Hematology Amboss

Normal Values

- Hemoglobin
    - Men: 13 - 17
    - Women: 12 - 15
- Red cell count (million per micro liter)
    - Men: 4.3 - 5.9
    - Women: 3.5 - 5
- Hematocrit
    - Men: 40% to 54%.
    - Women: 36% to 48%.
- WBC count: 4,500 to 11,000 cells per microliter (μL)
- Retic count: 0.5 - 2. 5%
- MCV: 80 - 100
- MCH: 27 - 33 pg
- MCHC: 33 - 37 g/dL
- RDW: 11.5 - 14.5

RBCs

- **Iron deficiency anemia (IDA)** is the most common form of anemia worldwide and is caused by inadequate intake, decreased absorption (e.g., atrophic gastritis, inflammatory bowel disease), increased demand (e.g., during pregnancy), or increased loss (e.g., gastrointestinal bleeding, menorrhagia) of iron.
- Best initial test for IDA: ↓ serum ferritin
- **Mentzer index (MCV/RBC ratio):** a ratio < 13 suggests thalassemia; a ratio > 13 suggests IDA
- Beta thalassemia usually due to point mutation
- Alpha thalassemia usually due to deletion
- **Hemolysis evaluation**: ↓ Haptoglobin, ↑ LDH, ↑ reticulocytes
- Beta-thalassemia minor should be strongly suspected if **HbA2 is > 3.5%.**
- mutations on the α- or β-globin chains result in alpha or beta thalassemia
- The key feature in all forms of **thalassemia** is microcytic hypochromic anemia (which may be very mild in minor forms), but more severe forms may also manifest with hemolysis, splenomegaly, delay in growth and development, and skeletal deformities.
- Avoid splenectomy in patients < 5 years old due to the risk of overwhelming postsplenectomy sepsis.
- All patients receiving transfusion therapy should be periodically evaluated for iron overload disease and subsequent organ damage
- Features of extramedullary hematopoiesis may be present in certain severe, chronic forms of anemia (e.g., thalassemia, myelofibrosis).
    - ° Hepatosplenomegaly
    - ° Paravertebral mass

    - ° Widening of diploic spaces of the skull
- **Fanconi anemia** - Hereditary autosomal recessive disorder due to a DNA crosslink repair defect resulting in bone marrow failure
- **Methemoglobinemia** is a condition in which more than 1% of hemoglobin contains iron in its oxidized form (Fe+3) and cannot participate in oxygen delivery.
- The ferric iron in methemoglobin has a high affinity for cyanide, thus, amyl nitrite-induced methemoglobin is used as a competitive inhibitor in the treatment of cyanide poisoning
- Methylene blue is the first-line treatment for acquired methemoglobinemia.
- The most common underlying cause of vitamin B12 deficiency is **pernicious anemia**, an autoimmune disorder characterized by the absence of intrinsic factor (IF). IF is a protein that is crucial for vitamin B12 absorption.
- **Schilling test**: a test to determine the cause of vitamin B12 deficiency
- **homocysteine**: elevated in both vitamin B12 deficiency and folate deficiency
- **Methylmalonic acid (MMA)** is normal in folate deficiency and elevated in vitamin B12 deficiency
- Maternal folate deficiency: fetal spina bifida/anencephaly
- Hemolytic anemia is characterized by the breakdown of red blood cells
- Hemolysis can either be caused by abnormalities in RBCs (hemoglobin, the RBC membrane, or intracellular enzymes), which is called **intrinsic hemolytic anemia**, or by external causes (immune-mediated or mechanical damage), which is called **extrinsic hemolytic anemia**.
- Hemolysis can be further categorized depending on whether it occurs inside the blood vessels (**intravascular hemolysis**), in the reticuloendothelial system (**extravascular hemolysis**), or both
- Hemoglobinuria - Mostly occurs in intravascular hemolysis
- Hemosiderinuria - Late marker of severe intravascular hemolysis; Lends urine a brown color
- Urobilinogen - Mostly occurs in extravascular hemolysis
- **Typical biochemical findings in hemolysis** include ↓ haptoglobin, ↑ LDH concentration, ↑ indirect bilirubin concentration, peripheral blood smear abnormalities (e.g., ↑ reticulocytes, schistocytes, spherocytes, polychromasia), and urinalysis abnormalities (e.g., hemoglobinuria, hemosiderinuria, and urobilinogen).
- **Autoimmune hemolytic anemias (AIHAs)** are a collection of disorders characterized by the destruction of RBCs through antibody-mediated hemolysis (extravascular and/or intravascular). There are two broad types, categorized by the temperature at which the antigen-antibody reactions maximally occur: cold agglutinin hemolytic anemia (**cold AIHA**) and warm agglutinin hemolytic anemia (**warm AIHA**). AIHAs can be either idiopathic or secondary to another disease e.g., infectious, lymphoproliferative, or autoimmune diseases.
- **Cold AIHA**
    - • Mostly IgM antibodies
    - • Maximal reaction at cold temperatures (< 4°C)
    - • Can present with acute, severe episodes associated with cold exposure

    - Include acrocyanosis (Painful cyanosis of the extremities), livedo reticularis, Raynaud phenomenon, and skin ulcerations
    - PBS: marked agglutination of RBCs
- **Warm AIHA**
    - Mostly IgG antibodies
    - Maximal reaction at warm temperatures (≥ 37°C)
    - Typically gradual onset
    - PBS: abundant spherocytes
- Heinz bodies and bite cells in G6PD deficiency
- Target cells in sickle cell disease, thalassemia, and hemoglobin C disease
- Teardrop cells in thalassemia
- Smudge cells (Gumprecht shadows) in chronic lymphocytic leukemia (CLL)
- Hemoglobinuria - Mostly occurs in intravascular hemolysis
- Hemosiderinuria - Late marker of severe intravascular hemolysis
- Urobilinogen - Mostly occurs in extravascular hemolysis
- **Direct Coomb's test** - This is a key test in the workup of hemolytic anemia.
- Pyruvate kinase catalyzes the last step of glycolysis (i.e., irreversibly converts phosphoenolpyruvate into pyruvate)
  Absence of pyruvate kinase → ATP deficiency in RBC
  ATP deficiency disrupts the cation gradient along the RBC membrane → rigid RBCs → ↑ hemolysis (extravascular)
- Toxin-mediated RBC destruction, e.g., from a snake bite, oxidizing agents (e.g., dapsone, nitrofurantoin, phenazopyridine, primaquine)
- RBC destruction by intracellular pathogens, e.g., in malaria, babesiosis, bartonellosis, rickettsiosis
- Splenectomy is not recommended for cold AIHA. It is not effective as most extravascular hemolysis occurs in the liver.
- **Paroxysmal nocturnal hemoglobinuria (PNH)** is a hemolytic anemia caused by an acquired defect of the phosphatidylinositol glycan anchor (PIGA) gene, which leads to dysfunction of a red cell membrane protein (glycosylphosphatidylinositol) that is normally responsible for protecting RBCs from complement-mediated destruction.
- PNH is An acquired genetic defect of the hematopoietic stem cell characterized by a triad of hemolytic anemia, pancytopenia, and thrombosis
- PNH can also occur in patients with aplastic anemia and MDS
- Glucose-6-phosphate dehydrogenase **(G6PD) deficiency** leads to an impaired regeneration of reduced glutathione, an important antioxidant, which makes RBCs more susceptible to oxidative stress and can result in episodic hemolytic anemia.
- G6PD deficiency is usually asymptomatic, but a sudden surge in oxidative stress (e.g., after infection, consumption of fava beans, or various drugs) may lead to a life-threatening hemolytic crisis.
- As with sickle cell anemia, carriers of the G6PD deficiency may be less severely affected by malaria, especially if the disease is caused by Plasmodium falciparum.

- **Hereditary spherocytosis** (HS) is an autosomal dominant disease that is caused by red blood cell (RBC) membrane protein defects, which render the RBCs more vulnerable to osmotic stress and hemolysis.
- Moderate HS, which is the most common form, usually presents in infancy or childhood with the classic triad of anemia, jaundice, and splenomegaly.
- Frequently affected proteins in HS - spectrin, ankyrin, band 3, protein 4.2
- **Acute chest syndrome (ACS)** is a potentially fatal complication of sickle cell anemia caused by vaso-occlusion of the pulmonary vasculature.
- **Sickle cell disease** - Mutations in the hemoglobin β chain lead to the formation of hemoglobin S, which polymerizes when deoxygenated. Deoxygenated HbS results in sickle-shaped erythrocytes that can occlude blood vessels and cause ischemia.
- Acute complications of sickle cell disease include vascular occlusion events (e.g., vasoocclusive pain crisis, stroke, acute chest syndrome), severe anemia (e.g., sequestration, aplastic anemia), and invasive infections from encapsulated organisms (e.g., bacteremia, pneumonia, meningitis, osteomyelitis).
- **Sickle cell anemia** - The most severe clinical presentation of sickle cell disease. Genotypes include homozygous HbSS and compound heterozygous HbSβ0thal (a form of sickle beta thalassemia).
- The body increases the production of fetal hemoglobin (HbF) to compensate for low levels of HbA in sickle cell disease.
- Vasoocclusive crises (sickle cell pain crisis) - Characterized by recurrent episodes of severe throbbing or sharp pain. Typically affects the limbs, chest, and back and lasts for ~ 7 days
- Dysmorphic erythrocytes in sickle cell disease and hereditary spherocytosis are susceptible to parvovirus B19 infection, which can temporarily suppress bone marrow erythropoiesis leading to Aplastic crisis.
- Hemoglobin S (HbS) - Glutamic acid replaced by valine
- Hemoglobin C - Glutamic acid replaced by lysine

Sickle cell anemia

- Most common cause of death - acute chest syndrome and multiorgan failure
- Most common clinical manifestation - Pain crises
- First manifestation - Hand foot syndrome (dactylitis)

RBC parameters	Common causes of elevation	Common causes of reduction
Hemoglobin (Hb) Men: 13 - 17	• Polycythemia vera • Relative polycythemia (e.g., due to severe dehydration) • Increased erythropoietin (EPO) synthesis, e.g., due to: • Chronic hypoxia (e.g., in	• Anemia

Women: 12 - 15	COPD or CHF) ● Malignancies (paraneoplastic effect) ● EPO doping	
Hematocrit (Hct)	● Same as for elevated Hb and RBC count	● Anemia
Mean corpuscular volume (MCV) 90 ± 10 fL	Megaloblastic anemia, e.g., caused by: ● Vitamin B12 deficiency ● Folate deficiency Nonmegaloblastic macrocytic anemia, e.g, caused by: ● Liver disease ● Alcohol use ● Diamond-Blackfan anemia ● Hypothyroidism	Microcytic anemia, e.g., due to: ● Iron deficiency ● Thalassemia
Mean corpuscular hemoglobin (MCH) 30 pg		Hypochromic anemia, e.g., due to: ● Iron deficiency ● Thalassemia
Mean corpuscular hemoglobin concentration (MCHC) 33%		● Spherocytosis
Reticulocyte count	Increased erythropoiesis, e.g. secondary to: ● Hemolysis ● Blood loss	Insufficient erythropoiesis, e.g., caused by ● Aplastic anemia ● Chronic kidney disease
Red blood cell distribution width (RDW)	● Anisocytosis ● Vitamin B12 deficiency ● Folate deficiency ● Iron deficiency ● Sideroblastic anemia ● Anemia of chronic disease ● Hereditary spherocytosis ● Conditions associated with reticulocytosis (e.g., recent hemorrhage)	

PLATELETS AND COAGULATION DISORDERS

- Bleeding disorders are a group of heterogeneous conditions characterized by defects in hemostasis that lead to an increased susceptibility to bleeding (also known as hemorrhagic diathesis).
- They are classified into **disorders of primary hemostasis** (when caused by a platelet abnormality), **disorders of secondary hemostasis** (when caused by defects in the extrinsic and/or intrinsic pathway of the coagulation cascade), and **hyperfibrinolysis** (when there is increased clot degradation).
- Hemostasis is the physiological process by which a bleeding stops. Its final result is a thrombus (blood clot), which consists of blood cells and fibrin strands.
- A helpful way of remembering the coagulation factors of the extrinsic pathway is 3 + 7 = 10: Tissue factor (factor III) and factor VII form a complex that activates factor X of the common pathway.
- A helpful way of remembering the coagulation factors of the common pathway is 10/5 = 2 × 1: Factors Xa and Va form a complex that cleaves prothrombin (factor II) to thrombin (IIa). Factor IIa then cleaves fibrinogen (I) into insoluble fibrin monomers (Ia).
- Activated protein C and its cofactor protein S form the activated protein-C complex (APC complex), which inhibits factors Va and VIIIa.
- Anti thrombin - Degrades thrombin and factors IXa and Xa; Activates tissue plasminogen activator (tPA)
- Fibrinolysis - degradation of the fibrin network of thrombi by the enzyme plasmin
- Factor VIII deficiency (hemophilia A)
- Factor IX deficiency (hemophilia B)
- Factor XI deficiency (hemophilia C)
- vitamin K-dependent coagulation factors II, VII, IX, and X
- A thorough physical examination is essential for diagnosing bleeding disorders and should include the inspection of the entire skin, mucosa (esp. oral cavity), and joints.
- Superficial, petechial bleeding indicates defects of primary hemostasis, whereas large, palpable ecchymoses and deep tissue bleeding suggest defects of secondary hemostasis!
- Coagulation panel including fibrinogen level: Elevated PT/INR and/or aPTT are suggestive of disorders of secondary hemostasis.
- DIC is characterized by thrombosis, hemorrhage, and organ dysfunction and can be a medical emergency that requires immediate treatment.
- Contraindication of platelet transfusion: uremic platelet dysfuntion (Platelet transfusion is not indicated due to rapid inactivation of transfused platelets)
- Desmopressin: stimulates vWF release from endothelial cells → Best initial treatment for mild or moderate symptoms (typically type 1 and, sometimes, type 2)

- Platelet aggregation inhibitors (e.g., aspirin, NSAIDs, clopidogrel) and intramuscular injections are contraindicated in von Willebrand disease because they further increase the risk of bleeding!
- Thrombocytopenia is a platelet count below the normal range (< 150,000/mm3)
- Common causes of impaired platelet production include bone marrow failure, infection, malignancy, and chemotherapy/radiation. Additional etiologies include hereditary syndromes, such as Wiskott-Aldrich syndrome and Alport syndrome.
- increased peripheral platelet turnover may be caused by autoimmune conditions, (e.g., immune thrombocytopenia), drugs (e.g., heparin), and other conditions (e.g., TTP/HUS, DIC/sepsis).
- Infections causing impaired platelet production: CMV, EBV, hepatitis C virus, HIV, mumps and rubella, parvovirus B19, rickettsia, VZV, dengue
- **Thrombotic thrombocytopenic purpura (TTP)**
    - It is a thrombotic microangiopathy, a condition in which microthrombi, consisting primarily of platelets, form and occlude the microvasculature (i.e., the arterioles and capillaries).
    - TTP occurs primarily in adults and is typically due to acquired autoantibodies against a proteolytic enzyme (ADAMTS13) that cleaves von Willebrand factor (vWF). The classic pentad of findings (fever, neurological abnormalities, thrombocytopenia, microangiopathic hemolytic anemia, and impaired renal function) is seen in a minority of patients.
    - Pathophysiology of TTP: ADAMTS13 deficiency → excess vWF → microthrombus formation → blockage of small vessels → RBC fragmentation (hemolysis) and end-organ damage
    - While PT and aPTT are normal or only mildly elevated in TTP and HUS (no consumption coagulopathy), they are markedly elevated in disseminated intravascular coagulation (consumption of platelets and all coagulation factors).
- **HUS** predominantly affects children and is caused by bacterial toxins, most commonly the Shiga-like toxin of enterohemorrhagic Escherichia coli (E. coli)
- **Hemophilias** are disorders of blood clotting and consequently may lead to serious bleeding.
- Hemophilia presents with hemarthrosis (bleeding into joints) and muscular or soft tissue hematomas.
- Hemophilia usually affects males, as it is primarily an X-linked recessive disease.
- Petechial bleeding is a common sign of platelet disorders, but NOT of coagulation disorders such as hemophilia.
- Desmopressin - Indication: mild hemophilia A
- Desmopressin triggers the release of vWF from endothelial cells, which leads to an increase in factor VIII plasma concentration
- **Antiphospholipid syndrome** (APS) is an autoimmune disease associated with increased risk of thrombosis due to the presence of procoagulatory antibodies.
- **Disseminated intravascular coagulation (DIC)** is a disorder characterized by systemic activation of the extrinsic clotting cascade with microthrombi formation, platelet consumption, and subsequent exhaustion of all clotting factors.

- DIC - a syndrome characterized by thrombosis, hemorrhage, and organ dysfunction caused by systemic activation of the clotting cascade, which leads to platelet consumption and exhaustion of clotting factors
- Laboratory studies can be highly variable. However, typical findings in overt decompensated DIC include thrombocytopenia, prolonged PT and aPTT, decreased fibrinogen levels, and elevated D-dimer levels
- Common causes of acute DIC include septic shock, acute pancreatitis, burns, snake bites, transplant rejection
- Common causes of chronic DIC include malignancies, aneurysms, retroperitoneal hematomas, intrauterine fetal death
- The mnemonic “STOP Making Trouble!” helps to recall the etiology of DIC: S – Sepsis/Snakebites, T – Trauma (acute traumatic coagulopathy), O – Obstetric complications, P – Pancreatitis, M – Malignancy, T – Transfusion.
- **Warfarin** is ineffective and possibly harmful in DIC, as it inhibits production of protein C and protein S, which can worsen coagulopathy
- Fibrinolytic therapy (e.g., tPA) should be avoided in all types of DIC.
- **D-dimer**: fibrin degradation product that correlates with activity of coagulation and fibrinolysis
- Increased serum D-dimer levels occur in deep vein thrombosis (DVT), pulmonary embolism (PE), and disseminated intravascular coagulation.
- Virchow triad (stasis, hypercoagulability, endothelial damage)
- A **deficiency of protein S or protein C** results in overactivity of factors V and VIII (factor Va and factor VIIIa) → increases thrombotic events.
- **Budd-Chiari syndrome** is a rare condition resulting from hepatic vein obstruction that leads to hepatomegaly, ascites, and abdominal discomfort. It is most commonly due to a thrombotic occlusion secondary to a chronic myeloproliferative neoplasm (e.g., polycythemia vera), but may be caused by other conditions associated with hypercoagulable states.
- **Reye syndrome** is a rare type of hepatic encephalopathy that is associated with aspirin use for viral illness in children < 19 years
- In order to detect heparin-induced thrombocytopenia, platelets must be continuously monitored during heparin therapy and a baseline should be established before commencing treatment.

WBC DISORDERS

- **Acute leukemias** are malignant neoplastic diseases that arise from either the lymphoid or myeloid cell line. Acute lymphoblastic leukemia **(ALL)** is the most common childhood malignancy, whereas acute myeloid leukemia **(AML)** primarily affects adults
- Acute leukemias are characterized by the proliferation of immature, nonfunctional WBCs (blasts) in the bone marrow, which impairs normal hematopoiesis. This leads to pancytopenia, which manifests with symptoms and signs of anemia (decreased RBCs), clotting disorders (decreased platelets), and immunocompromise (decreased fully functional, mature WBCs).

- Immature blasts enter the bloodstream → infiltration of other organs (particularly the CNS, testes, liver, and skin)
- Patients with acute leukemia, especially those with AML, may develop extremely high WBC counts, increasing the risk of leukostasis and disseminated intravascular coagulation (DIC).
- Adult T-cell leukemia/lymphoma is linked to infection with HTLV
- Mature B-cell ALL (also known as **Burkitt leukemia**)
- B-ALL is usually positive for CD10, CD19, CD20
- T-ALL is usually positive for CD2–CD8, especially CD3
- The majority of subtypes of AML are positive for CD13, CD33, CD34, CD117, and HLA-DR
- **Philadelphia translocation**: present in ~ 20–30% of adults with ALL but only ~ 5% of children with ALL
- Myelogenous leukemias (e.g AML) are myeloperoxidase (MPO) positive.
- All patients with ALL should undergo screening for CNS infiltration.
- **CML** is caused by a reciprocal translocation between chromosomes 9 and 22, resulting in the formation of the Philadelphia chromosome, which contains the BCR-ABL1 fusion gene
- CML: Philadelphia chromosome = favorable
- ALL: Philadelphia chromosome = unfavorable
- The most effective treatment for CML is targeted therapy with tyrosine kinase inhibitors (TKIs).
- CML can cause extreme leukocytosis (often > 100,000/mm3) and is frequently associated with basophilia.
- Leukocyte alkaline phosphatase (LAP): Low LAP is a typical finding and can help distinguish CML from other types of leukemia and leukemoid reactions
- Identification of the BCR-ABL1 fusion gene is the hallmark of CML and confirms the diagnosis.
- The Philadelphia chromosome is a specific genetic abnormality resulting from a translocation between chromosomes 9 and 22, producing the BCR-ABL1 fusion gene.
- Targeted therapy: tyrosine kinase inhibitors
  Hydroxyurea or IFN-α may be used to reduce leukocyte counts and control symptoms associated with extreme leukocytosis or thrombocytosis.
- **Chronic lymphocytic leukemia (CLL)** is a type of B-cell malignancy that manifests with lymphocytic leukocytosis.
- **Small lymphocytic lymphoma (SLL):** a type of B-cell lymphoma with the same genetic and molecular markers as CLL that manifests primarily in the lymph nodes, bone marrow, and other lymphatic tissue rather than with leukocytosis
- Weight loss, fever, night sweats, fatigue (B symptoms)
- Lymphadenopathy is a typical finding in lymphoid malignancies such as CLL and helps to differentiate CLL from CML, a myeloid malignancy.
- **Hodgkin lymphoma (HL)** is a malignant lymphoma that is of B-cell origin

- Risk factors of Hodgkin lymphoma include Epstein-Barr virus (EBV) infection and immunodeficiency (e.g., HIV infection), autoimmune diseases (rheumatoid arthritis, sarcoidosis)
- **Pel-Ebstein fever**: intermittent fever with periods of high temperature for 1–2 weeks, followed by afebrile periods for 1–2 weeks; relatively rare but very specific for HL
- Biopsy Findings in Hodgkin Lymphoma: Hodgkin cells and Reed-Sternberg cells, or popcorn cells
- **Reed-Sternberg cells** (RSCs): Large cells with binuclear/bilobed nuclei with dark centers of chromatin and pale halos, which result in an owl-eye appearance on histopathologic examination. They are CD15/CD30-positive
- Hodgkin cells: mononuclear, malignant B lymphocytes
- popcorn cells: a variant of a Reed-Sternberg cell characterized by polylobulated nuclei that resemble popcorn.
- First-line treatment for all Hodgkin Lymphoma patients is typically ABVD: doxorubicin (Adriamycin®), bleomycin, vinblastine, dacarbazine.
- Lymphomas are malignancies that arise from lymphocytes and are classified as either Hodgkin lymphomas (characterized by Reed-Sternberg cells) or non-Hodgkin lymphomas (NHLs), which comprise all other types of lymphoma.
- The most common low-grade B-cell lymphoma is follicular lymphoma, while the most common low-grade T-cell lymphomas are cutaneous T-cell lymphomas, such as mycosis fungoides.
- The 4 T's of anterior mediastinal masses: Thymoma, Teratoma (and other germ cell tumors), Thyroid neoplasm, and Terrible lymphoma.
- R-CHOP therapy for Non Hodgkin lymphoma
- **Multiple myeloma (MM)** is a malignant plasma cell dyscrasia characterized by uncontrolled proliferation and diffuse infiltration of monoclonal plasma cells in the bone marrow.
- Malignant plasma cells in multiple myeloma produce monoclonal proteins (also known as M proteins or paraproteins) such as abnormal immunoglobulins (e.g., IgG, IgA) and free light chains (e.g., Bence Jones proteins).
- Evaluation of a patient with suspected multiple myeloma (e.g., a patient with CRAB criteria) should include CBC, CMP, immunoglobulin studies, LDH, β2 microglobulin, bone imaging, and a bone marrow biopsy.
- CRAB criteria for organ damage due to a plasma cell disorder: Calcium increased, Renal insufficiency, Anemia, Bone lesions
- **Bence Jones proteinuria** is suggestive of plasma cell disorders (e.g., multiple myeloma, Waldenstrom macroglobulinemia).
- **Waldenstrom macroglobulinemia** - A type of non-Hodgkin lymphoma associated with abnormal production of monoclonal IgM antibodies
- **Amyloidosis** is a collective term for the extracellular deposition of abnormal proteins, in a single organ (localized amyloidosis) or throughout the body (systemic amyloidosis).
- The combination of raccoon eyes, macroglossia, and the shoulder pad sign is highly specific for AL amyloidosis (but only present in ~ 10 % of patients)

- The main feature of AA amyloidosis at diagnosis is renal dysfunction (e.g., CKD, nephrotic syndrome). Cardiac involvement is rare.
- **Mucosa-Associated Lymphoid Tissue (MALT) lymphoma** (also called MALToma or extranodal marginal zone lymphoma) is a B-cell non-Hodgkin lymphoma (NHL) that typically affects elderly patients in the 7th and 8th decades.
- **Gastric MALTomas** are frequently associated with Helicobacter pylori (H. pylori) infection, whereas nongastric MALTomas are rather associated with autoimmune conditions (e.g., Sjögren syndrome, Hashimoto's thyroiditis).
- **Mycosis fungoides** is an indolent, CD4+ cutaneous T-cell lymphoma that presents on the skin
- Histopathological evidence is essential for diagnosis of mycosis fungoides and typically consists of atypical lymphocytes found in the upper dermis or aggregates of such cells within the epidermis (known as Pautrier microabscesses).
- Pautrier microabscesses: aggregates of atypical CD4+ T-cells within the epidermis that are indicative of mycosis fungoides
- **Myeloproliferative neoplasms (MPNs)** are a group of disorders characterized by a proliferation of normally developed (nondysplastic) multipotent hematopoietic stem cells from the myeloid cell line
- **Cytoreduction for splenomegaly**: Options include hydroxyurea, cladribine, and interferon alpha.
- **Primary myelofibrosis** - any MPN leading to bone marrow fibrosis, extramedullary hematopoiesis, and splenomegaly
- A **"dry tap"** refers to the failure to obtain a fluid or tissue sample during a medical procedure, commonly seen in bone marrow aspirations, lumbar punctures, and joint fluid aspirations
- **Essential thrombocythemia** - isolated uncontrolled proliferation of platelets not caused by another condition (e.g., reactive thrombocytosis, another MPN)
- **Neutropenic fever** is an oncologic emergency common in patients receiving chemotherapy. A decrease in a patient's absolute neutrophil count (ANC) can lead to potentially life-threatening infections, and the risk of serious infection is directly associated with the extent and duration of neutropenia.

MSK AMBOSS

- **Rheumatoid arthritis (RA)** is a chronic, systemic, inflammatory autoimmune disorder that primarily affects the joints (e.g., causes pain, swelling, synovial destruction, deformities), but may also manifest with extraarticular features (e.g., rheumatoid nodules, pulmonary fibrosis).
- The diagnosis is clinical and may be supported by laboratory tests (e.g., rheumatoid factor, anticitrullinated peptide antibodies) and imaging studies (e.g., the presence of synovitis on ultrasound and, later in the disease course, bone erosions and/or joint space narrowing on x-rays).
- RA - Morning stiffness (often > 30 min) that usually improves with activity

- **Osteoarthritis** is a disabling joint disease characterized by degeneration of the joint complex (articular cartilage, subchondral bone, and synovium) that can have various causes, most notably advanced age and overuse. It mainly affects weight-bearing joints and joints that are heavily used, such as the hip, knee, hands, and vertebrae.
- Osteoarthritis pain - Pain during or after exertion (e.g., at the end of the day) that is relieved with rest; Morning joint stiffness usually lasting < 30 minutes
- **Asymmetric arthritis** → common in conditions like psoriatic arthritis, gout, reactive arthritis, or osteoarthritis.
- **Symmetric arthritis** → more typical of rheumatoid arthritis, lupus.
- **Vasculitides** are a heterogeneous group of rare autoimmune diseases characterized by blood vessel inflammation (vasculitis). Inflammation can lead to ischemia, necrosis, and/or hemorrhage, with subsequent end-organ damage.
- **Thromboangiitis obliterans (TAO), also known as Buerger disease**, is an inflammatory, nonatherosclerotic, vasoocclusive disease affecting small and medium-sized vessels of the extremities. TAO most commonly affects adult males with a significant history of tobacco consumption (e.g., smoking, chewing, vaping). In susceptible individuals, tobacco exposure causes inflammation of the tunica intima, with the formation of a highly cellular thrombus that occludes the affected vessel. Patients frequently present with intermittent claudication, Raynaud phenomenon, and migratory superficial thrombophlebitis. Eventually, critical limb ischemia develops and the patient presents with rest pain, absent pulse in the extremities, and/or digital ulcerations.
- Osteomalacia and rickets are caused by insufficient calcium, phosphate depletion, and/or direct inhibition of bone mineralization. The most common cause of both disorders is vitamin D deficiency.
- Patients with osteomalacia usually present with bone pain and tenderness, while patients with rickets exhibit bone deformities and impaired growth. Over time, both conditions may lead to bowing of the long bones and/or pathological fractures.
- **Caplan's syndrome** - RA + Pneumonoconiosis + lung nodules
- **Felty's syndrome** - RA + splenomegaly +neutropenia
- **Reiter's syndrome** - Reactive arthritis + Non gonococcal urethritis + Conjunctivitis
- **Jaccoud's Arthropathy** - a rare manifestation of SLE

Peripheral Smear

- Dacryocytes (Tear drop cells) - Extramedullary hematopoiesis
    - Conditions associated with bone marrow infiltration e.g. Myelofibrosis
    - Thalassemia
    - Splenomegaly
- Schistocytes
    - Microangiopathic hemolytic anemia (HUS, DIC, TTP)
    - Mechanical damage: Artificial cardiac valves
- Spherocytes
    - Hereditary Spherocytosis
    - Autoimmune hemolytic anemia
    - ABO incompatibility in infants

    - Hemolytic transfusion reaction
- Target cells
    - Thalassemia
    - Hemoglobin C and S
- Bite cells and Heinz bodies - G6PD Deficiency
- Howell Jolly bodies - Asplenia

SURGERY

Fluid therapy

- **Crystalloids**: solutions that contain small molecular weight solutes (e.g., minerals, dextrose)
- **Colloids**: solutions that contain larger molecular weight solutes (e.g., albumin and starch)
- **Balanced IV fluid solutions**: crystalloids or colloids that do not significantly alter the homeostasis of the extracellular compartment
- Tx of Hypovolemic hyponatremia - Administer isotonic saline to re expand the contracted intravascular volume
- Tx of euvolemic hyponatremia (water retention alone) - Tx is water restriction to 0.5 to 1 liter per day
- SIADH - water restriction to 1 liter per day
- Hypernatremia - Infusion of isotonic 5% dextrose or hypotonic 0.45% saline at an initial rate of 50-70 mL/hour. However in older institutionalized patients, it is more likely that the disorder has developed slowly, and extreme caution should be exercised in lowering plasma sodium to avoid risk of cerebral edema.
- Bolus in shock: 20 mL/kg normal saline

Maintenance Fluid Requirement (Holliday–Segar Formula)

- 100 mL/kg for first 10 kg
- 50 mL/kg for next 10 kg
- 20 mL/kg for remaining weight

Management of hyperkalemia

- 10ml of 10% calcium glucose to protect myocardium
- 50 ml of 50% glucose with 10 units of insulin or salbutamol 0.5mg in 100ml of 5% glucose is given. This will push potassium from ECF into cells.
- Acidosis is corrected by $NaHCO_3$, but excessive sodium bicarbonate may aggravate acidosis as it is converted in CO_2 inside the cells.
- Ion exchange resins are given to neutralize excessive potassium
- Dialysis in severe form

Burns

- 1st Degree - Superficial burns
- 2nd Degree - Superficial partial-thickness burns
- 3rd Degree - Deep partial-thickness burns
- 4th Degree - Full-thickness burns

Important Points

- Wallace Rule of 9
    - Head and neck 9%
    - Each upper limb 9%
    - Each lower limb 18%
    -
    - Each side of torso 18%
    - Perineum 1%
- Parkland Formula calculates fluid to be replaced within first 24 hours
    - TBSA x wt x 4
    - half is given in 8 hours
- Muir and Barklay formula is the most common colloid based formula
    - TBSA x wt x 0.5

PREPARATION OF THE SURGICAL SITE

- Removal of metals and other foreign bodies e.g rings and piercings
- Hair removal from the surgical site - makes postoperative plaster or dressing changes relatively pain free. But it causes microabrasions and can potentially cause cellulitis and SSI.
- Timing of hair removal - on the operating table after a dose of prophylactic antibiotic is given
- Skin antisepsis - use of alcohol-based antiseptic solution is recommended
- Draping

Basic haemostatic methods and the principles of electrosurgery

- Bleeding encountered during an operation can be arterial, venous or capillary
- Surgical haemorrhage is categorised as
    - primary (during the operation),
    - reactionary (24–48 hours postoperatively)
    - secondary (days to weeks postoperatively).
- Reactionary haemorrhage is usually a consequence of a slipped ligature or when a vessel injury is missed with bleeding temporarily stopped owing to a combination of vasoconstriction and vasodilation
- Secondary haemorrhage is often a manifestation of a deep-seated infection eroding into a blood vessel.

ELECTROSURGERY

- Electrosurgery employs high-frequency electrical current to assist in making surgical incisions, dissection of tissue and achieving haemostasis.

Safe electrosurgery

- Always check diathermy setting before use
- Use the safest, lowest diathermy current setting
- Be careful when diathermy is used near other metallic instruments
- Employ the diathermy intermittently and for brief spells
- Use bipolar diathermy and advanced vessel-sealing devices where appropriate
- Smoke extractors to remove bio-aerosolised particles are essential

DRAINS IN SURGERY

- To allow fluid that might collect in a body cavity to drain freely to the surface. The fluid to be drained may include blood, serum, pus, urine, faeces, bile, lymph or air. Abdominal drains are usually placed in the pelvis to drain collections as this is the most dependent area. Other locations are usually dictated by the pathology and procedure performed.
- For wound irrigation in certain circumstances.
- Their use can be regarded as prophylactic or therapeutic, depending on the circumstance warranting their insertion.
- Corrugated drain: Passive
- Romovac / Jackson-Pratt: Active (suction)
- Active drains reduce infection risk

Asepsis & Antisepsis

- Asepsis: Absence of microorganisms
- Antisepsis: Destruction/inhibition of microorganisms on living tissue
- Sterilization: Complete destruction of all forms of life including spores
- Disinfection: Destruction of pathogenic organisms (not spores)
- Best antiseptic for skin preparation: Chlorhexidine + alcohol
- Best antiseptic for wounds: Povidone-iodine (diluted)
- Most effective hand scrub: Chlorhexidine or povidone-iodine

Sterilization Methods

- Autoclaving (steam under pressure) - Kills spores
- Ethylene oxide - Heat-sensitive items
- Dry heat (hot air oven) - 160°C for 2 hrs
- Glutaraldehyde - High-level disinfectant

Surgical Instruments

- Cutting Instruments
    - Scalpel blade No. 10: Skin incisions
    - Blade No. 11: Stab incisions, abscess drainage
    - Blade No. 15: Fine incisions (plastic surgery)
- Grasping Instruments

    - Toothed forceps: Skin (prevents slippage)
    - Non-toothed forceps: Viscera, bowel
- Hemostatic Forceps
    - Mosquito: Small vessels
    - Kelly: Medium vessels
    - Kocher: Tough tissue (has tooth)
- Needle Holders
    - Mayo-Hegar: Most commonly used
    - Olsen-Hegar: Has scissors + needle holder

Sutures

- Absorbable Sutures
    - Catgut - enzymatic absorption
    - Vicryl (polyglactin) - 60–90 days absorption
    - PDS (Polydioxanone) - slow absorption i.e. Complete absorption at 180 days
- Non-Absorbable Sutures
    - Silk - used for ligation
    - Nylon - used for skin (Degraded by 15–20% per year)
    - Prolene (Polypropylene) - Non-absorbable - used for vascular, hernia
- Best suture for skin: Nylon
- Best for bowel anastomosis: Vicryl
- Best for fascia: PDS / Prolene
- Vascular anastomoses require smooth, non-absorbable, non-elastic material.
- Biliary anastomoses require an absorbable material that will not promote tissue reaction or stone formation
- Bowel anastomosis is usually performed using polyglactin, PDS or polypropylene

Surgical Needles

- Cutting needle: Skin
- Reverse cutting: Tough skin (less tear)
- Round body: Bowel, muscle
- Skin → Cutting needle
- Viscera → Round body needle

Knots

- Square knot: Most secure
- Surgeon’s knot: Extra turn for tension
- Granny knot: Weak
- Knot should be flat
- Adequate tension
- Short ends increase security

Types of Wounds

- Clean: Old infected wounds

- Clean-contaminated: Fresh trauma
- Contaminated: Controlled GI entry
- Dirty: Old infected wounds

Types of wound healing

- Clean wounds with a good blood supply heal by primary intention and so closure simply requires accurate apposition of the wound edges.
- However, if a wound is left open, it heals by secondary intention through the formation of granulation tissue, which is tissue composed of capillaries, fibroblasts and inflammatory cells. Wound contraction and epithelialisation assist in ultimate healing, but the process may take several weeks or months.
- Delayed primary closure or tertiary intention is utilised when there is a high probability of the wound being infected. The wound is left open for a few days and if the infective process is resolved then the wound is closed to heal by primary intention. Skin grafting is another form of tertiary intention healing.

Pre-operative Care

- NPO time: 6 hrs for solids, 2 hrs clear fluids
- Consent: Mandatory before surgery
- Prophylactic antibiotics: Within 1 hour before incision

Shock

- Hypovolemic (most common surgical shock)
- Septic
- Cardiogenic
- First fluid in shock: Crystalloids (Normal saline / Ringer lactate)

Laparoscopic surgery Advantages

- Less postoperative pain
- Shorter hospital stay
- Early ambulation
- Less wound infection
- Better cosmetic results

Laparoscopic surgery Disadvantages

- Expensive equipment
- Long learning curve
- Loss of tactile sensation

Pneumoperitoneum

- Gas Used: Carbon dioxide (CO_2) → gas of choice
- Normal Intra-abdominal Pressure: 12–15 mmHg (Adults)
- Why CO_2?

    - Non-combustible
    - Highly soluble in blood
    - Rapidly excreted via lungs
- Techniques for creation of pneumoperitoneum
    - Closed technique (Veress needle) – Most common
    - Open technique (Hasson) – Obese, previous surgery
- Energy Devices
    - Monopolar cautery: Cutting + coagulation
    - Bipolar cautery: Safer near vessels
    - Harmonic scalpel: Ultrasonic energy (no electric current)

Ports & Trocars

- Common Port Sites
    - Umbilical port: Camera port
    - Epigastric port: Working port
    - Subcostal ports: Retraction
- Sizes
    - Camera port: 10 mm
    - Working ports: 5 mm
- Telescope Angles
    - 0° scope: Straight view
    - 30° scope: Most commonly used
- Image System
    - Camera head
    - Light source (Xenon/LED)
    - Monitor - Monitor at eye level

Complications of Laparoscopy

- Access-related
    - Bowel injury
    - Vascular injury (most dangerous)
- Gas-related
    - CO_2 embolism (rare but fatal)
    - Subcutaneous emphysema
- Post-operative
    - Shoulder tip pain (phrenic nerve irritation)

Contraindications to Laparoscopy

- Absolute
    - Uncorrected coagulopathy
    - Severe cardiopulmonary disease
- Relative
    - Previous multiple abdominal surgeries
    - Severe abdominal distension

Robotic Surgery

- Computer-assisted surgery where a surgeon controls robotic arms from a console.
- Common System - Da Vinci robotic system
- Advantages of Robotic Surgery
    - 3D vision
    - Tremor filtration
    - Better ergonomics
    - Greater precision
- Disadvantages of Robotic Surgery
    - Very expensive
    - No tactile feedback
    - Longer setup time

Post-Operative Care

- Care given to a patient after surgery to ensure safe recovery, prevent complications, and restore normal function.
- Goals
    - Maintain airway, breathing, circulation
    - Pain control
    - Early detection of complications
    - Early mobilization & nutrition
- Immediate Post-Operative Period (0–24 hours)
    - Vital signs: Pulse, BP, RR, temperature
    - Oxygen saturation (SpO_2)
    - Urine output
    - Adequate urine output: ≥ 0.5 ml/kg/hr (adults)
- Airway & Respiratory Care
    - Most common immediate post-op complication: Atelectasis
    - Oxygen therapy given routinely
    - Encourage deep breathing & incentive spirometry
- Causes of Post-op Atelectasis
    - Pain
    - Shallow breathing
    - Immobilization
- Cardiovascular Care
    - Monitor for hypotension → bleeding / hypovolemia
    - Tachycardia is earliest sign of shock
    - ECG monitoring in high-risk patients
- Fluid & Electrolyte Management
    - First IV fluid post-op: Crystalloids (Normal saline / Ringer lactate)
    - Avoid fluid overload
- Pain Management
    - Multimodal analgesia preferred

    - NSAIDs + paracetamol
    - Opioids used cautiously
    - Epidural analgesia after major abdominal surgery
- Post-Operative Nutrition
    - Early oral/enteral feeding preferred
    - Start within 24 hours if possible
    - Parenteral nutrition only if enteral not possible
- Tubes, Drains & Catheters
    - Nasogastric (NG) Tube - Routine use NOT recommended. Remove immediately or within 24 hours
    - Urinary Catheter - Remove early to prevent UTI
    - Drains - Remove as early as possible
- Wound Care
    - Inspect wound daily
    - Signs of infection: redness, warmth, discharge
    - Wound Infection Timing: Usually appears day 3–5 post-o
- Thromboprophylaxis
    - Early mobilization
    - Compression stockings
    - Low molecular weight heparin (LMWH)
- Gastrointestinal Care
    - Post-op ileus is common
    - Early mobilization reduces ileus
    - NG decompression only if indicated
- Important Numbers & One-Liners
    - Urine output ≥ 0.5 ml/kg/hr
    - Most common immediate complication → Atelectasis
    - First sign of shock → Tachycardia
    - NG tube removal → Immediate / within 24 hours
    - Fever day 1 → Atelectasis
    - Avoid tubes, drains, and prolonged fasting

ATLS (Advanced Trauma Life Support)

- A systematic approach to the early assessment and management of trauma patients to identify and treat life-threatening injuries in order of priority.
- Treat the greatest threat to life first.
- Order of Trauma Care
    - Primary survey (ABCDE)
    - Resuscitation
    - Secondary survey (Head-to-toe)
    - Definitive care
- Primary Survey – ABCDE
- A – Airway with Cervical Spine Protection
    - First priority in trauma

    - Assume cervical spine injury until proven otherwise
    - Jaw thrust preferred (not head tilt)
    - MCQ: Most important first step → Airway
- B – Breathing
    - Life-threatening chest injuries (MCQ FAVORITES)
        - Tension pneumothorax
        - Open pneumothorax
        - Massive hemothorax
        - Flail chest
    - MCQ: Treatment of tension pneumothorax → Immediate needle decompression
- C – Circulation with Hemorrhage Control (VERY HIGH YIELD)
    - Most common cause of shock in trauma: Hypovolemic (hemorrhagic)
    - Control external bleeding
    - Insert 2 large-bore IV cannulas
    - First fluid → Crystalloids (Ringer lactate)
    - Earliest sign of shock → Tachycardia
- D – Disability (Neurological Assessment)
    - GCS assessment
    - Pupillary response
    - MCQ: Normal GCS = 15
- E – Exposure & Environment
    - Completely expose patient
    - Prevent hypothermia
- Adjuncts to Primary Survey
    - Pulse oximetry
    - ABGs
    - ECG monitoring
    - FAST scan
    - Portable X-ray (Chest & Pelvis)
- FAST Scan
    - Focused Assessment with Sonography for Trauma
    - Detects free fluid (blood)
    - Performed during Primary survey (C – Circulation)
    - Cannot differentiate blood from other fluid
- Secondary Survey
    - Detailed head-to-toe examination
    - History using AMPLE:
        - Allergies
        - Medications
        - Past illness
        - Last meal
        - Events
- Glasgow Coma Scale (GCS)
    - Eye opening (4)

    - Verbal response (5)
    - Motor response (6)
- Shock in Trauma
    - Hypovolemic (most common)
    - Cardiogenic
    - Neurogenic
    - Septic
    - Warm shock with hypotension & bradycardia: Neurogenic shock
- Abdominal Trauma
    - FAST positive + unstable patient → Immediate laparotomy
    - Stable patient → CT scan

ORTHOPEDICS

Tuberculous Arthritis (TB Arthritis)

- Chronic granulomatous infection of joints caused by Mycobacterium tuberculosis
- Usually secondary to pulmonary or extrapulmonary TB
- Spread is hematogenous
- Common in developing countries
- Seen in children & young adults
- MCQ: TB arthritis is monoarticular, RA is polyarticular
- Risk factors:
    - Immunosuppression
    - Malnutrition
    - HIV
    - Previous TB
- Commonly Affected Joints (MCQ FAVORITE)
    - Hip – MOST COMMON
    - Knee
    - Ankle
    - Elbow
    - Wrist
- Pathogenesis
    - Hematogenous spread to synovium
    - Synovitis → pannus formation
    - Cartilage destruction
    - Bone erosion → joint space narrowing
- Clinical Features
    - Insidious onset
    - Chronic pain
    - Swelling
    - Restricted movements
    - Muscle wasting

    - Minimal warmth
    - Cold abscess may be present
    - 📌 MCQ: TB arthritis has less pain & less redness than pyogenic arthritis
- Classic Deformities / Signs
    - Flexion, adduction & internal rotation of hip
    - Night pain and stiffness
    - Sinus formation in advanced disease
- Radiological Features (Phemister's Triad – VERY IMPORTANT)
    - Juxta-articular osteopenia
    - Peripheral erosions
    - Gradual narrowing of joint space
    - 📌 MCQ: Phemister triad is seen in → Tuberculous arthritis
- Early X-Ray Findings
    - Soft tissue swelling
    - Osteopenia
- Late X-Ray Findings
    - Joint space narrowing
    - Marginal erosions
    - Subluxation / ankylosis
- Laboratory Findings
    - Raised ESR
    - Mild anemia
    - Mantoux test → positive
    - Synovial fluid:
        - Straw colored
        - High protein
        - Lymphocyte predominance
- Diagnosis (MCQ Focus)
    - Gold standard: Synovial biopsy showing caseating granuloma
    - AFB culture
    - PCR for TB (CBNAAT)
    - MCQ: Most confirmatory test → Synovial biopsy
- Medical Management (Mainstay)
    - Anti-tuberculous therapy (ATT)
    - Intensive phase: 2 months (HRZE)
    - Continuation: 7–10 months (HR)
    - Total duration: 9–12 months
- Surgical Indications
    - Diagnostic biopsy
    - Abscess drainage
    - Severe deformity
    - Non-responsive disease

📌 MCQ: Treatment of choice → ATT

- MCQs Rapid Review (One-Liners)

    - Most common joint → Hip
    - Radiological triad → Phemister triad
    - Confirmatory diagnosis → Synovial biopsy
    - Nature of disease → Chronic, monoarticular
    - Best treatment → Anti-tuberculous therapy
    - Type of abscess → Cold abscess

Pott's disease (Tuberculous spondylitis)

- Tuberculosis of the spine, most commonly affecting the thoracic > lumbar vertebrae.
- Caused by Mycobacterium tuberculosis spreading hematogenously (usually from lungs)
- Pathology / Spread
    - Starts in anterior part of vertebral body → caseation → collapse.
    - Intervertebral disc involved late (unlike pyogenic spondylitis).
    - Can spread under anterior/posterior longitudinal ligaments → skip lesions.
    - Cold abscess formation (e.g., psoas abscess).
- Common Sites
    - Thoracic (most common)
    - Lumbar
    - Cervical (least common but dangerous)
- Clinical Features
    - Chronic back pain (most common)
    - Local tenderness, stiffness
    - Constitutional symptoms: fever, weight loss, night sweats
    - Gibbus deformity (sharp kyphosis) due to vertebral collapse
    - Neurological deficits (Pott's paraplegia) due to:
        - Cord compression
        - Epidural abscess
        - Vertebral collapse
- Cold Abscess Tracking (MCQ favorite)
    - Cervical → retropharyngeal abscess
    - Thoracic → paravertebral abscess
    - Lumbar → psoas abscess (may present in groin)
- High-yield MCQs
    - Most common spinal TB site → Thoracic spine
    - Disc involvement in TB → Late
    - Imaging of choice → MRI
    - Sharp angular kyphosis → Gibbus
    - Lumbar TB abscess → Psoas abscess
    - Cause of paraplegia → Cord compression

Poliomyelitis

- Acute viral infection caused by Poliovirus (Enterovirus)
- Primarily affects anterior horn cells of spinal cord
- Leads to lower motor neuron (LMN) paralysis

- MCQ: Structure affected → Anterior horn cells
- Types (MCQ FAVORITE)
    - Abortive polio – mild, no paralysis
    - Non-paralytic polio – aseptic meningitis
    - Paralytic polio
        - Spinal (most common)
        - Bulbar
        - Bulbo-spinal
    - 📌 MCQ: Most common type → Spinal poliomyelitis
- Mode of Spread - Feco-oral route
- Age Group - Mostly children < 5 years, Unvaccinated population
- Pathology - Destruction of motor neurons results in:Muscle weakness, Muscle wasting, Flaccid paralysis
- MCQ: CSF glucose in polio → Normal
- MCQ: IM injections in polio → Contraindicated
- Acute Stage
    - Fever
    - Headache
    - Myalgia
    - Neck stiffness
    - Followed by paralysis
- Paralytic Stage
    - Asymmetric acute flaccid paralysis worsens over hours to days
    - No sensory loss
    - Areflexia
    - Muscle wasting
    - Fasciculations
- Sensory loss in polio → Absent
- Type of paralysis → LMN flaccid paralysis
- MCQ: Most commonly affected limb → Lower limb
- Characteristic Features (MCQ GOLD)
    - Asymmetrical involvement
    - Proximal muscles > distal
    - Lower limbs more commonly affected than upper limbs
    - No bladder/bowel involvement
- Deformities Caused by Polio (EXAM FAVORITE)
    - Foot drop - Foot remains in plantarflexed position
    - Equinus deformity - Foot is fixed in plantarflexion (heel does not touch the ground).
    - Calcaneus deformity - Foot is fixed in dorsiflexion (heel bears excessive weight).
    - Genu recurvatum - Hyperextension of the knee beyond normal limits while standing.
    - Scoliosis - A lateral curvature of the spine with vertebral rotation
    - Limb length discrepancy

    - 📌 MCQ: Most common deformity → Foot drop
- Stages of Poliomyelitis (Orthopaedic View)
    - Acute stage
    - Recovery stage
    - Residual paralysis stage
    - 📌 MCQ: Correct time for surgery → Residual paralysis stage

MCQs Rapid Fire (One-Liners)

- Virus → Poliovirus
- Nerve cell affected → Anterior horn cell
- Paralysis type → LMN
- Sensory loss → Absent
- Most common limb → Lower limb
- Most common deformity → Foot drop
- Surgery timing → Residual stage
- Common surgery → Tendon transfer

Congenital Talipes (Clubfoot)

- Congenital deformity of the foot where it is twisted out of normal position.
- Classic mnemonic: CAVE
    - C – Cavus (high medial longitudinal arch)
    - A – Adduction (forefoot toward midline)
    - V – Varus (hindfoot inverted)
    - E – Equinus (plantarflexion at ankle)
- Types
    - Idiopathic (most common) – Isolated deformity, usually bilateral.
    - Syndromic / Neuromuscular – Associated with: Spina bifida, Arthrogryposis, Cerebral palsy
- Clinical Features
    - Foot smaller than normal
    - Medial crease on plantar aspect
    - Forefoot adducted, hindfoot varus, equinus
    - “Tiptoe walking” if untreated
    - Most common type: Talipes Equinovarus.
- Non-surgical Management
    - Ponseti method: Gold standard
        - Weekly manipulation + casting
        - Achilles tenotomy often needed
        - Foot abduction brace for maintenance
    - French functional method: Daily physiotherapy, stretching, taping, splinting
- Surgical Management
    - Reserved for resistant cases or relapse
    - Soft tissue releases, tendon lengthening, osteotomies
- MCQ High-Yield Points
    - Most common congenital foot deformity → Clubfoot (talipes equinovarus)

    - First-line treatment → Ponseti casting
    - Male predominance, often bilateral
    - CAVE mnemonic describes the deformity

Quick ortho MCQ pearls

- Foot drop (Foot remains in plantarflexed position) → Peroneal nerve
- Toe walking → Equinus - Foot is fixed in plantarflexion (heel does not touch the ground).
- Heel walking → Calcaneus (Foot is fixed in dorsiflexion i.e. heel bears excessive weight)
- Knee hyperextension → Genu recurvatum
- Varus: Distal part deviates towards the midline.
- Valgus: Distal part deviates away from the midline.
- Rib hump → Scoliosis
- Functional scoliosis → LLD
- Coxa vara – Neck-shaft angle < 120°.
- Coxa valga – Neck-shaft angle > 135°.
- Hindfoot varus – Heel tilted inward (cavovarus).
- Hindfoot valgus – Heel tilted outward (planovalgus).
- Forefoot varus – Forefoot inverted relative to hindfoot.
- Forefoot valgus – Forefoot everted relative to hindfoot.
- Talipes equinovarus (Clubfoot) – Cavus, Adduction, Varus, Equinus.
- cavovarus (Cavus → high medial longitudinal arch) (Varus → hindfoot heel inversion)
- High arch (pes cavus)
- Cubitus varus
    - “Gunstock deformity”
    - Elbow deviates medially
    - Common after supracondylar fracture humerus
- Cubitus valgus
    - Elbow deviates laterally
    - Complication: ulnar nerve palsy
    - Seen after lateral condyle fracture

Cavovarus common causes

- Neurological disorders (most common)
    - Charcot–Marie–Tooth disease (classical association)
    - Old polio
    - Spinal cord lesions
    - Brain tumors, stroke
- Post-traumatic imbalance

T Score

- T-score compares a patient's bone mineral density (BMD) with that of a young healthy adult (20–30 years) of the same sex.
- Expressed in standard deviations (SD).
- ≥ –1.0 → Normal
- –1.0 to –2.5 → Osteopenia
- ≤ –2.5 → Osteoporosis
- ≤ –2.5 + fragility fracture → Severe (Established) osteoporosis
- 📌 MCQ: Osteoporosis defined as T-score → ≤ –2.5
- Sites Used for T-Score Measurement
    - Hip (femoral neck) – preferred
    - Lumbar spine
    - Distal radius
- 📌 MCQ: Gold standard test → DEXA scan
- T-score NOT used in:
    - Children
    - Premenopausal women

Fractures Important Points

- Most common hip fracture → Intertrochanteric
- Blood supply to femoral head → Medial circumflex femoral artery
- Most common complication of neck femur fracture → AVN
- Implant for intertrochanteric fracture → DHS
- Gold standard for femoral shaft fracture → Interlocking nail
- Classic sign of hip fracture → Externally rotated limb
- Intracapsular fracture → AVN risk
- Extracapsular fracture → Good healing
- Elderly + neck femur fracture → Hemiarthroplasty
- Fat embolism occurs 24–72 hours after fracture

Posterior Dislocation of Hip Joint

- Typical History
    - High-energy trauma (most common)
    - Road traffic accident
- Dashboard injury (knee hits dashboard)
- Symptoms
    - Severe hip pain
    - Inability to move limb
    - Inability to bear weight
- Most common hip dislocation → Posterior
- Limb position → Flexed, adducted, internally rotated
- Common nerve injury → Sciatic nerve
- Emergency complication → Avascular necrosis
- Time limit for reduction → Within 6 hours

- Anterior dislocation → limb is abducted & externally rotated
- Posterior dislocation → limb is adducted & internally rotated

Specific Fractures

- Colles fracture→ Distal radius, dorsal displacement, dinner-fork deformity
- Smith fracture → Reverse Colles, volar displacement
- a Colles fracture involves dorsal (backward) displacement, often from a fall on an outstretched hand, creating a "dinner fork" deformity; a Smith fracture is the opposite, with volar (forward/palm-side) displacement, typically from a fall on a flexed wrist, looking like a "garden spade".
- Galeazzi fracture–dislocation → Fracture of distal radius + DRUJ dislocation (DRUJ is distal radioulnar joint)
- Supracondylar fracture (children)→ Median nerve + brachial artery injury
- Boxer's fracture → Neck of 5th metacarpal
- Mallet fracture → Avulsion fracture distal phalanx (extensor tendon)
- Neck of femur fracture → AVN & non-union risk
- Intertrochanteric fracture → Most common hip fracture in elderly
- Subtrochanteric fracture→ High stress area, difficult union
- Pott's fracture → Bimalleolar ankle fracture
- Hangman's fracture → Fracture of pars interarticularis of C2

PRIME

Steps of Evidence-Based Medicine (5 A's)

- **Ask** - Formulate a clear, focused clinical question using PICO:
    - P – Patient / Problem
    - I – Intervention
    - C – Comparison
    - O – Outcome
- **Acquire** - Search for best available evidence:
    - PubMed
    - Cochrane Library
    - Google Scholar
    - Clinical guidelines
- **Appraise** - Critically assess evidence for: Validity, Importance, Applicability
- **Apply** - Apply evidence considering:Patient values, Clinical circumstances, Resources available
- **Assess** - Evaluate outcomes and performance.

Hierarchy (Levels) of Evidence

- Level I (Highest)
    - Systematic reviews
    - Meta-analyses
    - Randomized Controlled Trials (RCTs)
- Level II
    - Cohort studies
- Level III
    - Case-control studies
- Level IV
    - Case series
    - Case reports
- Level V (Lowest)
    - Expert opinion
    - Animal studies

Types of Studies in Evidence Based Medicine

- Experimental Studies
    - Randomized Controlled Trials (RCTs)
    - Clinical trials
- Observational Studies
    - Cohort
    - Case-control
    - Cross-sectional