Arterial blood gas analysis: Basic concept & Clinical correlation

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Learning objectives

At the end of the session we will able to know-

- Basic concept of Arterial blood gas analysis
- Acid-base homeostasis
- Role of buffers in maintenance of acid-base homeostasis
- Acid-base parameters in arterial blood gas analysis
- Acid-base disorders
- Diagnostic approach to acid-base disorders

Arterial blood gas (ABG) analysis

Arterial blood gas (ABG) analysis:

An **arterial blood gas (ABG) analysis** measures the amounts of **arterial gases**-

- Oxygen (O_2)
- Carbon dioxide (CO_2).

An **ABG test** measures the **arterial blood gas tension values** of –

- Partial pressure of oxygen (PaO_2)
- Partial pressure of carbon dioxide ($PaCO_2$)
- Blood pH
- Oxygen saturation (SaO_2)
- Serum HCO_3^-

Procedure of ABG analysis:

After a pulse is found, a blood sample is taken from the artery

Procedure of ABG analysis:

Collection of blood sample for ABG

Blood gas analyzer

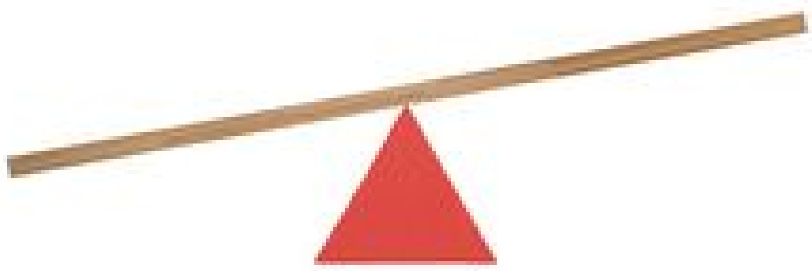
Acid-base homeostasis

Acid-base homeostasis:

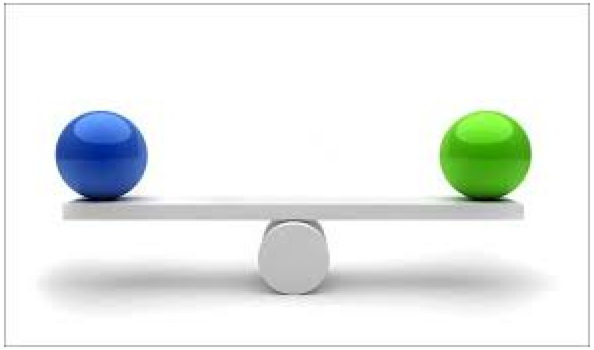
To achieve homeostasis
precise H^+ regulation
is essential.

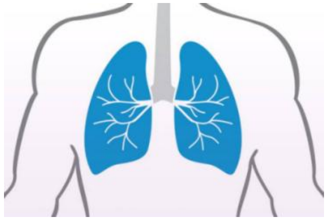
As almost all
the enzyme systems
in the body are
influenced by
H^+ concentration.

Therefore, changes in
H^+ concentration
alter virtually
all **cells** and **body functions**.

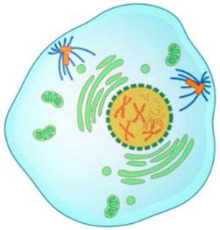


So, there must be a balance
between the
intake or production of H^+ and
the net removal of H^+
from the body.



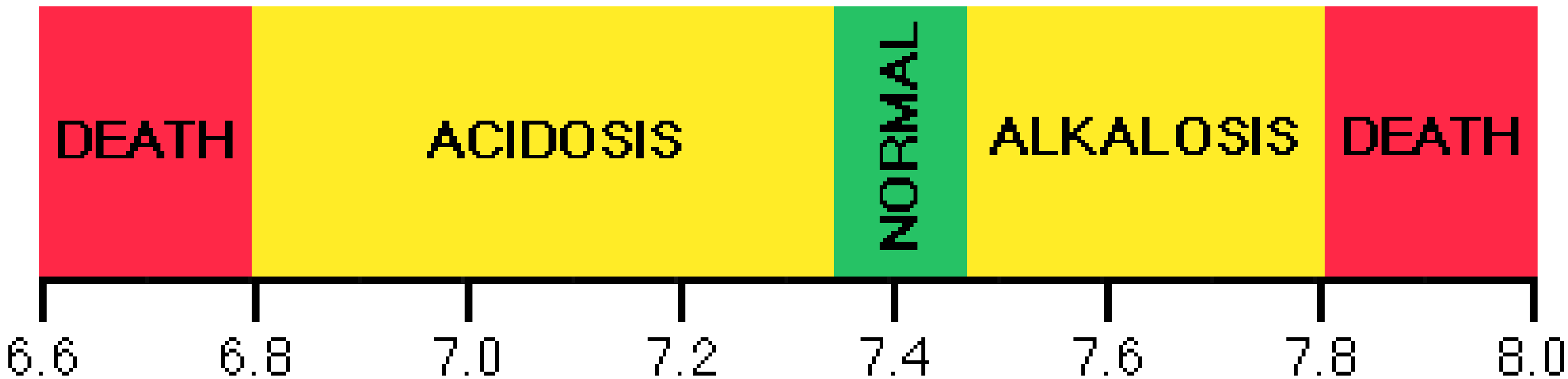


Besides the **kidneys**,
there are multiple
acid-base buffering mechanisms –
the **blood**, **cells** and **lungs**
that are essential in maintaining
normal **H^+ concentrations**
in both ECF & ICF.



pH scale

Normal blood pH level



Regulation of Acid –Base Homeotasis

During **metabolism,** body produces **metabolic acids** and **metabolic bases**.

There are two types of **metabolic acids** –

1. **Volatile acids:** CO_2 or H_2CO_3 -

   Route of excretion: Lungs

2. **Nonvolatile acids:** HCl, H_2SO_4, H_3PO_4, lactic acid, ketoacid

   Route of excretion: Kidney

Metabolic acid or **base** production depends on following factors –

1. Insulin status
2. Blood flow to tissues
3. Oxygen supply to tissues
4. Dietary habit:

Protein generates more **acid**
Fruits & **vegetables** generate more **base**.

Three primary systems regulate the **pH** of the body fluids to prevent **acidosis** and **alkalosis.**

1. **Body fluid buffer system**
   (1st line of defense, within seconds)

2. **Respiratory mechanism**
   (2nd line of defense, within few minutes)

3. **Renal mechanism**
   (3rd line of defense, hour to several days)

Role of buffer system in maintenance of p^H

Buffer systems:

1. Bicarbonate buffer system
2. Phosphate buffer system
3. Protein buffer system

1. Bicarbonate buffer system

Mechanism of action

1. Bicarbonate buffer system:

Mechanism of action

In acidosis,

$$HCl + NaHCO_3 \rightarrow H_2CO_3 + NaCl$$

(strong acid) (weak acid)

In alkalosis,

$$NaOH + H_2CO_3 \rightarrow NaHCO_3 + H_2O$$

(strong base) (weak base)

Since the dissociation of **H_2CO_3**
into **H^+** ion & **HCO_3^-** ion is very poor,
the change in **pH**
of the **body fluid** is
prevented.

2. Phosphate buffer system

Mechanism of action

2. Phosphate buffer system

Mechanism of action

In acidosis,

$$HCl + Na_2HPO_4 \rightarrow NaH_2PO_4 + NaCl$$

(strong acid) (weak acid)

In alkalosis

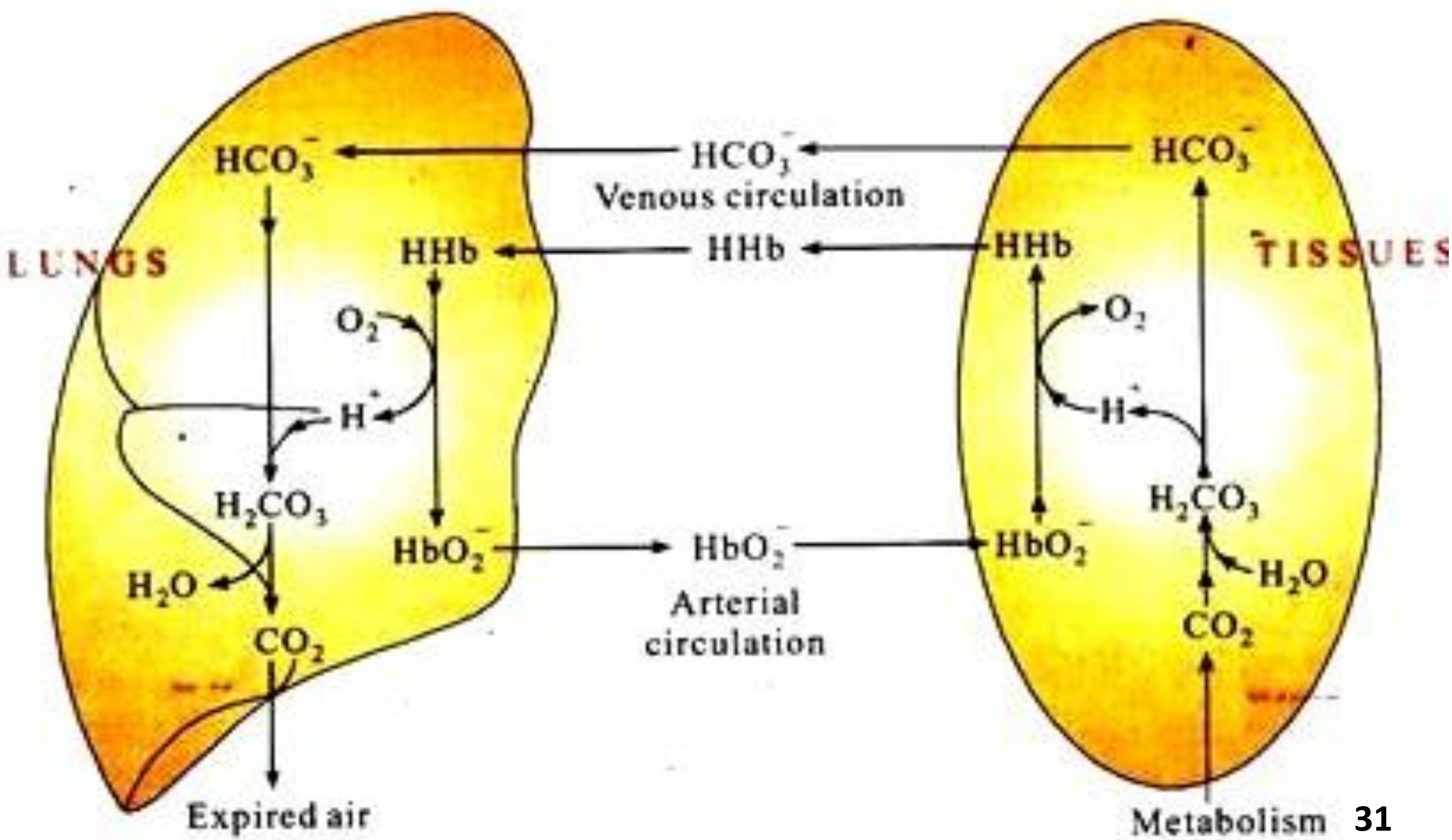
$$NaOH + NaH_2PO_4 \rightarrow H_2O + Na_2HPO_4$$

(strong base) (weak base)

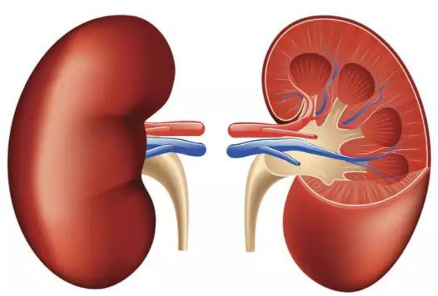
3. Protein buffer system

Mechanism of action

Hemoglobin buffer system



Role of kidney in acid-base balance



Role of kidney in acid-base balance:

Kidney controls **acid-base balance** by excreting either

acidic urine in **acidosis** &

alkaline urine in **alkalosis**.

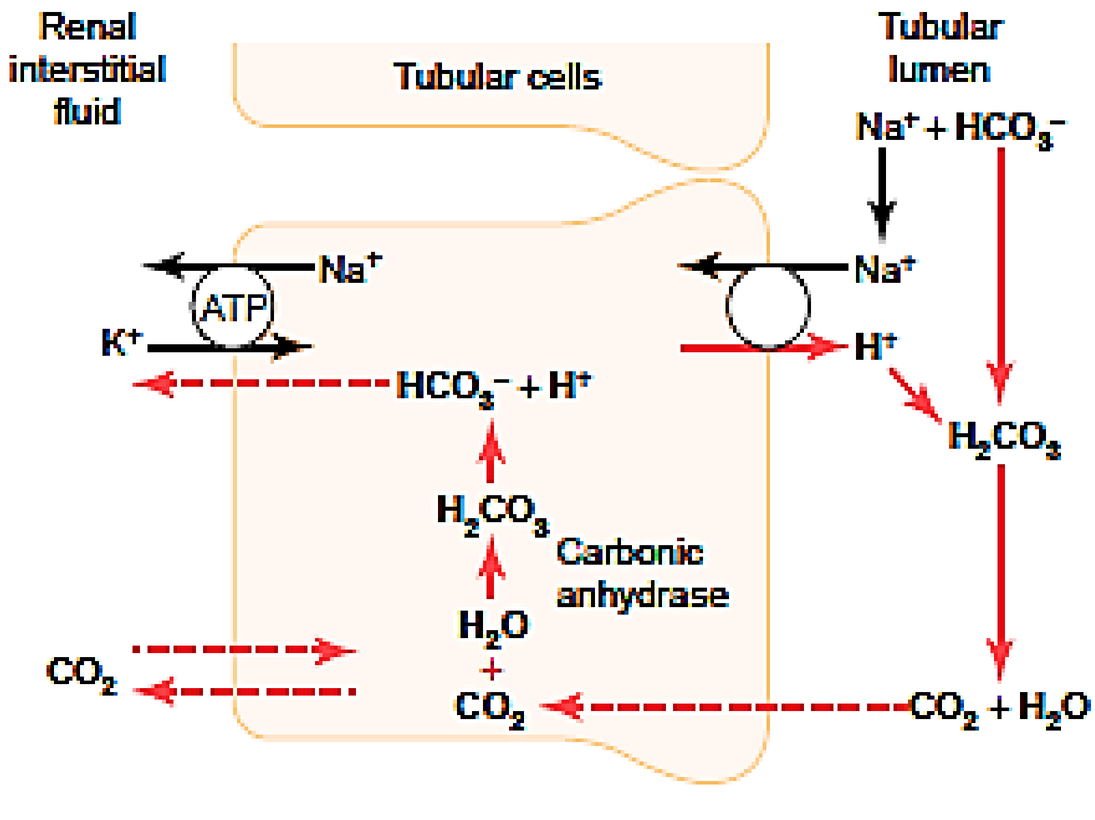
Normally, urinary pH is **acidic**.

1. Bicarbonate buffer mechanism

(Reabsorption of filtered bicarbonate)

Mechanism of action of HCO_3^- buffer:

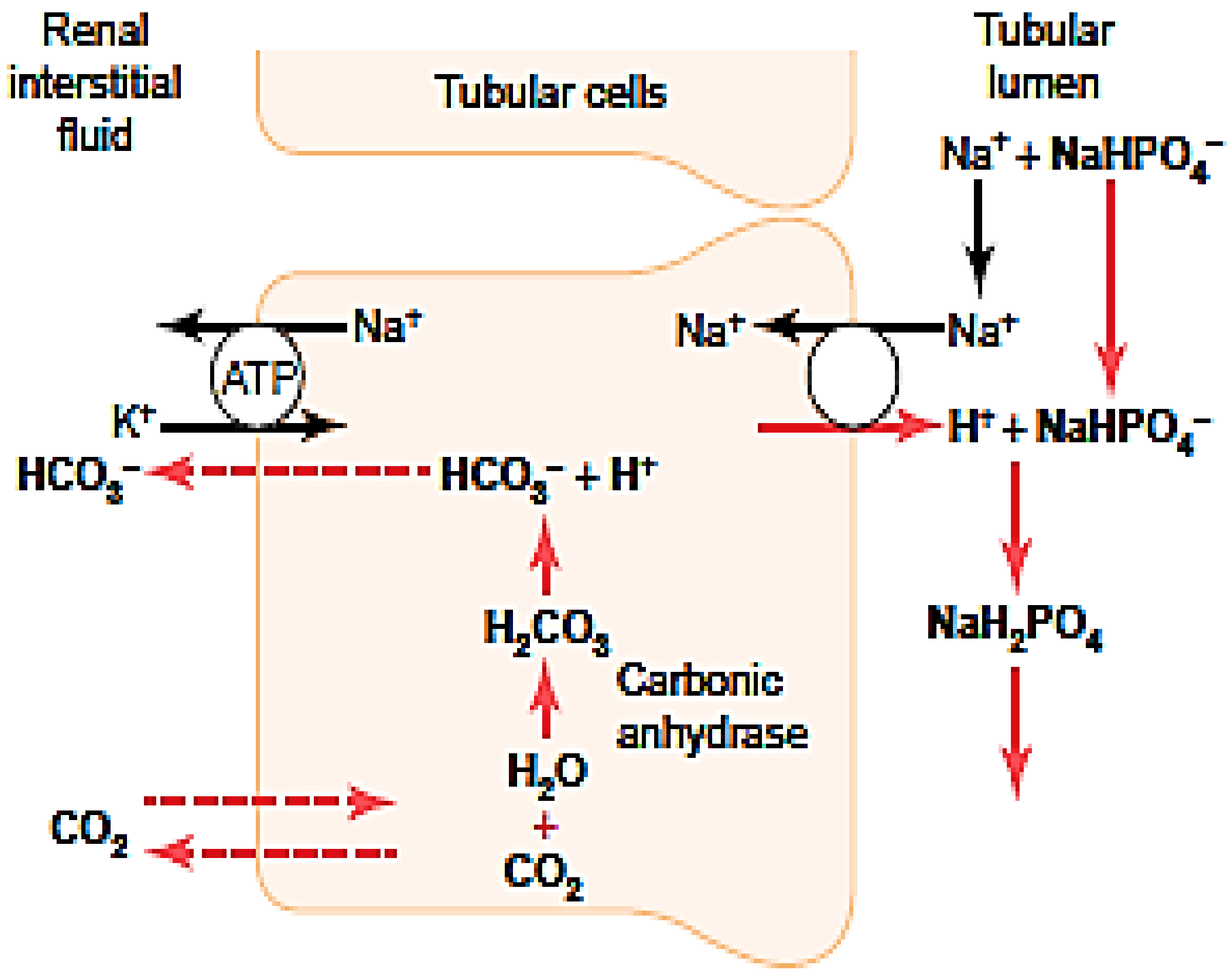
(H^+ ion secretion achieves HCO_3^- reabsorption)



2. Phosphate buffer (HPO_4^-/ H_2PO_4) mechanism

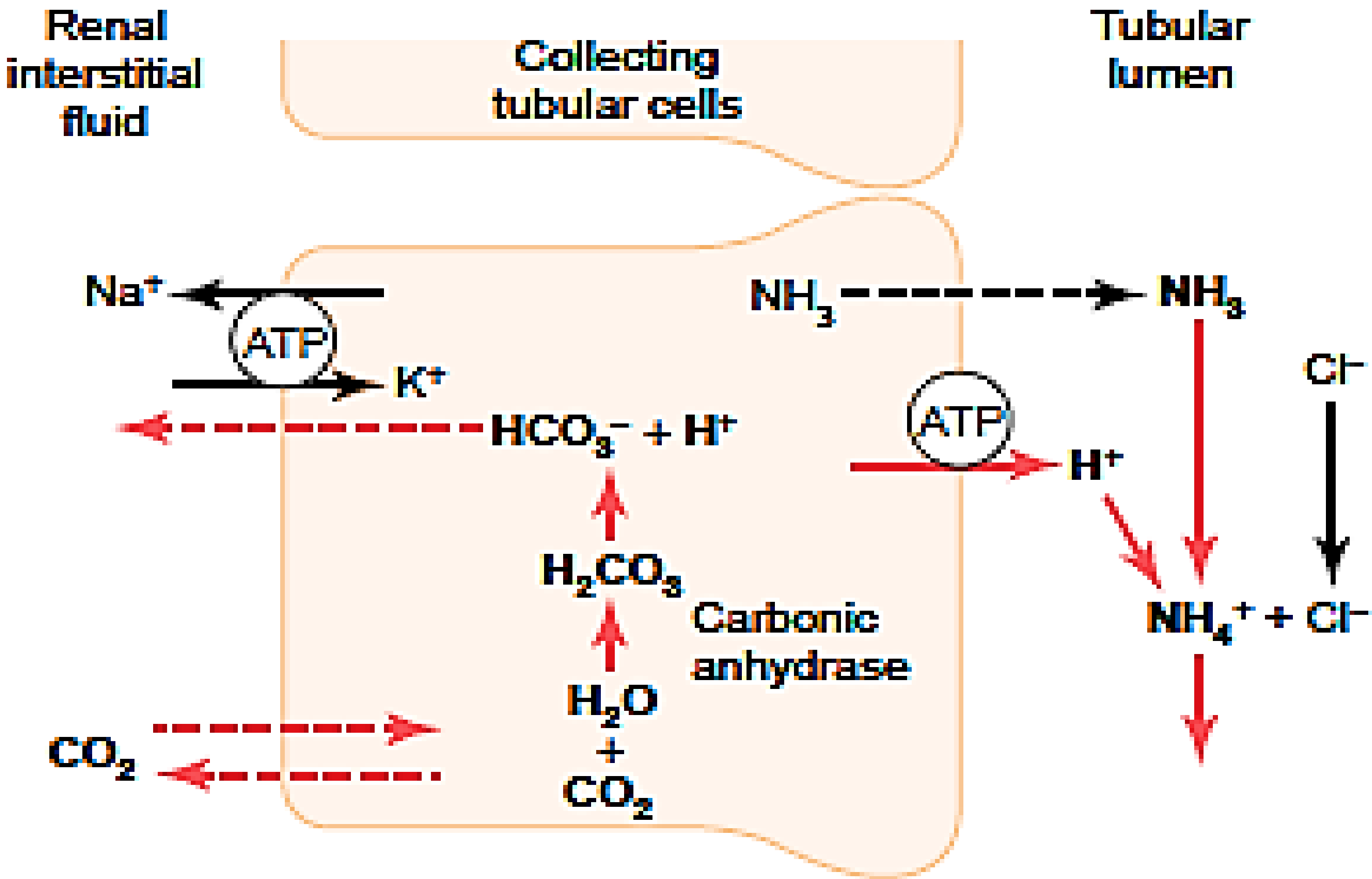
(Generation of new HCO_3^- & excretion of nonvolatile acids)

Mechanism of action of phosphate buffer:



3. Ammonia buffer (NH_3/NH_4) mechanism:

Buffering of secreted H^+ ion by ammonia buffer:



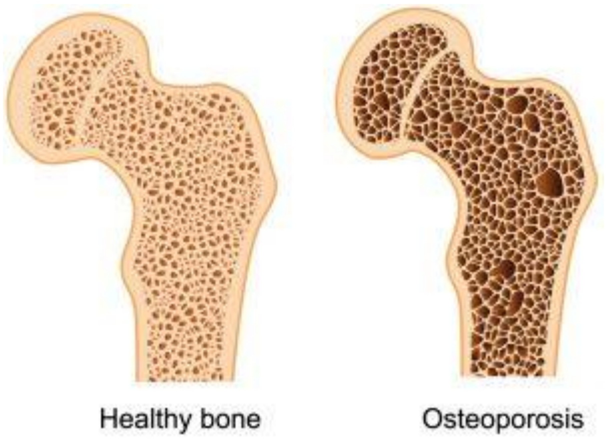
Bone buffer

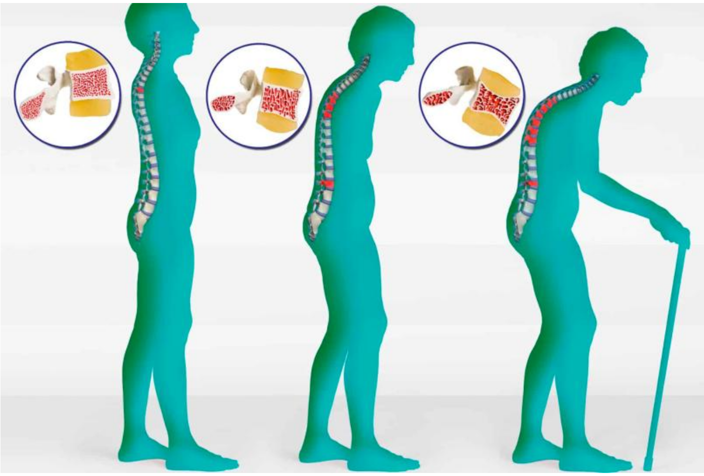
Bone buffer… cont.

Bone buffer is the alkaline **calcium-phosphate salt** of bone deposited in the form of **hydroxyapatite crystal (HAC)**.

In **chronic renal failure**,
bone buffer participates
in buffering activity
to maintain **body pH** but
at the cost of **bone demineralization**.

Patients suffer from Osteoporosis.





Normal acid-base parameters of blood

Normal acid – base parameters of blood

Parameters	Arterial blood	Venous blood
pH	7.4	7.38
PCO_2	40 mmHg	46 mmHg
PO_2	95 mmHg	40 mmHg
HCO_3^-	24 mmHg	26 mmHg
Base excess	± 2mmol/L	± 2mmol/L
Anion gap	8-16 meq/L	8-16 meq/L
O_2 saturation	> 95%	60-80%

Base excess

$[HCO_3^-]p - [HCO_3^-]std$

Base excess

It is the difference between the actual HCO_3^- concentration of an individual & the standard HCO_3^- concentration.

Base excess = $[HCO_3^-]p$ - $[HCO_3^-]$ std

Types of base excess:

1. Positive base excess-

high plasma HCO_3^- concentration.

2. Negative base excess-

low plasma HCO_3^- concentration.

Normal value: - 2 mmol/L to + 2 mmol/L

Positive base excess:

e.g. Metabolic alkalosis

Respiratory acidosis

Negative base excess:

e.g. Metabolic acidosis

Respiratory alkalosis

Anion gap

Anion gap

The plasma **anion gap** is based on the principle of the **'law of electrical neutrality'.**

Anion gap is
quantities of **anions**
not balanced by **cations**.

According to the **law of electrical neutrality** in plasma –

	Total cation = Total anion
	$Na^+ + K^+ + UC = Cl^- + HCO_3^- + UA$
	$Na^+ + K^+ - (Cl^- + HCO_3^-) = UA – UC$
So,	
Plasma anion gap	$= (Na^+) – (Cl^- + HCO_3^-)$
	= 144 – (108 + 24)
	= 144 – 132
	= 12mEq/L

Plasma anion gap = 8-16 mEq/L

Average = 12 mEq/L

Common unmeasured anions in plasma:

1. Plasma protein (mainly albumin)
2. Phosphate
3. Sulphate
4. Lactate
5. Keto acid anions
6. Other organic acid anions

Common unmeasured cations in plasma:

1. Calcium
2. Magnesium

Anion gap decreases in –

1. Hypoalbuminemia
2. Hypercalcemia
3. Hypermagnesemia
4. Hypergamma globulinemia

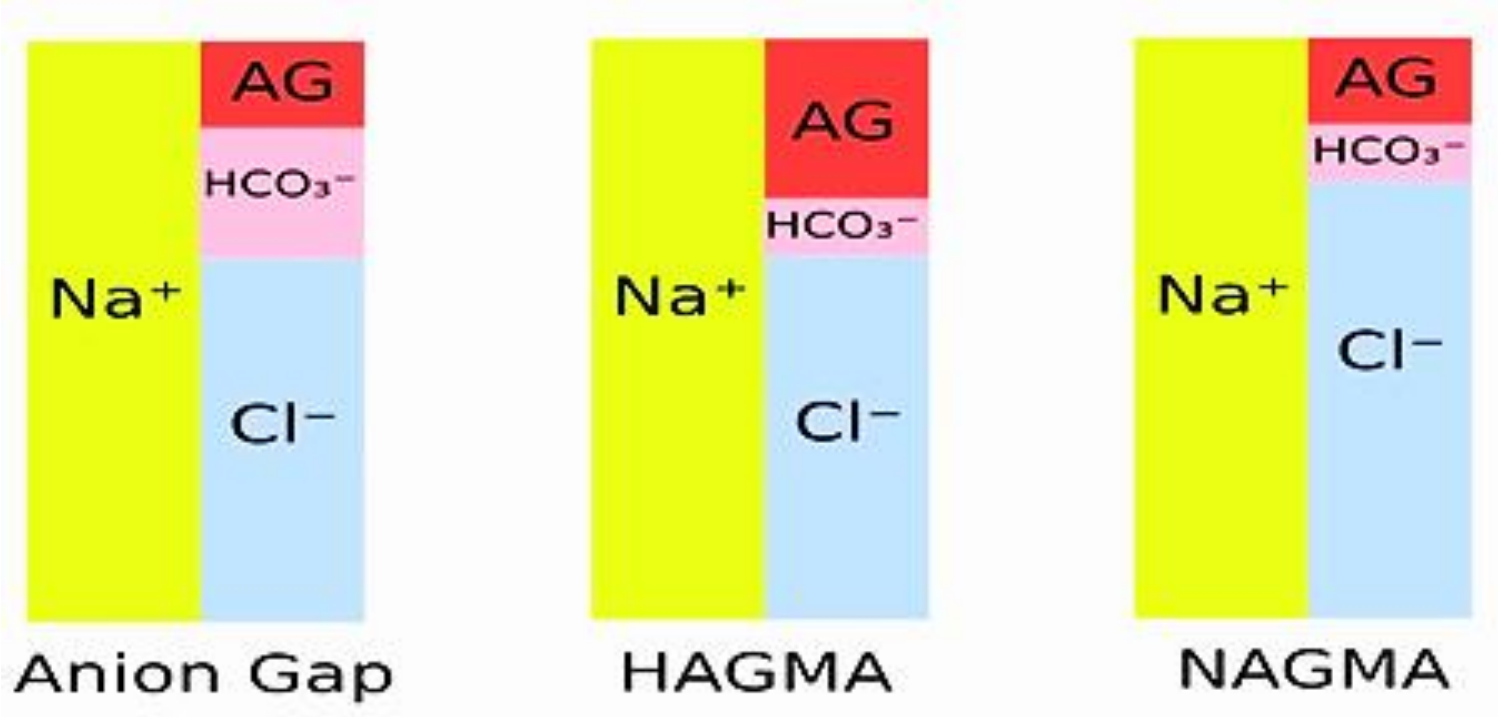
Anion gap increases in-

If it is more than 12 mEq/L

1. Renal failure
2. Lactic acidosis
3. Ketoacidosis
4. Intoxication
   - Alcohol poisoning
   - Salicylate poisoning
5. Hyperalbuminemia

High anion gap metabolic acidosis

Plasma anion gap: Types



Normal anion gap metabolic acidosis:
Hyperchlorimic metabolic acidosis

- **Diarrhoea- HCO_3^- loss in stool**
- **Renal tubular acidosis- HCO_3^- loss in urine**

↓

Kidney reabsorbs Cl^-

Anion gap = $[Na^+] - [\uparrow(Cl^-) + \downarrow(HCO_3^-)]$

= Normal

High anion gap metabolic acidosis:
unmeasured anions-

- Hyperphosphatemia
- Hyperalbuminemia
- IgA producing multiple myeloma

Increased unmeasured anions:

$[Na^+] - [\downarrow(Cl^-) + \downarrow(HCO_3^-)] = \uparrow$ Anion gap

Acid-base disorders

Acidosis:

It is a clinical condition in which **arterial pH** falls **bellow 7.4** due to accumulation of acid or loss of base from the body.

Alkalosis:

It is a clinical condition in which **arterial pH** rises **above 7.4** due to accumulation of base or loss of acid from the body.

Normally,

The ratio of $HCO_3 : CO_2 = 20 : 1$

In acidosis,

The ratio of $[HCO_3/CO_2]$ falls bellow 20/1

Acid-base disorders:

A. Respiratory:

Respiratory acidosis

Respiratory alkalosis

B. Metabolic:

Metabolic acidosis

Metabolic alkalosis

Respiratory acidosis

↑ PCO_2

Respiratory acidosis

It is the **acidosis** occurs due to **primary hypoventilatory disorder** of lungs leading to increased **PCO_2** and decreased **pH** in plasma.

Causes:

A. Pulmonary diseases:

1. Asthma
2. Chronic obstructive pulmonary disease (COPD):
    - a. Chronic bronchitis
    - b. Emphysema
3. Severe pneumonia
4. Severe pulmonary edema
5. Massive pulmonary embolism
6. Diffuse parenchymal lung disease

B. Respiratory center depression:

Sedatives

Anesthetics

Strock

Brain tumour

C. Mechanical hypoventillation

D. Neuromuscular defect:

Poliomyelitis

Guillain - Barre syndrome

Myasthenia gravis

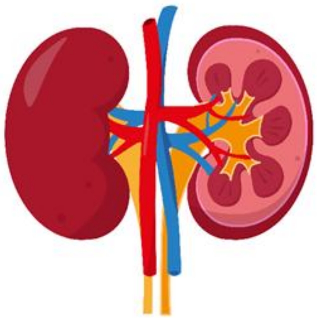
Spinal cord injury

Primary biochemical change:

↑ PCO_2

Renal compensation & correction:

- Complete **HCO_3^-** reabsorption from PCT
- New generation **HCO_3^-** in CD
- ↑ Ammonia formation



Respiratory alkalosis

↓ PCO_2

Respiratory alkalosis

It is the **alkalosis** occurs due to **primary hyperventilatory disorder** of **lungs** leading to **decreased** **PCO_2** and **increased** **pH** in **plasma**.

Causes:

A. CNS mediated stimulation of respiratory center:

1. Neurological disease: a. **Encephalitis**
   b. **Meningitis**
2. Poisoning: **Salicylate**
3. Psychological: a. **Hysteria**
   b. **Anxiety**
4. Septicemia
5. Hepatic encephalopathy

B. Tissue hypoxia mediated stimulation of respiratory center:

1. Congestive cardiac failure
2. Severe anemia
3. Congenital cyanotic heart disease
4. Pulmonary diseases:
   a. Pneumonia
   b. Pulmonary edema
   c. Pulmonary embolism
   d. Interstitial lung disease

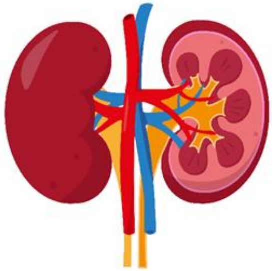
Primary biochemical change:

↓ PCO_2

Renal compensation & correction:

a. Inhibition of HCO_3^- reabsorption from PCT

b. Renal HCO_3^- excretion



Metabolic acidosis

pH < 7.35

Metabolic acidosis

It is the **acidosis** **occurs** due to primary decrease in **plasma** **HCO_3^-** following **gain of acid** other than **H_2CO_3** or loss of **base**.

Metabolic acidosis is characterized by –

Reduction in plasma **HCO_3^-** **&**

Consequent rise in **$[H^+]$**

The PCO_2 decreased secondarily by hyperventilation.

Four main causes of metabolic acidosis-

1. Lactic acidosis
2. Renal tubular acidosis
3. Ketoacidosis
4. Diarrhoea

Aetiological types of metabolic acidosis:

A. Normal anion gap metabolic acidosis:

1. Loss of base through GIT-
   - Diarrhea
   - Intestinal fistula
   - Pancreatic fistula
   - Biliary fistula
   - Ileostomy
   - Colostomy
2. Renal tubular acidosis
3. Hypoaldosteronism
4. Aldosterone antagonist abuse

B. High anion gap metabolic acidosis:

1. Renal failure
2. Lactic acidosis
3. Ketoacidosis
4. Intoxication:

   Alcohol, salicylate

Primary defect: ↓ HCO_3^- with normal PCO_2

Compensation: ↓ PCO_2 by hyperventilation

Correction:

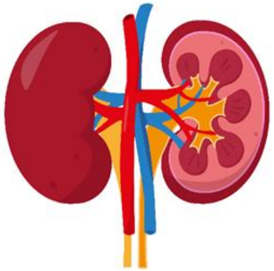
It is corrected by kidney by the following mechanism-

a. **Raise of HCO_3^- by –**

Conservation of HCO_3^-

Generation of new HCO_3^-

b. **Excretion of acidic urine.**



Metabolic alkalosis

pH >7.45

Metabolic alkalosis

It is the **alkalosis** occurs due to primary increase in **plasma HCO_3^- concentration** following **gain of base** or **loss of acid** other than **H_2CO_3**.

Causes:

A. Loss of acid from GIT:

Persistent vomiting

Excessive nasogastric suction

B. Loss of acid through renal route:

Diuretic abuse

Hyperaldosteronism

Cushing syndrome

C. Exogenous alkali administration

Primary biochemical change:

↑ HCO_3^- with normal PCO_2

HCO_3^- : PCO_2 → ↑

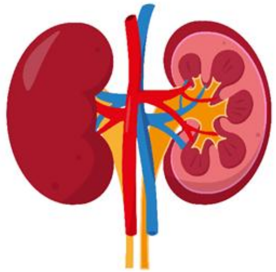
Compensation:

↑ PCO_2 by hypoventilation

Correction:

↑ HCO_3^- excretion

↓ HCO_3^- reabsorption in PCT



Characteristics of primary acid- base balance

Characteristics of primary acid- base balance:

	pH	H^+	PCO_2	HCO_3^-
Normal	7.4	40 meq/L	40 mmHg	24 meq/L
Respiratory acidosis	↓	↑	↑↑	↑
Respiratory alkalosis	↑	↓	↓↓	↓
Metabolic acidosis	↓	↑	↓	↓↓
Metabolic alkalosis	↑	↓	↑	↑↑

Salient feature of simple acid-base disorder:

Types	Primary defect	Unaffected component	HCO_3/PCO_2 ratio	pH
Respiratory acidosis	↑PCO_2	HCO_3^-	↓	↓
Respiratory alkalosis	↓PCO_2	HCO_3^-	↑	↑
Metabolic acidosis	↓HCO_3^-	PCO_2	↓	↓
Metabolic alkalosis	↑HCO	PCO_2	↑	↑

Salient feature of simple acid-base disorder…cont.

Types	Compensation	Mechanism of compensation
Respiratory acidosis	↑HCO_3^-	Renal HCO_3^- generation
Respiratory alkalosis	↓HCO_3^-	Renal HCO_3^- excretion
Metabolic acidosis	↓PCO_2	Hyperventilation
Metabolic alkalosis	↑PCO_2	Hypoventilation

Compensation of acid-base disorders

Compensation of **simple acid - base disorders** follow the '**same direction rule'** –

'Up yields up and down yields down'

Compensation of metabolic acidosis:

1. To counteract the **acidemia** of **metabolic acidosis**, body system creates the scenario of **respiratory alkalosis** by **decreasing PCO_2**

$$pH \propto \frac{HCO_3^- \downarrow}{PCO_2} \quad \textit{Initial} \qquad pH \propto \frac{HCO_3^- \downarrow}{PCO_2 \downarrow} \quad \textit{Compensated}$$

Compensation of metabolic acidosis

2. To counteract the **alkalemia** of **metabolic alkalosis**, body system creates the scenario of **respiratory alkalosis** by **increasing PCO_2**

$$pH \propto \frac{HCO_3^- \uparrow}{PCO_2} \quad \textit{Initial}$$

$$pH \propto \frac{HCO_3^- \uparrow}{PCO_2 \uparrow} \quad \textit{Compensated}$$

Compensation of respiratory acidosis

3. To counteract the **acidemia** of **respiratory acidosis**, body system the scenario of **respiratory alkalosis** by **increasing bicarbonate**

$$pH \propto \frac{HCO_3^-}{PCO_2 \uparrow} \quad \textit{Initial}$$

$$pH \propto \frac{HCO_3^- \uparrow}{PCO_2 \uparrow} \quad \textit{Compensated}$$

Compensation of metabolic alkalosis

4. To counteract the **alkalemia** of **respiratory alkalosis**, body system creates the scenario of **respiratory acidosis** by **decreasing bicarbonate**

$$pH \propto \frac{HCO_3^-}{PCO_2 \downarrow} \quad \textit{Initial} \qquad pH \propto \frac{HCO_3^- \downarrow}{PCO_2 \downarrow} \quad \textit{Compensated}$$

Acid-base disorder & serum potassium

Acid-base disorder & s. potassium…cont.

Acid-base disorders cause **potassium** to shift **into** and **out of cells**, a phenomenon called **"internal potassium balance"**.

Acid-base disorder & s. potassium…cont.

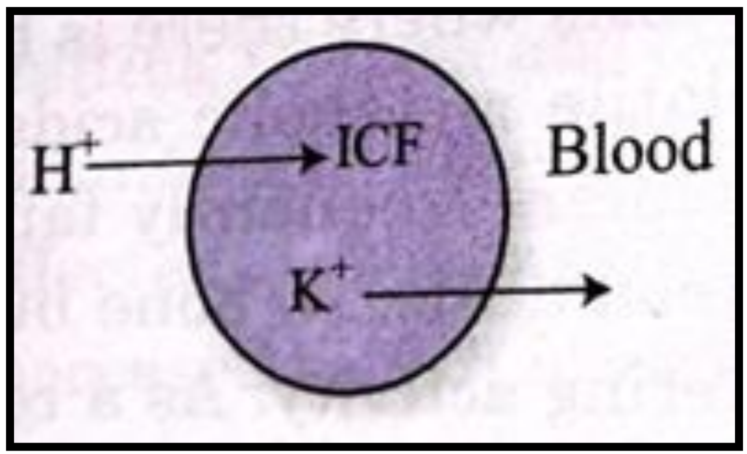
The **plasma potassium concentration** will **rise** by **0.6 mEq/L** for **every 0.1 unit reduction** of the **extracellular pH**.

Acid-base disorder & s. potassium…cont.

In acidosis,

hyperkalemia occurs
because of shifts of **potassium**
from the **intracellular**
to the **extracellular compartment**
to maintain **electrical neutrality**.

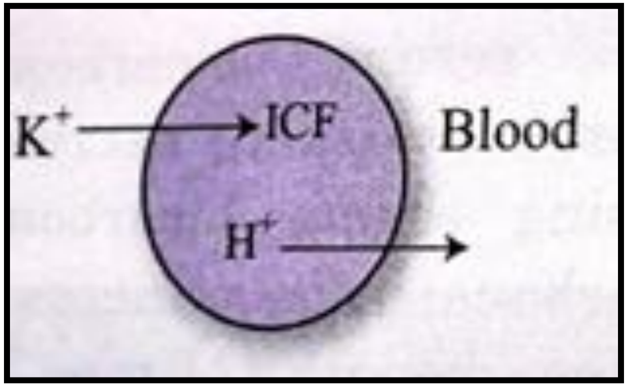
Acid-base disorder & s. potassium…cont.



Efflux of potassium out of the cell in acidosis leading to hyperkalaemia

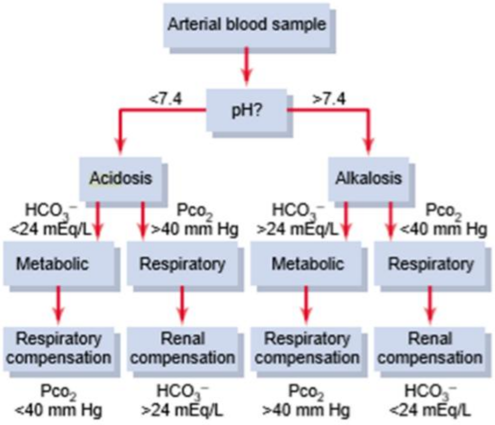
In alkalosis,

the opposite effect occurs, often leading to **hypokalemia**.



Influx of potassium into the cell in alkalosis leading to hypokalemia

Diagnostic approach to Acid-Base disorders



Diagnostic approach to acid-base disorders:

1. Clinical evaluation
2. Laboratory investigations
3. Assessment of acid-base parameters:
   - pH
   - PCO_2
   - PO_2
   - HCO_3^-
   - Base excess
   - Anion gap
   - Oxygen saturation
4. Assessment of compensatory response if simple acid-base disorder.

Arterial blood sample

↓

pH?

< 7.4

↓

Acidosis

- HCO_3^- < 24mEq/L
  ↓
  Metabolic
  ↓
  Respiratory compensation
  PCO_2 <40mmHg

- PCO_2 > 40mmHg
  ↓
  Respiratory
  ↓
  Renal compensation
  HCO_3^- >24mEq/L

> 7.4

↓

Alkalosis

- HCO_3^- > 24mEq/L
  ↓
  Metabolic
  ↓
  Respiratory compensation
  PCO_2 > 40mmHg

- PCO_2 < 40mmHg
  ↓
  Respiratory
  ↓
  Renal compensation
  HCO_3^- < 24mEq/L

Laboratory report based data interpretation

Problem-1:

pH = 7.3, HCO_3^- = 16 mmol/L, PCO_2 = 30 mmHg

$$pH = 7.3 \propto \frac{HCO_3^- \ (16)}{PCO_2 \ (30)}$$

pH = 7.3, HCO_3^- = 16 mmol/L, PCO_2 = 30 mmHg

Solution:

1. pH is decreased ⟶ **Acidosis**
2. HCO_3^- is decreased ⟶ **Metabolic acidosis**
3. PCO_2 is decreased ⟶ **not respiratory acidosis**
4. Compensation:

   As PCO_2 is decreased ⟶ **compensated metabolic acidosis**

Diagnosis: Compensated metabolic acidosis

Problem-2:

pH = 7.5, HCO_3^- = 40 mmol/L, PCO_2 = 50 mmHg

$$pH = 7.5 \propto \frac{HCO_3^- \ (40)}{PCO_2 \ (50)}$$

pH = 7.5, HCO_3^- = 40 mmol/L, PCO_2 = 50 mmHg

Solution:

1. pH is increased → Alkalosis
2. HCO_3^- is increased → Metabolic alkalosis
3. PCO_2 is increased → not respiratory alkalosis
4. Compensation:

   As PCO_2 is increased → compensated metabolic alkalosis

Diagnosis: Compensated metabolic alkalosis

Problem-3:

pH = 7.3, HCO_3^- = 35 mmol/L, PCO_2 = 70 mmHg

$$pH = 7.3 \propto \frac{HCO_3^- \ (35)}{PCO_2 \ (70)}$$

pH = 7.3, HCO_3^- = 35 mmol/L, PCO_2 = 70 mmHg

Solution:

1. pH is decreased → Acidosis
2. HCO_3^- is increased → not metabolic acidosis
3. PCO_2 is decreased → respiratory acidosis
3. Compensation:

   As HCO_3^- is increased → compensated respiratory acidosis

Diagnosis: Compensated respiratory acidosis

Problem-4:

pH = 7.55, HCO_3^- = 16 mmol/L, PCO_2 = 22 mmHg

$$pH = 7.55 \propto \frac{HCO_3^- \ (16)}{PCO_2 \ (22)}$$

pH = 7.55, HCO_3^- = 16 mmol/L, PCO_2 = 22 mmHg

Solution:

1. **pH is increased** → Alkalosis
2. **HCO_3^- is decreased** → not metabolic alkalosis
3. **PCO_2 is decreased** → respiratory alkalosis
4. **Compensation:**

   As HCO_3^- is decreased → compensated respiratory alkalosis

Diagnosis: **Compensated respiratory alkalosis**

Problem-5:

pH = 7.2, HCO_3^- = 26 mmol/L, PCO_2 = 52 mmHg

$$pH = 7.55 \propto \frac{HCO_3^- \ (26)}{PCO_2 \ (52)}$$

pH = 7.2, HCO_3^- = 26 mmol/L, PCO_2 = 52 mmHg

Solution:

1. pH is decreased → Acidosis
2. HCO_3^- is near normal → Uncompensated disorder
3. PCO_2 is high → Respiratory acidosis
3. Compensation:

   Not compensated

Diagnosis: Acute (uncompensated) respiratory acidosis

Problem-6:

pH = 7.7, HCO_3^- = 21 mmol/L, PCO_2 = 27 mmHg

$$pH = 7.55 \propto \frac{HCO_3^-\ (21)}{PCO_2\ (27)}$$

pH = 7.7, HCO_3^- = 21 mmol/L, PCO_2 = 27 mmHg

Solution:

1. pH is increased → Alkalosis
2. HCO_3^- is near normal → uncompensated disorder
3. PCO_2 is low → Respiratory alkalosis
4. Compensation:

Not compensated

Diagnosis: Acute (uncompensated) respiratory alkalosis

Problem-7:

pH = 6.9, HCO_3^- = 12 mmol/L, PCO_2 = 55 mmHg

$$pH = 7.55 \propto \frac{HCO_3^-\ (12)}{PCO_2\ (55)}$$

pH = 6.9, HCO_3^- = 12 mmol/L, PCO_2 = 55 mmHg

Solution:

1. **pH is decreased** ⟶ **Acidosis**
2. **HCO_3^- is decreased** ⟶ **Metabolic acidosis**
3. **PCO_2 is increased** ⟶ **Respiratory acidosis**
4. **Compensation:**

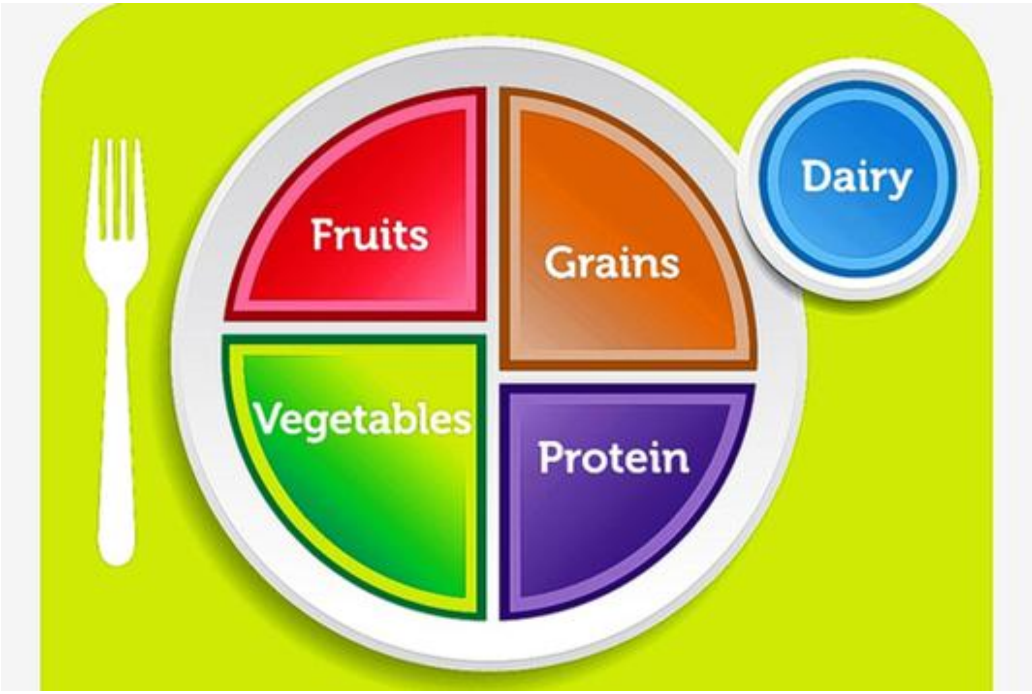
   Not compensated

Diagnosis: **Metabolic acidosis with respiratory acidosis**

Take home message

Take home message

Take home message



Thank You