Sem 3 Revision

Lipoprotein metabolism

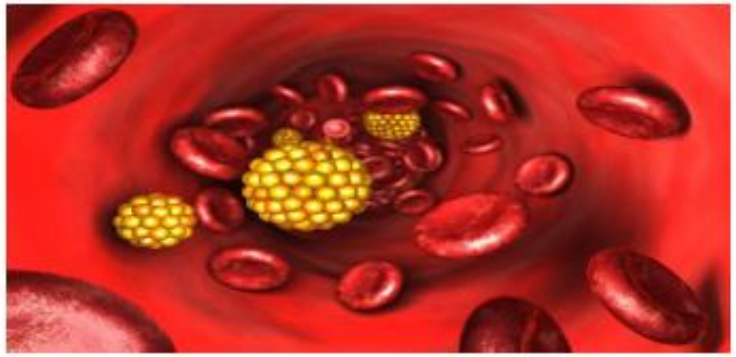
Plasma lipoproteins

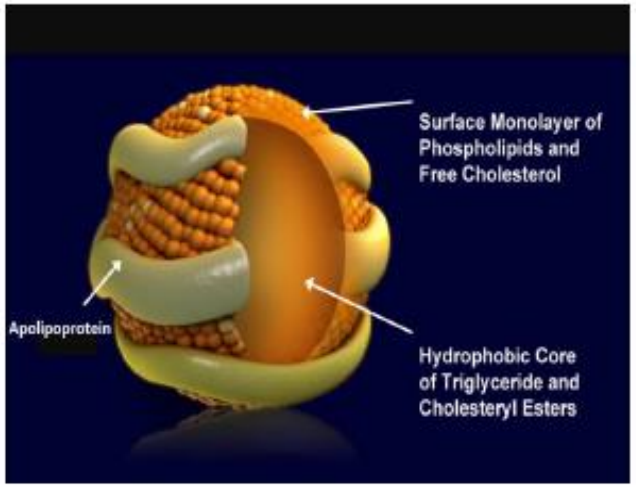
❖Functions:

➢ The primary function of plasma lipoproteins is to

transport lipids in circulation.

• Lipids are insoluble in water, so it bind to proteins to form water-soluble lipoproteins to be transported in the blood.





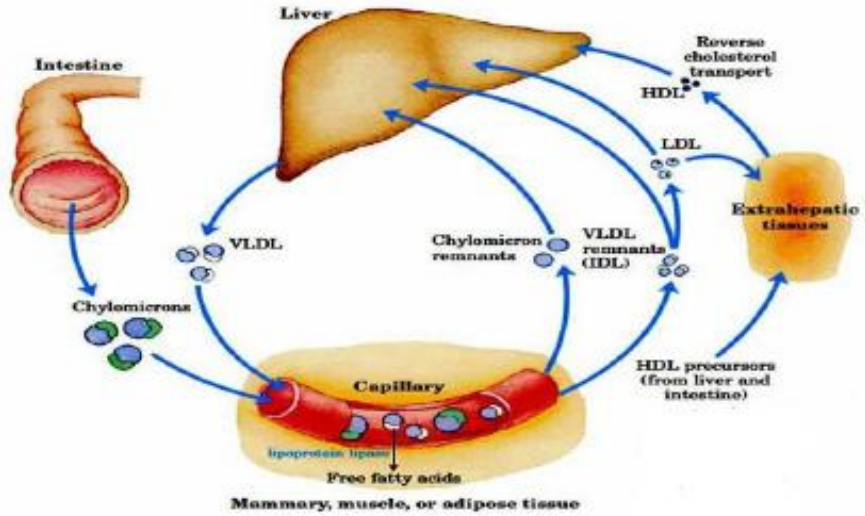
Lipoprotein metabolism

Plasma lipoproteins

❖ Functions:

➢ These lipoproteins play a key role in:

- Transport of dietary lipids from the small intestine,
- Transport of lipids from the liver to peripheral tissues,
- Transport of lipids from peripheral tissues to the liver.



Lipoprotein metabolism

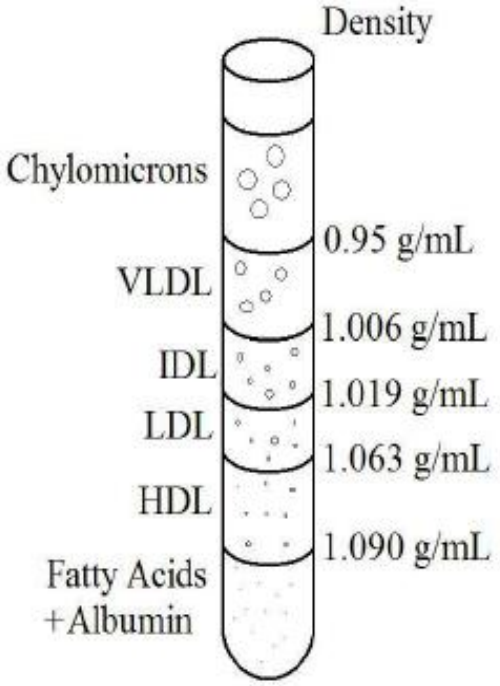
Plasma lipoproteins

❖Classification:

- Lipoproteins differ in **lipid and protein composition**, **size**, **density** and **electrophoretic mobility**.

- Based on density, the main 4 types are:

1) **Chylomicrons.**
2) **Very low density lipoproteins (VLDL).**
3) **Low density lipoproteins (LDL).**
4) **High density lipoproteins (HDL).**



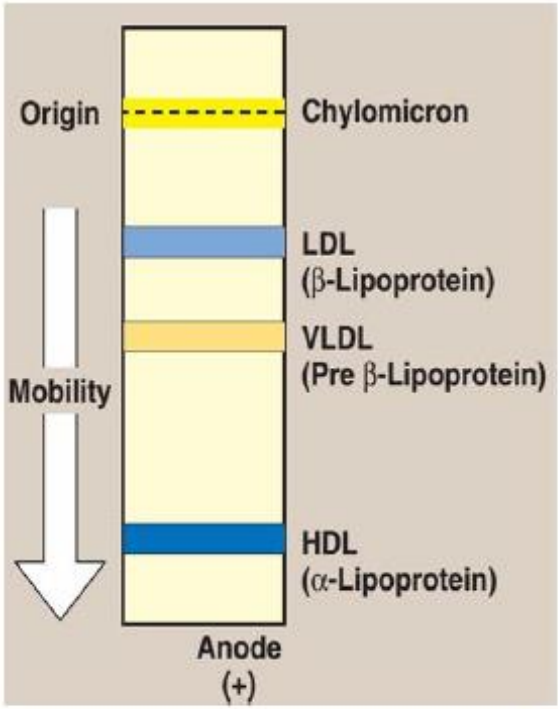
Lipoprotein metabolism

Plasma lipoproteins

❖ Classification:

- According to electrophoretic mobility, the main 4 types are:

1) Chylomicrons.
2) β-lipoproteins (LDL).
3) Pre β-lipoproteins (VLDL).
4) α-lipoproteins (HDL).



Lipoprotein metabolism

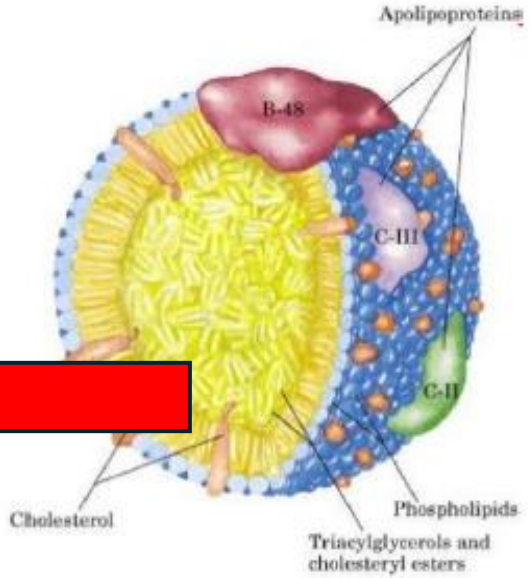
Chylomicrons

✓ **Site of synthesis:** intestinal mucosa.

✓ **The largest lipoproteins.**

✓ **Composition:**

- Lipid core (TG 85-90% and CE).
- Outer layer of phospholipids and cholesterol.
- Apolipoprotien, mainly **apoB-48** (1-2%).



✓ **Function:** transport

dietary TG from intestine to peripheral tissues

(liver, muscle and adipose tissue).

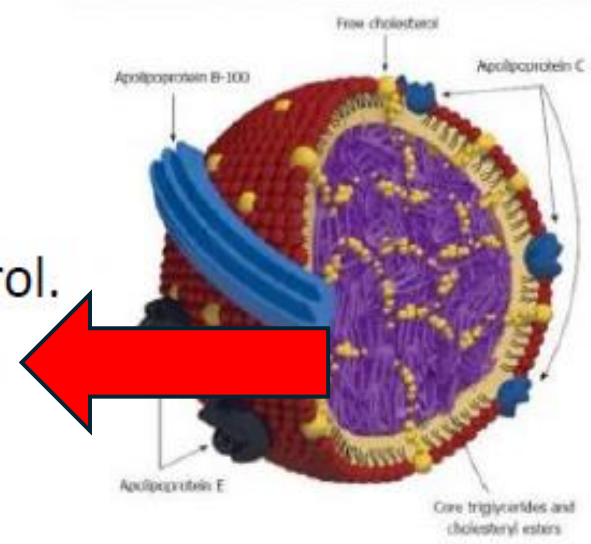
Lipoprotein metabolism

VLDL
(Pre β-lipoproteins)

✓ **Site of synthesis:** liver.

✓ **Composition:**

- Lipid core (TG 55% and CE).
- Outer layer of phospholipids and cholesterol.
- Apolipoprotien, mainly **apoB-100** (7-10%).



✓ **Function:** transport

TG from liver to extrahepatic tissues

(muscle and adipose tissue).

Lipoprotein metabolism

LDL
(β-lipoproteins)

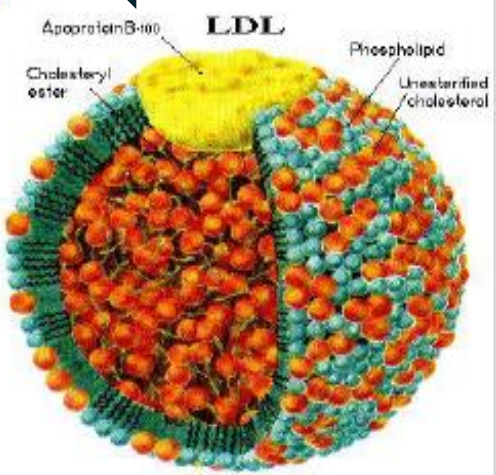
✓ **Site of synthesis:** in blood from VLDL.

✓ **Composition:**

- Lipid core (TG 13% and CE 48%).
- Outer layer of phospholipids (28%) and cholesterol.
- Apolipoprotien, mainly **apoB-100** and **apoE** (20%).

✓ **Function:** transport

cholesterol from liver to extrahepatic tissues.



Lipoprotein metabolism

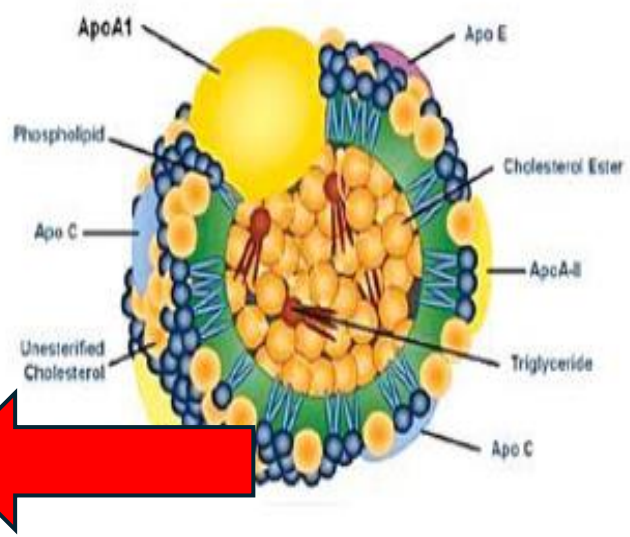
HDL
(α-lipoproteins)

✓ **Site of synthesis:** liver and intestine.

✓ **The smallest lipoproteins.**

✓ **Composition:**

- Lipid core (TG 3% and CE 15%).
- Outer layer of phospholipids (25%) and cholesterol.
- Apolipoprotien, mainly **apoA** (50%).



Lipoprotein metabolism

HDL
(α-lipoproteins)

✓ **Functions:**

1. A source of apoC and apoE required in the metabolism of chylomicrons and VLDL.
2. Uptake of free cholesterol from extrahepatic tissues and esterifying it using lecithin cholesterol acyl transferase (LCAT) enzyme, activated by apoA1.
3. Carry cholesterol ester to liver (reverse cholesterol transport).

$$\text{cholesterol} + \text{lecithin} \xrightarrow{\text{LCAT}} \text{cholesteryl ester} + \text{lysolecithin}$$

Hyperlipidemia

Causes of fatty liver

Causes

1. ↑ Fat in diet→ ↑ uptake by liver if uptake > mobilization by VLDL

   ↑ Fatty liver

2. ↑ CHO diet →

   First CHO → stored as glycogen If excess → lipogenesis

   If lipogenesis > VLDL synthesis → fatty liver

3. ↓ FA oxidation → direct FA → TG

4. ↑ Mobilization of Fat from adipose tissue to liver as in

   1-↓ CHO diet 2- DM 3- Starvation

5. ↓ Mobilization of fat from liver to adipose tissues due to ↓ apo B_{100} →↓ VLDL → Accumulation of TG in liver due to impaired lipid transport form liver.

6. Liver poisons: chloroform, Puromycin, CCL_4→↓ Apo proteins

Hyperlipidemia

Examples

1. Choline → lecithin, plasmalogen, sphingomyelin
2. Methionine → choline synthesis (-CH_3)
3. Folic acid & Vit B_{12} : transfer -CH_3 gp → choline
4. Insoitol → Phosphatidyl inositol
5. Essential FA → FA No 2 in phospholipids
6. Glycine → choline
7. Betaine → choline
8. Vit E & selenium → ↓ FA peroxidation
9. Adequate diet → proteins– vitamins

Lipotropic factors

- substances which facilitate mobilization of fat from liver = prevent fatty liver

Mechanism :

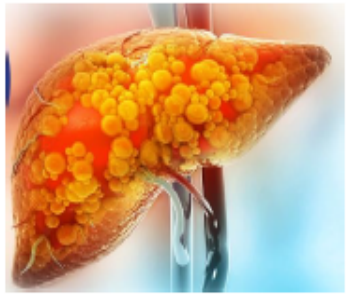
- Fat to be mobilized from liver must be in the from of phospholipid
- So lipotropic factors help phospholipids synthesis

Hyperlipidemia

Hypolipoproteinemias

1- Abetalipoproteinemia:

- Defect in lipid transport to apoB.
- ↓ or absent chylomicrons, VLDL and LDL.
- ↓ plasma lipids.
- Accumulation of TG in the intestine → Steatorrhea, and liver → fatty liver.



2-Hypobetalipoproteinemia:

- Gene mutation → ↓ formation of apoB.
- ↓ chylomicrons, VLDL and LDL.
- ↓ plasma lipids.

Hyperlipidemia

Hypolipoproteinemias

3-Familial α-lipoprotein deficiency (Tangiers disease):

- Gene mutation affects the efflux of cholesterol from the cells.
- ↓ or absent HDL and apoA.
- Accumulation of CE in tissues.

4-Familial LCAT deficiency:

- Genetic mutation in LCAT → absent enzyme.
- ↓↓ HDL (remains nascent).
- Accumulation of free cholesterol in tissues.

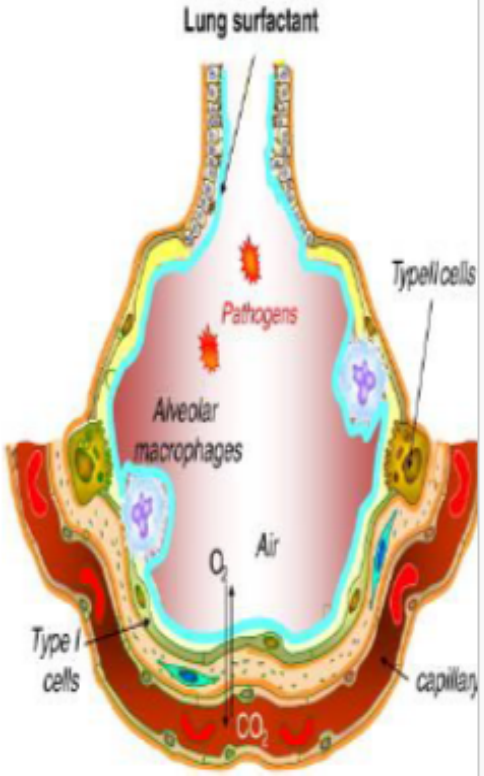
cholesterol + lecithin → cholesteryl ester + lysolecithin

Lung Surfactant

Lung Surfactant

❖ Definition:

- ✓ Lung surfactant is a surface acting material (absorb to the surface) that lowers the surface tension in alveoli.
- ✓ It coats alveoli and small airways to reduce surface tension over the air water interface→ prevents the alveolar collapse during exhalation.



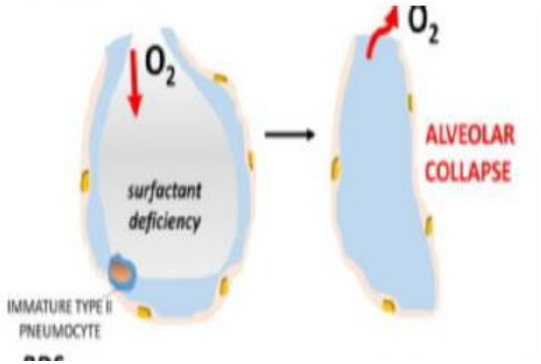
- ➢ Site of secretion: type II alveolar cells.

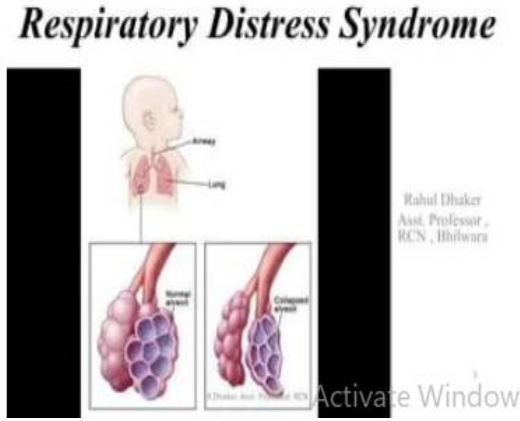
Lung Surfactant

Surfactant insufficiency

- Insufficiency of lung surfactant in preterm newborn

→Respiratory distress syndrome (RDS)→ neonatal death





Surfactant Insufficiency

- How to manage Respiratory distress syndrome (RDS)?

- ✓ **Shortly before delivery:** Glucocorticoids are given to mother → accelerate lung maturation.
- ✓ **Post natal:** Administration of natural or synthetic surfactant to newborn →prevent and treat infant RDS.

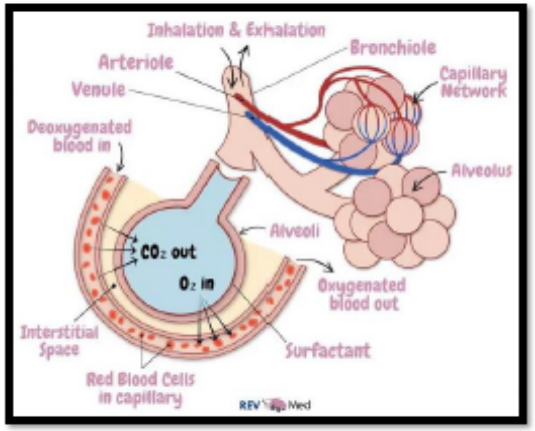
Metabolic functions of the respiratory system

Functions of the respiratory system

Respiratory Function

↓

Gas exchange



Non-respiratory functions

- Defense mechanism
- Olfaction (sensation of smell) and Phonation
- Maintenance of water balance
- Regulation of body temperature
- Regulation of acid-base balance
- Metabolic and endocrine functions

Metabolic functions of the respiratory system

Metabolic Functions of the respiratory system

1. Act as an effective biochemical filter.
2. Endocrine function.
3. Metabolism of biologically active substances.
4. Lipid metabolism and pulmonary surfactant.
5. Collagen and Elastin protein synthesis.
6. Drug metabolism.

Metabolic functions of the respiratory system

1. Biochemical filter

- **Lungs act as an effective biochemical filter:**

1. They **protect** the systemic circulation from exposure to high levels of circulating vasoactive compounds.
2. They also **regulate** systemic arterial concentrations of exogenous substances.

Metabolic functions of the respiratory system

4. Lipid metabolism and pulmonary surfactant

1. De novo synthesis of fatty acids
2. Fatty acid oxidation
3. Lipid esterification.
4. Hydrolysis of lipoprotein
5. Synthesis of phospholipids especially phosphatidylcholine.
6. Synthesis and secretion of PG and other eicosanoids
7. Synthesis and secretion of lung surfactant.

Metabolism of non protein nitrogenous compounds

Def of azotemia

Non Protein Nitrogenous Compounds

A stepwise increase in three **nitrogenous components** (uric acid, urea, creatinine) in blood is believed to reflect a deteriorating kidney function.

Azotemia

Increase in the blood levels of NPN (creatinine, urea, uric acid) is referred to as **azotemia** & is the hallmark of kidney failure.

Metabolism of non protein nitrogenous compounds

- **Urea, uric acid** and **creatinine** are **excreted in urine.**
- Their **serum concentrations** can be used as **markers of renal function** because the serum concentration **increases** as renal function deteriorates.
- **Creatinine is a better marker of renal function than urea** because its blood concentration is **not significantly** affected by **non-renal factors**, thus making it a **specific indicator of renal function.**
- A number of "pre-renal" and "post-renal" factors significantly increase blood urea levels.

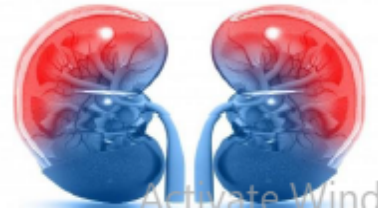
Metabolism of non protein nitrogenous compounds

Creatinine clearance test

- Creatinine is filtered at the glomerulus, but is neither secreted not reabsorbed by the tubules. So, its clearance gives the GFR.

- <u>This is a convenient method for estimation of GFR since:</u>

1) It is a normal metabolite in the body.
2) It does not require the intravenous administration of any test material.
3) Estimation of creatinine is simple.



Role of the kidney in vitamin D and calcium metabolism

Calcium

Vitamin D and calcium are essential for:

1. Bone health and mineralization
2. Muscle contraction and nerve transmission
3. Overall metabolic homeostasis

Calcium is also essential for:

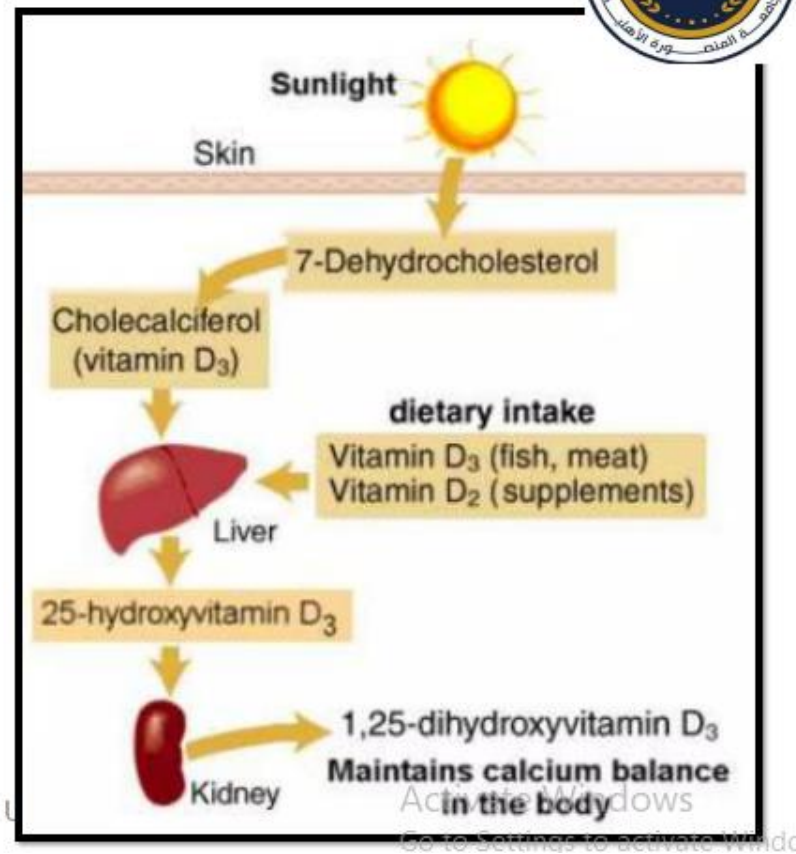
Blood clotting, hormone and enzyme action.

Role of the kidney in vitamin D and calcium metabolism

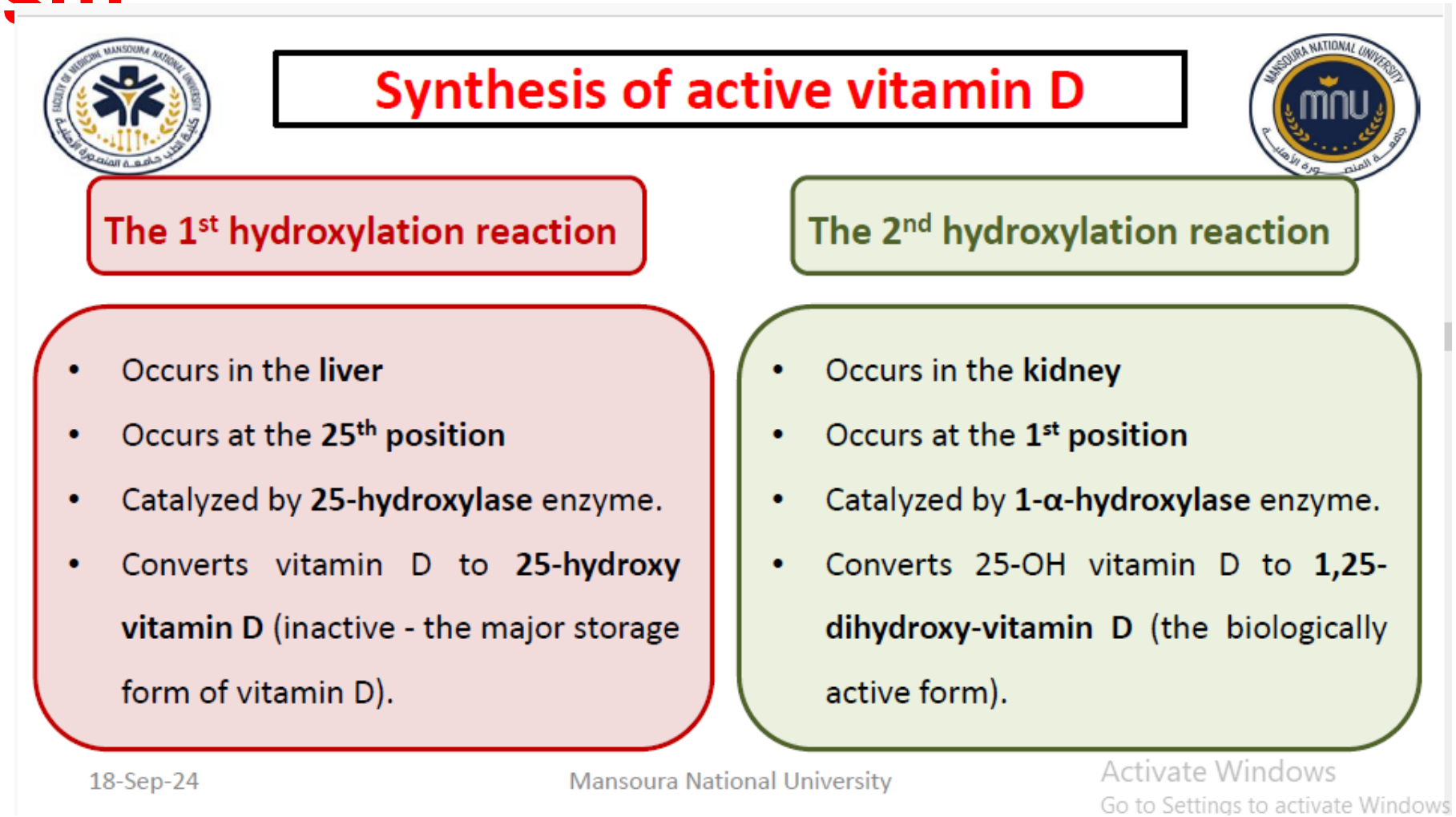
Synthesis of active vitamin D

Sources of vitamin D:

1. Sunlight (UV-B exposure): Converts 7-dehydrocholesterol (found in the skin) to vitamin D3 (cholecalciferol).
2. Diet: Provides both vitamin D2 (ergocalciferol) and D3 (cholecalciferol)



Role of the kidney in vitamin D and calcium metabolism



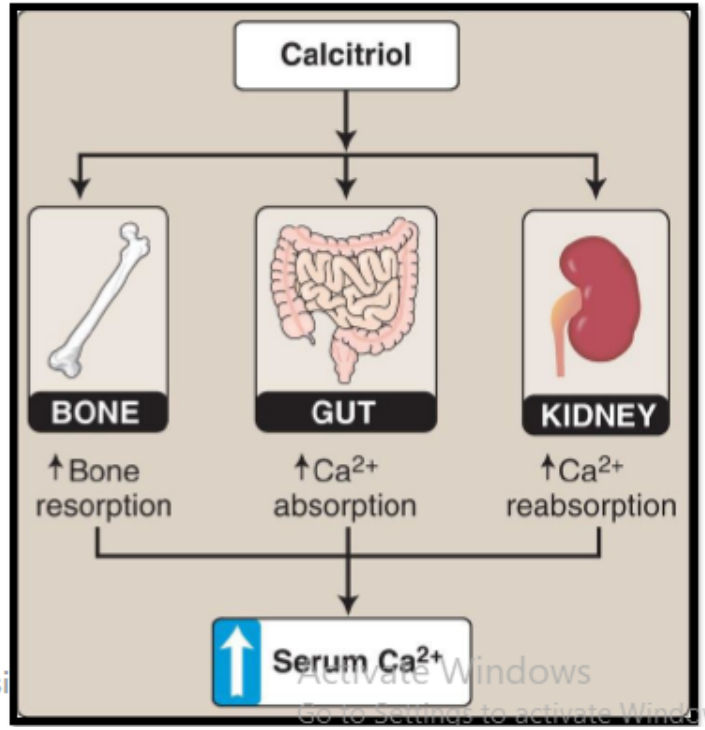
Role of the kidney in vitamin D and calcium metabolism

Function of vitamin D

- The principal function of vitamin D is to maintain plasma calcium concentration.

Vitamin D achieves this in three ways:

1. It increases intestinal absorption of calcium
2. It increases renal calcium reabsorption.
3. It increases calcium mobilization (resorption) from the bone.



Role of the kidney in vitamin D and calcium metabolism

Role of kidney in regulation of vitamin D and calcium metabolism

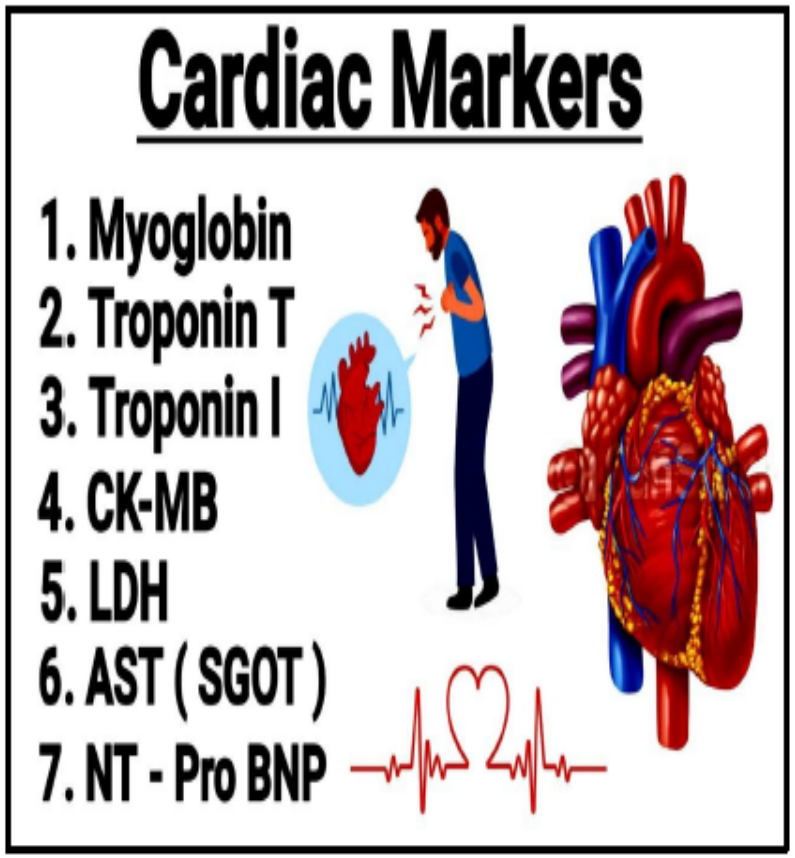
- Production of calcitriol in the kidneys is tightly regulated based on the body's needs for calcium and phosphate.
- The main site of vitamin D regulation is at the 1-α-hydroxylase enzyme in the kidney.
- <u>Several factors influence the activity of 1-α-hydroxylase:</u>

1. Parathyroid hormone (PTH).
2. Fibroblast Growth Factor 23 (FGF23).
3. Calcitriol Feedback Inhibition.


PRACTICAL

Cardiac biomarkers

Types of cardiac biomarkers



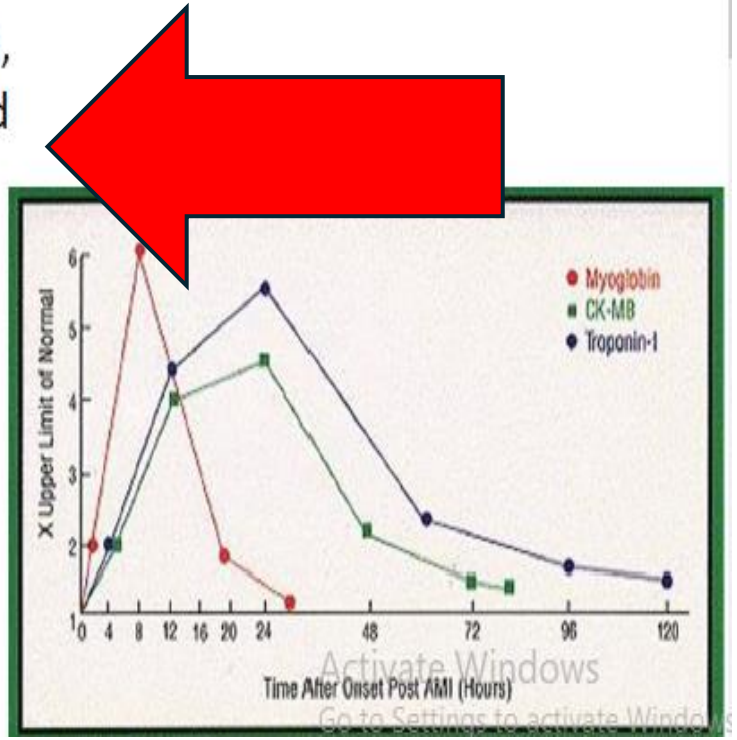
Include:

- Cardiac Enzymes
  - *1) Creatine Kinase (CK-MB)*
  - *2) Lactate dehydrogenase (LDH)*
  - *3) Aspartate transaminase (AST)*
- Cardiac proteins
  - *1) Myoglobin*
  - *2) Troponin (T and I) : current test of choice for cardiac damage*

Cardiac biomarkers

1- Cardiac troponin

- Troponin is a protein found in cardiac and skeletal muscles that plays a role in muscle contraction.
- Troponin I (TnI) and troponin T (TnT) are specific and sensitive biomarkers of cardiac muscle injury.
- They enter the blood soon after a heart attack, elevated within **3 - 4 hours**, peak in **12-24 h.**, and remain elevated in blood for **5 to 10 days.**
- Troponin **"I"** is expressed only in cardiac muscles, so it is highly specific for **MI.**
- Other possible causes of high troponin levels:
  - Renal failure.
  - Sepsis.
  - Pulmonary embolism.



Cardiac biomarkers

2- Myoglobin

- Myoglobin is an O2 binding protein found in cardiac and skeletal muscles ,used as an early biomarker of acute MI.

- ✓ Start to be released in **1-2 h.** after MI.
- ✓ Useful **in** early detection of MI**.**
- ✓ Peak within **8 – 12 hours**
- ✓ Persists for (return to normal after) **1-2 days.**

- It is not specific for cardiac necrosis as its level may be elevated in :
  - skeletal muscle diseases.
  - Chronic renal disease.

- Due to its rapid clearance, it is used to diagnose re-infarction when t
  elevated.

Cardiac biomarkers

Isoenzyme	Increases in	Shape
CK BB	Brain tumors	
CK MB	Heart diseases	
CK MM	Skeletal muscle diseases	

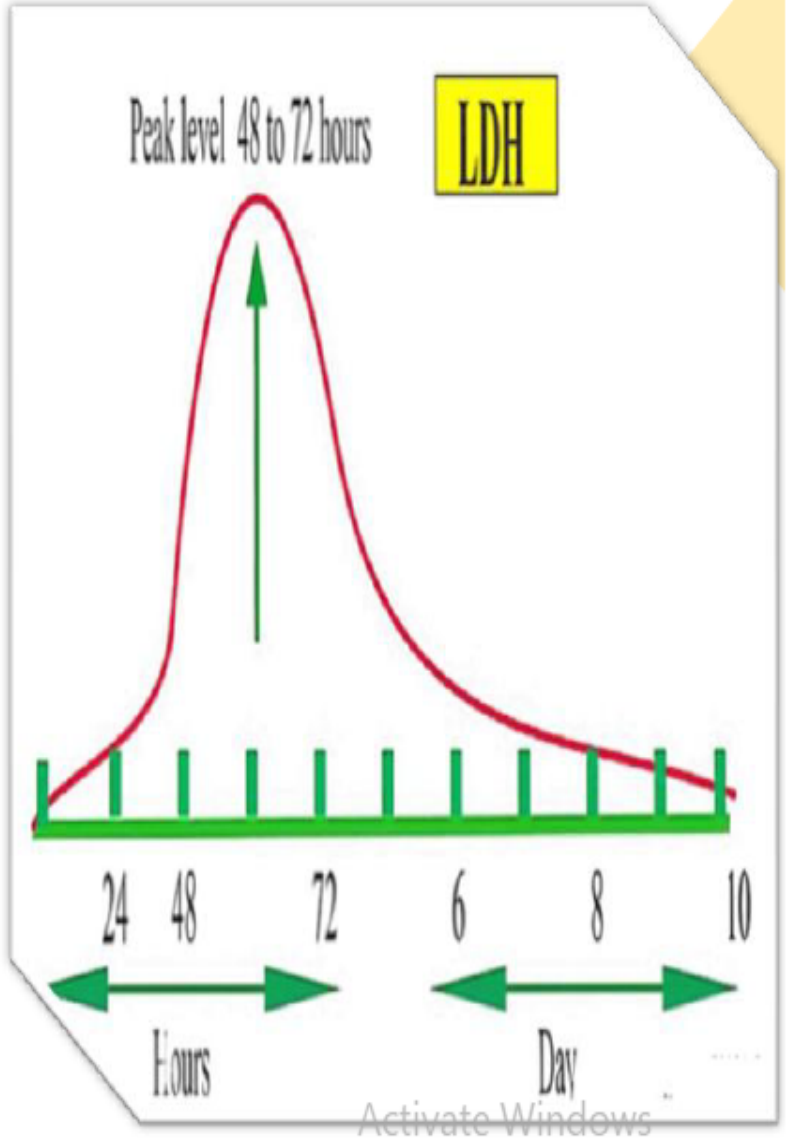
CK-MB

- **CK-MB:** This is iso-enzymeof CK which is more sensitive for heart damage.
- CK-MB **rises** **4 to 6 hours** after a heart attack, **peak** within **10 – 24 hours**, but it is generally **back to normal** in **2 - 3 days**.
- Since it has a **short** duration, it cannot be used for late diagnosis of acute MI but can be used to suggest **re-infarction or infarction extension** if its levels rise again.

Cardiac biomarkers

A rise in LDH-1 is highly significant in diagnosis of MI :

- Normally, concentration of LDH-1 is lower than LDH-2 in the blood, but after myocardial infarction, LDH-1 becomes elevated and exceeds the concentration of LDH-2 (increased LDH-1: LDH-2 ratio or *LDH-1/LDH-2 flip*) which suggests myocardial infarction.
- LDH isoenzyme levels start to increase **12-24 hours** following myocardial infarction, reach a peak in **2-3 days** and remain elevated for **8 to 14 days**, making it a late marker for myocardial infarction.



Cardiac biomarkers

- Lactate is Late.

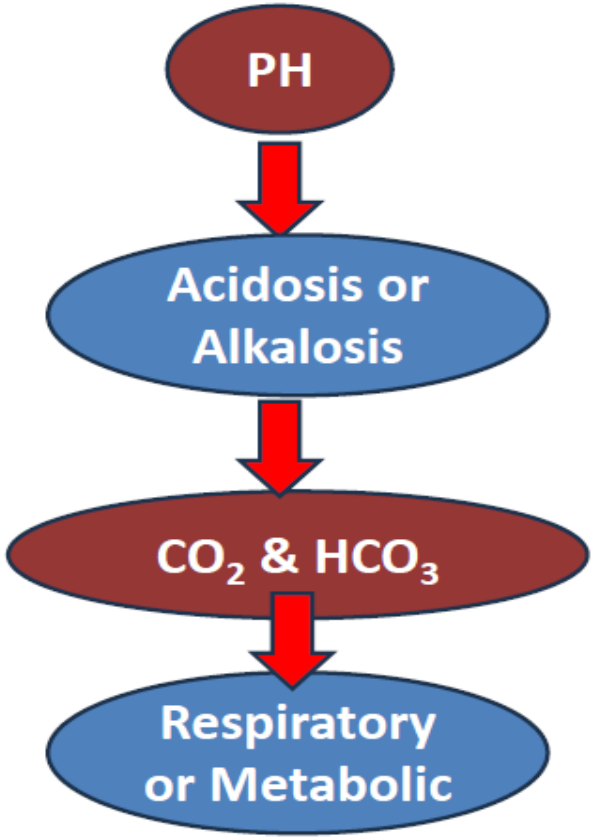
Late cardiac biomarker:
- Lactate dehydrogenase-1

- Ima Makes Cake early in the morning.

Early cardiac biomarkers:
- Myoglobin
- Creatine Kinase

ABG

Arterial Blood Gases (ABG)



Mansoura National University

Arterial Blood Gases (ABG)

Normal ABG values:

- pH: 7.35-7.45
- PaO_2: 75-100 mm Hg
- $PaCO_2$: 35-45 mm Hg
- HCO_3: 22-28 mEq/L
- SaO_2: 95-100%

ABG

Arterial Blood Gases (ABG)

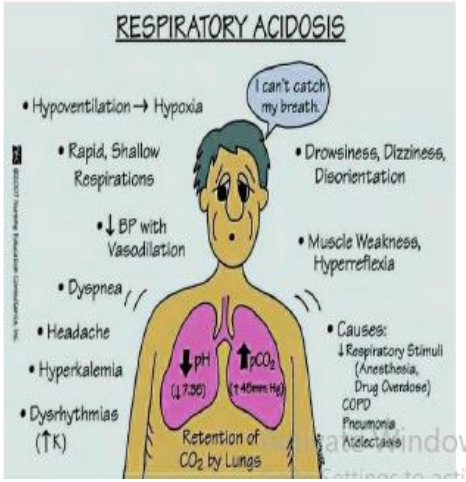
Respiratory Acidosis

A condition where pH is decreased (<7.35) due to accumulation of CO_2.

Causes:

Anesthesia, drug overdose,

COPD, pneumonia, atelectasis.



Arterial Blood Gases (ABG)

Respiratory Alkalosis

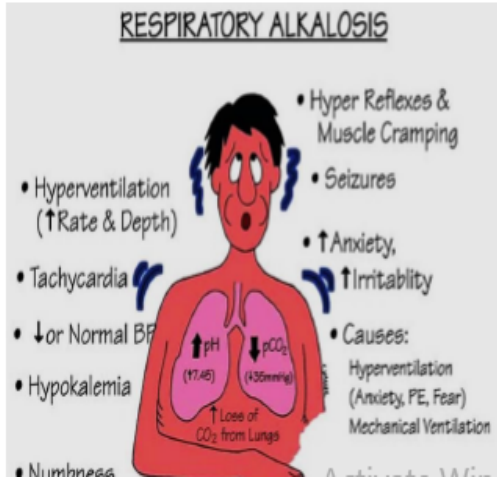
A condition where pH is increased (>7.45) due to hyperventilation (increased CO_2 removal from the lungs).

Causes:

Anxiety, fear,

pulmonary embolism,

mechanical ventilation.



ABG

Arterial Blood Gases (ABG)

Metabolic Alkalosis

A condition where pH is increased (>7.45) due to increased HCO_3^- (>28 mEq/L).

Causes:

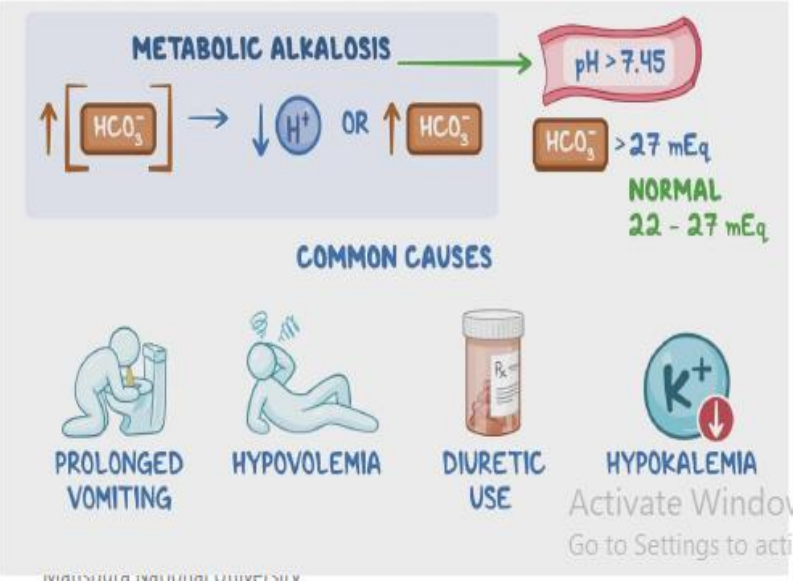
Vomiting,

NG suction,

diuretics,

antacids,

hypokalemia.



Arterial Blood Gases (ABG)

Metabolic Acidosis

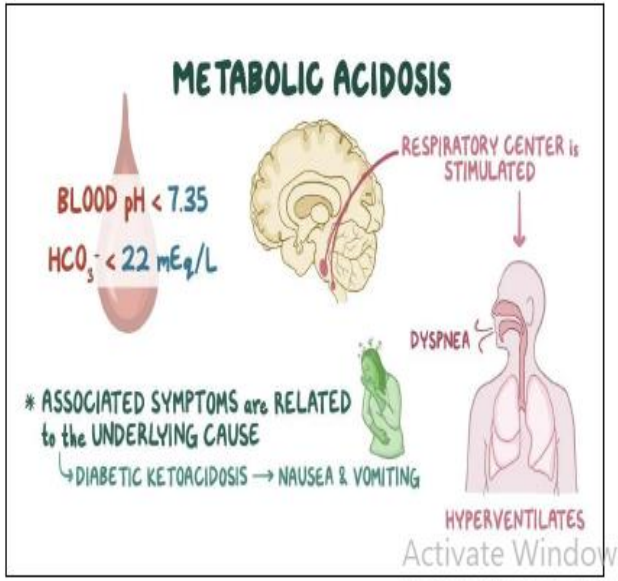
A condition where pH is decreased (<7.35) due to decreased HCO_3^- (<22 mmHg).

Causes:

DKA, lactic acidosis,

Renal tubular acidosis,

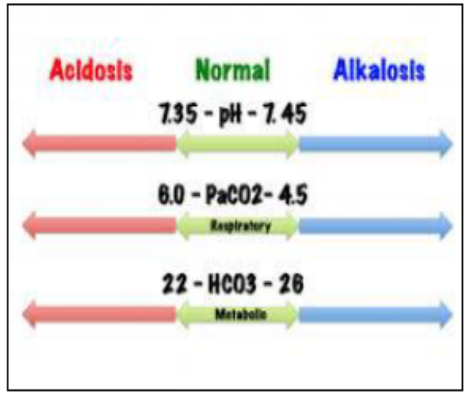
diarrhea.

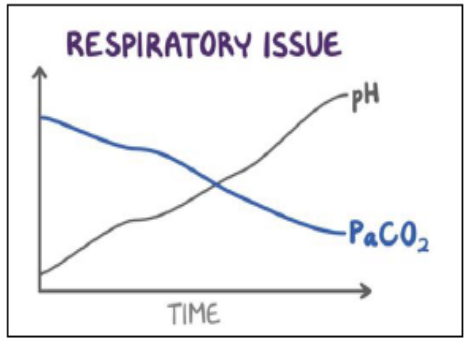


ABG

Interpretation of ABG

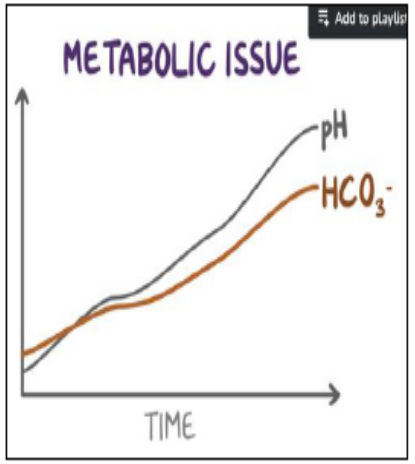
1. Look at the PH.
2. Look at the $PaCO_2$ & HCO_3^- to determine if it is due to a respiratory or renal problem.
   - If the cause is respiratory (due to change in CO_2) → $PaCO_2$ will move in the opposite direction to the pH.
   - HCO_3^- may also move in the opposite direction as a compensation.





Interpretation of ABG

- If the cause is metabolic (due to change in HCO_3^-)→ HCO_3^- will move in the same direction as the pH.
- $PaCO_2$ may also move in the same direction in an attempt at compensation.



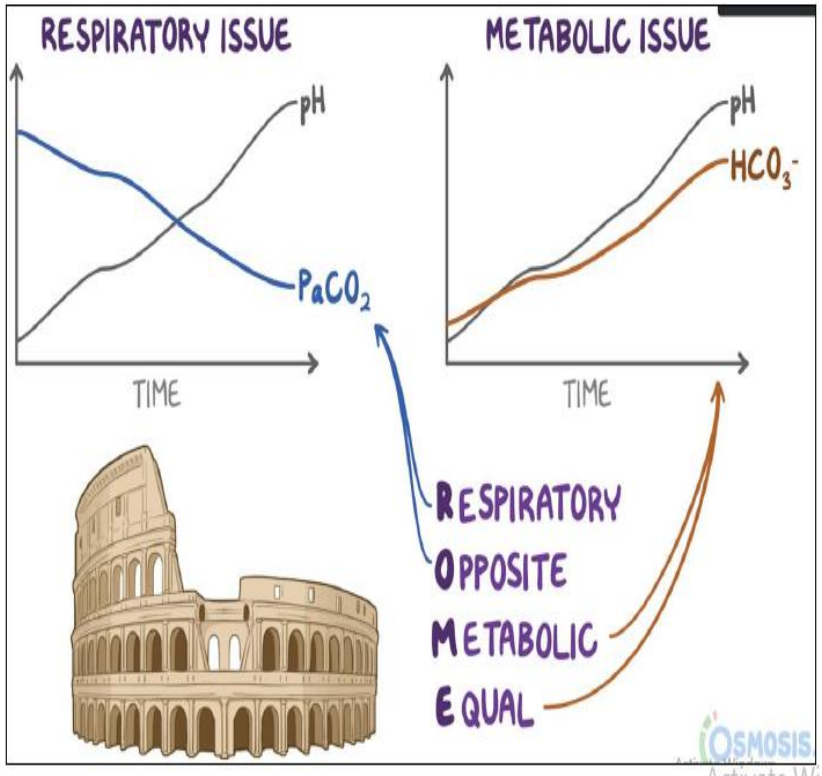
ABG

Interpretation of ABG

Whether the acid-base disorder is in the form of respiratory or metabolic, acidosis or alkalosis, it can be in one of 3 forms:

Uncompensated	The compensatory mechanism hasn't made concrete corrective steps yet, the pH is not normal (abnormal pH, only one of $PaCO_2$ & HCO_3^- is changed)	
Partially compensated	The compensatory mechanism has kicked in but has not managed to correct the pH (abnormal pH, both $PaCO_2$ & HCO_3^-)	If both $PaCO_2$ & HCO_3^- are changed, they always move in the same direction in non-complex scenarios. Either both ↑ ↑ Or both ↓ ↓
Compensated	The pH has returned to normal due to the effectiveness of the compensatory mechanism (normal pH, abnormal both $PaCO_2$ & HCO_3^-)	

Interpretation of ABG



ABG

Case scenario (Clinical correlate)

A 24-year-old female patient arrived at the emergency department with a complaint of shortness of breath. She had a history of anxiety and had been unable to pay for her medications this month. On examination her lungs were clear, and she was not feverish, but she had tachycardia. The physician seeing to her suspected anxiety-induced hyperventilation, so he ordered an ABG to assess for changes in the acid-base balance. Her ABG report was as follows:
pH= 7.48, $PaCO_2$= 30mmHg, HCO_3^-= 24 mEq/L.

Q: What is the type of acid-base imbalance here?

Uncompensated respiratory alkalosis

ABG

Case scenario (Clinical correlate)

A 72-year-old woman presents to the emergency department with a three-day history of vomiting and diarrhea. Her medical history is significant for hypertension which is typically well-controlled with hydrochlorothiazide. She reports no fever, chest pain or difficulty breathing. Temperature is 37.0 °C, heart rate is 108/min, respiratory rate is 12/min, blood pressure is 96/60 mmHg and oxygen saturation is 100% on room air. An ABG was ordered for her showing the following:

pH= 7.45, $PaCO_2$= 48 mmHg, HCO_3^-= 31 mEq/L.

Q: What is the type of acid-base imbalance here?

Fully Compensated metabolic alkalosis

ABG

Case scenario (Clinical correlate)

Patient:	Case 13		
Age:	3 years		
Sample type:	arterial		
Blood gas values			
pH	7.48		[7.35 - 7.45]
pCO_2	3.33	kPa	[4.5 - 6.0]
pO_2	14.7	kPA	[9.5 - 14.0]
Acid-base status			
HCO_3^-	18	mmol/l	[22.0 - 30]
BE	0.9	mmol/l	[-2 to +2]

Partially Compensated respiratory alkalosis

ABG

Case scenario (Clinical correlate)

Patient:	Case 8		
Age:	5 years		
Sample type:	arterial		
Blood gas values			
pH	7.3		[7.35 - 7.45]
pCO_2	4.9	kPa	[4.5 - 6.0]
pO_2	22.4	kPA	[9.5 - 14.0]
Acid-base status			
HCO_3^-	16	mmol/l	[22.0 - 30]
BE	-4.5	mmol/l	[-2 to +2]

Uncompensated metabolic acidosis

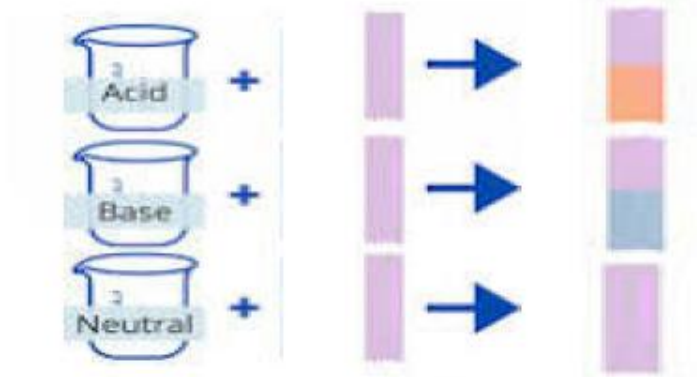
PH

A purple litmus paper changes color from violet to:

red when exposed to an **acid.**

blue when exposed to an **alkaline.**

No change means that solution is **neutral**



Be careful. Do not dip the whole paper in solution

PH

Advantages and limitations of litmus paper

- Although use of litmus paper is **simple** and **quick** but it **doesn't** determine PH as **numerical value**, it just provide simple color change which tells that solution is acidic, alkaline or neutral (**Qualitative**).

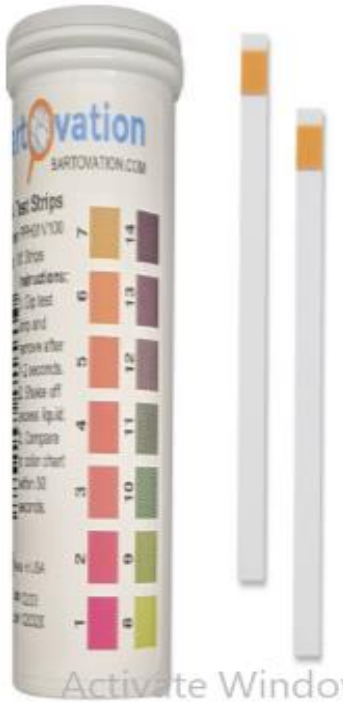
$NaOH \cdot H_2O$

H_2SO_4

PH

Advantage and limitations of PH test strips

- ✓ **Simple** and **quick** method.
- ✓ A pH test strip is a **semi-quantitative** test (not exact pH value).
- ✓ This semi-quantitative method is generally enough to indicate if the body have problem or not.



PH

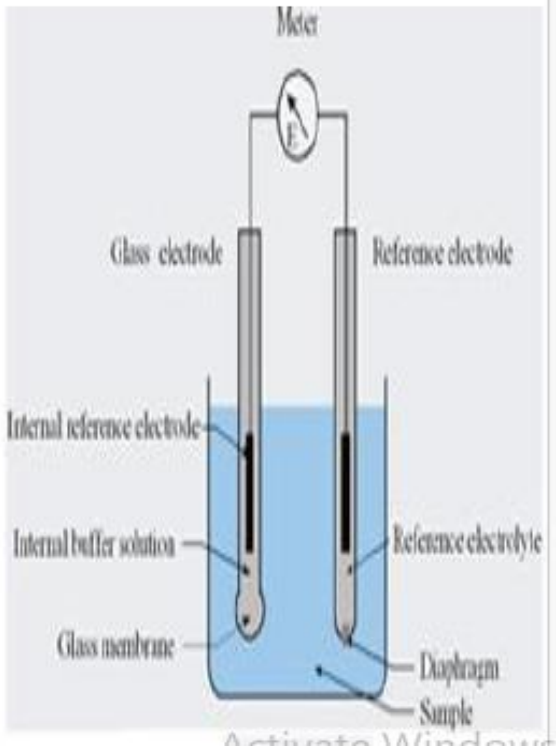
PH meter

A pH meter is an instrument used to measure hydrogen ion activity in solutions to determine acidity/alkalinity of a solution.

PH

Components of PH meter

1) **PH electrodes:** that actually measures the pH of the solution.
2) **Meter:** that displays the pH reading.
3) **Temperature probe:** Some pH meters also have a temperature probe to measure the temperature of the solution as the pH reading can be affected by temperature.
4) **Power source.**
5) **Sample chamber:** where the solution is placed, and the electrodes are inserted into it.



PH

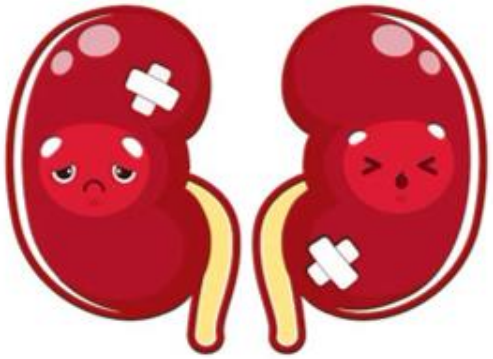
The body fluid PH

1- Blood: between 7.35 and 7.45
2- Stomach: 1.5 to 3.5.
3- Saliva:6.5 to 7
4- Urine: 4.5 to 8 (average = 6.5)

Renal function test

Increased serum creatinine

- Impaired renal function
- Very high protein diet
- Very large muscle mass: (Athletes, acromegaly)
- Rhabdomyolysis/crush injury
- Drugs: Trimethoprim, Probenecid



Metabolism of non protein nitrogenous compounds

- **Urea, uric acid** and **creatinine** are **excreted in urine.**
- Their **serum concentrations** can be used as **markers of renal function** because the serum concentration **increases** as renal function deteriorates.
- **Creatinine is a better marker of renal function than urea** because its blood concentration is **not significantly** affected by **non-renal factors**, thus making it a **specific indicator of renal function.**
- A number of "pre-renal" and "post-renal" factors significantly increase blood urea levels.

Urine analysis

Why it's done?

Urine analysis

Urine analysis is a simple and non-invasive test that is done for several reasons:

1. To **check your overall health**.
2. To **screen for** or **monitor** certain common health conditions such as diabetes, kidney disease and liver disease.
3. To **diagnose urinary tract infections (UTIs)**.

N.B: Other tests such as **pregnancy testing** and **drug screenings**, might rely on a urine sample, but these tests look for substances that **aren't included** in a typical urine analysis.

Urine analysis

1- Volume

Normal urine volume: 1- 2.5 L/day.

Polyuria:

> 2.5 L/day.

Causes:

1.Physiological

Increased water ingestion

2. Pathological

Diabetes Mellitus

Diabetes Insipidus

Oliguria:

< 400ml/day.

Causes:

1.Physiological

Decreased water ingestion

2.Pathological

Dehydration

Acute glomerulonephritis

Renal failure

Anuria:

< 100ml/day.

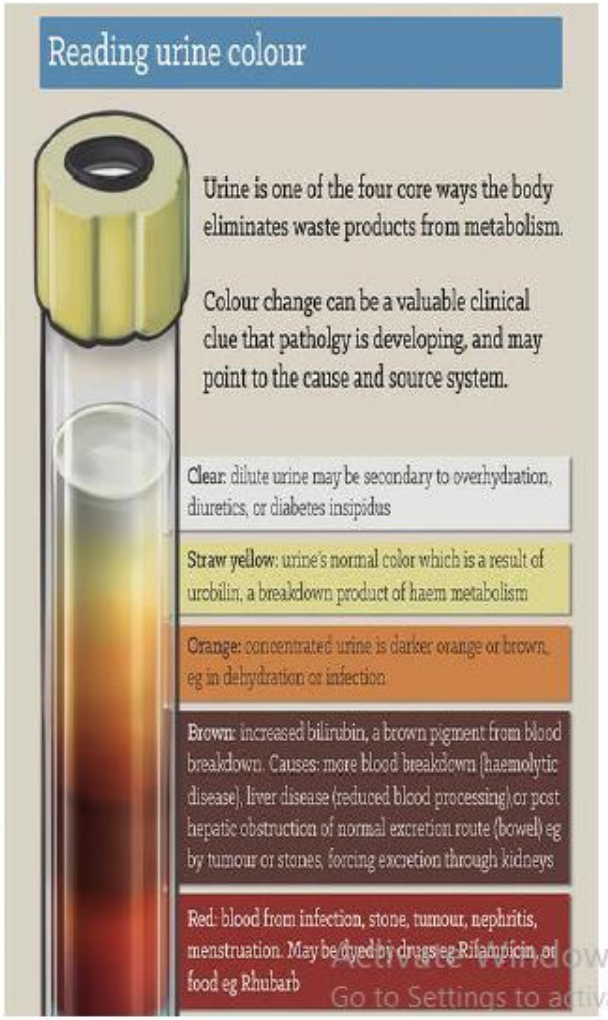
Causes:

Acute renal failure

Urine analysis

2- Color

Normal	Pale yellow (Amber yellow) (due to Urobilin)	
Colorless	• Overhydration • Diuretics • Diabetes insipidus	
Orange	Concentrated urine • Dehydration • Infection	
Brown	Hemolytic diseases Liver diseases Bile duct obstruction	
Reddish	Blood (Hematuria)	



Urine analysis

3- Aspect

Clear

Normal urine

Glucose (Glucosuria)

Acetone (Ketonuria)

Turbid

Protein (Proteinuria)

Pus (Pyuria)

Blood (Hematuria)

Bile (Choluria)

Urine analysis

4- Odor

Normal odor	**Faint aromatic**
On long standing	**Ammoniacal** (Urea decompose to ammonia)
Abnormal odor	**Foul or Offensive** Pus or infection
	Acetone odor Diabetes Mellitus
	Characteristic (protein –blood –bile)

Urine analysis

5- pH

Normal urine pH: ranges from **4.5 to 8.0**.

Average urine pH: **6.0** (slightly acidic).

Tested by: 1. Litmus paper

2. **Urine dipsticks**

Litmus paper

Dipsticks

Acidic urine (<4.5)	Alkaline urine (> 8.0)
1. Metabolic acidosis.	1. Metabolic alkalosis.
2. Respiratory acidosis.	2. Respiratory alkalosis.
3. Ketosis (DM, starvation).	3. Urinary tract infection (UTI).

Urine analysis

6- Specific gravity (SG)

Urine Specific Gravity = Density of Urine / Density of distilled H_2O

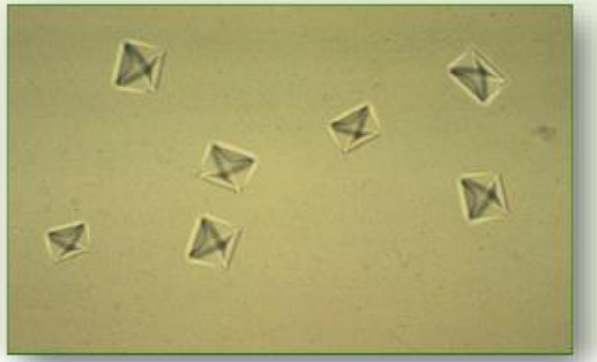
- It is a measure of **the total concentration of all chemical particles** in the urine.
- Main contributors to specific gravity of urine are **urea** and **sodium**
- It provides information on the **ability of the kidneys to concentrate the urine.**
- It is measured by **urine dipsticks**.
- Normal S.G.: 1.005 - 1.030.

Increase in S.G.	Decrease in S.G.
1. Low water intake 2. Diabetes mellitus 3. Proteinuria	1. High water intake 2. Diabetes insipidus 3. Renal tubular damage 4. Renal failure

Urine analysis

Calcium oxalate crystals

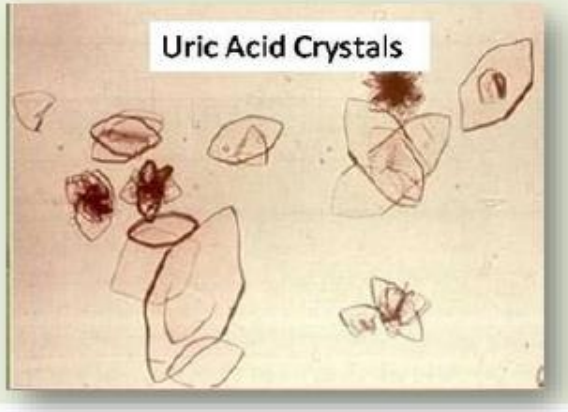
- The most common type.
- **Shape:** Square with a characteristic "X" mark (Envelop).
- **Found in**: acidic urine.
- **Significance:**
  - ✓Found in **low urine volume**
  - ✓Associated with **high oxalate** intake (foods as spinach)
  - ✓Commonly form **kidney stone**

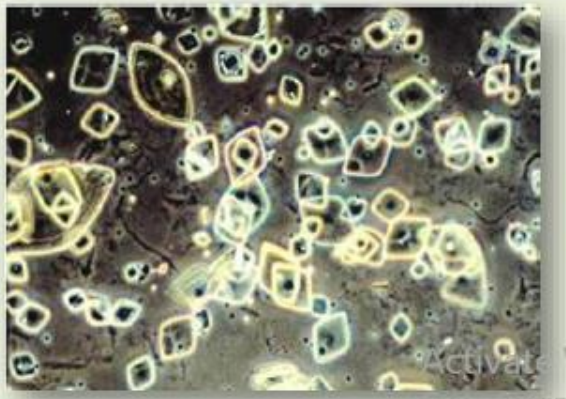


Urine analysis

Uric acid crystals

- **Shape:** Needle shaped diamond or wedges.
- **Found in:** acidic urine.
- **Significance:**
  - ✓In normal urine: caused by a purine-rich diet due to increase in uric acid in urine (hyperuricosuria).
  - ✓In disease : Gout, kidney stone, chemotherapy.

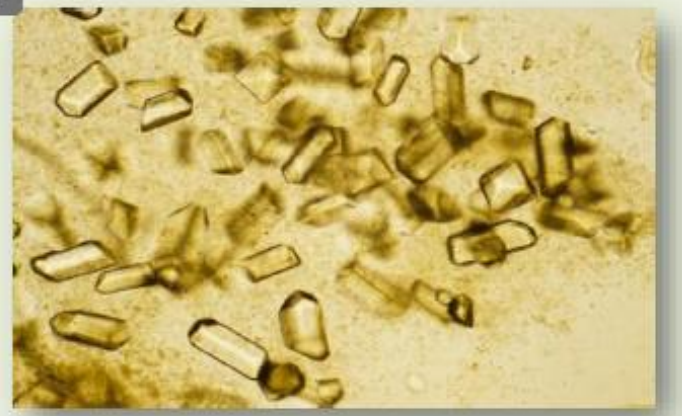


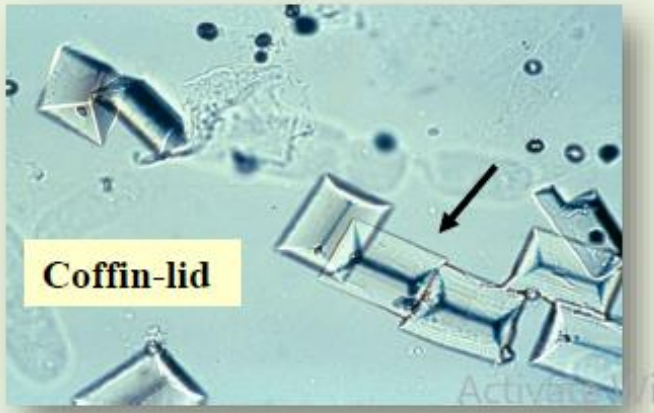


Urine analysis

Triple Phosphate crystals

- **Shape:** rectangular prisms, coffin-lid appearance.
- **Found in:** alkaline urine.
- Also called struvite crystals.
- **Composed** of magnesium, ammonium and phosphate.
- **Significance:**
  - ✓ Can indicate **urinary tract infection (UTI).**





Urine analysis

Cystine crystals

- **Shape:** hexagons.
- **Found in:** acidic urine.
- **Significance:**
  - ✓ Can indicate **cystinuria** (there is a defect in proximal tubular reabsorption of cystine).