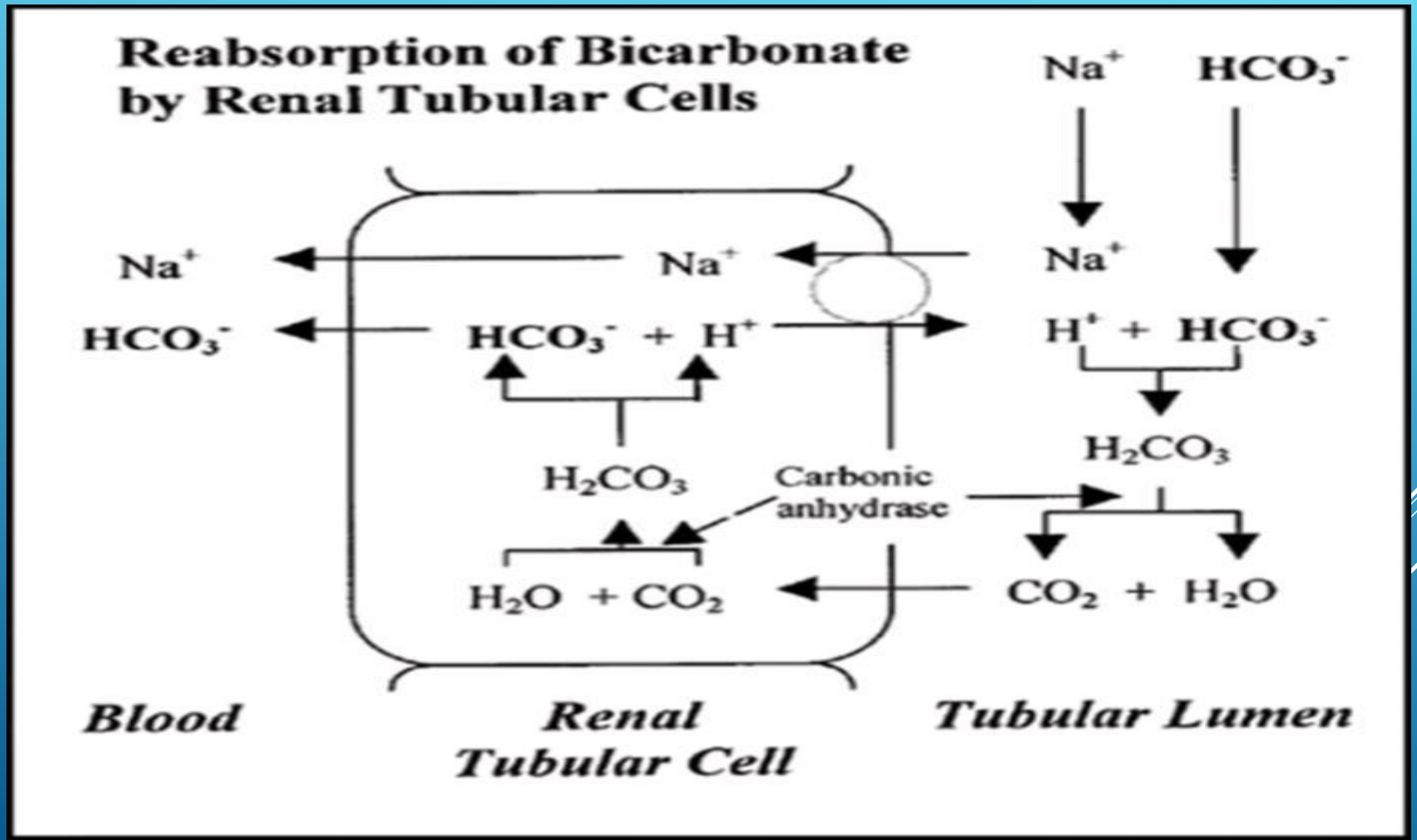
- - Ammonia is produced in renal tubular cells by the action of the enzyme glutaminase on the amino acid glutamine
- - Ammonia is unionized and so rapidly crosses into the renal tubule down its concentration gradient.
- - The ammonia combines with H+ to form the ammonium ion, which being ionized does not pass back into the tubular cell.




Ammonia Buffering in the Renal Tubule
Glutamine
Glutaminase
Glutamate
Glutamate + NH3
NH3
H+
H+ + NH3
Na+
Na+
Na+
ATPase
K+
K+
NH4+
Blood
Renal Tubular Cell
Tubular Lumen

B) REGENERATION OF BICARBONATE

- Bicarbonate ions are freely filtered by the glomerulus.
- **- The concentration of bicarbonate in the tubular fluid is equivalent to that of plasma.**
- **- If bicarbonate were not reabsorbed the buffering capacity of the blood would rapidly be depleted.**
- - Filtered bicarbonate combines with secreted H+ ions forming carbonic acid.
- - Carbonic acid then dissociates to form CO2 and water by carbonic anhydrase.
- - This CO2 readily crosses into the tubular cell down a concentration gradient.
- - Inside the cell the CO2 recombines with water, again under the influence of carbonic anhydrase, to form carbonic acid.
- - The carbonic acid further dissociates to bicarbonate and H+ ions.
- - The bicarbonate passes back into the blood stream whilst the H+ passes back into the tubular fluid

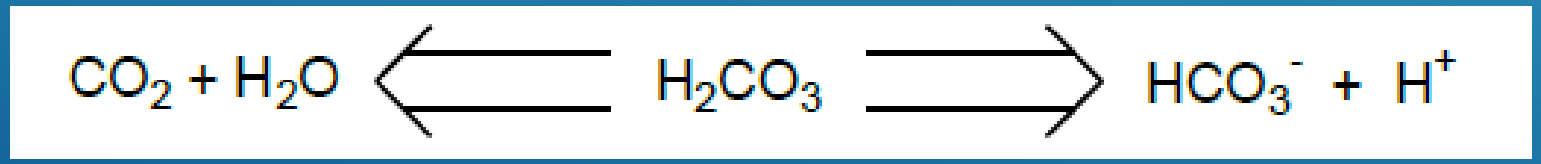

Reabsorption of Bicarbonate by Renal Tubular Cells
Na+
HCO3-
Na+
HCO3- + H+
H2CO3
Carbonic anhydrase
H2O + CO2
Na+
H+ + HCO3-
H2CO3
CO2 + H2O
Blood
Renal Tubular Cell
Tubular Lumen

C) ELECTROLYTES BALANCE

- **-** Sodium/Potassium: sodium reabsorption and hydrogen ion excretion are interlinked. Sodium reabsorption is controlled by the action of **aldosterone** on ion exchange proteins in the distal tubule. These ion exchange proteins exchange sodium for hydrogen or potassium ions. Thus, **changes in aldosterone secretion may result in altered acid secretion.**

- Chloride: The number of positive and negative ions in the plasma must balance at all times. Aside from the plasma proteins, bicarbonate and chloride are the two most abundant negative ions (anions) in the plasma. **In order to maintain electrical neutrality any change in chloride must be accompanied by the opposite change in bicarbonate concentration**. Therefore, the chloride concentration may influence acid base balance.

DISORDERS OF HYDROGEN ION HOMEOSTASIS

- Disturbance of the body's acid-base balance results in the plasma containing either too many H+ (**acidaemia or acidosis**) or too few H+ (**alkalaemia or alkalosis**).
- - These disturbances may be due to respiratory causes or non-respiratory (metabolic) causes.
- **a) Metabolic acid-base disorders:** affect directly the HCO3 concentration
- - Metabolic acidosis: decreased HCO3 concentration
- - Metabolic alkalosis: increased HCO3 concentration
- **b) Respiratory acid-base disorders: affect directly the PCO2**
- - Respiratory acidosis: increased PCO2
- - Respiratory alkalosis: decreased PCO2
- 

alkalosis acidosis

$$[H^+] \text{ is proportional to } \frac{PCO_2}{[HCO_3^-]}$$

- **Thus,**
- **Increase in [H+]** could be due to 1. adding H+, 2. removing bicarbonate or 3.increasing PCO2.
- **Decrease in [H+]** could be due to 1. removing of H+, 2. adding bicarbonate or 3. lowering PCO2.

- Since [H+] and [HCO3-] are regulated by the renal system, whereas PCO2 is regulated by the respiratory system, blood pH is controlled by our normal pattern of respiration and normal functioning of kidneys.

Compensation

- **a) Renal Compensation (slow to take effect/ 2 to 3 days)**

- For the primary respiratory disorders, renal tubules attempt to compensate by increase or decrease the H+ secretion

- Respiratory acidosis → compensated by increased tubular H+ secretion

- Respiratory alkalosis → compensated by decreased tubular H+ secretion

- **b) Respiratory Compensation (quick to take effect)**

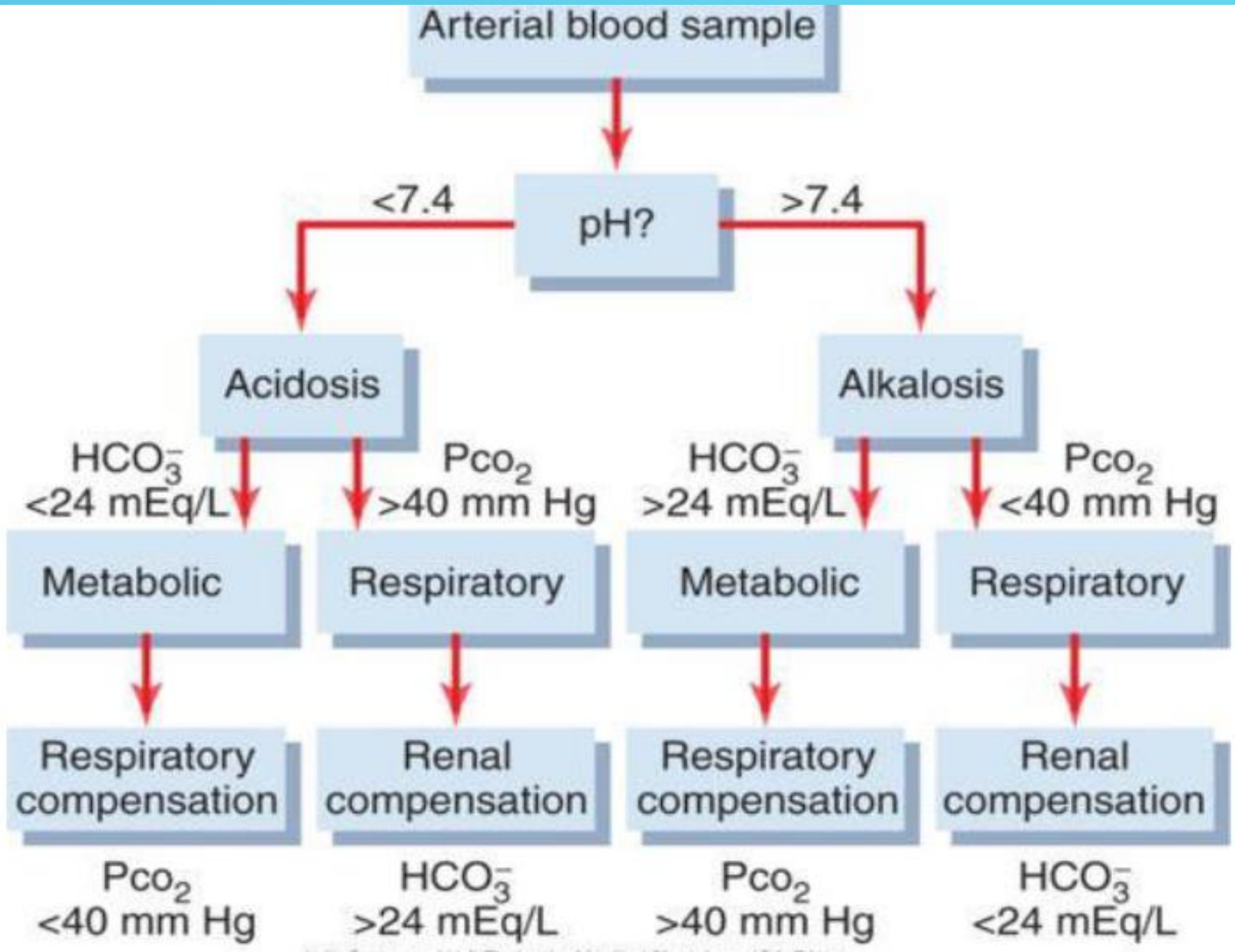
- For the primary metabolic disorders, lungs attempt to compensate by increase or decrease CO2 exhalation

- Metabolic acidosis → compensated by hyperventilation (*Kussmaul* breathing) → higher rate of excreting CO2 → ↓ PCO2 → ↓ acidosis.

- Metabolic alkalosis → compensated by hypoventilation → lower rate of excreting CO2 → ↑ PCO2 → ↓ alkalosis.

Compensation

Primary disorder	Compensatory response
↑ PCO_2 (Respiratory acidosis)	↑ H^+ secretion and ↑ HCO_3 reabsorption
↓ PCO_2 (Respiratory alkalosis)	↓ H^+ secretion and ↓ HCO_3 reabsorption
↓ HCO_3 (Metabolic acidosis)	Hyperventilation and ↓ PCO_2
↑ HCO_3 (Metabolic alkalosis)	Hypoventilation and ↑ PCO_2


Arterial blood sample
pH?
<7.4
>7.4
Acidosis
Alkalosis
HCO_3^- <24 mEq/L
Pco_2 >40 mm Hg
HCO_3^- >24 mEq/L
Pco_2 <40 mm Hg
Metabolic
Respiratory
Metabolic
Respiratory
Respiratory compensation
Renal compensation
Respiratory compensation
Renal compensation
Pco_2 <40 mm Hg
HCO_3^- >24 mEq/L
Pco_2 >40 mm Hg
HCO_3^- <24 mEq/L

Metabolic acidosis:

- 1) excess H+ production: due to increase organic acids (usually lactic or pyruvic) as a result of anaerobic metabolism. This may result from local or global tissue hypoxia. Increased ketone bodies in diabetic ketoacidosis
- 2) ingestion of acids : the most common drugs and chemicals that induce metabolic acidosis are biguanides (e.g. metformin), alcohols, salicylates, methanol, ethylene glycol, cyanide and carbon monoxide
- 3) inadequate excretion of H+: this results from renal tubular dysfunction and usually occurs in conjunction with inadequate reabsorption of bicarbonate. Any form of renal failure may result in metabolic acidosis.
- 4) excessive loss of bicarbonate: gastro-intestinal secretions are high in sodium bicarbonate. The loss of small bowel contents or excessive diarrhea results in the loss of large amounts of bicarbonate resulting in metabolic acidosis. This may be seen in such conditions as *cholera*

- **Lactic acidosis**: is a medical condition characterized by the buildup of in the blood, leads to lowering blood pH (acidosis). Over-production of lactic acid is due to shifting of cellular metabolism from aerobic metabolism to anaerobic metabolism which finally result in the formation of lactate. Under normal conditions, body cells generally metabolize glucose aerobically. Under exceptional conditions where tissues are deprived from oxygen or have defective oxidative metabolism, they shift to anaerobic metabolism of glucose whose end-product is lactate.
- the end-product of aerobic glycolysis is pyruvate which further metabolized into CO2 and H2O, whereas the end-product of anaerobic glycolysis is lacate which diffuses to blood, causing a reduction in blood pH).
- **Diabetic Ketoacidosis (DKA):** is a serious complication of insulin- deficiency induced hyperglycemia, where the body produces ketone bodies as an alternative source of energy for cells which are deprived from glucose. Ketone bodies involve acetoacetate and β-hydroxybutyrate, which are acidic in nature, thus when moving to blood, they lower blood pH resulting in acidemia.

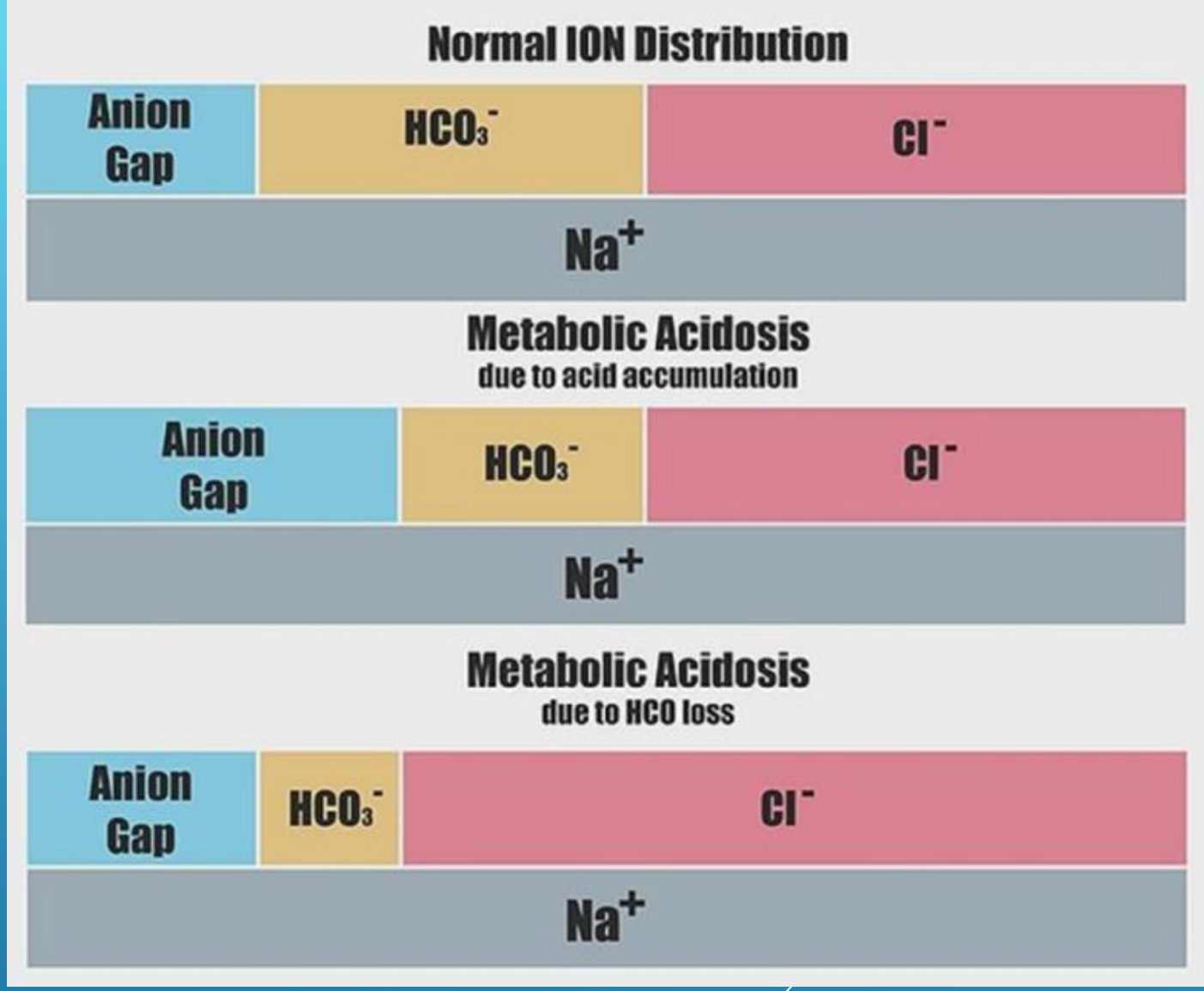
Anion gap

- The body fluids are electrochemical neutral (anions = cations)
- - It is not easy to measure all the anions in the body.
- Total concentration of anions and cations in plasma must be equal to maintain electrical neutrality, but only certain cations (Na+ with or without K+) and anions (Cl- , HCO3) are routinely measured in clinical laboratory.
- - The anion gap is the difference in the measured cations and the measured anions in serum, plasma, or urine. So the anion gap is usually measured by calculating the difference between the sum of the two *main* cations; Na+ and K+, and the sum of the two main anions; Cl+ and HCO3-.
- - The concentrations are expressed in units of (mmol/L)
- **= ([Na+] + [K+]) − ([Cl−] + [HCO3−])**

- potassium has become widely accepted, as potassium concentrations, being very low, usually have little effect on the calculated gap.
- **= [Na+] − ([Cl−] + [HCO3−]) normal is 6 - 18 mmol/L**
- Clinicians use the anion gap to identify the cause of metabolic acidosis.
- In a healthy person, the normal range of the anion gap is **6 - 18 mmol/L.**
- **-** If the gap is greater than normal, metabolic acidosis is diagnosed.
- **-** When the [HCO3] increases or decreases, other ions must take its place to maintain electrochemical neutrality.
- a) If HCO3 decrease and the anion gap increase this means increased levels of other acids (sulphuric, lactic, acetoacetate or salicylate) (its metabolic acidosis due to increased H+ production)
- b) If HCO3 decrease and the anion gap does not change this means Cl- substitutes for HCO3 (its metabolic acidosis due to HCO3 loss).
-

- if metabolic acidosis is a result of acid production (such as the case in lactate, acetoacetate, salicylate… (**organic acidosis**), the acidosis here is NOT substituted; i.e., with **ELEVATED anion gap**.

- In metabolic acidosis induced by HCO3- loss, the loss is substituted with Cl- (**hypercholremic acidosis**), and the acidosis here is with **NORMAL anion gap**.,



Metabolic acidosis with an elevated anion gap is seen in:

- 1- Diabetic ketoacidosis: altered metabolism of fatty acids, as a consequence of the lack of insulin, causes endogenous production of acetoacetic and β-hydroxybutyric acids.
- 2- Lactic acidosis: this results from a number of causes, particularly acute hypoxic states such as respiratory failure lactic acidosis develops within minutes and is life-threatening.
- 3- Certain cases of overdosage or poisoning including:
- Salicylate overdose where build-up lactate occurs.
- Methanol poisoning when formate accumulates.
- Ethylene glycol poisoning where oxalate is formed.

- Salicylates raise the gap to 20.
- Renal failure raises gap to 25.
- Diabetic ketoacidosis raises the gap to 35-40.
- Lactic acidosis raises the gap to > 35 (>50).

Hyperchloraemic acidosis is seen in:

1- Chronic diarrhea

2-Fluids containing bicarbonate are lost from the body.

3-Renal tubular acidosis, cells are unable to excrete hydrogen ions efficiently, and bicarbonate is lost in the urine.

Metabolic Alkalosis:

- 1) Excess H+ loss: Gastric secretions contain large quantities of hydrogen ions. Loss of gastric secretions, therefore, results in a metabolic alkalosis. This occurs in prolonged vomiting for example, pyloric stenosis or anorexia nervosa preventing parallel loss of bicarbonate-rich secretions from the duodenum

- 2) Excessive reabsorption of bicarbonate: Bicarbonate and chloride concentrations are linked. If chloride losses are excessive then bicarbonate will be reabsorbed to maintain electrical neutrality.

e.g) Chloride may be lost from the GIT in prolonged vomiting. Chloride losses may also occur in the kidney usually as a result of diuretic drugs. These drugs cause increased loss of chloride in the urine resulting in excessive bicarbonate reabsorption.

- 3) Ingestion of alkalis (overdose of sodium bicarbonate)

- 4- **Potassium deficiency:** hypokalemia results in alkalosis in the following mechanism: ↓plasma [K+] → Na+ and H+ move into the cell to maintain cellular electroneutrality → ↓ ECF [H+] → alkalosis.
-

Respiratory Acidosis:

- Most commonly due to decreased alveolar ventilation causing decreased excretion of CO_2. Less commonly it is due to excessive production of CO_2 by aerobic metabolism.
- 1) Inadequate CO2 excretion: The causes of decreased alveolar ventilation are numerous such as airway obstruction, central causes (stroke, trauma in brain) and lung diseases (severe asthma).
- 2) Excess CO2 production: This may occur in syndromes such as malignant hyperpyrexia, hyperthyroidism.

- **Respiratory acidosis** occurs within minutes or hours. The primary problem in acute respiratory acidosis is alveolar hypoventilation. If airflow is completely or partially reduced, the PCO2 in the blood will rise immediately and the [H+] will rise quickly. A resulting low PO2 and high PCO2 causes coma. If this is not relieved rapidly, death results.
- uncompensated because the renal compensation requires long time,

- **Chronic respiratory acidosis** usually results from chronic obstructive airways disease (COAD) and is usually a long standing condition, accompanied by maximal renal compensation. In a chronic respiratory acidosis the primary problem again is usually impaired alveolar ventilation, but renal compensation contributes markedly to the acid–base picture. The kidney increases hydrogen ion excretion and ECF bicarbonate levels rise, blood [H+] tends back towards normal
- compensated by kidneys.

Respiratory alkalosis:

- Usually these are acute conditions, and there is no renal compensation.
- - It is common than respiratory acidosis
- - Result from the excessive excretion of CO2 this is commonly seen in hyperventilation due to:

1- anxiety (**Hysterical over-breathing)**

2- High altitude.

3-**Mechanical over-ventilation** in an intensive care patient raised intracranial pressure, or hypoxia, both of which may stimulate the respiratory center.

- - Treatment: Increasing the inspired CO2 by making the patient re-breathe into a paper bag.

ASSESSMENT OF HYDROGEN IONS STATUS

$$H^+ = 24 \times \frac{pCO_2}{HCO_3^-}$$

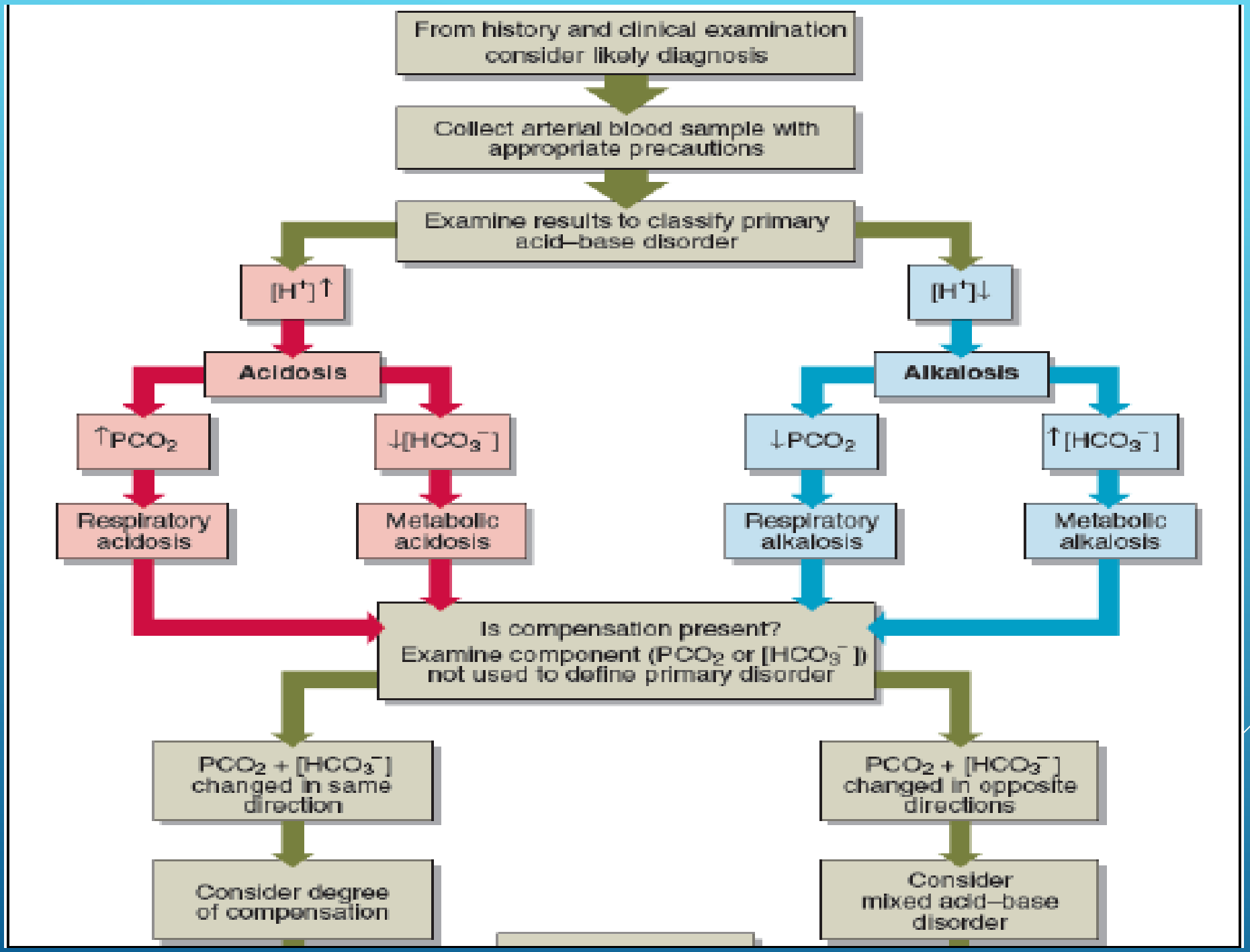
- Specimen for blood gases analysis:
- 1) Arterial blood is used
- 2) Heparin is used as anticoagulant
- 3) Exclude air from the syringe before and after the blood is collected.
- 4) The sample should be placed in ice during transit
- - The assessment of acid-base status is carried out by measuring [H+], [HCO3] and PCO2 however, in practice, we measure [H+] and PCO2 and there is no need to measure HCO3 as it can be calculated when the other 2 variables are known

▪ The practical approach to the interpretation of blood gas results:

- 1) Look at the [H+]. Decide if an acidosis or an alkalosis
- 2) **If acidosis,** decide what is the primary cause:
- - Increased PCO_2 → Respiratory acidosis
- - Decreased HCO_3 → Metabolic acidosis

- 3) **If alkalosis,** decide what is the primary cause:
- - Decreased PCO_2 → Respiratory alkalosis
- - Increased HCO_3 → Metabolic alkalosis.

4) Having decided on the primary acid-base disorder, look to see if there is compensation. There will be a change in the other component (the one which was not used to determine the primary disorder) in the direction which compensates for the primary disorder.


From history and clinical examination consider likely diagnosis
Collect arterial blood sample with appropriate precautions
Examine results to classify primary acid–base disorder
[H+]↑
[H+]↓
Acidosis
Alkalosis
↑PCO2
↓[HCO3−]
↓PCO2
↑[HCO3−]
Respiratory acidosis
Metabolic acidosis
Respiratory alkalosis
Metabolic alkalosis
Is compensation present?
Examine component (PCO2 or [HCO3−]) not used to define primary disorder
PCO2 + [HCO3−] changed in same direction
PCO2 + [HCO3−] changed in opposite directions
Consider degree of compensation
Consider mixed acid–base disorder

MIXED ACID–BASE DISORDERS

- **A patient may have both a metabolic and respiratory acidosis, such as the chronic bronchitis patient who develops renal impairment. In such a patient with a raised [H+] and PCO2. both expected findings in *primary* respiratory and *primary* metabolic acidosis.**

- **Other examples of mixed acid–base disorders commonly encountered are:**

1- Chronic respiratory disease + hypokalemia.

2- Intoxication with an acid-producing toxin + renal failure.

Clinical manifestations of acid-base imbalance:

- Mild acidosis may not cause any symptoms or it may be associated with nonspecific symptoms such as fatigue, nausea, and vomiting. Acute metabolic acidosis may also cause an increased rate and depth of breathing, confusion, and headaches, and it can lead to seizures, coma, and in some cases death. In case of severe acidosis, HCO_3^- may be intravenously given to people with dangerously low blood pH levels
- Symptoms of alkalosis are often due to associated potassium (K^+) loss and may include irritability, weakness, and muscle cramping.

- **Kussmaul breathing** is a deep, rapid and labored breathing pattern often observed in severe metabolic acidosis, particularly diabetic ketoacidosis (DKA) and kidney failure.

Clinical cases

- Case # 1

A 60-year-old man was admitted to hospital with severe abdominal pain that had begun 2.5 h earlier. He was not taking any drugs. On examination, he was shocked and had a distended, rigid abdomen; neither femoral pulse was palpable. **Investigations**

Test	Result	Reference values
pH	7.05	7.35 – 7.45
PCO_2	26.3 mmHg	35 – 46 mmHg
Serum HCO_3	7 mmol/L	22 – 30 mmol/L

- Case # 2

A 45-year-old man was admitted to hospital with a history of persistent vomiting. On examination, he was obviously dehydrated and his respiration was shallow. Gastroscopy showed pyloric stenosis, thought to be due to scarring caused by peptic ulceration. **Investigations**

Test	Result	Reference values
pH	7.56	7.35 – 7.45
PCO_2	54 mmHg	35 – 46 mmHg
Serum HCO_3	45 mmol/L	22 – 30 mmol/L

- Case # 3

As part of a class experiment in physiology, a medical student volunteered to have a sample of arterial blood taken. The demonstrator took some time to explain the procedure to the class, during which time the student became increasingly anxious. As the blood was being drawn the student complained of tingling in her fingers and toes. **Investigations**

Test	Result	Reference values
pH	7.52	7.35 – 7.45
PCO_2	26.4 mmHg	35 – 46 mmHg
Serum HCO_3	25 mmol/L	22 – 30 mmol/L

➢ Case # 5

A young woman was admitted to hospital unconscious, following a head injury. A skull fracture was demonstrated on radiography and a computerized tomography (CT) scan revealed extensive cerebral contusions. The respiratory rate was increased, at 38/min. Three days after admission, the patient's condition was unchanged. **Investigations**

Test	Result	Reference values
pH	7.5	7.35 – 7.45
PCO_2	29.3 mmHg	35 – 46 mmHg
Serum HCO_3	18 mmol/L	22 – 30 mmol/L

➢ Case # 6

An elderly man was admitted to hospital in a confused state. He was dyspnoeic and had a cough productive of sputum. He was unable to give a coherent history, but one of the casualty officers knew him to be an insulin-treated diabetic patient with a long history of chronic bronchitis. **Investigations**

Test	Result	Reference values
pH	7.18	7.35 – 7.45
PCO_2	55.3 mmHg	35 – 46 mmHg
Serum HCO_3	38 mmol/L	22 – 30 mmol/L

THE END