Interpretation of Laboratory data

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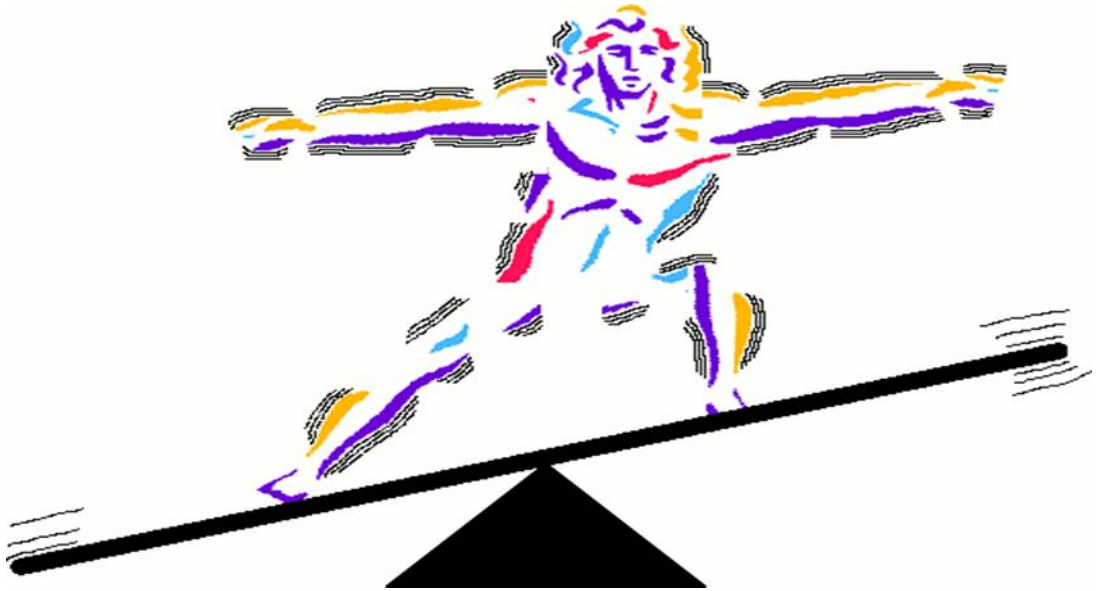
BIOCHEMICAL DATA

- The homeostasis of various elements, water and acid–base balance are closely linked, both physiologically and clinically.

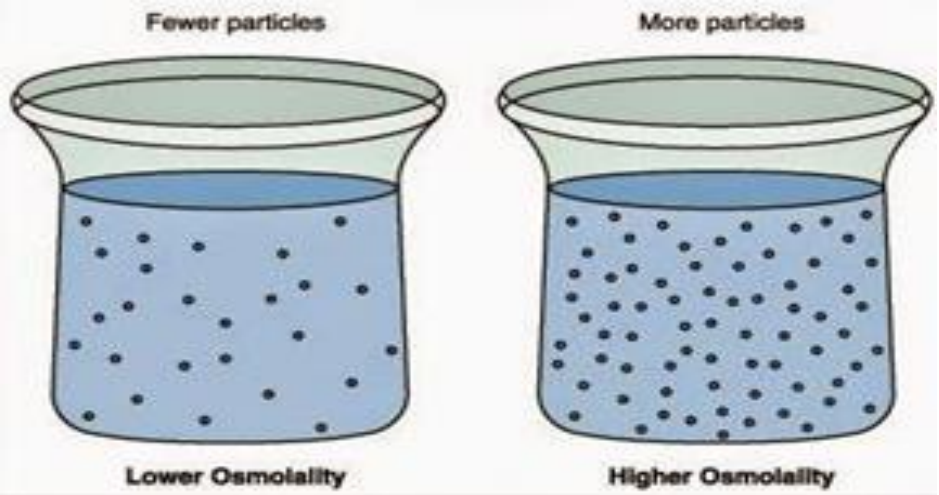
- Standard biochemical screening includes several measurements which provide a picture of fluid and electrolyte balance and renal function.



Sodium and water balance

- Sodium and water metabolism are closely interrelated both physiologically and clinically, and play a major role in determining the osmolality of serum.

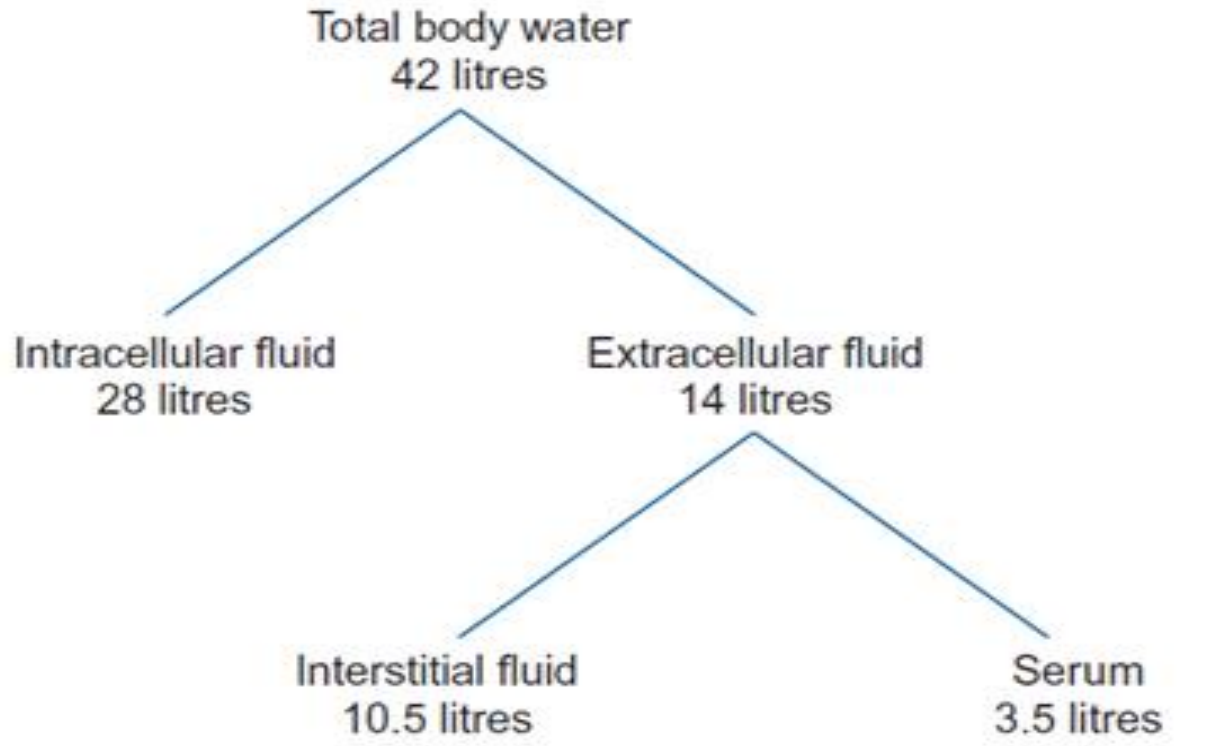
- A serum osmolality test measures the amount of chemicals dissolved in the liquid part (serum) of the blood. Chemicals that affect serum osmolality include sodium, chloride, bicarbonate, proteins, and sugar (glucose).



Sodium and water balance

- Water constitutes approximately
  - **60%** of body weight in **men**
  - **55%** of body weight in **women** (women have a greater proportion of fat tissue which contains little water)
- Approximately
  - **two-thirds** of body water is found in the **intracellular fluid (ICF)**
  - **one-third** of body water in the **extracellular fluid (ECF)**
- Of the ECF **75%** is found within interstitial fluid and 25% within serum.

Sodium and water balance



Approximate distribution of water in a 70 kg man.

Sodium and water balance

- In general, water permeates freely between the ICF and ECF.
- Water movement from one compartment to the other is controlled by osmotic pressure: water moves into the compartment with the higher osmotic concentration.
- The osmotic content of the two compartments is generally the same, that is, they are **isotonic**, which ensures normal cell membrane integrity and cellular processes.
- The kidneys are an exception to the rule.

Sodium and water balance

- The osmolality of the ECF is largely determined by **Sodium , Chloride,** and **Bicarbonate**
- Glucose and urea have a lesser, but nevertheless important, role in determining ECF osmolality.
- Protein, especially albumin, makes only a small (0.5%) contribution to the osmolality of the ECF but is a major factor in determining water distribution between the two compartments.
- The contribution of proteins to the osmotic pressure of serum is known as the **colloid osmotic pressure** or **oncotic pressure**.
- The major contributor to the osmolality of the ICF is **potassium**.

Water depletion

- Water depletion will occur if intake is inadequate or excessive loss of water.

- Excessive loss of water through the kidney is unusual except in
  - **diabetes insipidus**
  - **overuse of diuretics**
  - **fever**
  - **diarrhea & sever vomiting**

- Severe water depletion may induce cerebral dehydration causing confusion, fits, coma and circulatory failure.

Water depletion

- The underlying cause for the water depletion should be identified and treated.

- Replacement water should be given orally, nasogastric tube, intravenously or subcutaneously as necessary with 5% D\W or, in patients with associated sodium deficits, isotonic saline.

- **Hypernatraemia** should be corrected slowly: not more than half of the water deficit should be corrected in the first 12–24 h.

Water excess

- Water excess is usually associated with dilutional hyponatraemia and with an impairment of water excretion that caused by
  - **renal failure**
  - **syndrome of inappropriate secretion of the antidiuretic hormone arginine vasopressin (SIADH)**

Water and ECF osmolality

- A loss of water from the ECF will increase its osmolality and result in the movement of water from the ICF to ECF.

- This increase in ECF osmolality will stimulate the hypothalamic thirst centres to promote a desire to drink while also stimulating the release of vasopressin or ADH.

- ADH increases the permeability of the renal collecting ducts to water and promotes water reabsorption with consequent concentration of urine.

Water and ECF osmolality

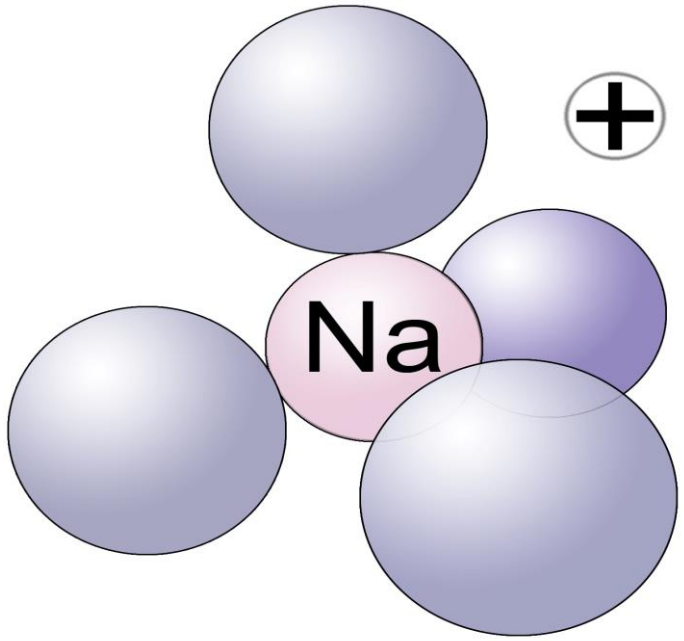
- Secretion of ADH is also stimulated by
  - **Angiotensin II**
  - **stress (pain)**
  - **Exercise**
  - **drugs such as morphine, nicotine, carbamazepine and vincristine.**

- If the osmolality of the ECF falls, there is no desire to drink and no secretion of ADH.

- Consequently, a dilute urine is produced which helps restore ECF osmolality to normal.

Sodium distribution

- The normal serum range is **135-145 mmol/L**.
- In contrast, the ICF concentration of sodium is only about **10 mmol/L**.



Sodium depletion

- Inadequate oral intake of sodium is rarely the cause of sodium depletion.
- Sodium depletion commonly occurs with water depletion, resulting in dehydration or volume depletion.
- The normal response of the body to the hypovolaemia includes
  - ↑ **Aldosterone secretion (which stimulates renal sodium reabsorption)**
  - ↑ **ADH secretion if ECF volume depletion is severe**

Sodium depletion

- The serum sodium level can give an indication of depletion, but it must be borne in mind that the serum sodium may be:
  -  where there is sodium and water loss but with predominant water loss, as occurs in excessive sweating
  - **normal**, where there is isotonic sodium and water loss, as occurs from burns or a haemorrhage
  -  when there is sodium loss with water retention as would occur if an isotonic sodium depletion were treated with a hypotonic sodium solution.

Sodium excess

- Sodium and water retention causes edema.

- Excess sodium level may be due to:
  - ↑ intake (not common but it can be associated with excessive IV saline infusion).
  - ↓ excretion
  - excess mineralocorticoid (Cushing syndrome or Conn's syndrome which is an aldosterone-producing adenoma).
  - secondary hyperaldosteronism associated with disorders such as congestive heart failure, nephrotic syndrome, hepatic cirrhosis with ascites or renal artery stenosis.

Hypernatraemia

- The signs and symptoms of hypernatraemia include
  - muscle weakness
  - Confusion

- Drug-induced hypernatraemia is often the result of a
  - **Diabetes insipidus-like syndrome (unresponsive to ADH)**
  - The affected patient presents with **polyuria**, **polydipsia** or **dehydration**
  - **Lithium and phenytoin are the most commonly implicated drugs.**
  - **The syndrome is usually reversible on discontinuation.**

Hypernatraemia

- Hypernatraemia can be caused by a number of other drugs by a variety of mechanisms including:
  - **Adrenocorticotrophic hormone**
  - **Anabolic steroids**
  - **Androgens**
  - **Corticosteroids**
  - **Lactulose**
  - **Oestrogens**
  - **Oral contraceptives**
  - **Sodium bicarbonate**

Drugs known to cause hyponatraemia

- Tricyclic antidepressants
- Amphotericin
- Angiotensin converting enzyme inhibitors
- Carbamazepine
- Cisplatin
- Clofibrate
- Cyclophosphamide
- Lithium
- Miconazole
- NSAIDs
- Opiates
- Tolbutamide
- Vasopressin
- Vincristine
- Diuretics
- Heparin

Potassium

- About 10% of the body potassium is bound in red blood cells (RBCs), bone and brain tissue and is not exchangeable.
- The remaining 90% of total body potassium is free and exchangeable with the vast majority having an intracellular location.
- Only 2% of the exchangeable total body potassium is in the ECF, the compartment from where the serum concentration is sampled and measured.
- Consequently, the measurement of serum potassium is not an accurate index of total body potassium, but together with the clinical status of a patient it permits a sound practical assessment of potassium homeostasis.

Main causes of Hypokalemia

Transcellular movement into cells

- The shift of potassium from the serum compartment of the ECF into cells accounts following intravenous or, less frequently, nebulised administration of ß-adrenoreceptor agonists such as salbutamol.

- Parenteral insulin also causes a shift of potassium into cells, and is used for this purpose in the acute management of patients with hyperkalaemia.

Main causes of Hypokalemia

Loss from the gastro-intestinal tract

- In patient with chronic diarrhea, considerable amount of potassium may be lost causing hypokalemia.
- Like wise abuse of laxative increase GI potassium loss causing hypokalemia.
- Persistent vomiting can also contribute to hypokalemia.

Main causes of Hypokalemia

Loss from the kidneys

- Hyperaldosteronism or Cushing's syndrome, can increase urinary potassium loss and cause hypokalaemia.
- Nephrotoxic antibiotics such as gentamicin may increase the excretion of potassium as a result from renal tubular damage.
- Corticosteroids can mimic aldosterone and increase potassium loss.
- The most commonly used groups of drugs that can cause hypokalaemia are thiazide and loop diuretics.

Clinical features of Hypokalemia

- The patient with moderate hypokalaemia may be asymptomatic, but the symptoms of more severe hypokalaemia include
  - **Muscle weakness**
  - **Hypotonia**
  - **Paralytic ileus**
  - **Depression and confusion**
  - **Arrhythmias**

Clinical features of Hypokalemia

- Although hypokalaemia tends to make antiarrhythmic drugs less effective, the action of **digoxin**, in contrast, is potentiated leading to increased signs of toxicity.

- **Insulin secretion** in response to a rising blood glucose concentration requires potassium and this mechanism may be impaired in hypokalaemia.

- Hypokalaemia is managed by giving either oral potassium or intravenous dilute potassium solutions, depending on its severity and the clinical state of the patient.

Hyperkalaemia

- **Hyperkalaemia** may arise from excessive intake (orally it is rare), but is common in an inappropriate use of parenteral infusion contain potassium.

- **Hyperkalaemia** is a common problem in patients with **renal failure** due to their inability to excrete a potassium load.

- The combined use of **potassium-sparing diuretics** such as amiloride, triamterene or spironolactone with an angiotensin converting enzyme (**ACE**) **inhibitor**, which will lower aldosterone, is a recognized cause of hyperkalaemia, particularly in the elderly.

- **Addison's disease** where there is a deficiency of aldosterone also decrease renal potassium loss and contribute to **hyperkalaemia**.

Hyperkalaemia

- The majority of body potassium is intracellular.
  - ***Severe tissue damage***
  - ***catabolic states***
  - ***Hypoxia***
  - ***diabetic ketoacidosis***

- May result in **hyperkalaemia** due to potassium moving out of and sodium moving into cells.

- **Haemolysis** during sampling or a delay in separating cells from serum will result in potassium escaping from blood cells into serum and causing an ***artefactual hyperkalaemia***.

Clinical features of hyperkalaemia

- Hyperkalaemia can be **asymptomatic** but **fatal**.

- It has many effects on the heart. ECG changes may precede
  - **ventricular fibrillation**
  - **cardiac arrest**

Clinical features of hyperkalaemia

- **In emergency management** of a patient with hyperkalaemia (>6.5 mmol/L ± ECG changes), **calcium gluconate (or chloride)** at a dose of 10 mL of 10% solution is given intravenously over 5 min.

- This does not reduce the potassium concentration but antagonises the effect of potassium on cardiac tissue.

- Immediately thereafter, **glucose 50 g with 20 units soluble insulin**, for example, by intravenous infusion will lower serum potassium levels within 30 min by increasing the shift of potassium into cells.

- **Chronic hyperkalaemia**, in renal failure, is managed by a **low potassium diet**.

Calcium

- Calcium is present in serum bound mainly to the
  - albumin component of protein (46%)
  - complexed with citrate and phosphate (7%)
  - free ions (47%)

- Only the free ions of calcium are physiologically active.

- The serum calcium level is often determined by measuring total calcium **i.e. (free & bound)**

- **Parathyroid hormone (PTH)** is inhibited by increased serum concentrations of calcium ions. PTH is secreted in response to low calcium concentrations.

Calcium

- **In alkalosis**, hydrogen ions dissociate from albumin, and calcium binding to albumin increases, together with an increase in complex formation.
- If the concentration of ionized calcium falls sufficiently, clinical symptoms of hypocalcaemia may occur despite the total serum calcium concentration being unchanged.
- The reverse effect, that is, increased ionized calcium, occurs **in acidosis**.
- **Changes in serum albumin** also affect the total serum calcium concentration independently of the ionized concentration.
- Calcium metabolism is regulated by **1,25-dihydroxycholecalciferol (vitamin D)** which, when serum calcium is low, is secreted to promote gastro-intestinal absorption of calcium.

Hypercalcaemia

- Hypercalcaemia may be caused by a variety of disorders, the most common
  - Hyperparathyroidism
  - Multiple myeloma
  - Carcinomas which metastasise in bone
  - Thyrotoxicosis
  - Vitamins A and D intoxication
  - Acute renal failure
  - Renal transplantation
  - Acromegaly

- PTH measurement can be pivotal in the establishment of the cause of hypercalcaemia.

Hypercalcaemia

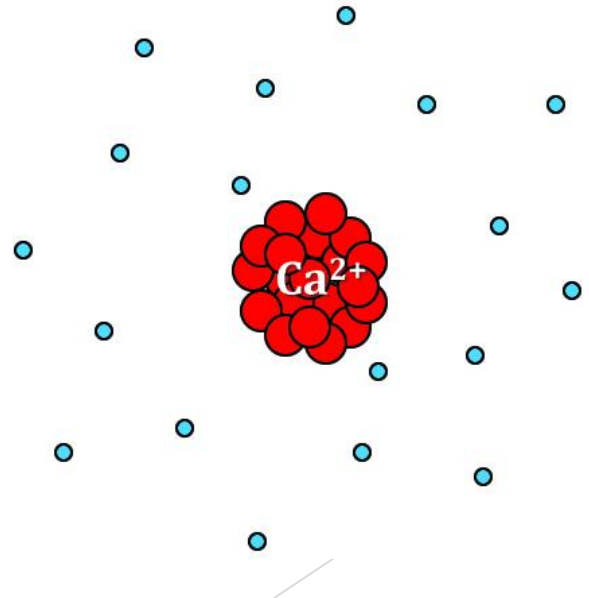
- Different medication may cause hypercalcaemia including:
  - Thiazide diuretics
  - Lithium
  - Tamoxifen
  - Calcium supplements

- An artefactual increase in total serum calcium may be seen as a result of tourniquet applied during venous sampling.

Hypercalcaemia

- Management of hypercalcaemia involves:

1. correction of any dehydration with **normal saline**
2. followed by **furosemide** which inhibits tubular reabsorption of calcium
3. **Bisphosphonates** are used to inhibit bone turnover



Hypocalcaemia

- Hypocalcaemia can be caused by a variety of disorders including
  - Hypoalbuminaemia
  - Hypoparathyroidism
  - Pancreatitis
  - Vitamin D deficiency caused by malabsorption, reduced exposure to sunlight, liver disease and renal disease.
  - Drug induced e.g. phenytoin, phenobarbital, aminoglycosides, phosphate enemas, calcitonin and furosemide.

Creatinine

- Serum creatinine concentration is largely determined by its
  - rate of production
  - rate of renal excretion

- It is frequently used to evaluate renal function. Normal range in adult is 80-120 ml/min.

- Creatinine undergoes complete glomerular filtration with little reabsorption by the renal tubules.

- Its clearance is, therefore, usually a good indicator of the glomerular filtration rate (GFR).

Creatinine

- As a general rule, if the serum creatinine doubles this equates to a 50% reduction in the GFR and consequently renal function.

- Individuals with a high muscle bulk produce more creatinine and, therefore, have a higher serum creatinine level compared to an otherwise identical but less muscular individual.

Measurement of creatinine clearance

- Creatinine clearance rates can be measured by
  - collecting urine for a specified period
  - collecting a blood sample for determination of serum creatinine at the midpoint of the concurrent urine collection time

$$CrCl\ (ml/min) = (UCr \cdot V\ urine)/(SCr \cdot T)$$

UCr: urine creatinine concentration (mg/dL)

V urine: volume of urine collected (ml)

SCr: serum creatinine collected at the midpoint of the urine collection (mg/dL)

T : time of the urine collection (minutes)

Estimation of creatinine clearance

- The most widely used of these formulas for adults aged 18 years and older is the method suggested by **Cockcroft and Gault**:

- **For males,**

$$CrClest = [(140 - age)\ BW] / (72 \cdot SCr)$$

- **For females,**

$$CrClest = [0.85(140 - age)BW] / (72 \cdot SCr)$$

CrClest: estimated creatinine clearance (mL/min)

Age (years)

BW: body weight (kg)

SCr: serum creatinine (mg/dL)

Urea

- The catabolism of dietary and endogenous amino acids in the body produces large amounts of **ammonia**.

- **Ammonia** is toxic and its concentration is kept very low by conversion in the liver to urea.

- **Urea** is eliminated in urine and represents the major route of nitrogen excretion.

- Urea levels vary widely with
  - ***Diet***
  - ***rate of protein metabolism***
  - ***liver production***
  - ***GFR***

Urea

- Production is decreased in situations where there is a
  - ***low protein intake***
  - ***some patients with liver disease***

- Thus, non-renal as well as renal factors should be considered when evaluating changes in serum urea concentrations.

Glycated haemoglobin

- Glucose binds to a part of the haemoglobin molecule to form a small glycated fraction.
- Normally, about 5% of haemoglobin is glycated, but this amount is dependent on the average blood glucose concentration over the lifespan of the red cells (about 120 days) and where red cell lifespan is reduced this leads to low glycated haemoglobin levels.
- The major component of the glycated fraction is referred to as HbA1C.
- Measurement of HbA1C is well established as an indicator of chronic glycaemic control in patients with diabetes.

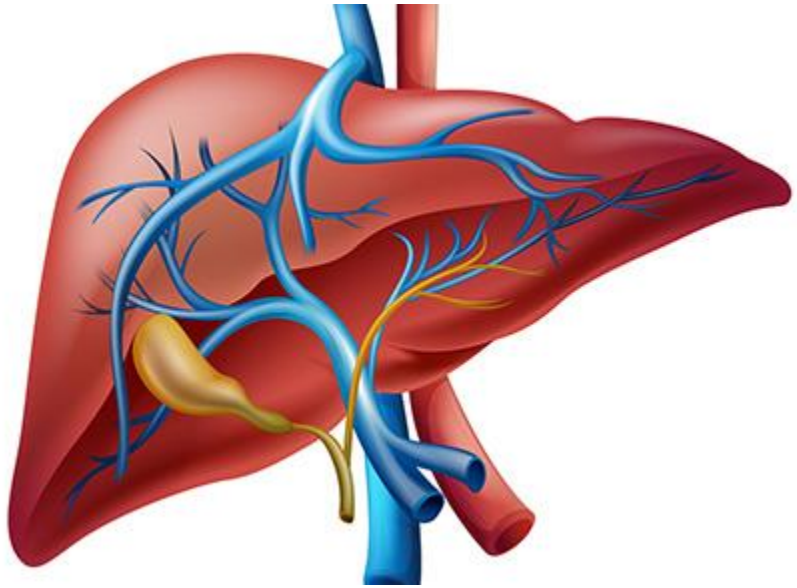
Uric acid

- It is end product of purine metabolism.
- Purines are produced by breakdown of nucleic acid from ingested meat or synthesized within the body.
- Two main factors contribute to elevated serum uric acid,
  - **increased rate of formation**
  - **reduced rate of excretion**
- Uric acid is poorly soluble and an elevation in serum concentration can result in deposition as monosodium urate in tissues or joints.
- Deposition precipitates an acute attack of gouty arthritis.

Liver function tests (LFTs)

- Routine LFTs give information mainly about the activity or concentrations of enzymes and compounds in serum rather than quantifying specific hepatic functions.

- Results are useful in confirming or excluding a diagnosis of clinically suspected liver disease, and monitoring its course.



Albumin

- Albumin is quantitatively the most important protein synthesized in the liver.
- About 60% is located in the interstitial compartment of the ECF.
- The concentration in the serum is important in maintaining its volume since it accounts for approximately 80% of serum colloid osmotic pressure.
- A reduction in serum albumin concentration often results in oedema.

Albumin

- Albumin has an important role in binding with **calcium**, **bilirubin** and many **drugs**.

- A reduction in serum albumin will increase free levels of agents which are normally bound and adverse effects can result if the ‘free’ entity is not rapidly cleared from the body.

- The serum concentration of albumin depends on:
    1. rate of synthesis
    2. volume of distribution
    3. rate of catabolism

Albumin

- Synthesis of albumin falls in parallel with
  - **increasing severity of liver disease**
  - **malnutrition states where there is an inadequate supply of amino acids to maintain albumin production.**
  - **Response to inflammatory mediators such as interleukin**

Albumin

- A low serum albumin concentration will occur when the volume of distribution of albumin increases, is the shift of albumin from serum to interstitial fluid causes dilutional Hypoalbuminaemia, for example,
  - in cirrhosis with ascites,
  - in fluid retention states such as pregnancy
  - parenteral infusion of excess protein-free fluid
  - postoperative patients
  - septicemia

Albumin

- Other causes of hypoalbuminaemia include catabolic states associated with a variety of illnesses and increased loss of albumin,
  - in urine from damaged kidneys (e.g., nephrotic syndrome)
  - via the skin following burns or a skin disorder such as psoriasis
  - from the intestinal wall in a protein-losing enteropathy
- An increase in serum albumin is rare and can be iatrogenic, for example, inappropriate infusion of albumin, or the result of dehydration or shock.

Bilirubin

- At the end of their life, RBCs are broken down by the reticuloendothelial system, mainly in the spleen.
- The haemoglobin molecules, which are subsequently liberated, are split into globin and haem.
- the iron in haem is reutilized, and the remaining of haem is degraded to bilirubin.

Bilirubin

- The liver produces 300 mg of bilirubin each day.
- However, because the mature liver can metabolize and excrete up to 3 g daily, serum bilirubin concentrations are not a sensitive test of liver function.
- As a screening test they rarely do other than confirm the presence or absence of jaundice.
- An elevation of serum bilirubin concentration above 50 μmol/L (i.e. approximately 2.5 times the normal upper limit) will reveal itself as jaundice, seen best in the skin and sclerae.

Bilirubin

- Elevated bilirubin levels can be caused by
  - **increased production of bilirubin e.g. hemolysis**
  - **ineffective erythropoiesis (premature death of red blood cells)**
  - **impaired transport into hepatocyte e.g. intrahepatic obstruction due to cirrhosis and in tumors**

Bilirubin

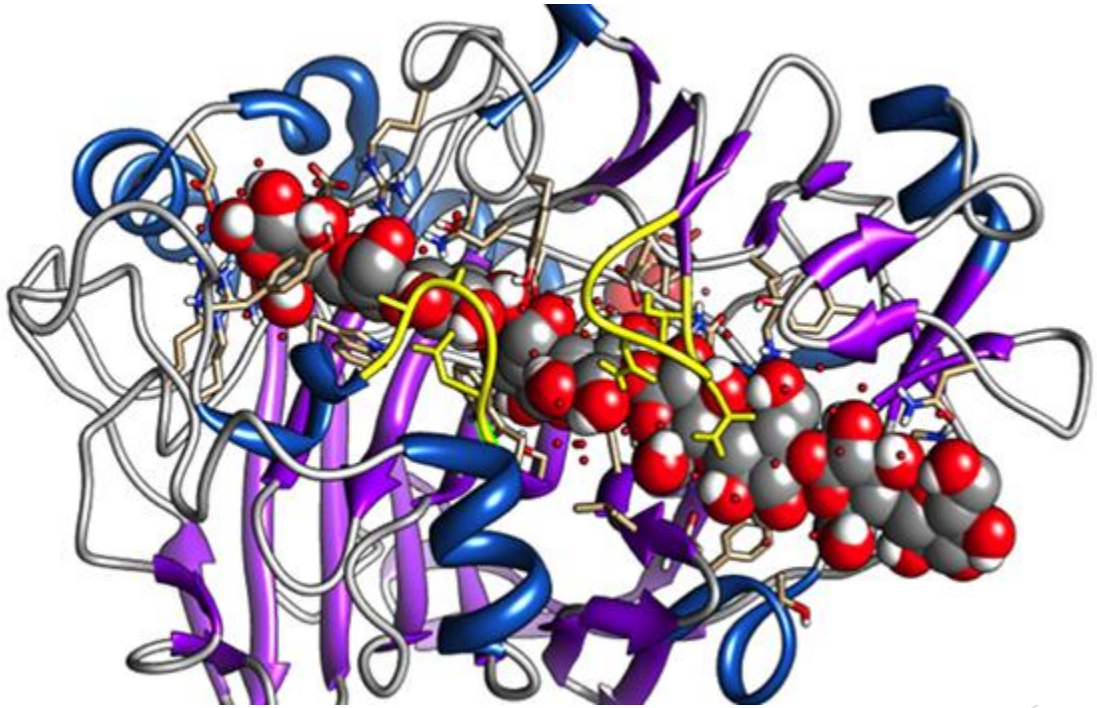
- The bilirubin is normally not filtered by the glomeruli and does not normally appear in the urine.

- Bilirubin in the urine (bilirubinuria) is usually the result of an increase in serum concentration of conjugated bilirubin and indicates an underlying pathological disorder.

Enzymes

- Enzyme concentrations in the serum of healthy individuals are normally low.

- When cells are damaged, increased amounts of enzymes are detected as the intracellular contents are released into the blood.



Enzymes

- It is important to remember that the assay of ‘serum enzymes’ is a measurement of **catalytic activity** and **not actual enzyme concentration**

- While the measurement of enzymes may be very specific the enzymes themselves may **not be specific to a particular tissue or cell**.

- Many enzymes arise in more than one tissue represent the damage to any one of the tissues which contain the enzymes.

Enzymes

- In practice, this problem may be clarified because some tissues contain two or more enzymes in different proportions which are released on damage.
- For example, alanine and aspartate transaminase both occur in cardiac muscle and liver cells, but their site of origin can often be differentiated, because there is more alanine transaminase in the liver than in the heart.
- In those situations where it is not possible to look at the relative ratio of enzymes, it is sometimes possible to differentiate the same enzyme from different tissues.
- Such enzymes have the same catalytic activity but differ in some other measurable property, and are referred to as **isoenzymes**.

Enzymes

- The measured activity of an enzyme will be dependent upon the time it is sampled relative to its time of release from the cell.
- If a sample is drawn too early after a particular insult to a tissue there may be no detectable increase in enzyme activity.
- If it is drawn too late, the enzyme may have been cleared from the blood.

Alkaline phosphatase

- Alkaline phosphatases are found in the
  - Hepatocytes
  - bone (reflect bone building)
  - intestinal wall
  - Placenta
  - Kidneys
  - leucocytes

Alkaline phosphatase can elevate in

- **Liver disorders** such as cholestasis, hepatitis, tumor, and drug-induced by ACE inhibitors or estrogens.
- **Pregnancy** due to release of the placental isoenzyme.
- During periods of **children and adolescents growth** in when the bone isoenzyme is released.
- **Bone disorders** such as osteomalacia and rickets, Paget's disease of bone, bone tumors, renal bone disease, osteomyelitis and healing fractures.
- Alkaline phosphatase may be raised in **inflammatory bowel disease**.

Transaminases

- The two transaminases of diagnostic use are
  - aspartate transaminase (**AST**; also known as aspartate aminotransferase)
  - alanine transaminase (**ALT**; also known as alanine aminotransferase)

- Highest concentration in **hepatocytes** and **muscle** cells.

Serum Transaminases are increased in

- **Liver disease**
- **Crush injuries**
- **Severe tissue hypoxia**
- **Myocardial infarction**
- **Surgery**
- **Trauma**
- **Muscle disease**
- **Pancreatitis**

- **Non-alcoholic fatty liver disease** is now the most common cause of mild alteration of aminotransferase levels in the developed world.

γ-Glutamyl transpeptidase

- Serum levels of γ-glutamyl transpeptidase can be increased with different disease or disorders.

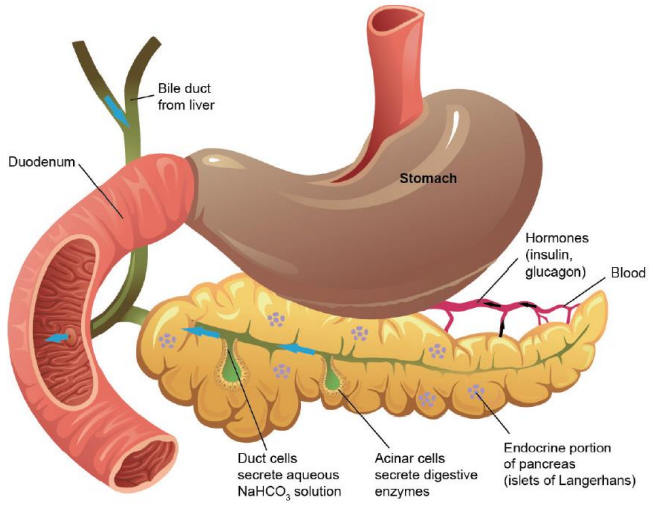
- Serum levels of γ-glutamyl transpeptidase activity can be raised by enzyme induction by certain drugs such as **phenytoin**, **phenobarbital**, **rifampicin** and **oral contraceptives**.

- Serum γ-glutamyl transpeptidase activity is usually raised in an individual with **alcoholic liver disease**.

- However, it can also be raised in **heavy drinkers of alcohol** who do not have liver damage, due to enzyme induction. Its activity can remain elevated for up to 4 weeks after stopping alcohol intake.

Amylase

- The **pancreas** and **salivary glands** are the main producers of serum amylase.
- Amylase can be increased with different disease or disorders.
- The serum amylase concentration rises within the first 24 h of an attack of pancreatitis and then declines to normal over the following week.



Cardiac markers - Troponins

- Cardiac troponins are the preferred biomarker for myocardial necrosis.

- Sampling of troponins at two time points, usually admission and 12 h from worst pain, is usually needed, although if it is entirely clear that there has been a myocardial infarction, particularly in a late presentation, a second sample may not be needed.

- Troponins do not discriminate between **ischemic** and **non-ischemic** mechanisms of myocardial injury, such as myocarditis, cardiac surgery and sepsis.

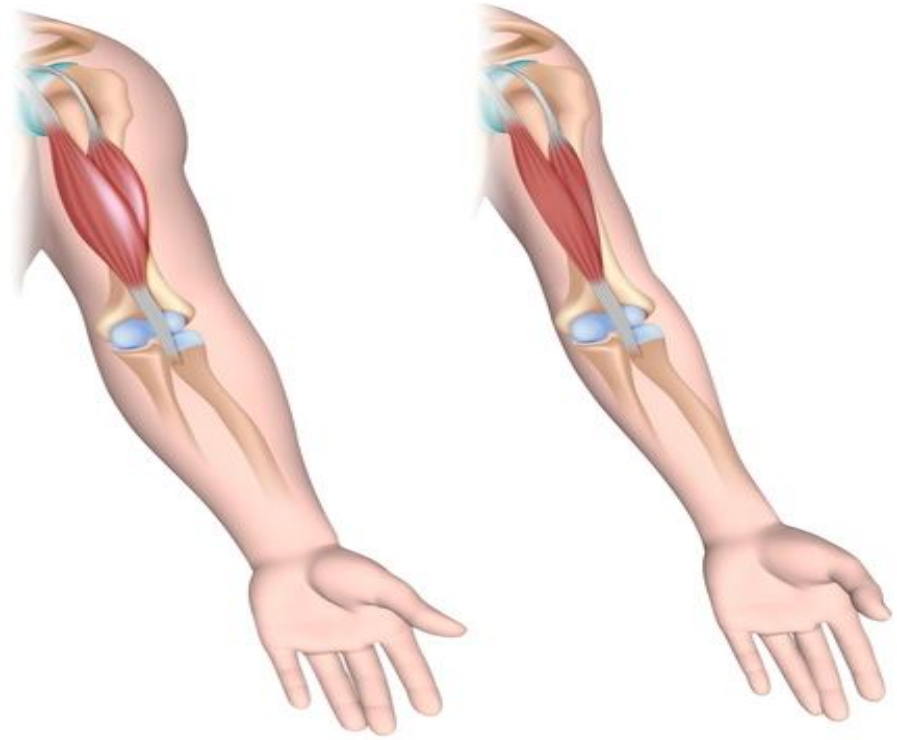
Cardiac markers - Creatine kinase (CK)

- CK is an enzyme which is present in relatively high concentrations in
  - **heart muscle**
  - **skeletal muscle**
  - **brain**
  - **smooth muscles and other tissue**

- Levels are significantly increased following
  - **shock and circulatory failure**
  - **myocardial infarction**
  - **muscular dystrophies**

Cardiac markers - Creatine kinase (CK)

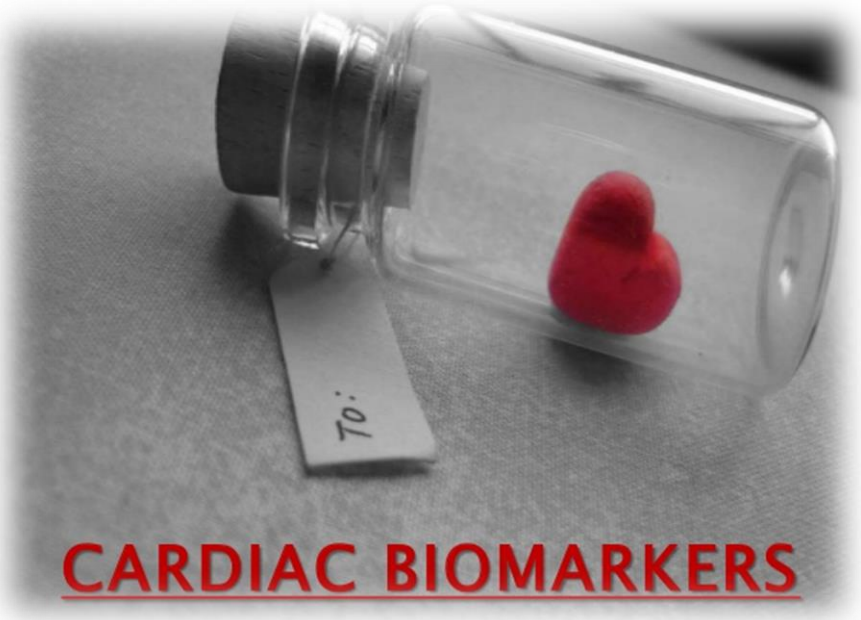
- The most important adverse effects associated with statins are myopathy and an increase in hepatic transaminases with **grossly elevated CK levels**.



Cardiac markers - Creatine kinase (CK)

- Following a myocardial infarction there is a characteristic increase in serum CK activity.

- Although measurement of CK activity was used in the past to detect myocardial damage, **cardiac troponin measurement is now the preferred biomarker**.



Cardiac markers - Lactate dehydrogenase (LD)

- Lactate dehydrogenase has five isoenzymes (LD1–LD5). Total LD activity is rarely measured because of the **lack of tissue specificity**.

LD isoenzyme correlation with clinical disorders

Isoenzymes elevation	Associated disorder
LD1 and LD2	• myocardial infarction • renal infarction • megaloblastic anemia
LD2 and LD3	• acute leukemia
LD3	• malignancies
LD5	• liver damage • skeletal muscle damage

Hematology data

RBC count

- A high RBC (erythrocytosis or polycythaemia) indicates increased production by the bone marrow and may occur as a physiological response to hypoxia, as in chronic airways disease, or as a malignant condition of red cells such as in polycythaemia rubra vera.

Reticulocytes

The reticulocyte count may be useful in assessing the response of the marrow to **iron**, **folate** or **vitamin B12** therapy. The count peaks at about 7–10 days after starting such therapy.

Hematology data

Mean cell volume (MCV)

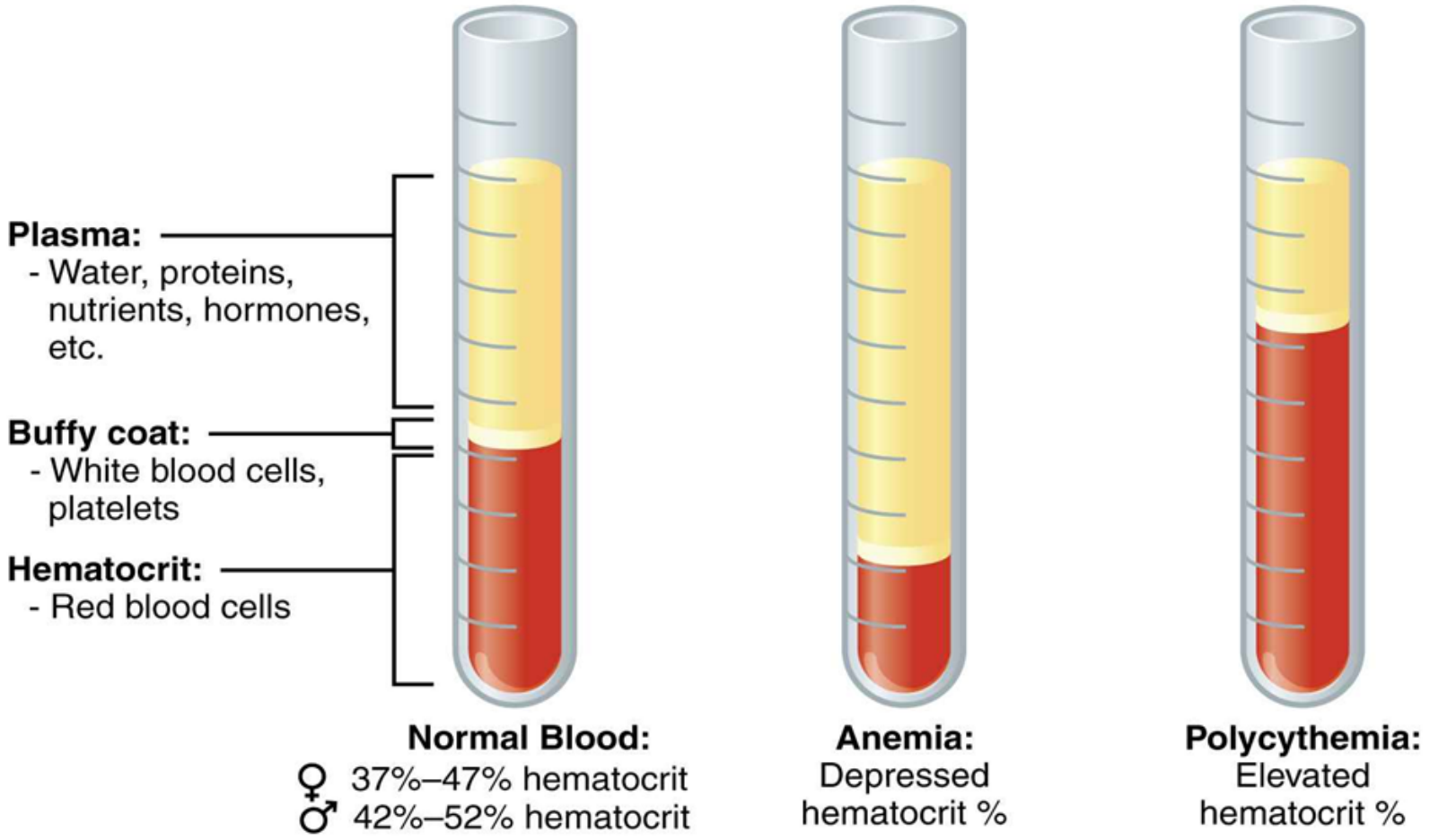
- The MCV is the average volume of a single red cell.
- Terms such as ‘microcytic’ and ‘macrocytic’ are descriptive of a low and high MCV, respectively.
- They are useful in the process of identification of various types of anaemias such as caused by iron deficiency (microcytic) or vitamin B12 or folic acid deficiency (megaloblastic or macrocytic).

Hematology data

Packed cell volume (PCV)

- The PCV or hematocrit is the ratio of the volume occupied by red cells to the total volume of blood.
- The PCV often reflects the RBC and will, therefore, be decreased in any sort of anemia. It will be raised in polycythaemia.
- It may, however, be altered irrespective of the RBC, when the size of the red cell is abnormal, as in macrocytosis and microcytosis.

Packed cell volume (PCV)



White blood cell (WBC) count

Neutrophils

- The neutrophil count increases in the presence of infection, tissue damage (e.g. infarction) and inflammation (e.g. rheumatoid arthritis, acute gout). Neutropenia, also described as agranulocytosis is associated with malignancy and drug toxicity, but may also occur in viral infections such as influenza, and hepatitis.

Basophils

- Basophilia occurs in various malignant and premalignant disorders such as leukemia and myelofibrosis.

White blood cell (WBC) count

Eosinophils

- apparent in many allergic conditions such as asthma, hay fever and drug sensitivity reactions as well as some malignant diseases.

Lymphocytes

- An increase in lymphocyte numbers occurs particularly in viral infections such as rubella, mumps, infectious hepatitis.

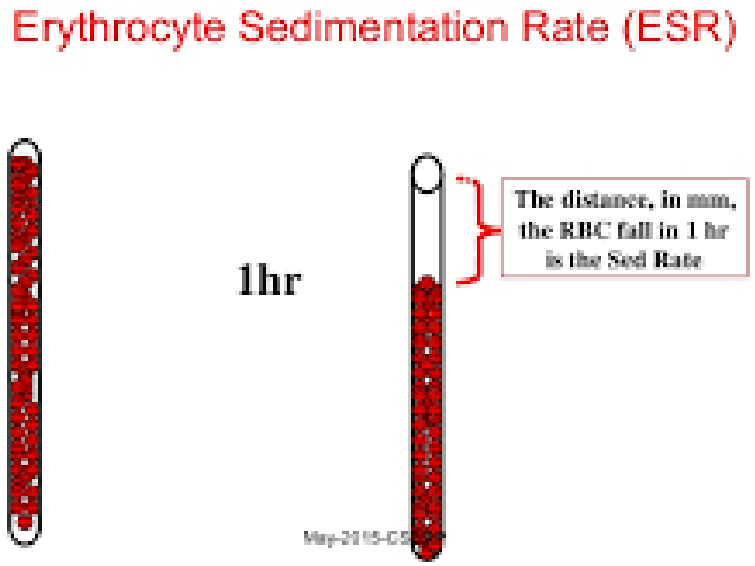
Monocytes

- Their numbers increase in some infections such as typhoid, bacterial endocarditis, and tuberculosis.

Erythrocyte sedimentation rate (ESR)

- The ESR is a measure of the settling rate of red cells in a sample of anticoagulated blood, over a period of 1 h, in a cylindrical tube.

- The ESR may be raised in the active phase of rheumatoid arthritis, inflammatory bowel disease, malignant disease and infection. The ESR is non-specific and, therefore, of little diagnostic value.



Easy or Tough?