Focused Review of Special Surgical Disciplines

- Urology
- Traumatology
- Ophthalmology
- Otorhinolaryngology
- Neurosurgery
- Anesthesiology
- Transplantology

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2nd Edition

Annotation:

English :

"Focused Review of Special Surgical Disciplines" provides a concise overview of key surgical specialties, including orthopedics and traumatology, urology, otorhinolaryngology, anesthesiology, ophthalmology, neurosurgery, and transplantology. The materials in this book are compiled from leading surgical textbooks, with a reference list provided at the end. As a continuation of the first book,
"Focused Review of Surgical
Diseases," this volume serves as a valuable guide for students preparing for licensing exams. It is specifically designed for senior medical students seeking a structured and comprehensive review of special surgical disciplines.

Кыргызча Аннотация:

"Focused Review of Special Surgical Disciplines"
китеби хирургиянын атайын тармактары боюнча кыскача баяндама берет. Ага ортопедия жана травматология, урология, оториноларингология, анестезиология, офтальмология, нейрохирургия жана трансплантология кирет. Китептин материалдары негизги медициналык текстбуктардан топтолгон жана алардын тизмеси китептин аягында берилген. Бул чыгарма "Focused Review of Surgical Diseases" китебинин уландысы болуп эсептелет жана лицензиялык экзамендерге даярдануу учун мыкты гид боло алат. Китеп жогорку курстагы медицина студенттерине арналган.

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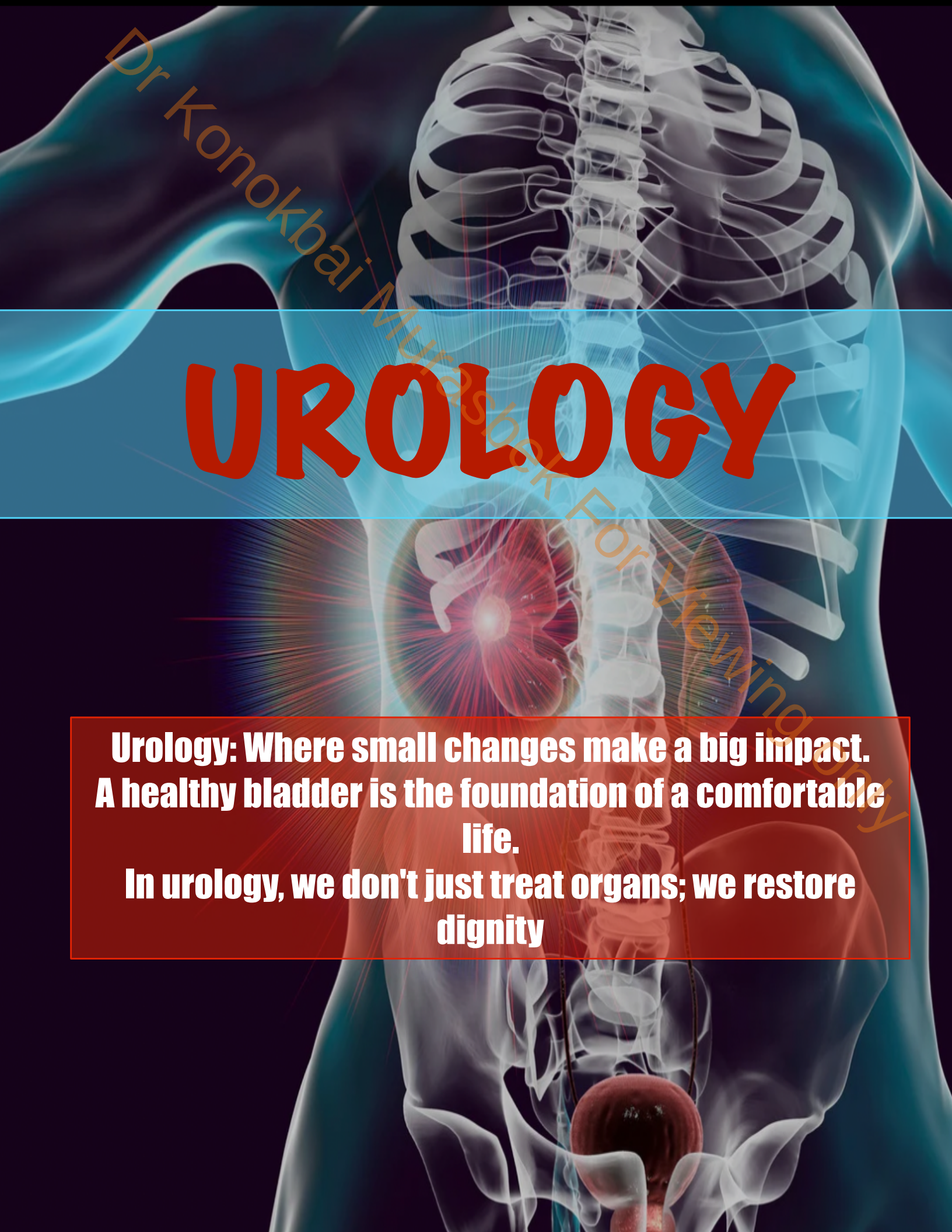


UROLOGY

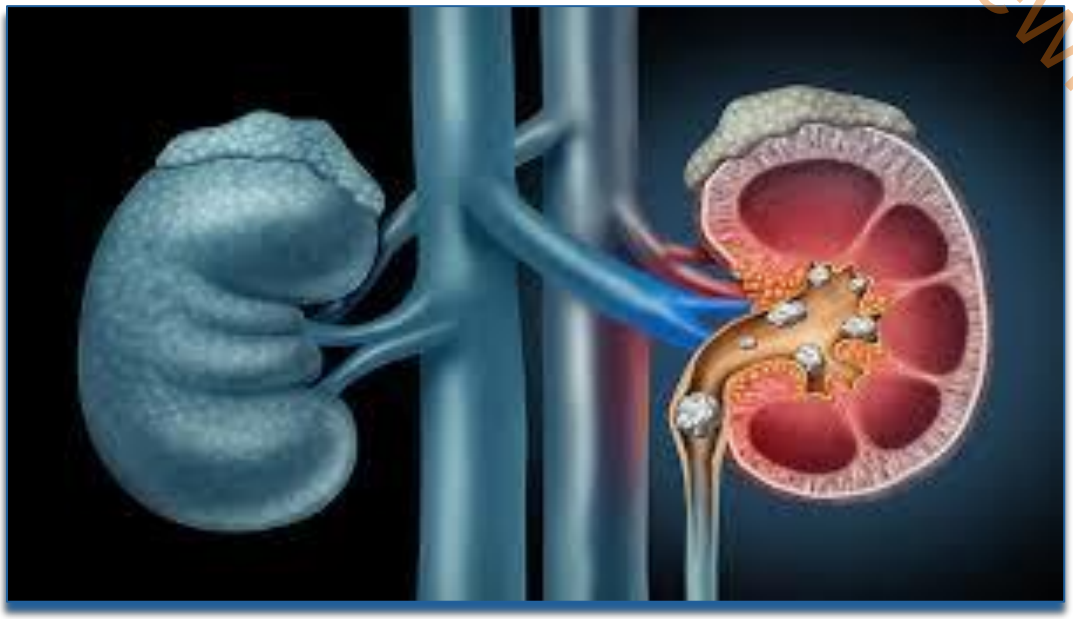
Urology: Where small changes make a big impact.
A healthy bladder is the foundation of a comfortable life.
In urology, we don't just treat organs; we restore dignity

Unit : 1

STONEDISEASE
KIDNEY AND URETERAL STONES

Etiology

- Male/Female ratio: 2-3:1 (infection stones are more common in women due to higher urinary tract infection rates).
- Directly related to body mass index.
- Stones are polycrystalline masses made up of varying amounts of crystalloid and organic matrix.
- Supersaturated urine is required for stone formation.
- Supersaturation depends on urine pH, ionic strength, solute concentration, and the ability to form complexes.



Crystal Component

- Stones primarily consist of a crystallized component.

Matrix Component

- The amount of matrix component varies depending on the type of urinary stone and constitutes 2-10% of the stone's weight.
- It is mainly composed of protein with small amounts of hexose and hexosamine.
- On a plain abdominal X-ray, matrix stones are often radiolucent and can be confused with filling defects such as blood clots, upper urinary tract tumors, and fungal bezoars.

Inhibitors (substances in urine that reduce stone formation):

- Nephrocalcin, uropontin, Tamm-Horsfall proteins.

Types of Stones and Their Characteristics

1. Calcium Stones

Factors Facilitating Calcium Stone Formation:

- **Hypercalciuria**: Defined as daily urinary calcium excretion exceeding 200 mg despite an appropriate diet. Types include absorptive, resorptive, and renal hypercalciuria.
- **Hyperoxaluria**: Urinary oxalate levels exceeding 40 mg. Causes include primary hyperoxaluria, malabsorption in inflammatory bowel diseases, celiac disease, intestinal resections, and excessive dietary oxalate or its precursors.
- **Hyperuricosuria**: Increased uric acid in urine is associated with calcium stones.
- **Hypocitraturia**: Low urinary citrate levels.
- **Low urine pH**.
- **Renal tubular acidosis (type 1 is the most common cause of stones)**.

2. Non-Calcium Stones

Struvite Stones:

- Composed of magnesium, ammonium, and phosphate (MAP).
- Often found as staghorn stones in the kidneys.
- Associated with urease-producing bacteria such as *Proteus*, *Pseudomonas*, *Providencia*, *Klebsiella*, *Staphylococcus*, and *Mycoplasma*.
- These bacteria alkalinize the urine.
- Urine pH in MAP stone patients is typically between 6.8-8.3 and rarely below 7.0.

- Intense diuresis does not prevent struvite stones.
- Removal of the stone is curative.

Uric Acid Stones:

- Low pH, low urine volume, and hyperuricosuria are the three main causes.
- Common in patients with gout, myeloproliferative diseases, and those treated with cytotoxic drugs for malignancies.
- High uric acid levels are often due to dehydration and excessive purine intake.
- Urine pH is consistently below 5.5 in these patients.
- Pure uric acid stones are radiolucent.

Cystine Stones:

- Caused by a hereditary defect in intestinal mucosal absorption and renal tubular reabsorption of dibasic amino acids, including cystine, ornithine, lysine, and arginine.
- Family history of stones, round and smooth-bordered stones resembling watch glasses on X-ray suggest cystine stones.
- A low-methionine diet is necessary.
- Cystine stones are resistant to ESWL (extracorporeal shock wave lithotripsy).
- They are the hardest stones.

Xanthine Stones:

- Caused by congenital xanthine oxidase deficiency.
- Radiolucent.

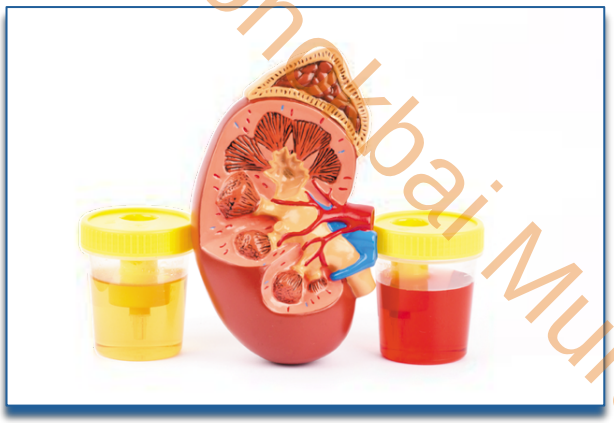
Medication-Induced Stones:

- Associated with drugs such as indinavir, triamterene, guaifenesin, and ephedrine.

Symptoms and Findings :

1.Pain

- There are two types of pain: renal colic and non-colic pain.
- Stretching of the collecting system or ureter usually causes renal colic. In contrast, non-colic pain results from stretching of the renal capsule.
- As the stone moves down the ureter, localized pain is felt in areas innervated by the ilioinguinal nerve and the genital branch of the genitofemoral nerve.



2.Hematuria- the presence of blood in the urine. It can be classified into two types:

Gross Hematuria-visible blood in the urine, causing it to appear pink, red, or cola-colored.

Microscopic Hematuria-blood is present in the urine but only detectable under a microscope.

3.Infection in urine tract (Lower UTI):

- Dysuria (burning sensation during urination).
- Increased frequency and urgency of urination.
- Cloudy or foul-smelling urine.
- Suprapubic pain.

Radiological Examinations:

Plain X-rays (KUB) and Targeted Ultrasonography

- These are the first imaging methods that should be requested.
- A plain X-ray of the kidneys, ureters, and bladder (KUB) combined with renal ultrasonography can be as effective as IVP in diagnosis.

Computed Tomography (CT)

- The best imaging method.
- Does not require the use of intravenous contrast.
- Uric acid stones can be visualized similarly to calcium oxalate stones.

Intravenous Pyelography (IVP)

- Provides detailed imaging of the urinary tract using contrast agents.

Retrograde Pyelography

- May be required for small radiolucent stones that are difficult to detect using other methods.

Nuclear Scintigraphy

- Injection of bisphosphonate markers allows for the identification of even small stones that are difficult to detect on conventional KUB X-rays.

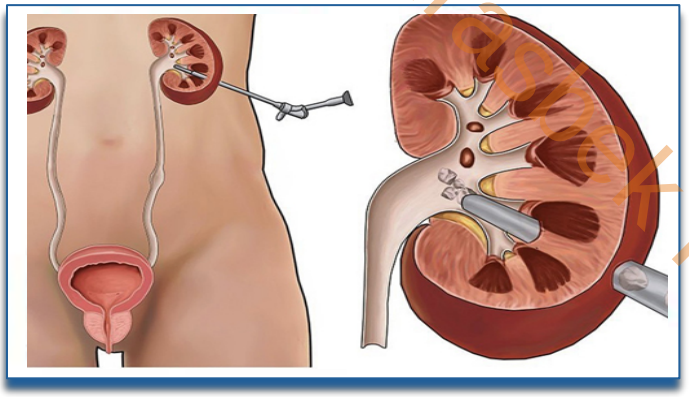
Treatment

Medical Treatment

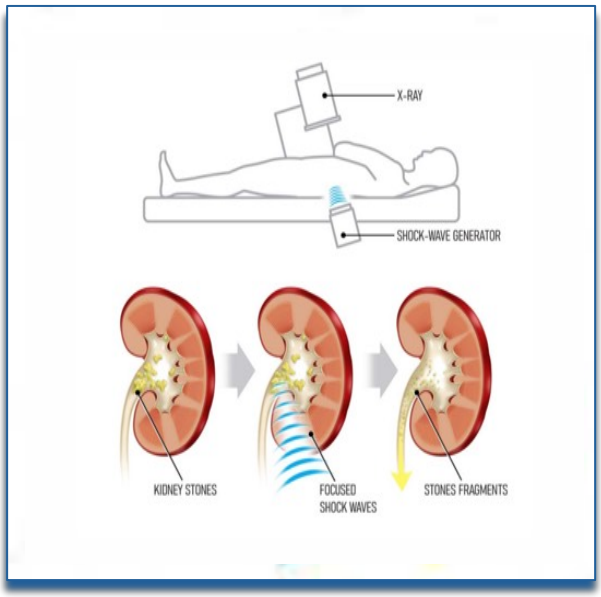
- Fluid Intake: At least two liters daily.
- Fruit Juices: Lemon and orange juice are recommended.
- Dietary Adjustments: Reduce animal protein and salt intake.

Interventional Treatment

- Staghorn Calculi: These are infection stones, and the primary approach should be **percutaneous nephrolithotomy** to prevent the spread of infection.



- Small Ureteral Stones (<5 mm): Since they are more likely to pass spontaneously, the best approach is to wait and observe.
- Larger Stones: **Extracorporeal Shock Wave Lithotripsy (ESWL)** or ureteroscopy may be performed.



Contraindications for Shock Wave Lithotripsy (SWL)

- Pregnancy
- Uncorrected Coagulopathy or Bleeding Diathesis
- Arterial Aneurysm Close to the Stone (renal or abdominal aortic aneurysm)
- Urinary System Obstruction Distal to the Stone
- Technical Problems in Targeting the Stone (e.g., skeletal anomalies)

Unit : 2

GENITOURINARY TUMORS

Bladder Carcinomas

General Information

- Bladder cancer is the second most common cancer of the genitourinary tract.

Risk Factors and Pathogenesis

- **Smoking**: Accounts for approximately 50% of cases in men and 31% in women.
- The etiological agents are thought to be aromatic amines such as alpha or beta naphthylamine, excreted in the urine of smokers.
- **Infection, instrumentation, and physical trauma** induced by stones increase the risk of malignancy.

Histopathology

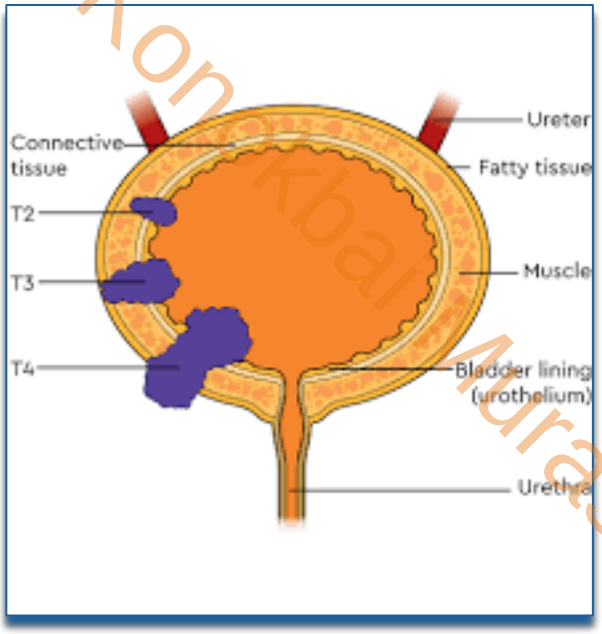
- The most common type is **transitional (urothelial) cell carcinoma**.

Symptoms

- **Painless clotted hematuria** is the typical presentation.
- Hematuria may be **microscopic or macroscopic** and is often intermittent rather than continuous.

Laboratory Findings

- **Routine tests**: Hematuria is the most frequently observed laboratory abnormality.
- **Urine cytology**: Cells from both normal and neoplastic urothelium can be easily identified. However, a marker with sufficiently high sensitivity and specificity has not yet been defined.
- **IMAGING**



Tumor Staging (Bladder Cancer)

- **Tx**: Primary tumor cannot be assessed.
- **T0**: No evidence of primary tumor.
- **Tis**: Carcinoma in situ (flat tumor).
- **T1**: Tumor invades the subepithelial connective tissue.
- **T2**: Tumor invades the muscle.
- **T3**: Tumor invades peri vesical tissue.
- **T4**: Tumor invades adjacent structures such as the prostate, uterus, vagina, pelvic wall, or abdominal wall.

Treatment

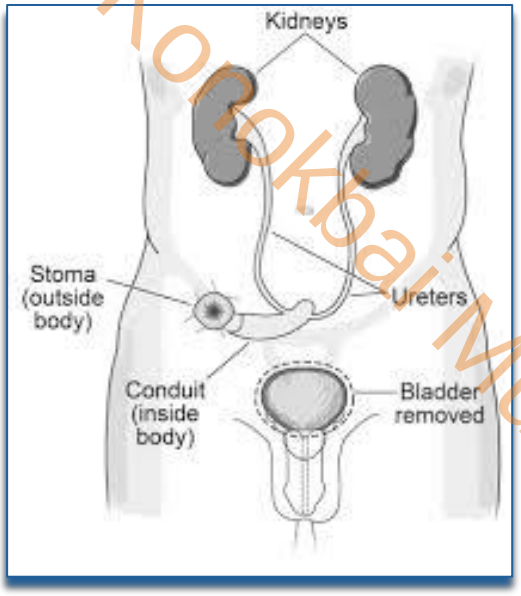
Intravesical Chemotherapy

- Therapeutic or chemotherapeutic agents are delivered directly into the bladder via catheter to avoid the morbidity of systemic administration.

1. **Calmette-Guérin Bacillus (BCG):**
    - Most commonly preferred.
2. **Mitomycin C:**
    - An anticancer alkylating antibiotic that inhibits DNA synthesis.
3. **Thiotepa:**
    - An alkylating agent.

Surgical Treatment

- **Transurethral Resection (TUR):**
    - The initial treatment for all bladder cancers.
    - Allows reasonably accurate estimation of tumor stage, grade, and the necessity for additional therapy.
- **Partial Cystectomy:**
    - Suitable for solitary, infiltrating tumors (T1-T3) located in the posterior lateral wall, dome of the bladder, or diverticular cancers.



- **Radical Cystectomy:**
  - Involves the removal of anterior pelvic organs.
  - In men: Includes the removal of surrounding fatty tissue, peritoneal attachments, prostate, and seminal vesicles.
  - In women: Includes the removal of the bladder, surrounding fatty tissue, peritoneal attachments, cervix, uterus, anterior vaginal wall, urethra, and ovaries.

Malignant Renal Tumors

Renal Cell Carcinoma (RCC)

- Accounts for approximately 85% of all primary malignant renal tumors.
- The male/female ratio is 2:1.
- Risk factors associated with RCC include tobacco exposure, obesity, and hypertension.
- Renal cell cancer is traditionally known to originate from the proximal tubular epithelium.
- Two aggressive subtypes of renal cell carcinoma, renal medullary carcinoma and collecting duct carcinoma, affect more distal parts of the nephron (In Robbins Pathology, it is stated that the "papillary subtype most commonly affects the distal tubule").

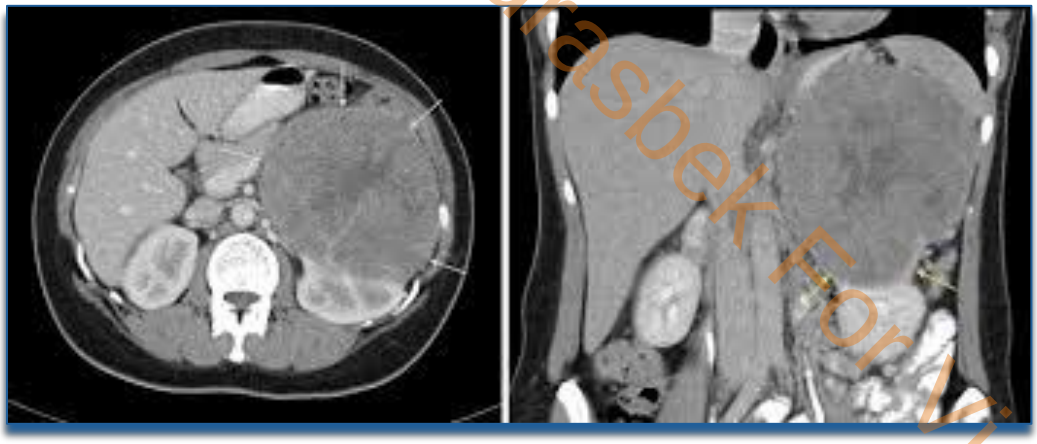
Clinical Findings

Incidental (050)

- Due to local tumor growth:
  - Hematuria
  - Flank pain
  - Abdominal mass
  - Perirenal hematoma
- Due to metastasis:
  - Persistent cough
  - Bone pain

- Cervical lymphadenopathy (LAP)
- Fever, weight loss, fatigue

- Paraneoplastic syndromes:
  - Hypercalcemia
  - Hypertension
  - Polycythemia
  - Stauffer syndrome



Treatment

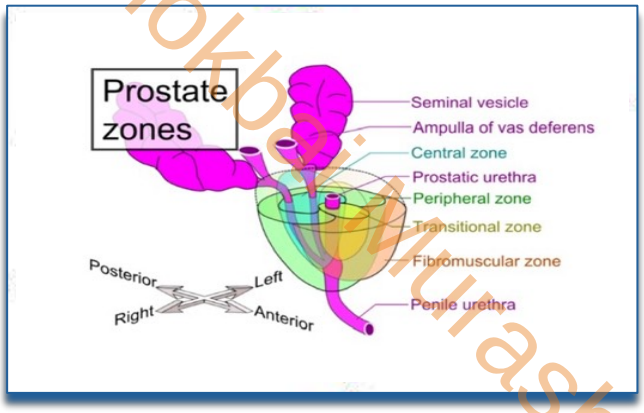
- The primary treatment for localized RCC (T1 and T2) is partial or radical nephrectomy.
- The goal is to remove the tumor and the surrounding normal tissue.
- Radical nephrectomy involves removal of the entire kidney along with the surrounding fascia (Gerota's fascia), the ipsilateral adrenal gland, the proximal upper segment of the ureter, and the lymph nodes surrounding the renal vessels.
- Approximately 30% of RCC patients are diagnosed with metastatic disease. **I- Surgical Treatment:** In the presence of metastatic disease, nephrectomy (cytoreductive nephrectomy) can be performed using open or laparoscopic techniques.

II- Radiotherapy

III- Biological Response Modifiers: Recombinant interferon-alpha, interleukin-2 (IL-2)

Benign Prostatic Hyperplasia (BPH)

BPH is the most common benign tumor in men, and its incidence is related to age.



Etiology

- The etiology of BPH is under endocrine control.
- BPH develops in the transitional (transitional epithelial cell) zone.
- In patients with significant smooth muscle components, they respond to alpha blocker therapy.
- Patients with BPH consisting of epithelial cells respond better to 5-alpha reductase inhibitors.
- Patients with a significant collagen component in the stroma may not respond well to either of the medical treatments.

Clinical Findings

- The symptoms of BPH can be divided into obstructive and irritative complaints.
- **Obstructive symptoms** include hesitancy, a reduction in the force and caliber of urine flow, a feeling of incomplete bladder emptying, double voiding (urinating again within 2 hours after the first urination), difficulty urinating, and post-void dribbling.
- **Irritative symptoms** include urgency, frequent urination, and nocturia.

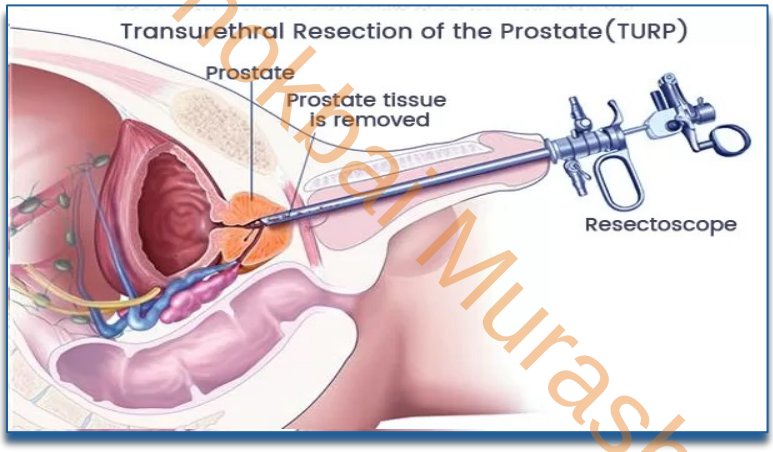
Treatment

Medical Treatment

- **Alpha blockers**: Phenoxybenzamine and prazosin
- **5-alpha reductase inhibitors**: Finasteride

Surgical Treatment

- **Conventional Surgical Treatment**



Transurethral resection of the prostate (TURP)

- Simple prostatectomies can be performed endoscopically in 95% of cases.
- Risks of TURP include retrograde ejaculation, impotence, and incontinence.
- Complications include bleeding, urethral stricture or bladder neck contracture, extravasation caused by perforation of the prostate capsule, and TUR syndrome (resulting from the absorption of hypotonic irrigation solutions if perforation is severe).
    - Clinical signs of TUR syndrome include nausea, vomiting, confusion, hypertension, bradycardia, and visual disturbances.
    - The risk of TUR syndrome increases with resection times exceeding 90 minutes. Treatment consists of diuresis and hypertonic saline in severe cases.

Open simple prostatectomy

- If the prostate is too large to be removed endoscopically, enucleation through open surgery is required.
- Open enucleation is typically considered for prostates weighing over 100 grams.

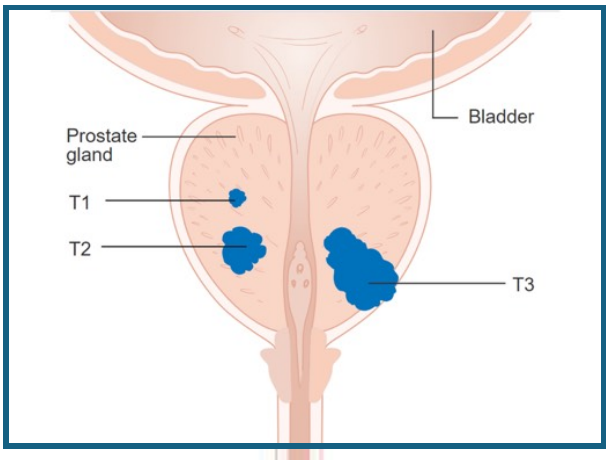
PROSTATECANCER

- Prostate cancer is the most commonly diagnosed cancer type in men.
- Among all cancers, prostate cancer exhibits the most rapid increase with age. Unlike most cancers, which peak in incidence at a certain age, the incidence of prostate cancer continues to rise with advancing age.
- At the age of 50, the lifetime risk of latent prostate cancer (incidentally detected at autopsy and unrelated to the cause of death) is approximately 40%.

- The likelihood of developing prostate cancer in men under 40 is 1/10,000, between 40-59 years is 1/103, and between 60-79 years is 1/8.

Pathology

- More than 95% of prostate cancers are adenocarcinomas.
- Between 60-70% of prostate cancer cases originate from the peripheral zone.
- The Denonvilliers fascia serves as a strong barrier, making rectal involvement rare.
- Lymphatic metastasis is most commonly identified in the obturator lymph node chain.
- Distant metastases are often seen in the axial skeleton bones, most frequently involving the lumbar spine.
- Other commonly affected areas, in descending order, include the proximal femur, pelvis, thoracic spine, ribs, sternum, skull, and humerus.
- Bone lesions in metastatic prostate cancer are typically osteoblastic in nature.



TX: Tumor cannot be assessed
T0: No evidence of primary tumor
Tis: Carcinoma in situ (Prostatic Intraepithelial Neoplasia - PIN)
T1a: Cancer is found in <5% of the tissue resected for benign disease, with normal Prostate Rectal Margin (PRM)
T1b: Cancer is found in >5% of the tissue resected for benign disease, with normal PRM
T1c: Detected only through elevated PSA levels, with normal PRM and transrectal ultrasound (TRUS)
T2a: Tumor is palpable via PRM or visible on TRUS and confined to one side of the prostate
T2b: Tumor is palpable via PRM or visible on TRUS and present on both sides of the prostate, but still confined to the prostate
T3a: Extracapsular extension on one or both sides of the prostate
T3b: Involvement of the seminal vesicles
T4: Tumor directly invades the bladder neck, external sphincter, rectum, levator muscles, or pelvic sidewall

Clinical Findings

- Most patients with early-stage prostate cancer are asymptomatic.
- Trouble urinating – commonly including frequent urination and slow or weak urine stream
- Erectile dysfunctions
- Blood in the urine or semen

- Advanced prostate tumors can metastasize to nearby lymph nodes and bones, particularly in the pelvis, hips, spine, ribs, head, and neck

Treatment

Localized Disease

- **Careful observation and active surveillance:** Although local cancer can progress, disease-specific mortality is generally low in early-stage prostate cancer under careful observation, ranging between 4% and 15% at 10 years.
- **Radical prostatectomy and radiotherapy** (external beam radiotherapy)
- **Cryosurgery and high-intensity focused ultrasound (HIFU)**

Metastatic Disease

- **Initial hormonal therapy**.

TESTICULAR TUMORS

Germ Cell Tumors

- Seminoma
- Embryonal carcinoma
- Yolk sac tumor
- Polyembryoma
- Trophoblastic tumors
- Choriocarcinoma
- Teratoma

Sex Cord-Gonadal Stromal Tumors

- Leydig cell tumor
- Sertoli cell tumor
- Granulosa cell tumor

Tumors with Both Germ Cell and Sex Cord-Gonadal Stromal Components

- Gonadoblastoma

Tumors of Lymphoid and Hematopoietic Origin

- Lymphoma
- Plasmacytoma

- Leukemia

Key Points:

- Testicular tumors are the most common malignancy in males aged 20-40 years.
- Germ cell tumors have a 2% likelihood of being bilateral.
- In men over 50, most testicular tumors are lymphomas, which tend to be bilateral.

Risk Factors for Testicular Tumors:

1. Cryptorchidism
2. Family history of testicular cancer
3. Personal history of testicular cancer
4. Intratubular germ cell neoplasia: Diagnosed through testicular biopsy conducted for various reasons, such as:
    - Contralateral testicular biopsy in patients with germ cell tumors
    - Biopsy during infertility evaluation
    - Rarely, testicular-sparing surgery
      Within five years, there is a 50% risk of developing a germ cell tumor. Hence, **radical orchiectomy and low-dose radiotherapy** are recommended.

GERM CELL TUMORS OF THE TESTIS

Seminoma

- The most common germ cell tumor of the testis.
- Two histological subtypes of pure seminoma have been defined.
- **85% of all seminomas** are classical seminomas.
    - Most frequently seen in the fourth decade of life.
- **Spermatocytic seminoma** accounts for **5-10% of all seminomas**.
    - Half of the patients with spermatocytic seminoma are over 50 years old, and it has a better prognosis.
    - In early-stage seminomas, treatment is debated. Close follow-up, primary radiotherapy, or primary chemotherapy with carboplatin have similar success rates.
    - In advanced-stage seminomas, chemoradiotherapy is generally used in combination.

Embryonal Cell Carcinoma

- Composed of undifferentiated malignant cells resembling primitive epithelium.
- Macroscopically, it appears **yellow-brown** with areas of hemorrhage and necrosis.
- Embryonal carcinoma is the **least differentiated germ cell tumor** and can transform into other germ cell tumors.

Yolk Sac Tumor

- Sometimes called **endodermal sinus tumor**.
- Rare in adults but more common in childhood.
- Hyaline globules are present in **84% of cases**, and **Schiller-Duval bodies** are seen in about half of the cases.
- Yolk sac tumors almost always secrete **alpha-fetoprotein (AFP)**, not beta-hCG.

Teratoma

- Can occur in both children and adults.
- Contains multiple germ cell layers at various stages of differentiation and maturation.
- **Mature teratomas** consist of benign elements derived from ectoderm, mesoderm, and endoderm.
- Microscopically:
    - **Ectoderm**: Squamous epithelial cells or neural tissue.
    - **Endoderm**: Intestinal, pancreatic, or respiratory tissue.
    - **Mesoderm**: Smooth or skeletal muscle, cartilage, or bone.

Choriocarcinoma

- Pure choriocarcinoma is rare.
- Clinically aggressive, characterized by **early hematogenous spread**.

Mixed Cell Type

- Most mixed cell type tumors (approximately **90-95% of all testicular tumors**) are a combination of **teratoma and embryonal carcinoma**, known as **teratocarcinoma**.

Metastatic Spread

- Except for choriocarcinoma, which spreads hematogenously early, germ cell tumors of the testis typically spread gradually via the **lymphatic system**.

- Common metastatic sites, in decreasing frequency, include:
    - Lungs
    - Liver
    - Brain
    - Bone
    - Kidneys
    - Adrenal glands
    - Gastrointestinal tract
    - Spleen

Clinical Findings

Symptoms:

- The most common symptom of testicular cancer is **painless testicular enlargement**.In most cases, a testicular mass or generalized enlargement is detected.
- A hydrocele may accompany the tumor, masking it.
- Transillumination of the scrotum can help differentiate between the two conditions.
- Patients with testicular cancer are most frequently misdiagnosed with epididymitis or epididymo-orchitis.
- The second most common misdiagnosis is hydrocele.

Imaging:

- The primary testicular tumor can be identified rapidly and accurately with **scrotal ultrasonography**.

Leydig Cell Tumors

Epidemiology and Pathology:

- These are the most common **non-germ cell tumors** of the testis, accounting for **1–3% of all testicular tumors**.
- Unlike germ cell tumors, they are **not associated with cryptorchidism**.
- Reinke crystals, pear-shaped cytoplasmic inclusions, are **pathognomonic** for Leydig cells.

Clinical Features:

- **Prepubertal children** often exhibit **virilization**, and the tumors are generally benign.
- **Adults** are usually asymptomatic, except for **gynecomastia** in **20–25%** of cases.
- Approximately **10% of tumors in adults** are malignant.
- Patients often have elevated levels of **serum and urinary 17-ketosteroids** and **estrogens**.

Treatment and Prognosis:

- The primary treatment for Leydig cell tumors is **radical orchiectomy**.

Sertoli Cell Tumors

Epidemiology and Pathology:

- **Sertoli cell tumors** are very rare, accounting for less than **1% of all testicular tumors**.

Clinical Features:

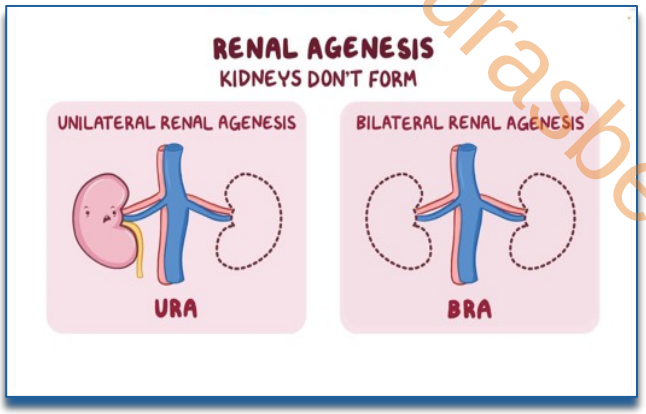
- The most common symptom is a **testicular mass**.
- **Virilization** is commonly observed in children, while **gynecomastia** occurs in approximately **30% of adults**.

Diagnosis

- In patients suspected of having a testicular tumor, the diagnostic procedure involves **radical orchiectomy** with excision of the **spermatic cord up to the internal inguinal ring**.
- Any lesser form of resection disrupts lymphatic drainage, increasing the risk of **local recurrence** and **nodal metastasis**.

Unit : 3

Congenital Anomalies of the Kidney



Agenesis

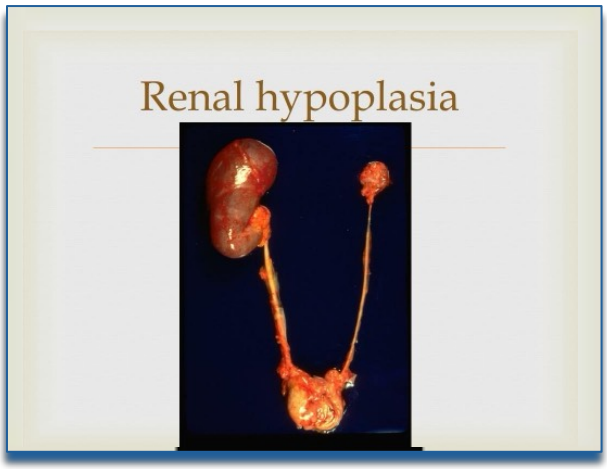
- **Bilateral:**

  ○ Leads to **oligohydramnios** during intrauterine life.

  ○ Associated with **pulmonary hypoplasia** and facial deformities (**Potter's face**).

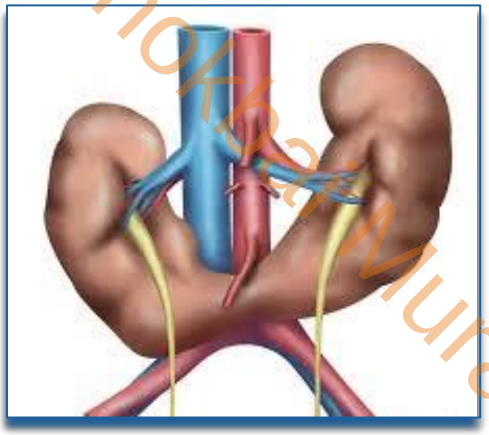
- **Unilateral:**

  ○ Asymptomatic; however, there is an increased risk of **infection**, **stones**, and **hydronephrosis** in the other kidney.



Hypoplasia

- Refers to a **small kidney**.
- The number of calyces is **less than 5**.
- Observed in conditions such as **fetal alcohol syndrome** and **intrauterine cocaine exposure**.



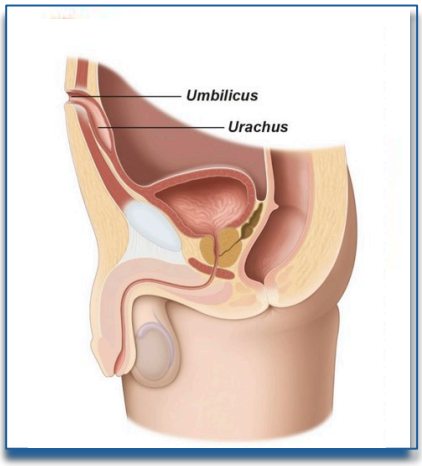
Renal Fusion

- The most common fusion anomaly is the **horseshoe kidney**.
- **Most commonly fused at the lower poles**.
- Often associated with **genitourinary** and **extra-urinary anomalies**.
- Increased risk of **obstruction**, **infection**, and **stones**.

Bladder Anomalies

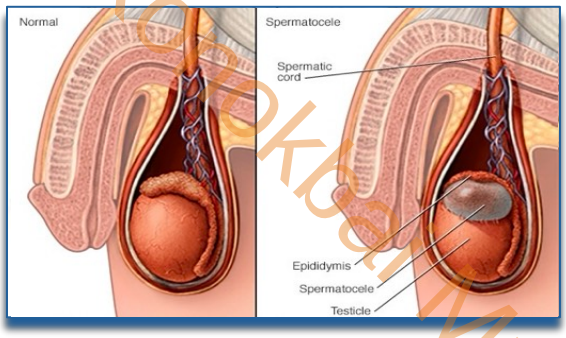
Exstrophy of the Bladder

- A **complete ventral defect** of the urogenital sinus.
- Most commonly associated with **epispadias**.
- May lead to the development of **adenocarcinoma** in the underlying tissue.



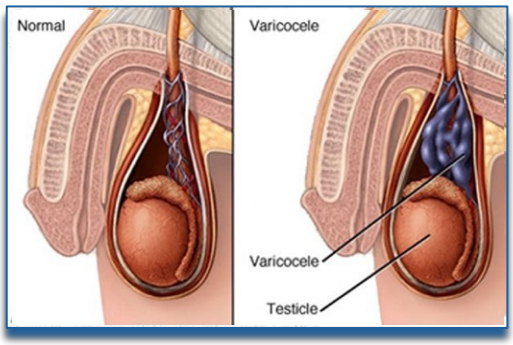
Persistent Urachus

- Occurs when the **urachus** fails to regress caudally from the bladder.
- **Adenocarcinoma** can develop from a **urachal cyst**.



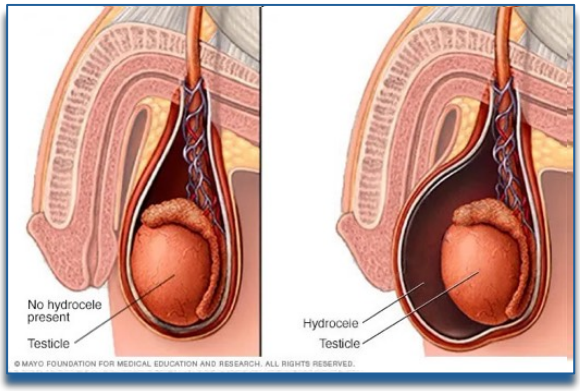
Spermatocele

- A **painless cystic mass** containing sperm, originating from the rete testis, efferent ductules, or epididymis.
- It is located superior and posterior to the testis and is **separate from the testis**.
- **No treatment** is required.



Varicocele

- A cystic dilation of the **pampiniform plexus veins**.
- More commonly occurs on the **left side** due to higher pressure in the renal vein and incompetent valves.
- Typically seen in **young individuals**.
- If present in **older individuals** or on the **right side**, a tumor should be investigated.
- **Treatment**: Ligation of the **spermatic vein** at the level of the internal inguinal ring.
- If untreated, it may lead to **testicular atrophy** and **infertility**.



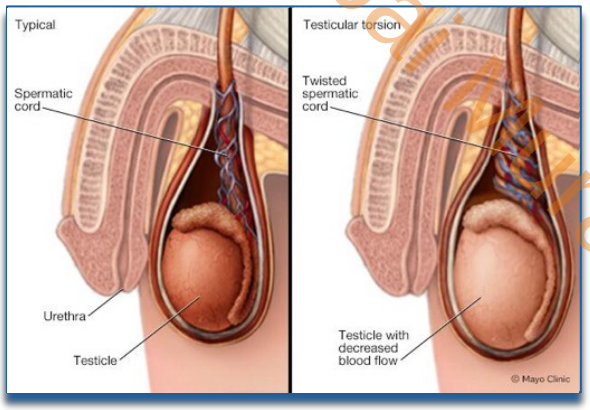
Hydrocele

- Refers to the accumulation of fluid within the **tunica vaginalis**.
- Commonly occurs after the age of **40**, but can also be seen in infants with a **patent processus vaginalis**.
- Can occur secondary to **epididymo-orchitis** or testicular tumors.
- Generally **painless**.
- If large and uncomfortable, it can be surgically removed.
- In children, it usually **resolves by age 1**. **Transillumination** is positive.

Hematocele

- Results from the **rupture of the tunica albuginea** due to trauma.
- **Does not transilluminate**.

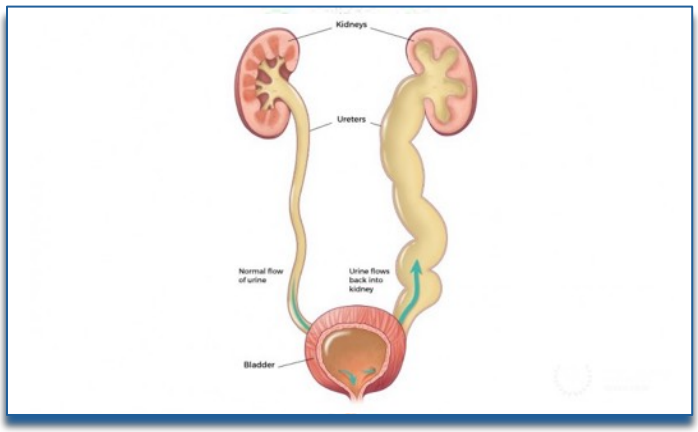
Testicular Torsion



- Occurs when the left testis rotates **counterclockwise**, and the right testis rotates **clockwise**.
- Most commonly seen between the ages of **12–18**.
- Testicular atrophy can develop within **3–4 hours** due to strangulation of testicular blood flow.
- More common in **undescended testes**.
- The testis appears **swollen and tender**.
- May mimic **acute epididymitis**, but in acute epididymitis, pain may decrease when the testis is elevated (**Prehn's sign**), though this sign has **low sensitivity and specificity**.
- **Doppler ultrasound** is necessary to confirm the presence or absence of blood flow.

Vesicoureteral Reflux (VUR)

- **Under normal conditions**, the ureterovesical junction allows urine to flow into the bladder while preventing its backflow into the ureter, especially during voiding.
- This mechanism protects the kidney from both high bladder pressure and contamination with infected bladder urine.
- When this **valve mechanism is inadequate**, the risk of urinary tract infection (UTI) significantly increases, making **pyelonephritis inevitable**.



Causes of Vesicoureteral Reflux

The causes are divided into **primary** and **secondary** types:

- **Primary Reflux**:
  - Occurs due to **structural congenital defects** of the ureterovesical junction.
  - Despite low-pressure urine storage in the bladder, reflux develops.
  - The root issue lies in the **disrupted length-to-diameter ratio** of the intravesical portion of the ureter.

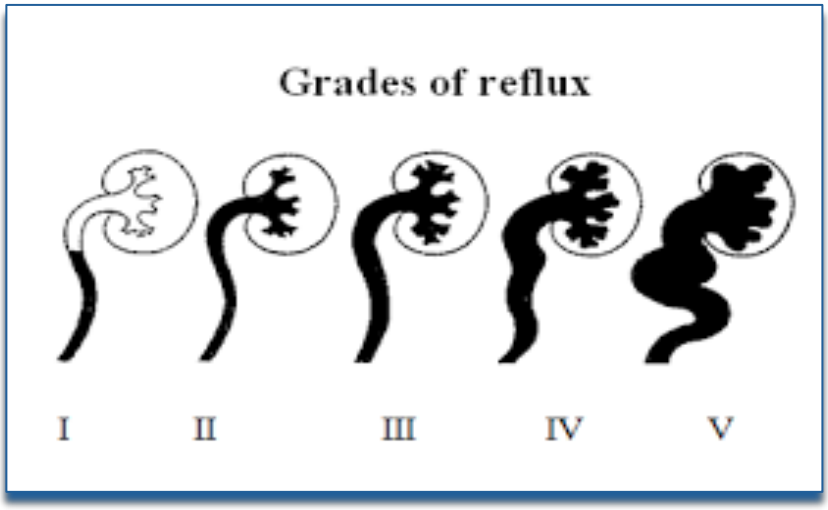
- **Secondary Reflux**:
  - Caused by any condition that leads to **bladder obstruction** and increased pressure.
  - The most common cause of bladder obstruction in children is a **posterior urethral valve**.
  - Among neurofunctional disorders that increase bladder pressure, **spina bifida leading to neurogenic bladder** should be considered.
- **Note**: Reflux is not the cause of urinary tract infections but **facilitates pyelonephritis**.

Conditions That May Facilitate Reflux

- Ureteropelvic junction obstruction
- Bladder diverticulum
- Ureteral duplication
- Renal anomalies (e.g., multicystic dysplastic kidney and renal agenesis)

Evaluation

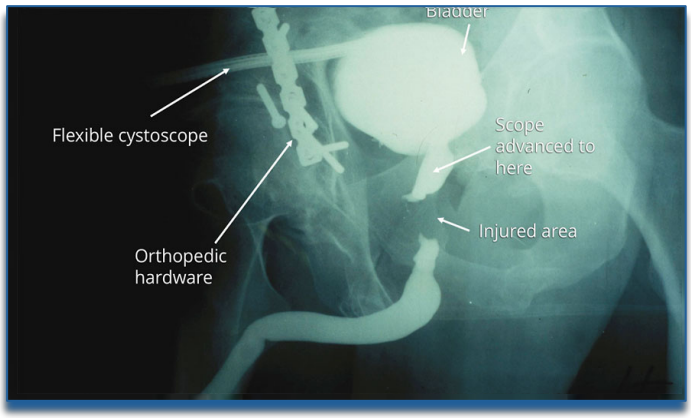
- **Ultrasound**: The primary imaging method used.
- **Scintigraphic methods**: Best for showing renal parenchymal damage.
- **Voiding cystourethrography (VCUG)**: The diagnostic method of choice.



Grade	Description
I	Urine refluxes into a **non-dilated ureter**.

Grade	Description
II	Urine refluxes into the **renal pelvis and calyces**, with no dilation.
III	**Mild to moderate dilation** of the ureter, renal pelvis, and calyces, with slight blunting of the fornices.
IV	**Moderate tortuosity** of the ureter with dilation of the pelvis and calyces.
V	Severe dilation of the ureter, pelvis, and calyces with **blurring of papillae** and significant tortuosity.

Urethral Injuries

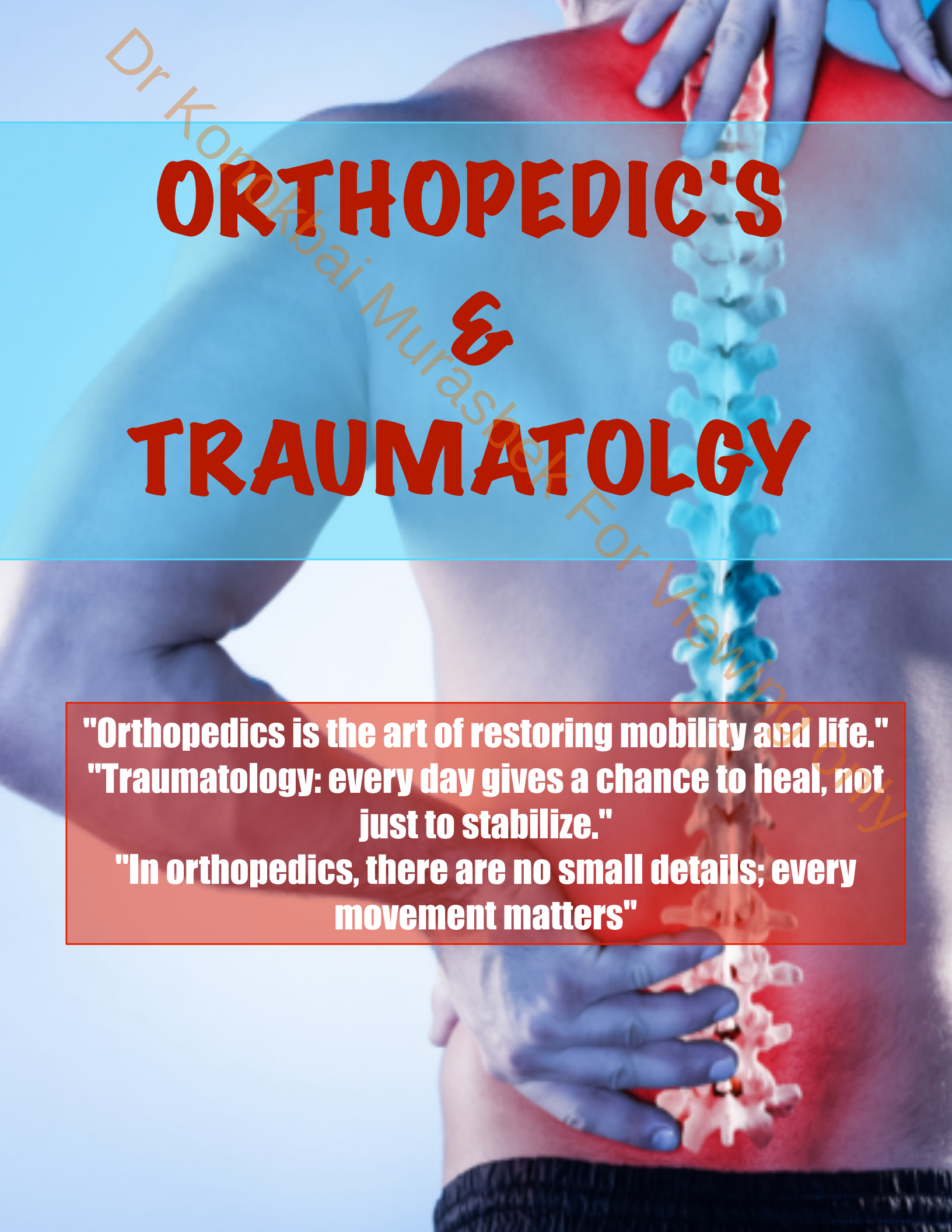


Posterior Urethral Injuries

- The **membranous urethra** passes through the pelvic floor and the voluntary sphincter, making it the most vulnerable part of the posterior urethra to trauma.
- When **blunt trauma** leads to pelvic fractures, the membranous urethra can separate from the prostatic apex at the **prostatomembranous junction**.
- The presence of **blood at the urethral meatus** is the most important sign of urethral injury.
- Detection of blood at the external urethral meatus indicates the need for **urgent retrography** to confirm the diagnosis of posterior urethral injury.

Anterior Urethral Injuries

- The anterior urethra lies **distal to the urogenital diaphragm**.
- **Saddle-type trauma** can cause laceration or contusion of the urethra.
- **Partial tears** can result from either spontaneous or **iatrogenic catheterization.**



ORTHOPEDIC'S & TRAUMATOLGY

"Orthopedics is the art of restoring mobility and life."
"Traumatology: every day gives a chance to heal, not just to stabilize."
"In orthopedics, there are no small details; every movement matters"

Unit : 1

DEVELOPMENTAL HIP DYSPLASIA (DDH)

General Information

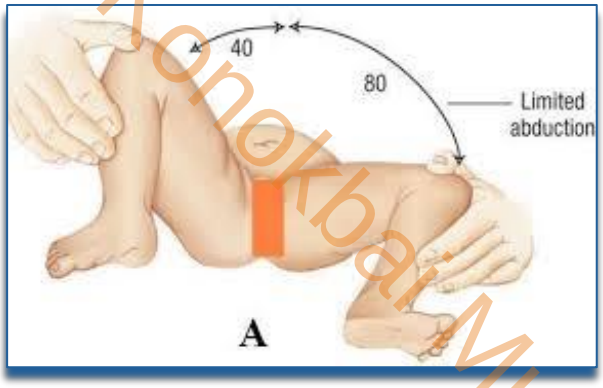
The term "developmental hip dysplasia (DDH)" encompasses all forms of the condition, including complete dislocation, partial dislocation (subluxation), and dysplasia. In DDH, the relationship between the femoral head and the acetabulum is disrupted or lost. As a result, structural abnormalities in the acetabulum and proximal femur worsen if treatment is delayed.

Factors Contributing to the Etiology of DDH:

- Mechanical and structural factors
- Ligamentous laxity
- Acetabular structures, including the capsule, labrum, pulvinar, ligamentum teres, and transverse acetabular ligament

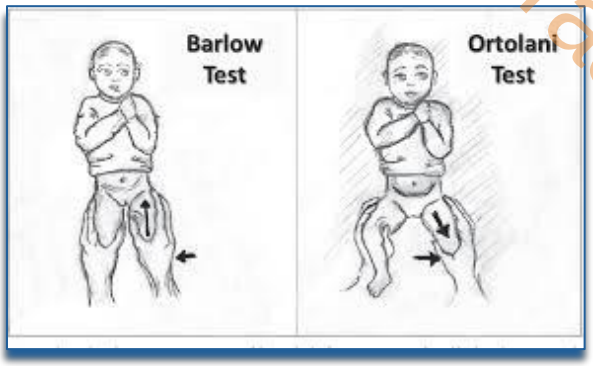
Additional risk factors include:

- Firstborn infants
- Female gender
- Mechanical factors: Breech presentation,Oligohydramnios,Multiple pregnancies, Postnatal positioning
- Positive family history
- Associated musculoskeletal anomalies, such as torticollis, metatarsus adductus, and pes calcaneovalgus
- Swaddling practices that force the hips into extension and adduction (the physiological position for newborn hips is flexion and abduction).

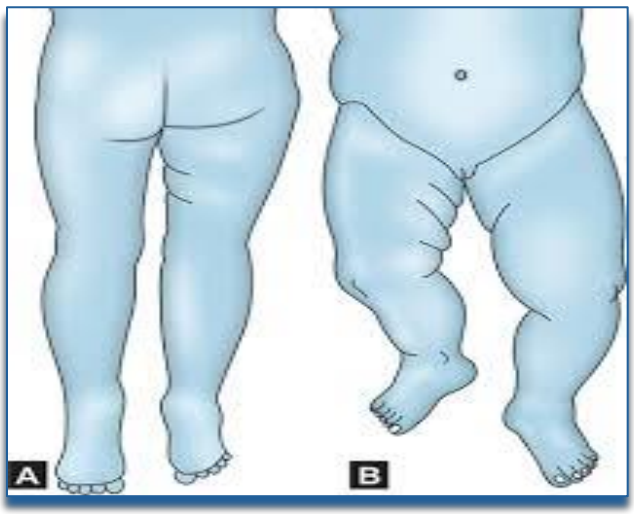


Clinical Evaluation:

Restricted abduction is a highly reliable diagnostic method, particularly in unilateral DDH cases, across all age groups. However, in bilateral dislocations, restricted abduction may be misleading.

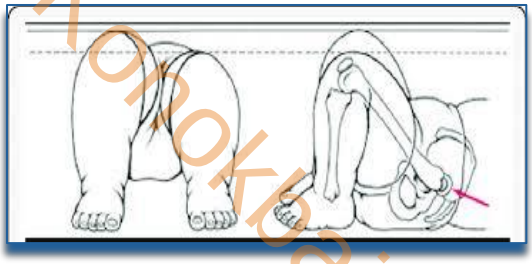


During the neonatal period, the **Ortolani test** (indicating reducibility of the hip) and the **Barlow test** (indicating dislocatability of the hip) can be used to assess the condition.



Post-Neonatal Period:

- The thighs and groin should be checked for asymmetry in skin folds.
-



- **Galeazzi-Allis Sign**: Refers to an inequality in knee height when the child lies on their back with hips and knees flexed. The knee on the dislocated side appears lower (Figure 3b).
- In bilateral dislocations, asymmetry of folds or the Galeazzi-Allis sign may be misleading.

In Walking Children:

- Recent studies indicate that children with DDH do not experience delays in walking.
- In unilateral dislocation, the affected side is shorter, and the body tilts toward the opposite side during walking (**Trendelenburg gait**).
- When standing on the affected side, the pelvis tilts toward the opposite side due to relative weakness of the **gluteus medius** muscle on the dislocated side (**Trendelenburg sign**).
- In bilateral dislocation, there is a **bilateral Trendelenburg gait** (waddling gait) and increased lumbar lordosis.

Radiology:

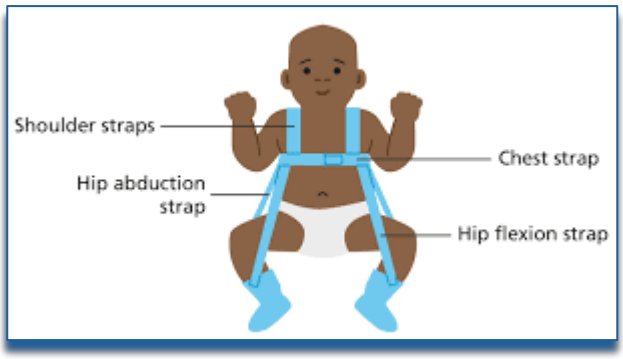
- During the first 4 weeks of life (neonatal period), physiological **ligament laxity** may be observed.
- In this period, relying solely on ultrasound (US) can be misleading; combining it with physical examination findings provides a more accurate assessment.
- Since the neonatal hip is predominantly made of cartilage, evaluation using plain radiography is challenging.
- In this period, hip ultrasonography (US) best demonstrates the relationship between the acetabulum and femoral head. Particularly in the first 6 months of life, hip US is considered the gold standard for radiological diagnosis.
- The key advantages of hip US are its ability to provide a quick and accurate diagnosis and its lack of ionizing radiation, making it safe for infants.

- Its disadvantages include the need for expertise to perform the procedure and its sensitivity, which can lead to overtreatment, especially in the early neonatal period.

The **anteroposterior (AP) pelvic radiograph** is extremely important and becomes the gold standard after 6 months of age. On the pelvic radiograph, the **Shenton-Menard line** is assessed. This is an arc-shaped line that starts from the lesser trochanter, continues along the femoral neck, and follows the upper border of the obturator foramen (inner border of the pubis). Normally, this line should form a continuous arc(green line in figure). However, in cases of developmental hip dysplasia (red line in figure), the continuity of this line is disrupted.

Treatment:

- The first **2–3 months of life** are especially critical for treatment.



During the **first 6 months**, treatment involves dynamic or static orthoses (such as the Pavlik harness) that keep the hip in abduction and flexion. The **Pavlik harness**, a dynamic method, is the preferred choice.

- After **6 months**, the success of conservative treatments decreases, and the child's treatment often requires hospitalization and procedures performed in the operating room.

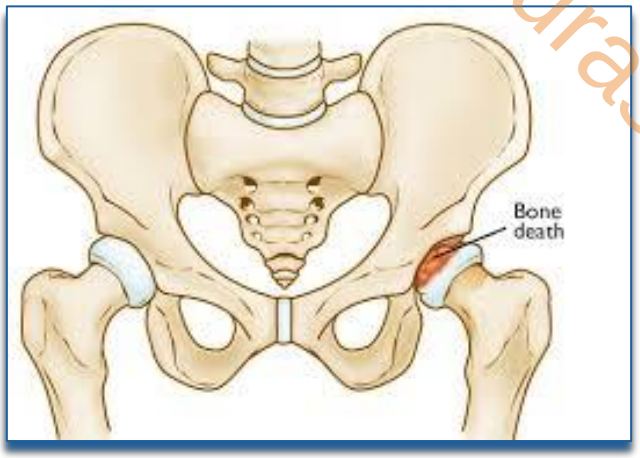
- Between **7–18 months**, closed or open reduction is performed either as a primary treatment or for hips where conservative methods failed during the first 6 months.
    - Closed reduction of the hip and subsequent immobilization with a cast (hyperabduction cast) is performed under general anesthesia.
    - Open reduction is performed if closed reduction fails. In open reduction, soft tissue obstacles both inside and outside the joint that prevent the femoral head from entering the acetabulum are surgically removed.
    - Cases unresponsive to open reduction may require **osteotomy surgery**, typically performed before the **18th month**.

Complications:

- **Avascular necrosis (AVN)** is the most significant long-term complication of treatment, often leading to degenerative joint disease in the future.
- AVN is entirely treatment-related and preventable.

OSTEOCHONDROSES

These are **aseptic (avascular) necroses of the ossification center**. There are several clinical types:



1.Perthes-Calvé-Legg Disease

- Aseptic necrosis of the femoral head (epiphysis), most commonly associated with trauma in the etiology.
- It is more frequently observed in **boys aged 4–8 years**.
- Clinical Symptoms:
  - Initially presents with pain in the hip and knee, along with limping.
  - Restricted abduction and internal rotation of the hip.
  - Pain and shortening in the thigh muscles may develop.

Radiological Findings

1. Synovitis Stage: (4 weeks)Swelling in the joint capsule, increased medial joint space (tear drop or U sign), and subchondral lytic line (crescent sign) are observed.

2. Avascular Necrosis Stage: (six months) Increased density in the femoral head, flattening of the epiphysis, and a cystic appearance in the metaphysis.

3. Fragmentation (Regeneration) Stage: (1–2 years).Sclerotic and lytic appearance in the epiphysis, shortening and flattening of the femoral neck, further flattening of the epiphysis, and displacement of the femoral head outside the acetabulum.

4. Residual Stage: The femoral head becomes flattened and enlarged (**coxa plana**).It may grow to the extent that it extends beyond the acetabulum (**coxa magna**). In some cases, acetabular depth decreases.

Prognosis

1. Younger age of onset is associated with a better prognosis (the most critical factor).
2. The less involvement of the femoral head, the better the prognosis.
3. Obesity negatively affects the outcome.
4. Stiffness in joint movements and the formation of flexion contractures worsen the prognosis.

Treatment

Conservative:

- Applied in young patients with partial involvement of the femoral head.
- Goal: To guide the femoral head into the acetabulum and reduce the load on the hip.
- Methods include bed rest, NSAIDs, traction, and orthopedic devices.

Surgical:

- Performed in patients older than 5 years with high-risk total involvement of the femoral head and neck.
- Goals: To improve blood supply and reduce intraosseous pressure.
- Surgical methods include: Varus osteotomy,Salter osteotomy,Chiari osteotomy,Cheilectomy.

2.Kohler Disease: Aseptic necrosis of the tarsal navicular bone.

3.Osgood-Schlatter Disease : Aseptic necrosis of the tibial tuberosity.

4.Freiberg Disease: Aseptic necrosis of the 2nd metatarsal head.

5.Sever Disease: Apophysitis of the posterior calcaneus.

6.Kienbock Disease : Aseptic necrosis of the lunate bone.

7.Scheuermann Disease (Adolescent Kyphosis)Structural kyphosis of the thoracic and thoracolumbar vertebrae.Onset age is typically 10–12 years.Anterior wedging occurs in the lower thoracic and upper lumbar vertebrae.

8.Panner Disease : Avascular necrosis of the capitellum of the humerus.

9.Johansson-Carson Disease: Aseptic necrosis of the inferior pole of the patella.

10.Preiser Disease : Aseptic necrosis of the scaphoid bone.

Unit : 2

OSTEOMYELITIS

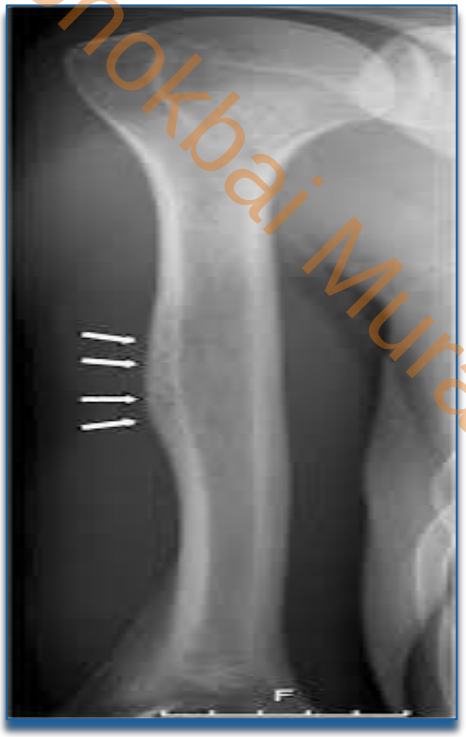
Osteomyelitis is an infection of bone tissues.

It can develop;

- Primarily due to the direct inoculation of microorganisms into the bone through trauma or surgical procedures.
- Secondarily via hematogenous spread or direct extension from surrounding soft tissues and joints.
- *Staphylococcus aureus* is the most frequently isolated microorganism, especially in acute hematogenous osteomyelitis.
- In patients with sickle cell anemia, there is an increased incidence of *Salmonella* osteomyelitis; however, the most common causative agent remains *S. aureus*.
- In intravenous drug users, the most common causative agent is *Pseudomonas aeruginosa*.
- In 2–4-week-old infants without other health issues, *Group B Streptococci* are the most commonly isolated organisms

Acute hematogenous osteomyelitis is the most common form and generally affects children. It can be classified into **acute**, **subacute**, and **chronic** osteomyelitis.

Classification



Acute Osteomyelitis

- Leukocytosis .
- Erythrocyte sedimentation rate (ESR) and C-reactive protein **(CRP)** levels are elevated.
- Radiographic findings: Soft tissue swelling appears within 1–3 days, but bone or periosteal damage may not be visible until 10–12 days.Differential diagnosis includes septic arthritis, Ewing sarcoma, osteosarcoma, juvenile arthritis, sickle cell crisis, Gaucher disease, and pathological fractures.
- Magnetic Resonance Imaging (MRI) shows early inflammatory changes in the bone marrow and soft tissues.
- **Technetium-99m** bone scintigraphy is positive in 90–95% of cases and is used to rule out acute osteomyelitis if negative.

Subacute Osteomyelitis

The most common form of subacute hematogenous osteomyelitis is **Brodie abscess**.

- Radiologically, it presents as lytic bone lesions surrounded by sclerotic tissue.
- It develops after bacteremia from an infection focus in the body.
- It is a localized form of osteomyelitis, often affecting the long bones of the lower extremities in young adults.
- Can be mistaken for a tumor radiologically, necessitating biopsy for confirmation.

Chronic Osteomyelitis

- Characterized by infected necrotic bone tissue (sequestrum) surrounded by soft tissue (involucrum).
- Sclerotic avascular bone tissue and scarred muscle and subcutaneous tissue are present around the infected focus.
- Necrotic bone areas may develop new bone formation and are infiltrated by lymphocytes, histiocytes, and plasma cells.
- Over time, it can perforate soft tissues and form a sinus tract that opens to the skin surface.

Features

- Chronic osteomyelitis of long bones can result from open or comminuted fractures, poorly treated fractures, or internal fixation devices.
- Systemic symptoms such as fever and pain are often absent.
- The gold standard for diagnosis is histological and microbiological examination via biopsy.
- ESR and CRP levels are elevated, but leukocytosis is seen in only 10–35% of cases.
- There is no definitive technique to confirm or exclude chronic osteomyelitis.
- Radiographs should be the initial investigation.
- **CT** provides excellent results for cortical bone but is less effective for soft tissue.
- **MRI** is more effective for soft tissue evaluation.

Treatment:

- Debridement of necrotic tissue
- Filling of dead spaces
- Long-term effective antibiotic therapy
- Hyperbaric oxygen therapy

Acute Septic Arthritis (Pyogenic Arthritis)

- It is an infection of the joint space.
- It most commonly occurs in children around 4 years of age.
- It is most frequently seen in the hip and knee joints.
- In adults, it may be seen in cases of diabetes, rheumatoid arthritis, the use of immunosuppressive medications such as steroids, and alcoholism.
- The most common causative agent is *S. aureus*.
- In patients with prostheses, the most common causative agent is *S. epidermidis*.

Clinical:

- Symptoms include fever, fatigue, joint pain, swelling, increased temperature, and restricted movement.

Diagnosis:

- Joint aspiration shows PMNL (polymorphonuclear leukocytes), increased total protein, and decreased glucose levels.

Radiology:

- Joint space widening
- Expansion of surrounding soft tissues
- Bulging in the capsule area
- Joint trabeculation destruction
- Osteoporosis in the surrounding bones

Treatment:

- Drainage
- Antibiotics
- Resting the joint in a fixed position

Pott's Disease (Vertebral Tuberculosis)

- It most commonly occurs in the thoracic and upper lumbar vertebrae.

- It initially settles in the vertebral bodies, causing destruction of the body. It then spreads to adjacent vertebrae.
- It can cause localized kyphosis (Gibbus).
- Typically spreads downward and anteriorly due to the effect of gravity.
- In the lumbar region, an abscess can form and may spread downward along the psoas muscle to the inguinal region.

Radiology:

- Narrowing of the joint space
- Subchondral bone destruction
- Cold abscess
- Localized kyphosis (Gibbus)

Treatment:

- Anti-TB medications, drainage, surgery

Spina Ventosa (TB Dactylitis):

- It is the most common form of tuberculosis found in the phalanges and causes swelling in the fingers.
- Radiologically, there is expansion in the phalanx, periosteal reaction, and cystic appearance in the bone.
- Treatment includes anti-TB therapy and immobilization.

FRACTURES AND DISLOCATIONS

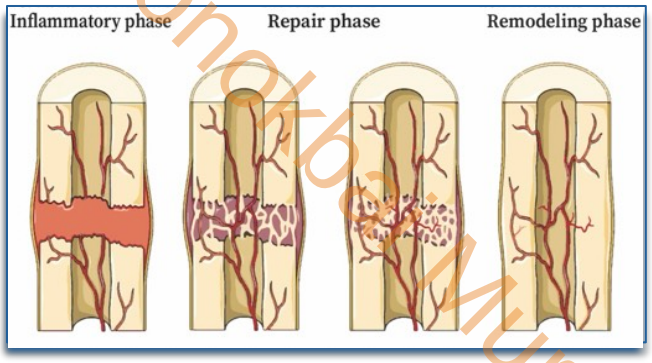
- **Fractures**: Disruption of the anatomical integrity of bones.
- Based on their relationship with the external environment, fractures are classified as **open** or **closed**. Based on the fracture line, they are classified as **complete** or **incomplete**.

Pediatric-Fractures;

Physiologically and anatomically differ from adult fractures:

- Growth plates in children are cartilaginous and do not appear on X-rays, so imaging the healthy side for comparison is necessary for diagnosis.
- The periosteum is thick, and fractures heal quickly.
- Due to the higher flexibility of their bones, **torus** and **greenstick fractures** are commonly observed.
- Deformities may develop with **epiphyseal fractures**.

Fracture Healing



1. **Inflammatory Phase**:
    - The endosteum, periosteum, and surrounding soft tissues are damaged at the fracture site. Blood, exudate, and lymph accumulate (fracture hematoma).
2. **Repair Phase**:
    - The hematoma organizes, and fibroblast infiltration is observed from the surrounding tissue (fibrous callus).
    - Within a week, osteoblasts and chondroblasts begin to form bone by releasing collagen and Ca (callus formation).
    - If bone ends are securely joined with internal fixation, direct healing occurs without callus formation, bridging the cortical bone directly.
3. **Remodeling Phase**:
    - Bone is resorbed and rebuilt simultaneously, leading to the formation of new bone.

Factors Affecting Fracture Healing

- Healing is influenced by **age** (faster in younger individuals), local factors, hormones, minerals, and vitamins.
- **Local factors** are among the most important:
    - Blood supply to the surrounding soft tissue.
    - Presence of infection (negative effect).
    - Immobilization of the fracture.
    - Type of bone (spongy bone heals more easily) and the extent of bone loss.
    - Development of avascular necrosis.
    - Presence of malignancy.
    - Interposition of soft tissue between bone ends.

Clinical Signs of Fractures

- Pain (due to periosteal tear).
- Loss of function.
- Deformity.
- Abnormal movement and crepitus.
- Neurovascular injuries.
- Radiological findings.

Early Fracture Complications

- Fat embolism.
- Development of infection (e.g., gas gangrene, osteomyelitis).
- Tetanus.
- Hemorrhagic shock.
- Compartment syndrome.
- Vascular or nerve injury.
- Post-traumatic reflex dystrophy.

Late Fracture Complications

- Non-union or malunion (most commonly due to inadequate reduction).
- Avascular necrosis.
- Osteoarthritis.
- Myositis ossificans.
- Late nerve paralysis.

FAT EMBOLISM SYNDROME

General Information

- Fat embolism refers to the presence of fat emboli in the circulation after long bone fractures or major trauma. When these emboli cause clinical symptoms, it is termed **fat embolism syndrome**. It can present in three clinical forms:

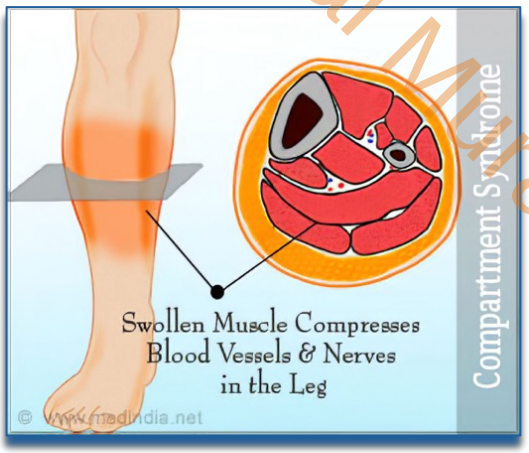
1. **Subclinical**:
    - Only a decrease in PaO_2 below 60 mmHg is observed.

2. **Clinical**:
    - The typical form, emerging 24–72 hours after trauma.
    - **Major findings**:
        - Respiratory failure (PaO_2 < 60 mmHg, PCO_2 > 55 mmHg).
        - Cerebral involvement.
        - Petechial hemorrhages.
        - Retinal findings.
    - **Minor findings**:
        - Fever (39–40°C).
        - Tachycardia.
        - Liver failure.
        - Oliguria or anuria.
        - Anemia, thrombocytopenia, leukocytosis.
        - ECG abnormalities.
    - **Diagnosis**: Fat globulins in combination with 1 major and 4 minor findings confirm the diagnosis.
3. **Fulminant**:
    - Appears within hours of trauma and has a severe clinical presentation.

- **Key criterion**: A PaO_2 level below 60 mmHg is the most important diagnostic factor in fat embolism syndrome.
- **Treatment**: Supportive therapy is the mainstay. With adequate treatment, the condition is generally self-limiting and resolves within 1–2 weeks.
- **Prevention**: The most critical measure to prevent fat embolism syndrome is the immobilization of fractures as soon as possible.

Unit : 3

COMPARTMENT SYNDROME



General Information

- **Compartment**: Refers to sections enclosed by osseofascial structures that contain muscles and neurovascular elements.
- **Compartment syndrome**: Occurs when increased intracompartmental pressure leads to perfusion impairment and subsequent ischemia.
- Most common cause in adults: Tibia fracture.
- Most common cause in children: Supracondylar humerus fracture.

Etiology:

- Trauma (fracture, edema, hematoma).
- Tumors.
- Burns.
- Snake bites.
- Angioneurotic edema.

Pathophysiology:

- Intracompartmental pressure exceeding 30 mmHg causes venous congestion.
- Arteriolar spasm leads to ischemia.
- The first symptom is deep, severe pain, unrelieved by simple analgesics or immobilization.
- In the early stage, due to arteriolar-level spasm, the pulse may still be palpable. However, distal perfusion deficits (DP signs) appear in advanced stages.
- Thus, the presence of a palpable distal pulse does not rule out compartment syndrome in the early stage.
- Circular casts must be checked within the first 24 hours for the development of compartment syndrome.

Treatment:

- Urgent surgical decompression is necessary. This is achieved through fasciotomy.

VOLKMANN ISCHEMIC CONTRACTURE

- A deformity resulting from compartment syndrome of the volar aspect of the forearm.
- Most commonly seen in children under 10 years old.
- Causes:
    - Tight casts and bandages that obstruct blood flow.
    - Immobilization of the elbow at an angle greater than 90 degrees.
    - Forearm crush injuries.
    - Arterial embolism.
    - Drug extravasation.
- Often seen in supracondylar humerus fractures.
- Most commonly affected artery: Brachial artery (A. brachialis).

Clinical Presentation:

- Begins with severe forearm pain a few hours after trauma. The pain worsens with finger extension.
- Symptoms on the volar forearm:
    - Increased warmth.
    - Swelling.
    - Redness.
    - Increased pain
- Within a few days:
    - Paralysis of flexor muscles and intrinsic hand muscles.
    - Sensory loss in radial and ulnar nerve dermatomes.
    - The volar forearm becomes firm, and the **5 P's** appear:
        - **Pain**.
        - **Pulselessness** (lack of pulse).

    - **Pallor** (paleness).
    - **Paresthesia**.
    - **Paralysis**.
- Deformity:
    - Contracture with metacarpophalangeal (MCP) joint extension and proximal (PIP) and distal interphalangeal (DIP) joint flexion.
    - Atrophy of the hand.

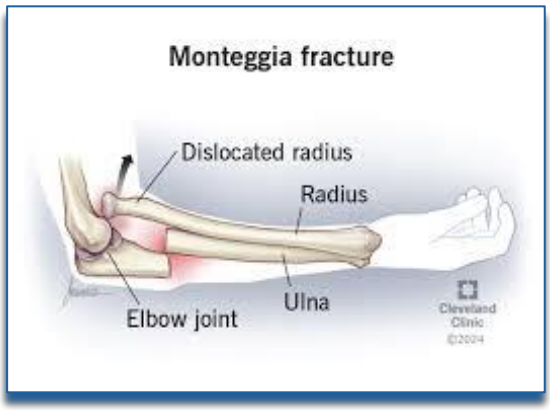
Treatment:

- Emergency surgical decompression.
- Efforts to correct the contracture.

Unit : 4

UPPER EXTREMITY FRACTURES AND DISLOCATIONS

Monteggia Fracture-Dislocation

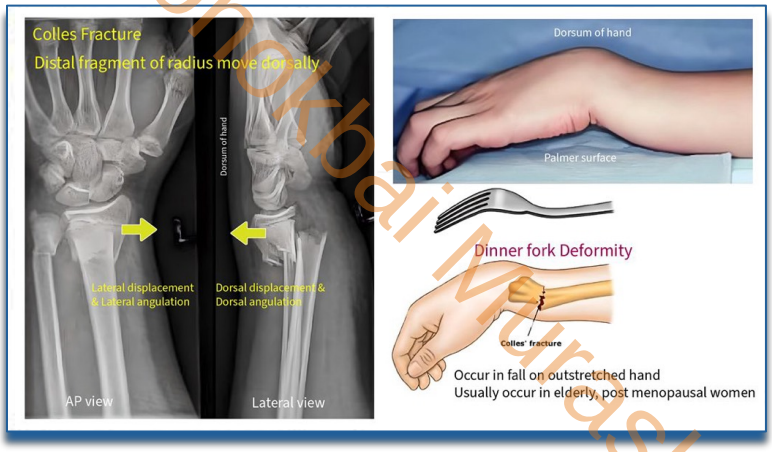


- Defined as a **fracture of the ulna** accompanied by **dislocation of the radial head**.
- **In children**: Closed reduction is preferred. However, if the ulna fracture is unstable or the radial head cannot be reduced, surgical intervention is performed.
- **In adults**: Open reduction and internal fixation are performed.
- The most common associated injury is to the **posterior interosseous nerve**.

Galeazzi Fracture-Dislocation

- Defined as a fracture of the radius with an associated dislocation of the distal radioulnar joint.

Colles Fracture



- A fracture that occurs up to 3 cm above the distal articular surface of the radius, where the distal fragment is displaced dorsally and radially, forming an angulation with its apex towards the volar side.
- It is a fracture of the extensor surface of the radius.
- Most commonly caused by falls onto an outstretched hand.
- Treatment involves reduction followed by immobilization with a long arm cast. Healing typically occurs within 6 weeks.

Smith Fracture

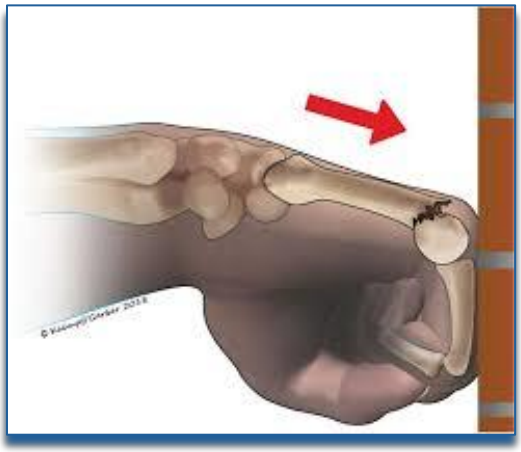
- Occurs due to a fall onto the back of the hand while the wrist is in palmar flexion.
- It is the opposite of a Colles fracture.
- The distal fragment of the radius is displaced volarly, while the proximal fragment shifts dorsally.
- Treatment involves reduction, with the forearm in supination and the wrist in dorsiflexion.

Bennett's and Rolando`s Fracture

It is a fracture-dislocation of the base of the first metacarpal.

Boxer's-Fractures

It is a fracture of the neck of the 4th and 5th metacarpals.



Baseball Fracture (Mallet Finger)

- It is an avulsion fracture caused by the pull of the extensor tendon attached dorsally to the base of the distal phalanx.
- There is no extension movement in the distal phalanx.

Unit : 5

BONE TUMORS

- In an adult patient, an epiphyseal lesion should suggest a giant cell bone tumor, whereas in a patient whose skeletal system has not yet matured, an epiphyseal lesion should suggest chondroblastoma.
- In diaphyseal lesions, keep in mind Ewing's sarcoma, osteomyelitis, osteoid osteoma, osteoblastoma, histiocytosis, lymphoma, fibrous dysplasia, and adamantinoma.
- In adult patients with vertebral lesions, metastasis, myeloma, and hemangioma should be considered. In the sacrum, chordoma and giant cell bone tumor should be considered.
- In young patients with lesions in the vertebral body, histiocytosis should be considered. If the posterior elements of the vertebra are involved, aneurysmal bone cyst, osteoblastoma, and osteoid osteoma should be considered in the differential diagnosis.

BENIGN BONE TUMORS

Osteoid Osteoma

- Occurs in the 2nd to 3rd decades of life and is three times more common in males.
- Involves the long bones of the lower extremity and the posterior elements of the spine.
- Affects the diaphysis and metaphysis.
- Causes pain that worsens at night and responds to nonsteroidal anti-inflammatory drugs (NSAIDs).
- Lesions show increased levels of cyclooxygenase and prostaglandins, explaining the severe pain relief with NSAIDs.

- Imaging reveals a radiolucent nidus smaller than 1.5 cm in diameter.
- Histology shows trabeculae surrounded by loose fibrovascular tissue.

Bone Island

- Also called **Enostosis**.
- Occurs in adults with an equal male-to-female ratio.
- Generally asymptomatic.
- Radiologically appears as small, rounded areas of increased density in spongy bone with spicules extending toward the periphery.
- A rare condition with multiple bone islands is called **Osteopoikilosis**.
- Managed with follow-up.

Chondroma

- Benign lesions of hyaline cartilage. The phalanges are the most commonly affected areas.
- It is the most common tumor of the small bones in the hands and feet.
- They are mostly asymptomatic and are detected incidentally.
- Since they mostly originate from the medullary canal, they are also referred to as **enchondromas**.

- Multiple enchondromatosis is known as **Ollier's disease**.
- When soft tissue hemangiomas are present over the lesion, it is referred to as **Maffucci syndrome**.
- In 25% of Ollier's disease cases, lesions undergo sarcomatous changes after the age of 40.
- Radiologically characterized by irregular calcifications, described as speckled, punctate, or popcorn-like appearance.

Osteochondroma

- Common benign bone tumors.
- Some define them as developmental malformations.
- The lesion is typically characterized by a bony mass with a stalk resulting from progressive endochondral ossification.
- It has a cartilage-capped head.
- Growth usually stops after skeletal maturation is complete.
- Most commonly appears in the **distal femur**, **proximal tibia**, and **proximal humerus**.
- Generally asymptomatic and detected incidentally.

- Malignant degeneration is very rare.

Nonossifying Fibroma

- Also known as metaphyseal fibrous defect, fibrous cortical defect, or fibroxanthoma.
- A common developmental anomaly seen in 35% of children.
- Usually detected incidentally.
- Occurs between the ages of 2 and 20, typically affecting the distal femur, tibia, and fibula.
- Appears as well-defined, lobulated lesions on direct radiographs.
- Mostly asymptomatic.

Cortical Desmoid

- Irregularities usually seen in boys aged 10–15 years at the posteromedial distal femoral metaphysis.
- Thought to be a stress lesion caused by the pulling force of the adductor magnus muscle.
- The lesion can be visualized with an oblique radiograph taken with the lower extremity externally rotated 20–45 degrees.
- May cause soft tissue swelling and pain.
- Typically managed with observation.

Benign Fibrous Histiocytoma

- Rare lesions more commonly found in soft tissue and less frequently in bone.
- Appear in the diaphysis or epiphysis of long bones and the pelvis.
- Typically seen in individuals aged 30–40 years.
- Characterized by well-defined lytic lesions on radiographs.

- Due to the possibility of local recurrence, extended curettage or resection is recommended.

Fibrous Dysplasia

- Developmental anomalies that can be monostotic (single bone) or polyostotic (multiple bones).
- Characterized by the replacement of normal bone and bone marrow with fibrous tissue and small bone spicules.
- **McCune-Albright Syndrome**: Associated with polyostotic fibrous dysplasia, skin pigmentation, and endocrine anomalies.
- **Mazabraud Syndrome**: Associated with polyostotic fibrous dysplasia and intramuscular myxomas.
- Radiologically, it shows the typical "ground-glass" appearance with sclerotic margins.

Surgical treatment is recommended in the presence of severe pain, pathological fractures, or significant deformity.

BENIGN/AGGRESSIVE BONE TUMORS

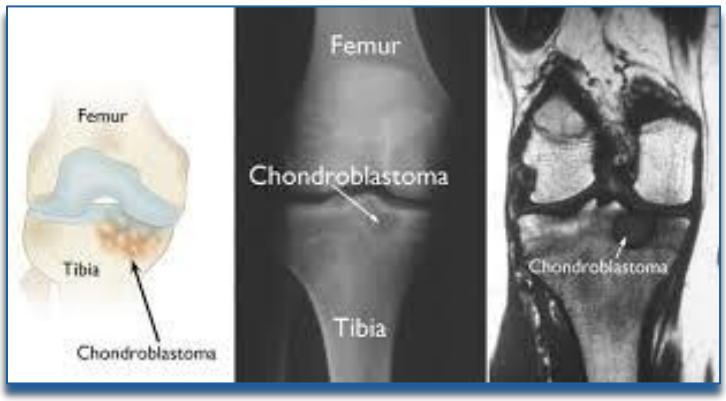
Some tumors described here are entirely benign in nature, while others may exhibit overt malignancy.

- While the majority can be treated with intralesional therapies, some require aggressive resection.
- Giant cell tumor of bone and chondroblastoma can develop pulmonary metastases and, in rare cases, may be fatal.

Giant Cell Tumor of Bone

- Occurs between the ages of 20-40, with a slight female predominance.
- Most common locations: distal femur, proximal tibia, and distal radius.
- Accounts for 5% of all bone tumors.
- Usually presents as a single lesion. Pulmonary metastases occur in about 3% of cases, with a mortality rate of 15% due to metastases.
- Malignant giant cell tumors make up less than 5% of cases.
- Most patients experience progressive pain.
- Radiological findings: lytic lesions located in the epiphysis of long bones, particularly in subchondral bone.
- These tumors are often locally aggressive. Recurrence after treatment is observed in 5-15% of cases.
- Surgical curettage is the treatment of choice.
- In cases unsuitable for surgery, **zoledronic acid** (inhibiting osteoclastic activity and inducing osteoclast apoptosis) can be used.
- Additionally, the monoclonal antibody **denosumab**, which limits osteoclastic activity, is FDA-approved for non-surgical cases.

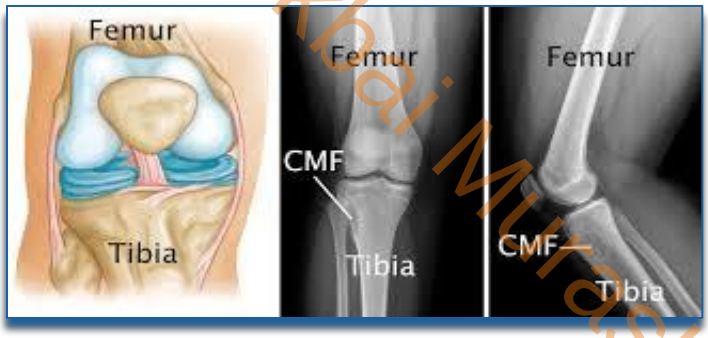
Chondroblastoma



- Occurs between the ages of 10-25.
- Accounts for less than 1% of all bone tumors.
- Affects the epiphysis of long bones, with a characteristic well-defined appearance on imaging.
- Surgical curettage is recommended.

- Pulmonary metastases occur in less than 1% of cases and are responsive to resection.

Chondromyxoid Fibroma



- Most commonly found in the proximal tibia.
- Patients typically present with pain.
- Resection is recommended.
- A rare type of tumor.

Osteoblastoma

- Most commonly located in the spine.
- Pain is usually the initial symptom.
- Painful scoliosis and neurological deficits may occur due to spinal involvement.
- Radiologically, the most common appearance is a bone-forming mass in the posterior part of the spine in young patients.
- Classic nidus formation is rare; the nidus seen in osteoblastoma is typically larger (>1.5 cm) than in other conditions.
- Surgery is recommended.

Langerhans Cell Histiocytosis

- A group of diseases affecting multiple systems. Isolated bone lesions are referred to as eosinophilic granulomas.
- **Hand-Schüller-Christian disease**: Skull involvement, exophthalmos, and diabetes insipidus.
- **Letterer-Siwe disease**: Fever, lymphadenopathy, hepatosplenomegaly, and multiple bone lesions.
- Orthopedic involvement is primarily in eosinophilic granulomas of the bone. Pain is a characteristic feature.

- Most commonly affects the vertebral bodies.
- Diagnosis is made through biopsy. Isolated disease can be monitored as spontaneous resolution is possible.
- Other treatment options include corticosteroid injections, radiotherapy, and curettage.

RADIOLOGY IN BENIGN TUMORS

- Narrow zone of transition is characteristic.
- Well-defined lesion margins.
- No Codman triangle formation.
- No periosteal reaction, such as the onion-skin appearance.
- No evidence of soft tissue extension.

MALIGNANT BONE TUMORS

Osteosarcoma

- Most commonly located at the distal femur, proximal tibia, and metaphysis (knee region).
- The second most common primary malignant bone tumor in adults. The most common non-hematologic primary bone tumor.
- Frequently seen in males aged 10–30.
- The most significant symptom is pain.
- The area where the tumor disrupts the cortex presents a triangular appearance (Codman's triangle, sunburst pattern).
- Most commonly metastasizes to the lungs.

Classification

- **Primary Osteosarcomas**:
    - Classical osteosarcoma, low-grade intramedullary osteosarcoma, parosteal osteosarcoma, periosteal osteosarcoma, high-grade osteosarcoma, telangiectatic osteosarcoma, and small-cell osteosarcoma.

- **Secondary Osteosarcomas**:
    - Usually seen in individuals over 50 years old.
    - Develop in patients with Paget's disease or those who have previously undergone radiotherapy.
    - Osteosarcoma in Paget's disease typically occurs in the 6th to 8th decades of life.

Grades

- High-grade: Classical osteosarcoma, small-cell osteosarcoma, high-grade osteosarcoma.
- Intermediate-grade: Periosteal osteosarcoma.
- Low-grade: Low-grade osteosarcoma, parosteal osteosarcoma.
- **Paget's disease**: The incidence of developing osteosarcoma is ~1%. Most commonly located in the pelvis.

Treatment

- Surgery, chemotherapy, and radiotherapy.

Chondrosarcoma

- The second most common malignant bone tumor of non-hematologic origin after osteosarcoma.
- **Primary chondrosarcomas**: Seen between the ages of 40–60.
- **Secondary chondrosarcomas**: Seen between the ages of 25–45.
- Most frequently located in the pelvis, proximal femur, and proximal humerus. Rarely seen in the hands but is the most common malignancy in that region.
- Clinical presentation includes slowly progressing and intensifying pain.

Risk Factors

- **Secondary chondrosarcomas**: High incidence in Ollier and Maffucci syndromes (~25%).
- Also develop from osteochondromas. Risk of malignancy is ~1% in solitary lesions and ~5% in multiple osteochondromas.

Radiology

- Appears as punctate, popcorn-like, or comma-shaped calcifications.

Ewing Sarcoma

- The third most common non-hematologic malignant bone tumor. The most common malignant bone tumor in children under 10.
- Commonly affects individuals aged 5–25.
- Begins in the metaphysis of long bones and extends into the diaphysis. Rare in the African population.

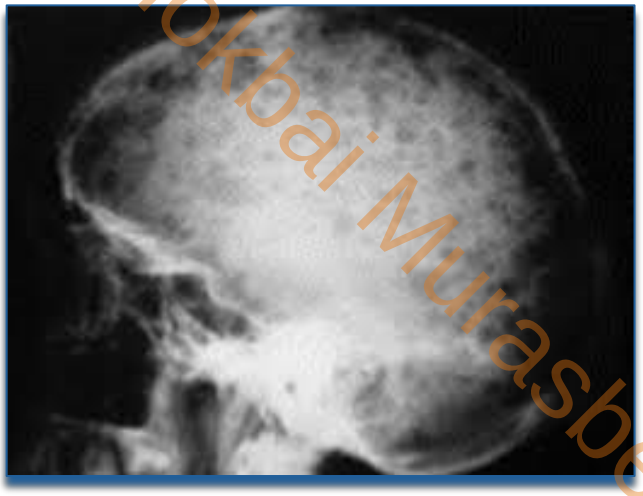
Clinical Presentation

- Pain, fever, erythema, and swelling often mimic osteomyelitis.
- Diagnosis is typically delayed, averaging 3–4 months after symptom onset due to late presentation and diagnostic delays.

Laboratory, Radiology and Histology

- Leukocytosis, elevated C-reactive protein, and increased erythrocyte sedimentation rate.
- Characteristic “onion-skin” appearance on radiographs.
- Histology shows small blue cells.
- Translocation t(11;22) (q24;q12) in 90% of cases.
- Positive for periodic acid-Schiff (PAS) staining due to intracellular glycogen.

Multiple Myeloma



- The most common primary malignant bone tumor.
- Typically seen in individuals aged 40–60.
- Pain.

Characteristics

- Causes bone fractures.
- Most commonly affects the vertebrae, followed by the hip and pelvic bones.
- Urinary findings: **Bence Jones proteinuria**.
- Blood findings: Increase in alpha-2, alpha-3, or gamma regions on electrophoresis.

Radiology

- "Punched-out" lytic lesions are observed in the bones, most commonly in the skull.

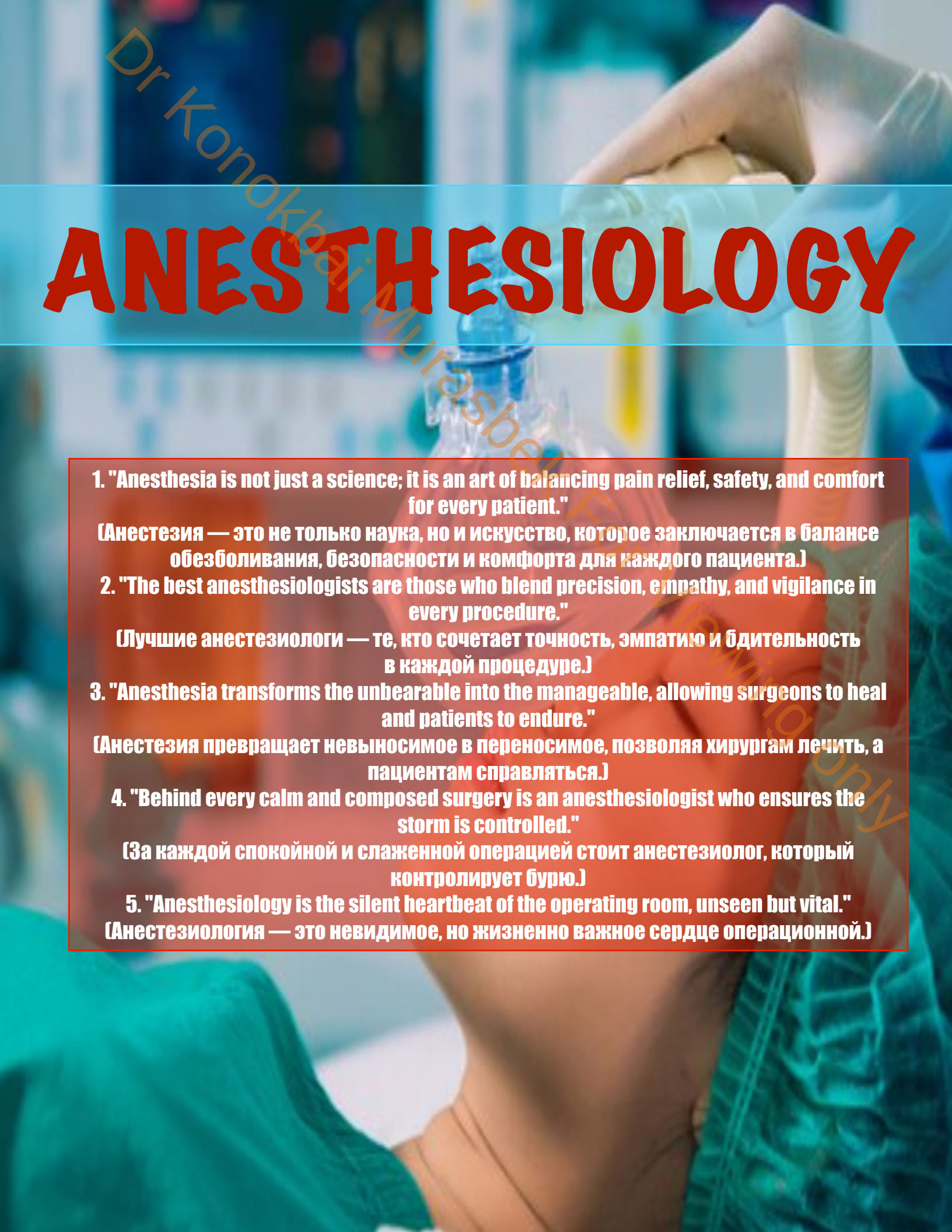
Metastatic Bone Tumors

- Bone metastases occur from the breast, prostate, lungs, kidneys, thyroid, and gastrointestinal system.
- The majority of bone metastases are osteolytic, but prostate and breast cancer metastases are osteoblastic.

Locations

- **Epiphysis**
  - Giant cell tumor
  - Chondroblastoma
  - Clear cell chondrosarcoma
- **Diaphysis**
  - Ewing sarcoma
  - Lymphoma
  - Adamantinoma

  - Fibrous dysplasia
  - Histiocytosis
- **Metaphysis-Diaphyseal**
  - Osteoma osteoma
  - Chondromyxoid fibroma
  - Non-ossifying fibroma
- **Metaphysis**
  - Osteosarcoma
  - Parosteal Osteosarcoma
  - Fibrosarcoma
  - Osteochondroma
  - Bone cyst
  - Osteoma
  - Chondrosarcoma



ANESTHESIOLOGY

1. "Anesthesia is not just a science; it is an art of balancing pain relief, safety, and comfort for every patient."
(Анестезия — это не только наука, но и искусство, которое заключается в балансе обезболивания, безопасности и комфорта для каждого пациента.)
2. "The best anesthesiologists are those who blend precision, empathy, and vigilance in every procedure."
(Лучшие анестезиологи — те, кто сочетает точность, эмпатию и бдительность в каждой процедуре.)
3. "Anesthesia transforms the unbearable into the manageable, allowing surgeons to heal and patients to endure."
(Анестезия превращает невыносимое в переносимое, позволяя хирургам лечить, а пациентам справляться.)
4. "Behind every calm and composed surgery is an anesthesiologist who ensures the storm is controlled."
(За каждой спокойной и слаженной операцией стоит анестезиолог, который контролирует бурю.)
5. "Anesthesiology is the silent heartbeat of the operating room, unseen but vital."
(Анестезиология — это невидимое, но жизненно важное сердце операционной.)

Unit : 1

GENERAL ANESTHESIA

Anesthesia is a state of analgesia and amnesia accompanied by suppressed responses of the central nervous system (CNS) to painful stimuli.

Neurolept Analgesia

- It is an analgesia achieved by intravenously combining a narcotic analgesic drug (fentanyl) with a neuroleptic drug (dehydrobenzperidol, droperidol, haloperidol).

Neurolept Anesthesia

- Neurolept anesthesia is achieved by adding nitrous oxide to the neurolept analgesia combination.
- This combination includes dopamine receptor blockers, making it contraindicated in patients with Parkinson's disease.

Balanced Anesthesia

- Balanced anesthesia is a combination of nitrous oxide, a narcotic (fentanyl), thiopental, and a neuromuscular blocker.

The **ASA (American Society of Anesthesiologists) classification** is a system used to assess and communicate a patient's preoperative physical status. It helps predict surgical risk and guide anesthetic planning.

1. **ASA I**: A healthy patient with no medical problems.
   Example: A fit, non-smoking individual with no systemic disease.
2. **ASA II**: A patient with mild systemic disease that does not limit daily activity.
   Example: Controlled hypertension or mild asthma.
3. **ASA III**: A patient with severe systemic disease that limits daily activity but is not incapacitating.
   Example: Poorly controlled diabetes or chronic obstructive pulmonary disease (COPD).
4. **ASA IV**: A patient with severe systemic disease that is a constant threat to life.
   Example: Recent myocardial infarction or severe heart failure.
5. **ASA V**: A moribund patient who is not expected to survive without the operation.
   Example: Ruptured abdominal aortic aneurysm.

6. **ASA VI**: A brain-dead patient whose organs are being removed for donor purposes.

PREMEDICATION

Premedication is the preparation of patients for surgery using medications.

Purposes of Premedication:

- Eliminate anxiety.
- Reduce secretions.
- Decrease various autonomic reflex responses.
- Provide amnesia.
- Provide analgesia.
- Induce sedation.
- Achieve antiemetic effects.
- Reduce anesthetic requirements by decreasing metabolic activity.
- Facilitate induction of anesthesia.
- Decrease gastric fluid volume and increased pH.
- Provide prophylaxis against allergic reactions.
- Relieve anxiety before regional anesthesia.
- Enhance the effect of local anesthesia.
- Address special situations (e.g., transdermal nitroglycerin in angina).

Medications Used in Premedication:

Antiemetics:

- **Anticholinergics**: Atropine, scopolamine.
- **H1 histamine antagonists**: Cyclizine, diphenhydramine, promethazine.
- **Dopamine antagonists**: Phenothiazines, butyrophenones, metoclopramide.

Sedatives and Hypnotics:

- **Barbiturates**: Pentobarbital, secobarbital.
- **Antihistamines**: Diphenhydramine, hydroxyzine.
- **Chloral derivatives**: Triclofos sodium, chloral hydrate.

Tranquilizers:

- **Butyrophenones**: Haloperidol, droperidol.
- **Phenothiazines**: Chlorpromazine, promethazine, promazine.
- **Benzodiazepines**: Diazepam, nitrazepam, temazepam.

Narcotic Analgesics:

- Morphine, pethidine (dolantin), fentanyl, buprenorphine.

Anticholinergics:

- Atropine, scopolamine, glycopyrrolate.

GENERAL ANESTHESIA

General anesthesia can be administered via various routes, but the most common are intravenous and inhalation.

Volatile Anesthetics and Guedel's Classification:

General anesthesia, particularly with volatile agents like diethyl ether, progresses through the following stages according to Guedel's classification:

1. **Analgesia Stage:**
    - The period from the induction of anesthesia to loss of consciousness.
    - Initially, analgesia occurs, followed by amnesia.
2. **Excitement Stage:**
    - The period from loss of consciousness to the onset of regular respiration.
    - Symptoms may include vomiting, breath-holding, hiccups, tachycardia, and arrhythmias.
    - Amnesia and loss of consciousness occur.
    - Respiration is irregular, and inhibitory Golgi type II neurons in the cerebellum are suppressed.
3. **Surgical Anesthesia Stage:**
    - Extends from the beginning of regular respiration to respiratory arrest and consists of four phases:

a)From the onset of regular respiration to the loss of eye movement.
b)From the loss of eye movement to the beginning of intercostal muscle paralysis.
c)From the beginning of intercostal muscle paralysis to its completion.
d)From complete intercostal paralysis to diaphragmatic paralysis.

- **Characteristics:**
  - Anesthesia is fully established, and superficial reflexes are lost.
  - Deep reflexes related to vital functions remain active.
  - Respiration is automatic and rhythmic.
  - Blood pressure and pulse return to normal.

4. **Medullary Depression Stage:**
   - Extends from diaphragmatic paralysis to cardiac arrest.
   - Pupils are fully dilated.
   - The vasomotor center and respiratory center are depressed.

Stages of General Anesthesia from an Application Perspective

1)Induction

- This is the initial phase of anesthesia.
- Induction can be administered through intravenous, inhalation, or intramuscular routes.

2)Maintenance

- This phase follows induction and is characterized by the deepening and stabilization of anesthesia.
- Surgical procedures are performed during this stage.
- The depth of anesthesia is maintained according to the requirements of the surgical procedure.

3)Emergence

- A critical phase where changes in consciousness occur, similar to induction.
- Toward the end of the surgical procedure, the dosage of anesthetics is gradually reduced.
- The most significant issues during emergence are related to respiration.

4)Recovery

- Awakening from anesthesia may be delayed due to factors related to anesthetic drugs and techniques, the patient, or the surgery:
  - Residual drug effects (overdose, delayed drug metabolism, etc.)
  - Duration and type of anesthesia

- Prolonged neuromuscular blockade (e.g., due to liver or kidney failure, plasma cholinesterase deficiency)
- Metabolic and endocrine disorders (e.g., hypothyroidism, hypo/hyperglycemia)
- Acid-base and electrolyte imbalances (e.g., hyperphosphatemia, hyponatremia)
- Hypothermia
- Hypo/hypercarbia
- Neurological complications (e.g., central anticholinergic syndrome, cerebral hypoxia, intracranial hemorrhage, embolism)

Airway Assessment

Difficult Airway

Indicators for difficult mask ventilation:

- Age 55 and above
- Body mass index > 26 kg/m^2
- Absence of teeth
- Presence of a beard
- History of snoring

Indicators for difficult airway:

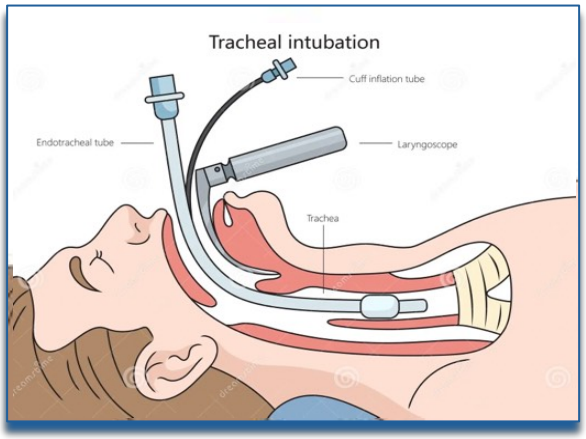
- Obstructive sleep apnea, history of snoring, obesity
- Increased neck circumference
- Thyromental distance less than 7 cm with head extension
- High Mallampati scores
- Large tongue
- Inability to protrude the mandible or advance lower teeth past upper teeth
- Facial and neck deformities from previous surgery
- Head and neck trauma
- History of head and neck radiation
- Congenital anomalies of the head and neck
- Rheumatoid arthritis

- Down syndrome
- Scleroderma
- Cervical spine diseases and previous cervical spine surgery

The **Mallampati classification** is a method used to assess the visibility of oral structures and predict the difficulty of airway management.

- **Class I:** Full visibility of soft palate, uvula, and tonsillar pillars.
- **Class II:** Soft palate and uvula are visible.
- **Class III:** Soft palate and the base of the uvula are visible.
- **Class IV:** Only the hard palate is visible; soft palate is not seen.

Higher classes (III and IV) indicate a potentially difficult airway for intubation or ventilation.



Endotracheal Intubation

Verification of the tracheal tube's placement:

- Presence of condensation or moisture inside the tracheal tube.
- Observation of tidal volume returning during expiration.
- Noticing the rise and fall of the chest.
- Auscultation of breath sounds.

- Visualization of the tube in the trachea on a lateral neck X-ray.
- Detection of end-tidal carbon dioxide (**the most definitive indicator**).
- Confirmation of placement through laryngoscopy.

Complications of Intubation

During laryngoscopy and intubation:

- **Incorrect placement**
    - Esophageal intubation

  - Bronchial intubation
  - Cuff placement in the larynx
- **Airway trauma**
  - Tooth damage
  - Laceration of the lips, tongue, or mucosa
  - Sore throat
  - Mandibular dislocation
  - Retropharyngeal dissection
- **Physiological reflexes**
  - Hypertension, tachycardia
  - Intracranial hypertension
  - Intraocular hypertension
  - Laryngospasm

Complications of Tracheal Extubation:

- Inadequate respiratory maintenance (e.g., residual anesthetic)
- Hypoxia (e.g., atelectasis)
- Upper airway obstruction (e.g., edema, residual anesthetic/decreased upper airway tone)
- Obstruction associated with the vocal cords (e.g., subglottic edema)
- Bronchospasm (airway irritation caused by the endotracheal tube)
- Aspiration (due to reduced gag and swallowing reflex)
- Hypertension
- Increased intracranial pressure
- Increased intraocular pressure
- Increased abdominal wall pressure (risk of surgical wound dehiscence)

POSTOPERATIVE PROBLEMS:

1)Respiratory Issues:

- **Airway Obstruction**
  - The primary cause is the tongue falling back in an unconscious patient.

- **Hypoventilation**
    - Postoperative pain, muscle spasms, paralysis, or drug effects can reduce respiratory control, leading to decreased ventilation effectiveness.
- **Hypoxia**
    - Causes include hypoventilation, ventilation-perfusion mismatch, and increased pulmonary shunts.
    - Conditions increasing shunting include atelectasis, pneumothorax, diffuse airway collapse, pulmonary edema, aspiration, pulmonary embolism, and shivering.
    - **Diffusion Hypoxia**: This occurs in patients who, while inhaling oxygen/nitrous oxide, switch to breathing room air. The nitrous oxide diffuses from tissues and rapidly leaves the bloodstream, diluting alveolar oxygen and carbon dioxide.
    - Administering pure oxygen for 1-2 minutes after discontinuing nitrous oxide prevents this.
- **Atelectasis**

    - The most common postoperative lung complication.
    - It is caused by pain, tight abdominal bandaging, and inability to clear bronchial secretions.
    - Symptoms include fever, tachypnea, tachycardia, and reduced breath sounds with rales heard over the atelectasis area.
    - Prevention involves effective pain control, early mobilization, coughing, and breathing exercises.
    - Respiratory therapy, postural drainage, expectorants, antibiotics, and humidified oxygen may be given.
- **Aspiration Pneumonia (Mendelson Syndrome)**
    - Hypoxia is the earliest and most reliable sign.
    - Tachycardia, tachypnea, bronchospasm, cyanosis, hypotension, and cardiovascular collapse may occur.
    - Chest X-ray may show irregular densities, particularly in the hilum.

- Respiratory support, oxygenation, and antibiotic therapy are applied.

- **Surgical Emphysema**

- Caused by excessive distension of alveoli due to the administration of pressurized gas.

- **Pneumothorax**
- **Pulmonary emboli**
    - Difficult and prolonged surgeries, long bed rest (e.g., hip fracture)
    - Venous thrombosis in the legs and pelvis
    - Postoperative symptoms (2-14 days): sudden chest pain
    - Cyanosis, fainting, hemoptysis, pleural pain, and dyspnea may occur.
    - Tachycardia, elevated central venous pressure, distension of neck veins, hypotension.
    - Chest X-ray may show right heart enlargement, diaphragm elevation, and shadows in the lower zones.
    - EKG may show signs of right heart hypertrophy.
    - **Treatment:** anticoagulation therapy, oxygen administration with sedation.

2)Air Embolism

- Occurs during surgeries that affect the neck, thorax, chest, and pelvic veins, heart surgeries, or uterine curettage.
- Diagnosis and treatment involve the introduction of air into the peritoneal and pleural cavities, bladder, uterus, and tubes.
- Caused by the sudden introduction of excessive air into vascular structures.
    - **Symptoms:** Sudden cyanosis, hypotension, tachycardia, neck vein distention, irregular breathing, tachypnea, hypopnea, and possible cardiac arrest.

- The patient is placed in the Trendelenburg position.
- Oxygen is administered, and the patient is turned to their left side.
- **Air is aspirated from the right heart via a catheter.**
- If necessary, the right thorax is opened to aspirate air from the heart and perform cardiac massage.

3) Postoperative Fever

Day1: Atelectasis
Day3: Catheter infection (thrombophlebitis or urinary tract infection)
Day5: Wound infection, if developed in the first 48 hours, caused by beta-hemolytic streptococci; on days 4-7, gram-negative bacteria, gram-positive cocci, and staphylococci are common.
Day 7: Intraabdominal abscess

4)Neurological Issues

- **Delayed recovery :** The most common causes are drug effects and cerebral hypoxia.
- **Convulsions :** Hypoxia, brain edema, cerebrovascular events, alkalosis, fever, hypo/hyperglycemia, hypokalemia, uremia, deep ether anesthesia, local anesthetics, eclampsia.
- **Postoperative cognitive dysfunction :** Awareness issues
- **Postoperative fatigue :**Excitement and hallucinations following anesthesia.Anxiety
- **Peripheral nerve injuries:**Most commonly affect the ulnar nerve, brachial plexus, and lower extremity motor nerves.
- **Ischemic spinal injury:**The most important cause is prolonged hypotension.

5) Central anticholinergic syndrome : The state of consciousness is normally maintained by continuous stimulation of the cerebral cortex by acetylcholine. Centrally acting anticholinergics (such as atropine) can lead to symptoms like amnesia, reduced concentration, agitation, dysarthria, and hallucinations.
This is more commonly seen in elderly individuals, especially those with reduced vision and hearing. Treatment involves increasing acetylcholine in the central nervous system (using physostigmine, salicylates).

6) Gastrointestinal Issues

- Aspirational Pneumonia (See Respiratory Problems)
- Regurgitation

- Nausea-Vomiting : If food was eaten within 5-6 hours before surgery, pyloric stenosis, peritoneal irritation, abdominal distension, gastric bleeding, esophageal and intestinal obstructions can prevent stomach emptying.
    - Head trauma, labor, pain, stress, poor general condition, and narcotics slow gastric emptying.
    - Anticholinergics, antihistamines, phenothiazines, butyrophenones, dopamine antagonists, and ondansetron may be used.
    - Postoperative Gastrointestinal Function after general anesthesia, stomach emptying can take up to 24 hours, small bowel movement takes 1-2 hours, and colon movement returns to normal after 48 hours.Narcotic use delays stomach emptying.
    - Neostigmine increases contractions, especially in the ileum.

7)CardiovascularIssues

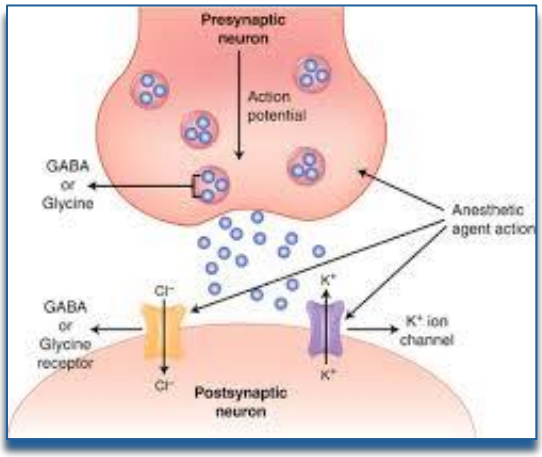
- The most common cause is a **hematoma** resulting from the removal of a cannula or needle without applying pressure to the vessel. Thrombosis and thrombophlebitis caused by intravenous agents and infusions. The most significant thrombotic complications are deep vein thrombosis and pulmonary embolism.
- **Arrhythmias and Cardiac Arrest**
- **Tachycardia** :
    - Can occur during anesthesia induction due to excitation and when anesthesia is superficial.
    - Can occur during surgery due to blood loss and shock.
    - Can be caused by atropine used in premedication.
    - Can result from hyperthyroidism or pheochromocytoma.
- **Bradycardia**
    - Sinus bradycardia, vagal stimulation.
    - Anoxia due to respiratory failure.
    - Myocardial depression caused by cardiotoxic drugs.
- **Myocardial Infarction** : often occurs on the third day after surgery and can be painless .Sudden hypotension, left heart failure, and arrhythmias during this period should raise suspicion for infarction.
- **Hypotension**
  Hypotension is the most common early complication.Causes include hypovolemia,

vasodilator therapy or the effects of anesthetics, deep anesthesia, anoxia, spinal anesthesia, myocardial infarction, hypoxia, pulmonary embolism and pneumothorax, vasovagal reflex, and anaphylactic reactions.

- **Hypertension :** May result from cyclopropane anesthesia, intracranial lesions, thyrotoxicosis, CO_2 retention, position changes or improper positioning, and pain.
- **Convulsions**: The specific treatment involves the use of medications such as meperidine/pethidine.

GENERAL ANESTHETIC DRUGS

Mechanism of Action of General Anesthetics



- They block ion channels (especially sodium channels) by altering the lipid matrix of the neuronal membrane.
- They produce general anesthesia (GA) by increasing **GABA-A receptor activity**. General anesthetics that perform this function include inhalation anesthetics, barbiturates, benzodiazepines, etomidate, and propofol.
- Ketamine, nitrous oxide (N_2O), cyclopropane, and xenon exert their effects by inhibiting the **NMDA receptors** of glutamate.

Termination of the Effect of General Anesthetics

- The most significant factor in terminating the effects of anesthetic agents is **redistribution**, which removes them from the brain.

Excretion and Metabolism

- Intravenous general anesthetics are metabolized in the **liver** and excreted from the body.
- Halogenated inhalation anesthetics are metabolized in the liver to varying degrees.
- There is a correlation between the degree to which these drugs are metabolized and their toxicity.
- **Methoxyflurane** and **halothane** are the general anesthetics that dissolve most in the blood and are therefore metabolized the most.
- The metabolism of these agents releases **fluoride**, which is toxic to the kidneys. Methoxyflurane, a halogenated ether derivative, is the general anesthetic that causes the most fluoride release.
- The metabolism of **halothane** produces **hepatotoxic trifluoroacetic acid** and bromide.

- Nitrous oxide, xenon, isoflurane, desflurane, and sevoflurane are metabolized to a much lesser extent.

PARAMETERS DETERMINING THE SPEED OF INDUCTION OF ANESTHESIA

- **Solubility in Blood (KI):** The lower the solubility, the faster the induction of anesthesia.
- **Minimum Alveolar Concentration (MAC) (B1):** A measure of anesthetic potency.
- **Alveolar Blood Flow (BT):** Higher flow facilitates faster induction.
- **Respiratory Rate (M):** The faster the respiratory rate, the quicker the induction of anesthesia.
- **Arterial-Venous Partial Pressure Difference (KI):** The greater the difference, the faster the induction of anesthesia.
- **Density of the Anesthetic Gas Mixture (KI):** Lower-density agents lead to faster anesthesia induction.

Solubility in Blood (Blood-Gas Partition Coefficient):

· The **less soluble** a general anesthetic is in blood, the **faster the induction** of anesthesia.

· Conversely, the **more soluble** it is in blood, the **slower the induction** of anesthesia.

· Same Rule for Recovery:

- The less soluble an anesthetic is in blood, the **faster the recovery** from anesthesia.
- The more soluble it is, the **slower the recovery** will be.

Minimum Alveolar Concentration (MAC):

- The alveolar anesthetic concentration at 1 atmospheric pressure that prevents response to painful stimuli in 50% of individuals.
- This value represents the potency of general anesthetics.
- The smaller the potency value, the stronger the drug.
- The lower the MAC value, the more potent the anesthetic agent. The strongest general anesthetic is **methoxyflurane**, while the **weakest** is **nitrous oxide**.

Inhalation Anesthetics

Halothane

- Causes bradycardia through Vago mimetic effects.
- Increases the heart's sensitivity to catecholamines (highest risk), leading to arrhythmias.

- The only modern inhalation anesthetic containing bromine.
- Reduces cardiac contractility dose-dependently, decreasing cardiac output and causing hypotension.
- Depresses respiration; decreases tidal volume and increases respiratory rate.
- Preferred in asthma patients due to its broncho dilatory effect.
- The anesthetic most likely to cause malignant hyperthermia.
- Causes hypothermia.
- Increases intracranial pressure.
- Reduces hepatic and renal blood flow.
- Decreases glomerular filtration rate.
- The most hepatotoxic agent. Obesity, female gender, and repeated doses increase the risk of fulminant hepatic necrosis. Not toxic to children.
- Reduces uterine contractions centrally, potentially leading to postpartum hemorrhage.

Enflurane

- Lowers mean arterial pressure and increases heart rate.
- Causes **bronchodilation** while maintaining airway reflexes.
- Should not be used in **epileptic patients** as it increases the tendency for seizures.

Isoflurane

- A potent vasodilator, causing **hypotension**.
- Causes **bronchodilation** without arrhythmogenic effects, making it suitable for asthma patients.
- Reduces cerebral oxygen consumption and does not increase intracranial pressure, making it preferred in **neurosurgery**.
- Very low potential for **hepatotoxicity** and **nephrotoxicity**.

Methoxyflurane

- The most potent general anesthetic (**lowest MAC value**).
- Does not increase sensitivity to catecholamines.
- **Nephrotoxic**, as its metabolism releases fluoride, causing **diabetes insipidus** and acute kidney toxicity (can form calcium oxalate stones in the kidneys).

Sevoflurane

- The least metabolized inhalation anesthetic in the liver.
- Does not increase myocardial sensitivity to catecholamines or cause tachycardia.
- Depresses respiration and causes **bronchodilation**.
- Does not irritate airways, making it suitable for **induction in children**.
- Safe for use in **pheochromocytoma**.
- Does not have significant organ toxicity, but its metabolites and released fluoride ions can be **nephrotoxic**, so it is not preferred in kidney failure.

Desflurane

- Among the fastest inhalation anesthetics for **induction and recovery**.
- Can irritate airways, causing **laryngospasm**, making it unsuitable for induction via mask.
- Exists as a gas at room temperature.
- Requires special vaporizers due to its high vapor pressure.
- Reacts with dry **baralyme**, producing carbon dioxide.
- Excreted unchanged through the lungs with minimal metabolism.

Nitrous Oxide (N_2O)

- A good **analgesic** but a weak anesthetic.
- Prolonged use (>1 hour) increases the incidence of postoperative nausea and vomiting.
- Non-toxic to the liver and kidneys.
- Has a **euphoric effect**.
- Does not cause **malignant hyperthermia**.
- Mildly depresses the myocardium, but this is counterbalanced by its sympathetic stimulant effect.
- Increases respiratory rate (**tachypnea**) and decreases tidal volume.
- Does not cause significant depression of respiratory or cardiovascular systems.
- No muscle relaxation effects, making it safe in childbirth without uterine relaxation.
- Has **anticonvulsant effects**.
- Increases cerebral blood flow and volume, slightly raising intracranial pressure.

- May cause **methemoglobinemia**.
- Inhibits vitamin B12-dependent enzymes, potentially causing **bone marrow depression**.
- **Teratogenic**
- During recovery, **diffusion hypoxia** can occur.
- Accumulates in gas-containing body cavities (e.g., middle ear, intestines, sinuses), causing volume or pressure increases.

Xenon

- An inert gas, present in low concentrations in the atmosphere.
- The anesthetic with the **lowest blood: gas partition coefficient**, enabling rapid induction and recovery.
- Low MAC value (0.7%) with both anesthetic and analgesic effects via NMDA receptor inhibition.
- Not metabolized, excreted unchanged by the lungs.
- Minimal effects on cardiovascular, neural, and respiratory systems.
- Does not trigger **malignant hyperthermia**.
- Limited use due to its **high cost**.

INTRAVENOUS ANESTHETICS

Used for induction but can also be applied for maintenance of surgical anesthesia.

Due to high lipophilicity, they quickly penetrate the brain and are redistributed away from the brain.

Ketamine

- Can be administered intravenously, intramuscularly, orally, and rectally.
- Causes significant enzyme induction in the liver; thus, frequent and repeated use may lead to tolerance.
- Noncompetitive NMDA receptor antagonist.
- Increases cerebral blood flow, metabolism, oxygen consumption, and cerebrospinal fluid (CSF) pressure.
- Does not cause nausea or vomiting.
- Creates **dissociative anesthesia** (a state resembling catatonia with analgesia and amnesia without significant loss of consciousness; the patient appears awake).

- Awakening from anesthesia may be turbulent (hallucinations, nightmares).
- Increases intraocular pressure and may cause nystagmus.
- **Increases blood pressure** and cardiac output, making it a good choice in shock patients.
- Should not be used in **myocardial infarction** as it increases myocardial oxygen consumption.
- Causes bronchodilation and minimal respiratory depression.
- Stimulates exocrine gland secretion, so it should be used with anticholinergics.
- May lead to mood and memory disturbances.
- **Has analgesic effects.**
- Can be used alone for minor surgeries.
- Useful in conditions where maintaining blood pressure is challenging, such as shock, poor general condition, or in the elderly.
- Used in open-heart surgery.
- Induction of anesthesia in small children.
- Despite causing myoclonus and increased EEG activity, it is anticonvulsant and can be a last-resort agent for **status epilepticus**.

Propofol

- Acts by binding to GABA receptors.
- Not recommended for patients with egg allergies.
- Its emulsion can support bacterial growth; strict sterility must be ensured (use within 6 hours).
- Causes pain at the injection site, which can be mitigated by using larger veins or mixing lidocaine with propofol.
- Anesthetic effect ends due to redistribution from central lipophilic tissues to peripheral compartments.
- Metabolized primarily in the liver; inactive, water-soluble metabolites are excreted via the kidneys.
- **Weak analgesic effect**.
- Causes dose-dependent hypotension and depresses the cardiovascular system.
- Suppresses laryngeal reflexes.
- Has antiemetic effects.

- Preferred for outpatient surgeries as it prevents postoperative nausea and maintains clear mental status.
- Does not cross the placenta, making it safe for use in pregnancy.
- Reduces cerebral blood flow, metabolism, and intracranial pressure.
- Decreases intraocular pressure.
- Prolonged high-dose infusions can cause **propofol infusion syndrome** in critically ill patients, characterized by heart failure, rhabdomyolysis, metabolic acidosis, renal failure, hyperkalemia, hypertriglyceridemia, and hepatomegaly.

Etomidate

- Structurally unique among anesthetics, contains an imidazole ring.
- Causes pain during injection (due to propylene glycol), which can be reduced by intravenous lidocaine.
- Enhances GABA receptor affinity but primarily acts on the beta subunit of GABA receptors.
- Preserves coronary hemodynamics.
- Decreases cerebral oxygen consumption, blood flow, and intracranial pressure.
- Reduces adrenal steroid synthesis (inhibits 11-beta-hydroxylase enzyme); should not be used in **adrenal insufficiency**.
- Contraindicated in **porphyria**.
- Causes subcortical disinhibition, leading to myoclonic involuntary movements during induction.

Midazolam

- A short-acting benzodiazepine.
- Causes **anterograde amnesia**.
- May cause paradoxical/disinhibitory reactions like excitation or aggression.
- Frequently used in adults and children for premedication.
- Does not have analgesic properties.
- **Flumazenil** is a specific competitive antagonist with high affinity for benzodiazepine receptor sites.

Thiopental

- Inhibits the reticular activating system in the brainstem.
- Enhances GABA receptor activity, prolonging chloride ion channel opening.

- Reduces cerebral oxygen consumption, blood flow, and intracranial pressure.
- Can cause severe laryngospasm and respiratory depression; use cautiously in respiratory-compromised patients.
- Highly alkaline (pH 9-10), may precipitate with acidic drugs and irreversibly block intravenous catheters.
- Extravasation can cause severe tissue necrosis and pain.
- Does not have analgesic effects and may even cause **hyperalgesia**.
- Intra-arterial administration causes endothelial damage, vasoconstriction, and thrombosis.
- Anesthetic effect terminates due to redistribution from central lipophilic brain tissues to lean peripheral muscle compartments.
- Metabolized in the liver, conjugated in bile, and slowly eliminated via the kidneys.
- Contraindicated in **porphyria**, **status asthmaticus**, and **barbiturate allergies**.

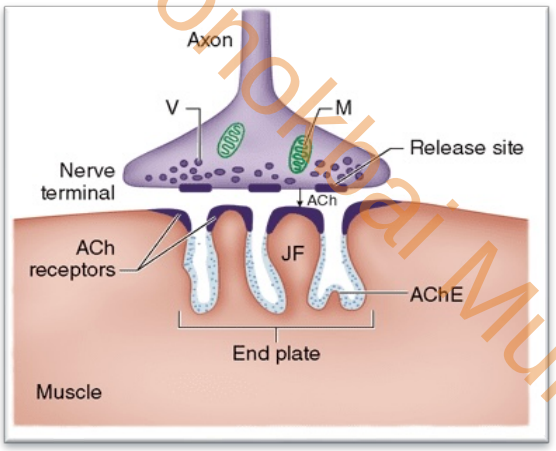
Dexmedetomidine

- Activates alpha-2 adrenergic receptors in the central nervous system, causing sedation.
- Provides sedation and analgesia without significant respiratory depression.
- Continuous infusion for more than 24 hours may cause **rebound hypertension**, **rebound excitability**, and arrhythmias.

NEUROMUSCULAR BLOCKERS

- **Muscle and nerve transmission can be blocked** by inhibiting acetylcholine synthesis, release, or interaction with receptors.
- These drugs specifically block **nicotinic receptors** at the neuromuscular junction.
- Sensitivity to these drugs increases in conditions such as **myasthenia gravis**, **hypokalemia**, **hyponatremia**, **hypocalcemia**, **hypermagnesemia**, and with certain antibiotics (aminoglycosides, polymyxin B, lincosamides, tetracyclines), inhalational anesthetics/local anesthetics, Class I antiarrhythmics, lithium, hypothermia, and acidosis.

Depolarizing Block

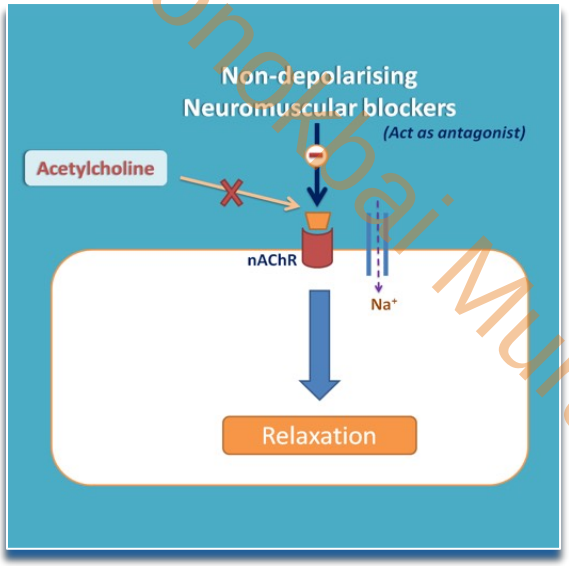


- This type of block is preceded by **fasciculations** due to depolarization.
- **Not antagonized by anticholinesterases**.
- Potentiated by **acetylcholine, isoflurane, enflurane, alkalosis, hypothermia**, and **Mg^{2+}**, while antagonized by **ether, halothane, and acidosis**.
- **Depolarizing agents:** decamethonium and succinylcholine.
- **Main disadvantages:** hyperkalemia and bradycardia.

Succinylcholine

- At high doses, it may show **non-depolarizing block** effects.
- Stimulates the vagus nerve, causing **bradycardia**.
- Increases **serum potassium levels**.
- Raises **intraocular pressure**.
- Causes **postoperative muscle pain**.
- Increases **salivary and gastric secretions**.
- Metabolized by **pseudocholinesterase** produced in the liver and present in plasma.
- In pseudocholinesterase genetic deficiencies:
    - Leads to **prolonged apnea**.
    - May result in **malignant hyperthermia**.
- Prolonged effects occur in **liver failure**.

Non-depolarizing Block



Prevents acetylcholine from reaching the receptor and inhibits depolarization.

- Fasciculations **do not occur** before the block.
- **Antagonized by anticholinesterases** (e.g., neostigmine, edrophonium, physostigmine, pyridostigmine).

Types of Non-depolarizing Agents

- **Long-acting:** Pancuronium, pipecuronium, doxacurium, gallamine, metocurine.
- **Intermediate-acting:** Atracurium, cisatracurium, vecuronium, rocuronium.
- **Short-acting:** Mivacurium, gantacurium.

Histamine Release Potential: Tubocurarine > metocurine > atracurium > mivacurium.

Pancuronium Bromide

- **Minimal or no histamine release**.
- **Crosses the placenta in small amounts**.
- Causes **vagal block**.
- Does not cross the blood-brain barrier.
- **Should not be used in renal failure**.
- Stimulates the myocardium, increasing **blood pressure** and **pulse rate**.
- Does **not accumulate** in the body.
- Duration of effect: **60 minutes**

Atracurium Besylate

- Eliminated via **Hoffman degradation** (spontaneous breakdown under physiological conditions).
- Does not affect the **cardiovascular system**.

- Does not alter **intracranial or intraocular pressure**.
- Suitable for use in **anuric patients**.
- Duration of effect: **15-35 minutes**

Cisatracurium

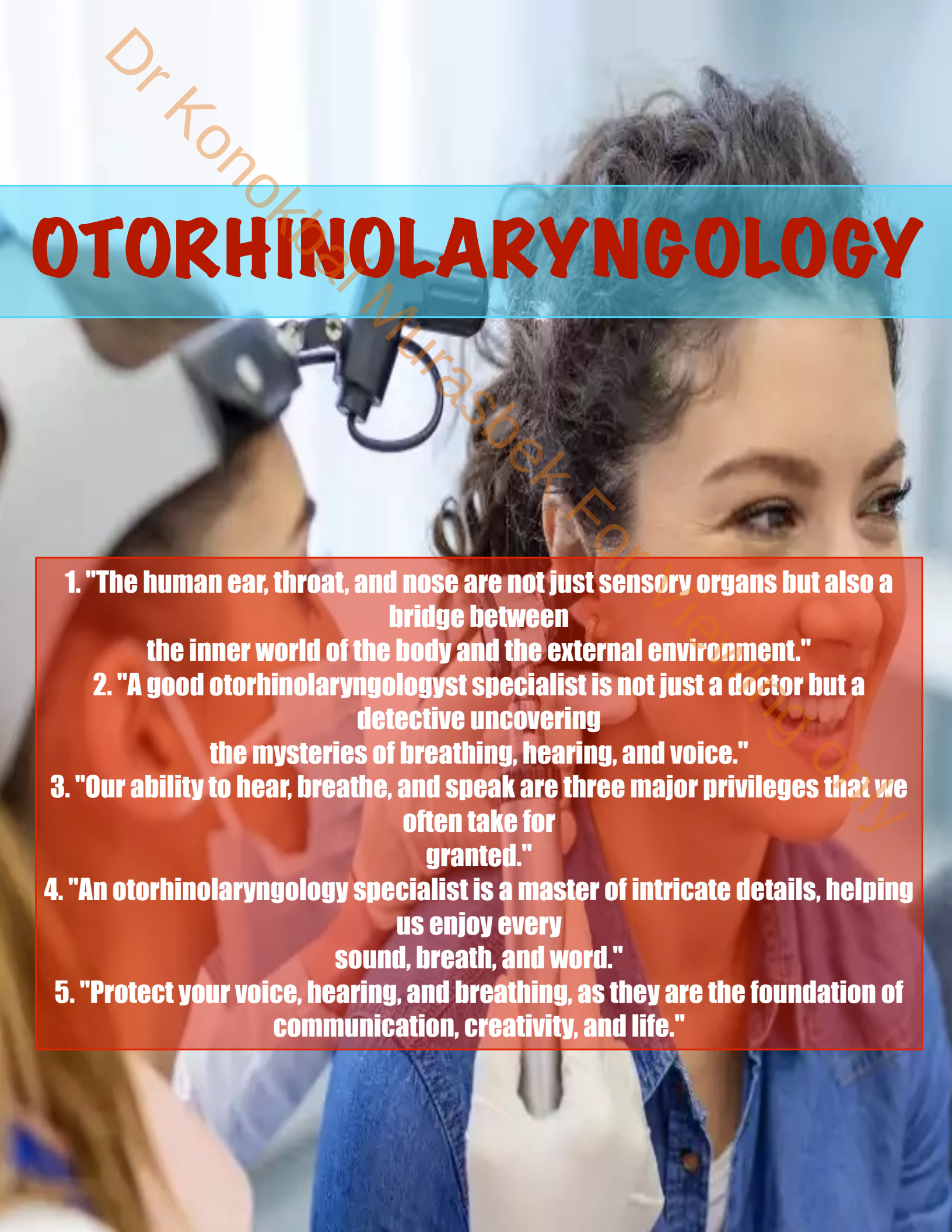
- A stereoisomer of atracurium.
- Causes **histamine release**, but does not lead to hemodynamic changes.
- 3-4 times **stronger** than atracurium.
- Metabolized via **Hoffman degradation**.
- **Not influenced** by end-organ disease or aging.
- Duration of effect: **20-29 minutes**

Vecuronium Bromide

- **No cardiovascular effects**—the only non-depolarizing muscle relaxant without such effects.
- Can be used in **renal failure**.
- Its effect is most enhanced by **enflurane**.
- Duration of effect: **20-30 minutes**

Rocuronium:

- Intubation dose: **0.6 mg/kg** achieves good-to-excellent intubation conditions in **60-90 seconds**.
- Duration of effect: **25-30 minutes**.
- **Sugammadex** is used to reverse its effects.



OTORHINOLARYNGOLOGY

1. "The human ear, throat, and nose are not just sensory organs but also a bridge between
the inner world of the body and the external environment."
2. "A good otorhinolaryngologyst specialist is not just a doctor but a detective uncovering
the mysteries of breathing, hearing, and voice."
3. "Our ability to hear, breathe, and speak are three major privileges that we often take for
granted."
4. "An otorhinolaryngology specialist is a master of intricate details, helping us enjoy every
sound, breath, and word."
5. "Protect your voice, hearing, and breathing, as they are the foundation of communication, creativity, and life."

Unit : 1

EAR DISEASES

EXTERNAL EAR CANAL DISEASES

Acute Localized Otitis Externa (Furuncle)

- The most common causative agent: *Staphylococcus aureus*.

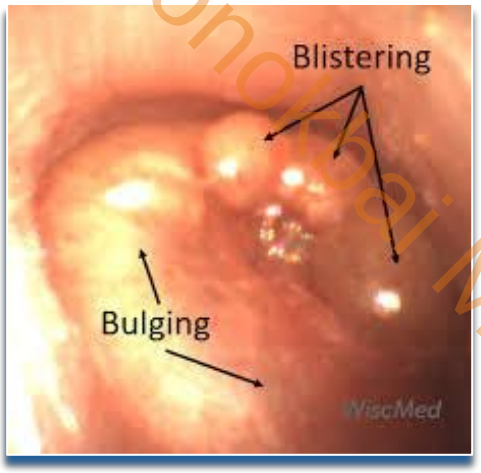
Acute Diffuse Otitis Externa (Swimmer's Ear)

- The most common causative agent: *Pseudomonas aeruginosa*.
- Symptoms: Pain, edema, and discharge in the external ear canal.
- Tenderness on tragus palpation.

Necrotizing (Malignant) Otitis Externa

- The most common causative agent: *Pseudomonas aeruginosa*.
- Typically affects elderly diabetics and immunosuppressed patients.
- Deep tissue involvement: Osteomyelitis of the temporal bone and skull base, meningitis, brain abscess.
- Progressive and fatal course.

Bullous Myringitis



Characterized by bullae on the tympanic membrane after an upper respiratory tract infection (URTI).

Although most common causes are viral, *Mycoplasma pneumoniae* is also significant.

Associated with coughing and ear pain.

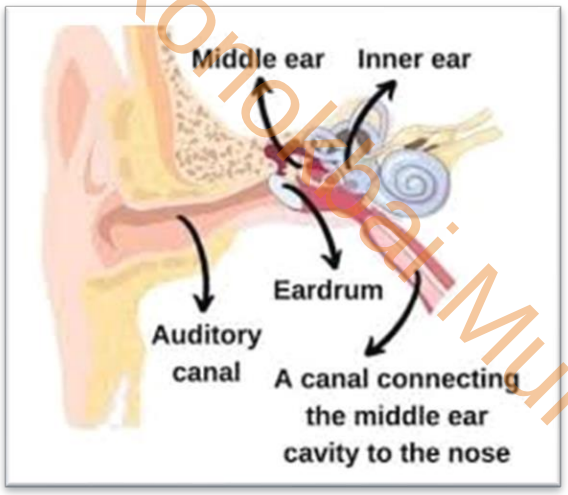
Fungal Otitis Externa (Otomycosis)

- The most common causative fungus: *Aspergillus Niger*.

OTITIS MEDIA AND MIDDLE EAR EFFUSIONS

Acute Otitis Media (AOM)

- Most common age of occurrence: 6-13 months.
- Most common causative agents: *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Moraxella catarrhalis*.
  - In children under five, the incidence of *Haemophilus influenzae* increases.
- Etiology: Eustachian tube dysfunction (obstruction) is the most important factor.
  - In children under seven, the Eustachian tube is shorter, more horizontal, less functional, and less mature.
- Transmission occurs via an ascending route following URTI.



Causes of Recurrent Acute Otitis Media:

- Adenoid hypertrophy.
- Immunodeficiency.
- Rhinitis.
- Cystic fibrosis.

Treatment:

- Antibiotics, anti-inflammatory medications, and paracentesis performed in the posterior-inferior quadrant.

Chronic Otitis Media

- Persistent middle ear infection lasting over three months with tympanic membrane perforation.
- Features: Perforated eardrum and intermittent ear discharge.
- Two types:
    - **Simple Chronic Otitis Media:** Treatment involves tympanoplasty surgery.
    - **Cholesteatomatous Otitis Media:** Cholesteatoma results from the invasion of external ear canal epithelium into the middle ear, extending to mastoid cells.
        - Treatment: Mastoidectomy.
        - Most common site: Posterior superior part of the tympanic membrane (pars flaccida).

Complications of Otitis Media:

Acute Mastoiditis: Redness in the mastoid region.

Subperiosteal Abscess: Behind the ear, hyperemia and swelling; the ear is pushed forward.

Labyrinthitis: Associated with vertigo and nystagmus.

Petrositis: Retro-orbital pain; when accompanied by abducens nerve palsy and acute/chronic otitis media with discharge, it is called Gradenigo syndrome.

- **Facial Paralysis:**
    - If due to acute otitis, treatment includes myringotomy and antibiotics.
    - If due to chronic otitis, surgery is the only option.
- **Sigmoid Sinus Thrombosis:** May present with spiking fever and signs of increased intracranial pressure.
- **Otitic Hydrocephalus:** Characterized by increased intracranial pressure and enlarged ventricles due to sigmoid sinus thrombosis.
- **Meningitis:** More common in children.
- **Subdural Abscess:** More frequent in sinusitis than otitis. It has a catastrophic course, often resulting in rapid loss of consciousness and death.

Acute and Chronic Otitis Media Complications:

1. **Intertemporal-Complications:**
   a. Mastoiditis (most common complication of otitis):
    - Subperiosteal abscess.

2.Bezold abscess: Sternocleidomastoid muscle region.

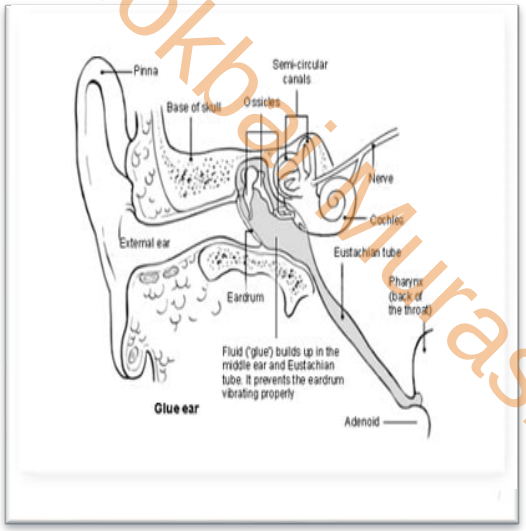
3.Citelli abscess: Posterior belly of digastric muscle.

4.Luc-abscess:Zygomaticregion.
b.Petrositis.
c.Labyrinthitis.
d.Facial-paralysis.
e. Deep neck infection.

Intracranial-Complications:
a.Extradural-abscess/granulation.
b.Sigmoid-sinus-thrombosis.
c.Brain-abscess.
d.Otitis-hydrocephalus.
e.Meningitis (most common intracranial complication of otitis).
f. Subdural abscess.

Otitis Media with Effusion (OME)

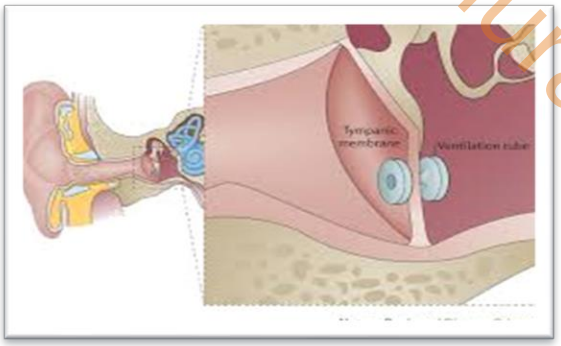
- Fluid accumulation in the middle ear without signs of infection like fever or pain.



- Middle ear mucosa undergoes metaplasia, increasing goblet cell numbers and secretions.
- Most common age: 3-8 years.
- Causes: Allergy, viral-bacterial infections, and Eustachian tube dysfunction.
- Most common symptom: Conductive hearing loss.
- Does not lead to deep neck infections.

- **Treatment :**
  Antibiotics
  Antihistaminic
  Anti inflammation
  Decongestant
  Mucolytic ventilation tube

Indications for Ventilation Tube in OME:



- Retractions in the tympanic membrane.
- Accompanying sensorineural hearing loss.
- Speech delay.
- Hearing loss exceeding 35 dB.
- Recurrent otitis attacks.
- Persistent effusion.
- Transition to autumn-winter seasons.

OME in Adults:

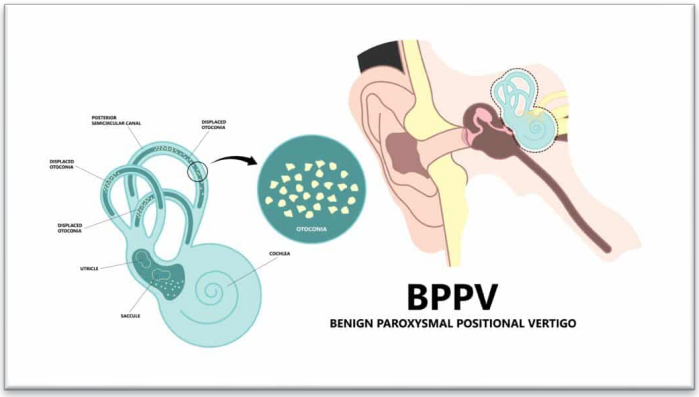
- Most commonly follows viral URTI.
- Nasal pathologies such as nasal polyposis, septal deviation, and rhinosinusitis are significant causes in adults.

Unit : 2

PERIPHERAL DISEASES CAUSING BALANCE DISORDERS

- **Benign Paroxysmal Positional Vertigo (BPPV) (most common)**
- **Vestibular Neuritis**
- **Meniere's Disease or Endolymphatic Hydrops**
- **Vestibular Ototoxicity**
- **Otosclerosis**
- **Cogan Syndrome**

Benign Paroxysmal Positional Vertigo (BPPV)



Etiology:

- The cause is unknown in many cases (head trauma? sudden head movements?).

Pathophysiology:

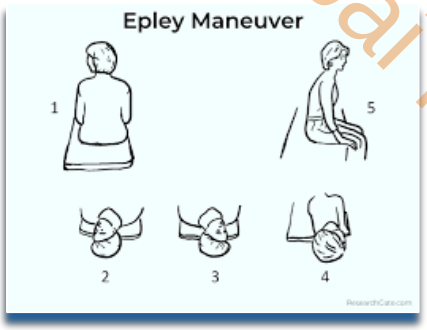
- **Canalithiasis:** Caused by otoconia dislodged from the utricle into the endolymph, most commonly in the posterior semicircular canal.
- Dizziness triggered by head movements lasting 10-20 seconds and resolving shortly.
- **Most common:** Posterior semicircular canal BPPV.

Diagnosis:

- Provocation tests: **Dix-Hallpike maneuver**, **modified Barany test**.

- Latency period (2-3 seconds) before nystagmus appears.
- **Horizontal rotatory nystagmus**, lasting less than 1 minute (fatigable nystagmus).
- Repeated testing reduces nystagmus.

Treatment: Repositioning maneuvers (Epley maneuver).

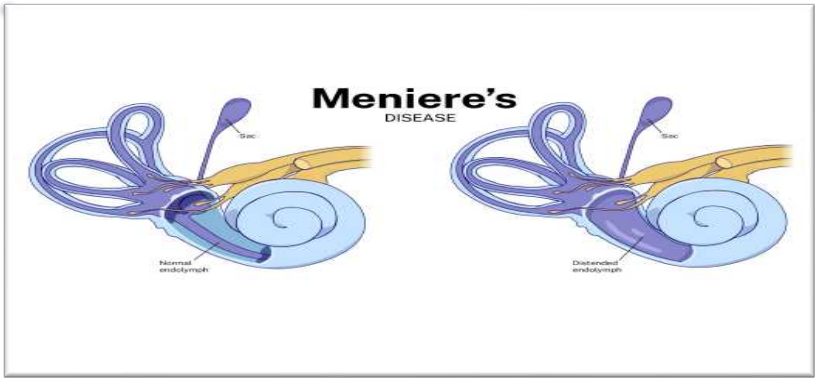


Vestibular Neuritis

- **History:** Often follows upper respiratory tract infections (URTI) or gastroenteritis.
- **Symptoms:** Severe dizziness, nausea, vomiting .
- **Findings:** Spontaneous nystagmus, horizonto-rotatory nystagmus .
- **Normal:** Neurological exam and hearing.
- Significant symptom improvement within 48 hours.
- No associated ear symptoms (tinnitus, hearing loss, fullness).

Meniere's Disease

Pathophysiology: Endolymphatic hydrops; either increased production or decreased absorption of endolymph.



- **Symptoms:**
  - Recurrent attacks every 2-3 months.
  - Vertigo that awakens the patient from sleep is typical.
  - Sensorineural hearing loss progresses with each attack.
  - Sudden drop attacks (Tumarkin crises) are characteristic.

Diagnostic Criteria:

1. Vertigo

2. Ear fullness
3. Hearing loss
4. Tinnitus

- **Findings:** Horizontal nystagmus is present but not a diagnostic criterion.

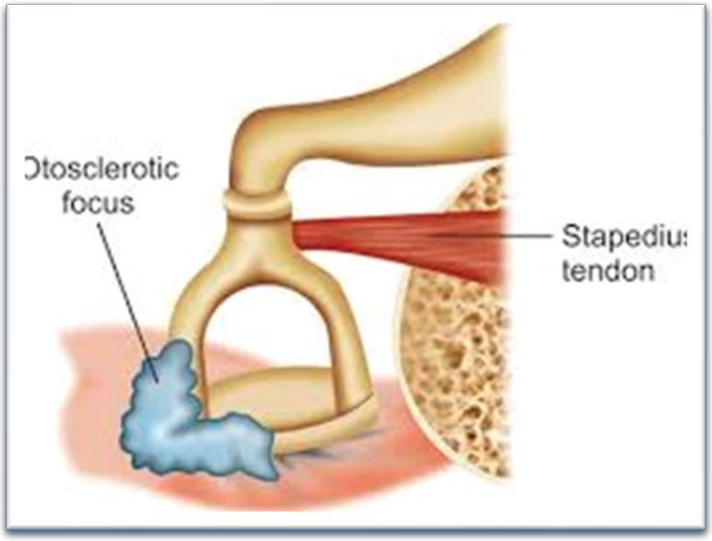
Treatment:

- **Dietary:** Reduce salt intake.
- **Medications:** Vasodilators, diuretics (e.g., acetazolamide).
- **Severe cases:** Medical treatment (intratympanic gentamicin) or surgical labyrinthectomy (vestibular nerve section).

Vestibular Ototoxicity

- **Ototoxic Drugs:**
    - **Antibiotics:** Aminoglycosides, vancomycin, erythromycin, tetracycline, capreomycin.
    - **Diuretics:** Loop diuretics.
    - **Chemotherapeutic Agents:** Nitrogen mustard, vincristine.
    - **Others:** Salicylates, quinine.

Otosclerosis



- **Definition:** Formation of spongy bone tissue in the otic capsule (surrounding the inner ear and labyrinth).
- **Common Site:** Typically bilateral, starting at the oval window (fissula ante fenestram).

Etiology:

- 90-95% autosomal dominant inheritance.
- Fluorine-zinc deficiency, puberty, menopause.
- Common in osteogenesis imperfecta.

- **Measles virus.**

Pathophysiology:

- **Oval window involvement:** Begins with conductive hearing loss and may progress to sensorineural hearing loss.
- **Cochlear involvement:** Leads to sensorineural hearing loss.

Symptoms:

- **Paracusis:** Improved hearing in noisy environments, seen in all conductive losses.

Diagnosis:

- Mixed sensorineural hearing loss.
- Acoustic reflex abnormality.
- **Carhart notch:** Drop in bone conduction at 2000 Hz on audiometry.
- **Schwartz sign:** Pink hue on the tympanic membrane due to vascular reflection over the promontory, seen early.
- Family history of symmetrical progressive hearing loss.

Treatment:

- Stapedectomy with prosthesis placement in oval window otosclerosis.
- **Complication:** Permanent sensorineural hearing loss.

Cogan Syndrome

- Develops after upper respiratory infections caused by **Chlamydia.**
- Characterized by interstitial keratitis, sensorineural hearing loss, and episodic vertigo.
- May be confused with Meniere's syndrome.

Middle Ear Tumors

The most common malignant tumor of the middle ear is squamous cell carcinoma.

The most common benign tumor of the middle ear is glomus jugulare.

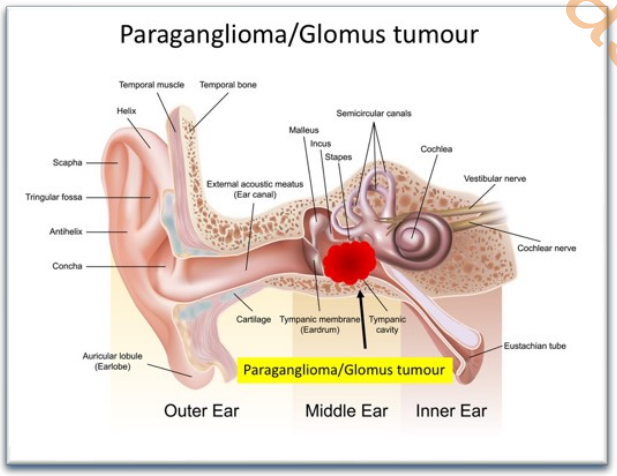
Glomus Tumors

During embryological development, paraganglial cells migrate into the sympathetic ganglia.

Carotid Body Tumors (Glomus Caroticum):

- Arise from the bifurcation of the common carotid artery.
- The most common glomus tumor in the head and neck region.
- Separates the internal and external carotid arteries.
- These tumors move laterally but do not move in the cranio-caudal (up and down) direction.

Vagal Paraganglioma (Glomus Vagale):



- Arises from the paraganglion cells in the perineurium of the vagus nerve.
- During surgery, the nerve is usually cut, so post-surgical vocal cord paralysis may develop.
- Paragangliomas (glomus tumors) are neuroendocrine tumors that arise from extra-adrenal paraganglia cells.
- Sometimes functional, they can secrete norepinephrine and dopamine.

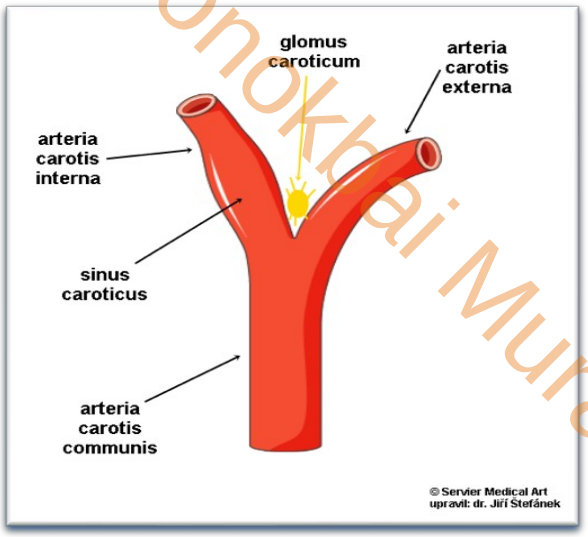
Glomus Tympanicum:

- Arises from the tympanic branch of the glossopharyngeal nerve (Jacobson's nerve).
- A pulsatile red lesion is seen behind the tympanic membrane.
- Patients often have pulsatile tinnitus.

Glomus Jugulare:

- Arises from the paraganglial cells in the adventitia of the jugular bulb.
- The tumor grows by eroding the temporal bone.
- The tumor's vascular structure often causes pulsatile tinnitus.
- If the tumor opens into the middle ear, it can appear as a blue-colored lesion behind the tympanic membrane.
- As the tumor grows, it may cause jugular foramen syndrome (involving cranial nerves IX, X, and XI).
- Rarely, it may involve cranial nerves VII and XII.

Carotid Body Tumors (Glomus Caroticum):



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Sudden Hearing Loss

It is sensorineural hearing loss that occurs within less than 12 hours.

Factors Affecting Prognosis:

- The severity of the hearing loss.
- The pattern of the hearing loss on the audiogram (flat, rising, or falling between frequencies).
- Vertigo.
- Age.

It is usually unilateral.

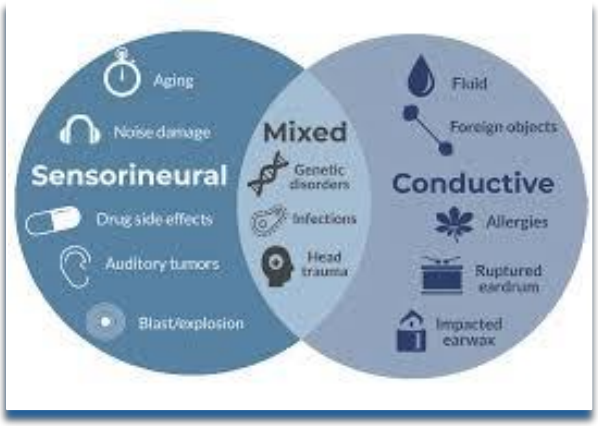
Causes:

- Viral infections
- Acoustic neuroma
- Meningitis
- Multiple sclerosis
- Lyme disease
- Sarcoidosis

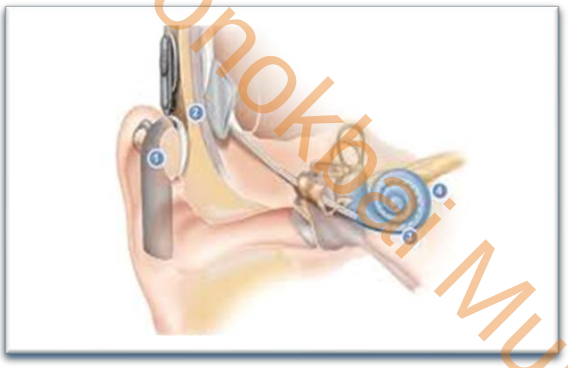
- Syphilis
- Head trauma
- Acquired immunodeficiency syndrome (AIDS)
- Meniere's disease
- Acoustic trauma
- Ototoxic drugs
- Vascular: Labyrinthine artery embolism, thrombosis, spasm, or hemorrhage
- Autoimmunity: Caused by antigen-antibody complexes in the perilymph.

Treatment: High-dose steroids and hyperbaric oxygen are administered.

Types of Hearing Loss



Cochlear implant



Indications for Cochlear Implants:

- Children who have not passed the age of learning speech (4-5 years old).
- Adults with bilateral hearing loss close to 90-100 decibels.

Contraindications:

- Narrow internal acoustic meatus (absence of the 8th cranial nerve).
- Michel aplasia (absence of the cochlea).
- Cochlear ossification (late sequelae of meningitis).

Unit :3

Rhinology

Rhinitis

Rhinitis refers to symptoms such as itching inside the nose, congestion, rhinorrhea, sneezing, and obstruction.

Allergic Rhinitis

Characterized by findings in the nose due to allergens. It is associated with IgE and often develops in response to pollen and house dust mites. This type benefits the most from antihistamines and topical steroids.

- **Seasonal allergic rhinitis:**
    - Symptoms worsen during spring seasons.
- **Perennial allergic rhinitis:**
    - Symptoms persist throughout the year.

Non-Allergic Rhinitis

Characterized by symptoms similar to allergic rhinitis but with negative allergy tests (prick test, specific IgE). It is divided into five groups:

1. **Idiopathic (Vasomotor) Rhinitis:**
    - Nasal autonomic dysfunction is present. Congestion and rhinorrhea occur due to inadequate sympathetic response to increased parasympathetic stimulation.
2. **Non-Allergic Rhinitis with Eosinophilia (NARES):**
    - Negative skin tests and specific IgE, with more than 20% eosinophils observed in a nasal smear.
3. **Irritative Toxic Rhinitis:**
    - Caused by exposure to chemical solvents and cigarette smoke. Symptoms arise from direct irritation of the mucosa rather than immunological mechanisms.
4. **Hormonal Rhinitis:**
    - The most common form is pregnancy rhinitis.

5. **Drug-Induced Rhinitis:**
    - The most significant type is **rhinitis medicamentosa**, caused by prolonged use of vasoconstrictive agents (e.g., oxymetazoline, xylometazoline - Iliadin, Otrivin).

- **Samter's Triad:**
    - The co-occurrence of aspirin hypersensitivity, asthma, and allergic rhinitis (with nasal polyps) in the same patient.

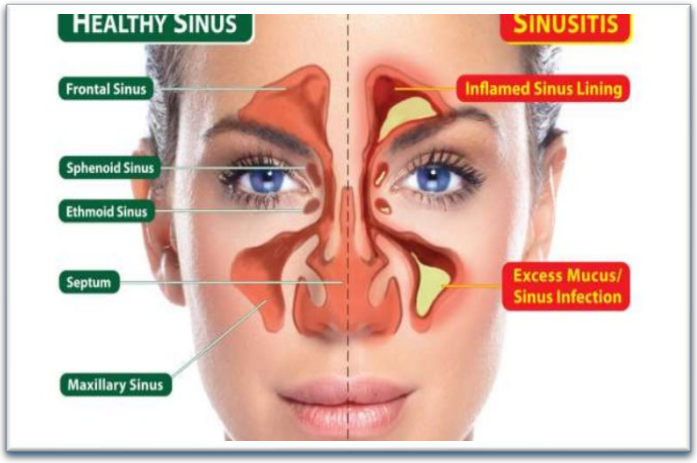
Chronic Atrophic Rhinitis (Ozaenae)

- In some cases, caused by **Klebsiella ozaenae**.
- Characterized by progressive atrophy of mucosa and turbinate bones, squamous epithelial metaplasia, thick secretions, crusting, and foul odor.

Sinusitis

At birth, the ethmoid and maxillary sinuses are present.

- **Children:** Most common is ethmoid sinusitis.
- **Adults:** Most common is maxillary sinusitis.



Etiology

- **Streptococcus pneumoniae**, **Haemophilus influenzae**, and **Moraxella catarrhalis**.

Predisposing Factors for Sinusitis

- Nasal allergies
- Septal deviation
- Ciliary dysfunction (e.g., immotile cilia, Kartagener syndrome)
- Adenoid hypertrophy

Rhinosinusitis Complications

Complications are categorized into three groups:

1. **Orbital Complications:**
    - **Most common cause:** Ethmoid sinusitis.
    - **Most common pathogens:** Streptococcus species.
    - **Complications:**

- **Preseptal cellulitis:** Most common complication. Characterized by eyelid edema and redness without affecting eye movements or visual acuity.
- **Orbital cellulitis:** Includes proptosis, chemosis, painful and restricted eye movements, and diplopia.
- **Subperiosteal abscess:** Abscess between periosteum and lamina papyracea.
- **Orbital abscess:** Progression of orbital cellulitis, characterized by severe exophthalmos, chemosis, complete ophthalmoplegia, and may lead to blindness.
- **Cavernous sinus thrombosis:** Can also be considered an intracranial complication. Notable for involving the contralateral eye. Treated with antibiotics and abscess drainage.

2. **Intracranial Complications:**
    - **Frontal sinusitis** more frequently leads to brain complications.
    - **Most common pathogen in meningitis:** Streptococcus pneumoniae.
    - **Complications:**
        - Meningitis
        - Epidural abscess (frontal sinusitis complication)
        - Subdural abscess (high mortality and morbidity)
        - Intracerebral abscess
        - Venous sinus thrombosis

3. **Bone Complications:**

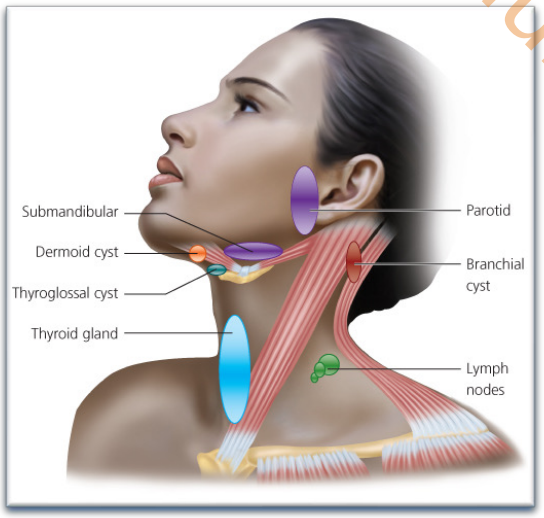
- **Frontal sinusitis** can lead to osteomyelitis.
- **Pott's Puffy Tumor:** Swelling due to pus accumulation under the periosteum.

Unit : 4

NECK DISEASES

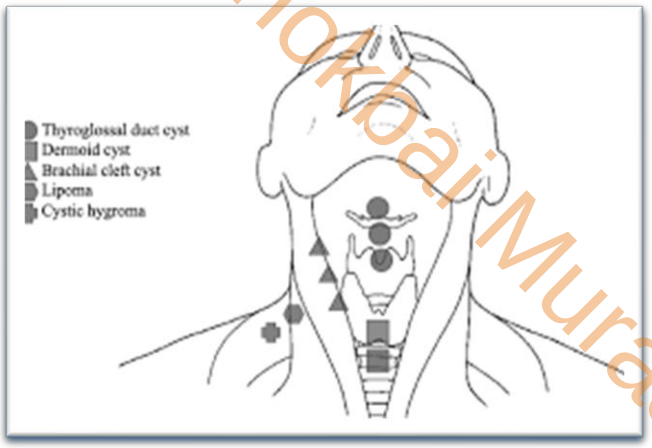
CONGENITAL NECK MASSES AND CYSTS

Lateral Neck Masses



1. **Branchial Anomalies**:
   - Depending on their contents, they are called branchial cysts or branchial sinuses.
   - Classified into six types based on their origin, with the **2nd branchial cyst** being the most common.
   - The 2nd branchial cysts appear as cysts deep within the sternocleidomastoid (SCM) muscle on the side of the neck.
2. **Laryngocele**:
   - Air-filled spaces originating from the ventricle's saccule due to increased intra-laryngeal pressure.
   - If it is within the larynx, it is called **internal laryngocele**, while if it protrudes through the thyrohyoid membrane, it is termed **external laryngocele**.
   - It is a lateral neck lesion.
3. **Thymic Cyst**:
   - During embryonic development, the thymus migrates from the lateral neck to the mediastinum. Residual thymic tissue along this path appears as a lateral neck mass.
4. **Pseudotumor of Infancy**:
   - Also known as **sternocleidomastoid tumor** or **fibromatosis coli**.
   - Spontaneously regresses, though around 20% progress to torticollis.

Midline Masses



1. **Thyroglossal Duct Cyst**:
   - Remnants of thyroid tissue migrating from the tongue root during embryonic development.
   - Appears as a midline cystic mass.
   - It is the **most common congenital neck mass**.
2. **Dermoid Cyst**:
   - Benign teratomas.
3. **Plunging Ranula**:
   - Caused by retention in the sublingual gland.
   - Appears at the floor of the mouth and extends into the neck.
4. **Cervical Teratomas**:
   - Can be midline or lateral in location.

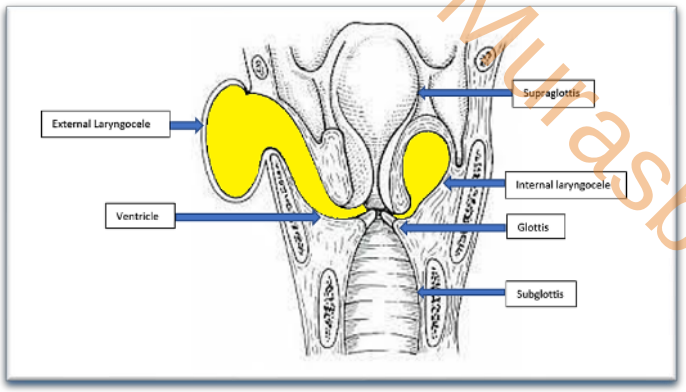
Other Lesions

1. **Lymphangioma (Cystic Hygroma)**:
   - Appears as a neck mass that transmits light.
   - Soft consistency, and aspiration yields a sticky fluid.
2. **Hemangioma**:
   - Vascular malformations.

Unit : 5

CONGENITAL LARYNGEAL DISORDERS

1.Laryngocele:

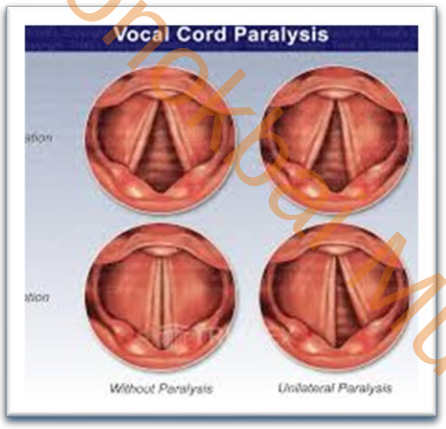


- Mass moves with coughing and becomes prominent during Valsalva.
- Commonly seen in those playing wind instruments due to increased intra-laryngeal pressure.

1.2 Laryngomalacia:

- Presents with inspiratory stridor and suprasternal retractions.
- Most cases resolve before 2 years of age.
- It is the **most common congenital laryngeal anomaly**.
- The leading laryngeal cause of respiratory distress and inspiratory stridor in infants.

Vocal Cord Paralysis:

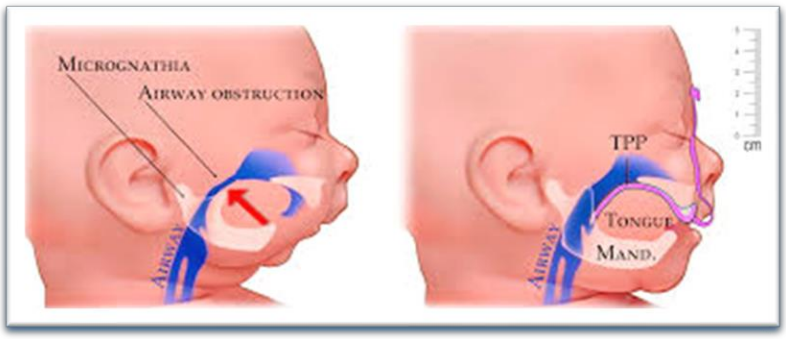


- The second most common cause of infant stridor after laryngomalacia.

Laryngeal Angiofibroma:

- A vascular lesion in children prone to bleeding, requiring caution during intubation.

PIERRE-ROBIN SYNDROME



- **Triad**: Cleft palate, micrognathia, and glossoptosis.
- Patients with cleft palate often exhibit velopharyngeal insufficiency and hypernasality.

PHARYNX DISEASES

1. **Vincent's Angina**:
    - Caused by spirochetes and fusiform bacteria.
    - Presents as foul-smelling ulcers covered by dirty gray membranes on the tonsils or tongue base.

2. **Tonsillectomy:**

Absolute Indications:

- Recurrent acute tonsillitis.
- Chronic tonsillitis.
- Peritonsillar abscess.
- Upper airway obstruction.
- Diphtheria carrier state.
- Suspicion of malignancy.

Relative Indications:

- Acute rheumatic fever.
- Cervical lymphadenitis.
- Glomerulonephritis.
- Frequent febrile convulsions.
- The tonsillar artery, a branch of the facial artery, is the primary blood supply. The exact artery most commonly involved in post-tonsillectomy bleeding is unclear.

3. **Adenoidectomy:**

Indications:

- Recurrent acute otitis media.
- Airway obstruction.
- Sinus ostium blockage by adenoids.
- Treatment-resistant otitis media with effusion.

Contraindications:

- Bleeding diathesis.
- Cleft palate.
- Polio epidemics.

Complications:

- Persistent nasal obstruction, mouth breathing, snoring, and hyponasal speech.
- Cor pulmonale (increased pulmonary artery pressure).

- Sleep apnea syndrome.
- Aspiration pneumonia.
- Chronic serous otitis media.
- Abnormal palate, teeth, and facial development ("adenoid face").

PERITONSILLAR, RETROPHARYNGEAL, AND PARAPHARYNGEAL ABSCESSES

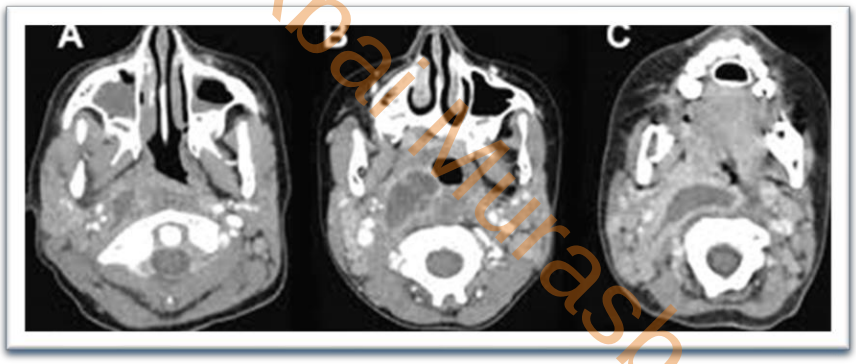
1. **Peritonsillar Abscess:**

- Common pathogens: Beta-hemolytic streptococci, pneumococci, and anaerobes.
- Presents with trismus and altered speech ("hot potato voice").
- Tonsillectomy is recommended due to the risk of recurrence.

2. **Retropharyngeal Abscess:**

- Can spread to the mediastinum and retroperitoneal region.
- Trismus is not observed.

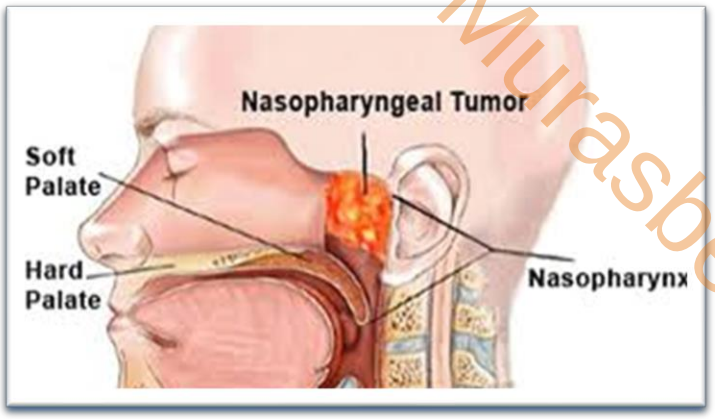
3. **Parapharyngeal Abscess:**
    - Often develops after tonsillitis or dental infections.
    - Trismus is present.



Unit : 6

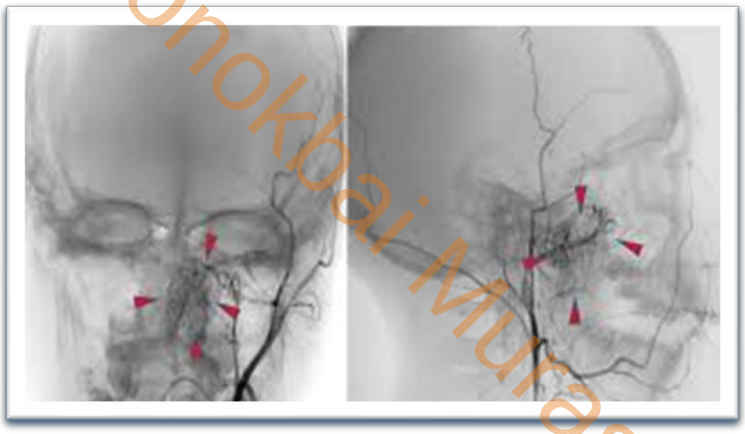
NASOPHARYNGEAL TUMORS

Nasopharyngeal Cancer



- **Most common site:** Rosenmüller fossa.
- **Etiology:** Includes salted fish (due to nitrosamine content) and Epstein-Barr Virus (EBV), with a definite relationship to EBV.
- **Histology:**
  - Non-keratinized carcinoma (differentiated or undifferentiated) (most common).
  - Keratinized squamous cell carcinoma.
  - Basaloid squamous cell carcinoma.
- **Clinical features:**
  - Painless neck mass (posterior cervical triangle): 76%.
  - Nasal symptoms (epistaxis, congestion, discharge, breathing difficulty): 73%.
  - Ear complaints (effusion otitis, tinnitus, etc.): 62%.
  - Cranial nerve symptoms (CN 3, 4, 5, 6): 20%.
- **Diagnosis:** Biopsy.
- **Treatment:**
  - Since the tumor is often undifferentiated, it responds well to radiotherapy (RT).
  - Surgery is challenging due to limited accessibility, so initial treatment is RT and chemotherapy (CT).
  - Surgery is not a treatment option.

Nasopharyngeal Angiofibroma

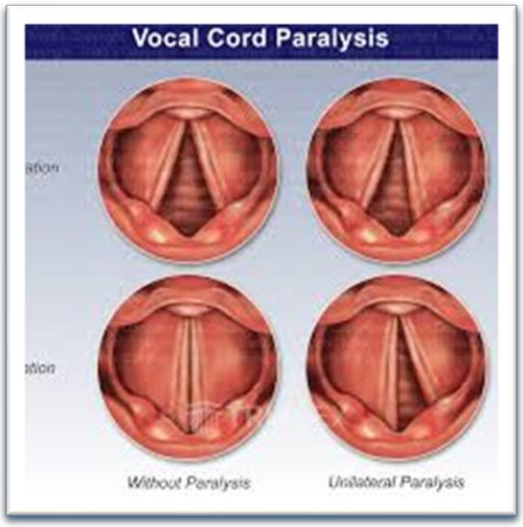


- A tumor originating from the periosteum of the occipital and sphenoid bones.
- Occurs in males aged 8–15 years.
- Presents with recurrent nosebleeds.
- Spontaneously regresses after puberty.

- **Treatment:** Primarily surgical.
- Does not occur in women or the elderly.
- Does not metastasize to the neck.

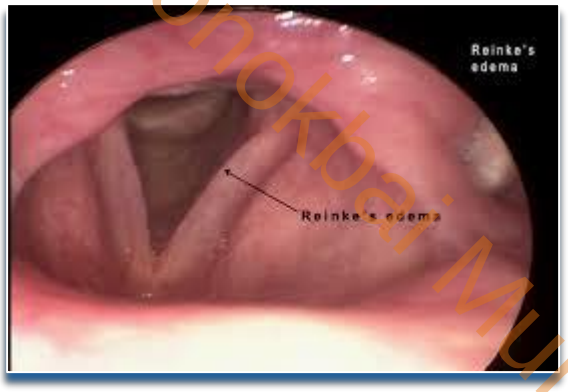
LARYNGEAL DISEASES AND TRACHEOSTOMY

Vocal Cord Paralysis



- **Most common cause:** Injury to the recurrent laryngeal nerve during thyroid surgery.
- **Unilateral paralysis:** Causes hoarseness; more common on the left side.
  - Caused by neck masses and lung pathologies affecting the left recurrent nerve.
  - Treated with "vocal cord medialization" to improve voice quality.
- **Bilateral paralysis:** Causes respiratory distress but not hoarseness.
  - Treated with "vocal cord lateralization" to relieve breathing difficulties.

Reinke's Edema

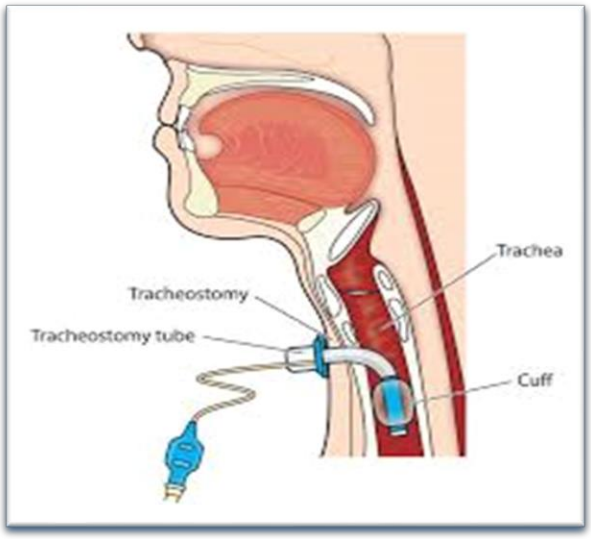


- A potential space between the vocal ligament and the vocal muscle.
- Formed by the superficial lamina propria.
- Typically seen in smokers, with edema causing voice deepening.
- **Treatment:** Ensure smoking cessation first; surgery if necessary.

Laryngeal Tumors

- **95% are epidermoid carcinoma.**
- **Subtypes:**
  - **Supraglottic cancer:** Presents with referred ear pain and bilateral neck lymph node involvement.
  - **Glottic cancer:** Causes hoarseness; has the best prognosis. Rarely metastasizes due to poor lymphatic supply of the vocal cords.
  - **Subglottic cancer:** Symptoms include stridor and dyspnea. It metastasizes to Delphian lymph nodes and has the worst prognosis.
- **Treatment:** Surgery, RT, and CT.

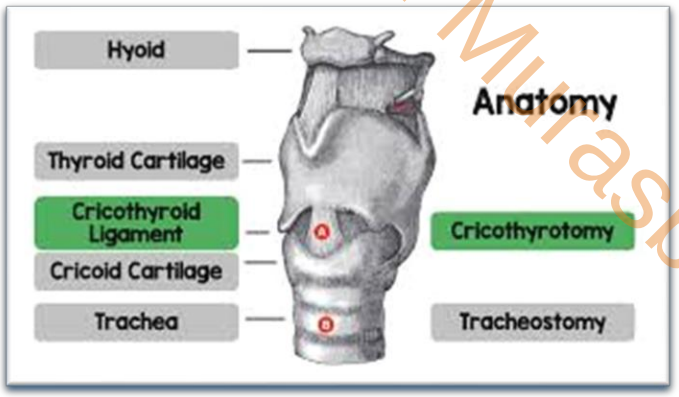
Tracheostomy



- Performed between the 2nd and 3rd cartilage.
- **Indications:**
  - Upper airway obstructions.
  - Conditions requiring reduced dead space in the upper airway.
  - Situations where deep suction is needed to clear secretions.
  - Preoperative or intraoperative management in upper airway surgeries.

- **Complications:**
  - **Intraoperative:** Bleeding, pneumothorax, mediastinal emphysema, esophageal injury, asphyxia.
  - **Postoperative:** Subcutaneous emphysema, atelectasis, pneumonia, tracheal granuloma, tracheal stenosis, bleeding, wound infection.

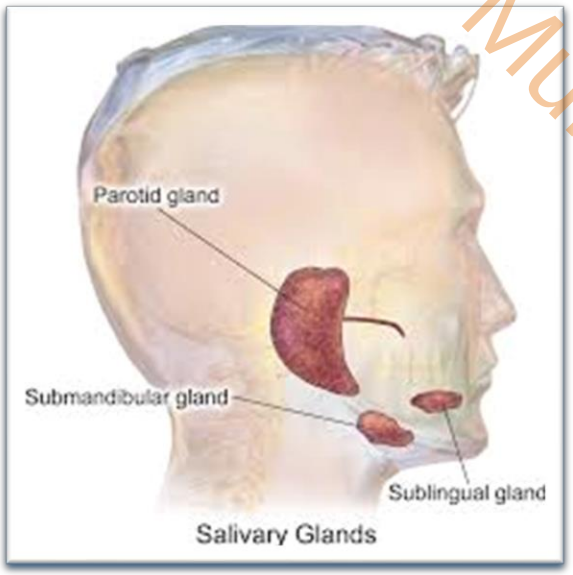
Cricothyrotomy



- Emergency airway access through the cricothyroid membrane.
- Preferred in patients who cannot be intubated.
- Rarely used due to potential laryngeal damage.

Unit : 7

SALIVARY GLAND DISEASES



Salivary Glands

Major Salivary Glands:

- **Parotid**
- **Submandibular gland**
- **Sublingual gland**

Minor Salivary Glands:

- Approximately 1000 minor salivary glands are dispersed throughout the oropharyngeal mucosa.
- Unlike the submandibular gland, the parotid gland contains lymphoid tissue.
- The submandibular gland produces the majority of the daily 1500 cc salivary secretion.
- The parotid gland secretes purely serous fluid.

Sialolithiasis (Salivary Stone Disease): Most common (80%) in the submandibular gland.

Salivary Gland Tumors

- **80%** occur in the parotid gland.
- **20%** of parotid tumors are malignant, whereas **65%** of tumors in the minor and sublingual glands are malignant.

Benign Tumors:

Pleomorphic Adenoma:

- Also known as **benign mixed tumor**.
- Most commonly develops in the **parotid gland**.
- Histologically, the tumor contains pseudopodia.
- **Most common salivary gland tumor**.

Warthin Tumor (Papillary Cystadenoma Lymphomatosum):

- Second most common tumor after pleomorphic adenoma.
- Four times more common in males; associated with smoking.
- Most common **bilateral salivary gland tumor**.

- Most frequently found in the **parotid gland**.

Oncocytoma: Occurs in older adults; rich in mitochondria.

Hemangioma: Benign vascular tumor.

Malignant Tumors:

Mucoepidermoid Carcinoma: Most common malignant salivary gland tumor, predominantly in the parotid gland.

Adenoid Cystic Carcinoma:

- Most common malignant tumor of the **submandibular** and **minor salivary glands**.
- Known as "cylindroma" due to its cylindrical cells.
- Perineural invasion occurs in 50% of cases.
- Frequently recurs after surgery due to perineural invasion.
- **Salivary gland tumor with neural progression**.

Malignant Mixed Tumor:

- Features both benign pleomorphic adenoma and malignant tumor tissue.
- Develops on the background of a pleomorphic adenoma that has been untreated for over 10 years.
- Also known as **carcinoma ex pleomorphic adenoma**.

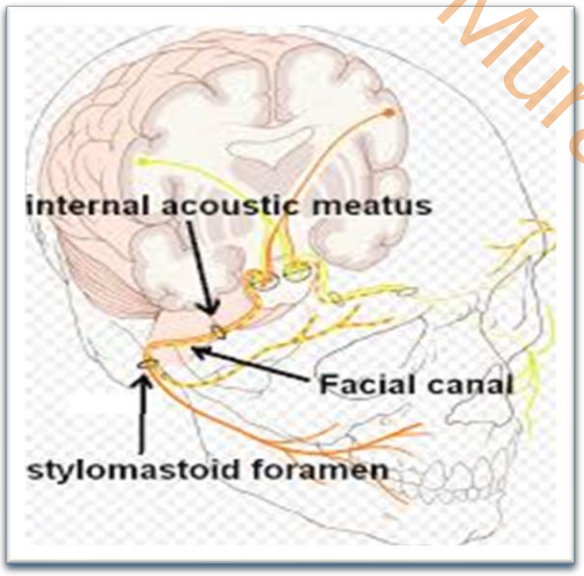
Frey Syndrome

- The skin overlying the parotid gland is innervated by sympathetic nerves.
- The parotid gland is innervated by parasympathetic fibers (auriculotemporal nerve).
- Following parotid surgery, parasympathetic fibers can extend to the skin and innervate it.
- This results in **sweating and flushing of the skin during eating**, a condition known as **Frey Syndrome**.

Unit : 8

FACIAL NERVE DISORDERS

Facial Nerve Overview

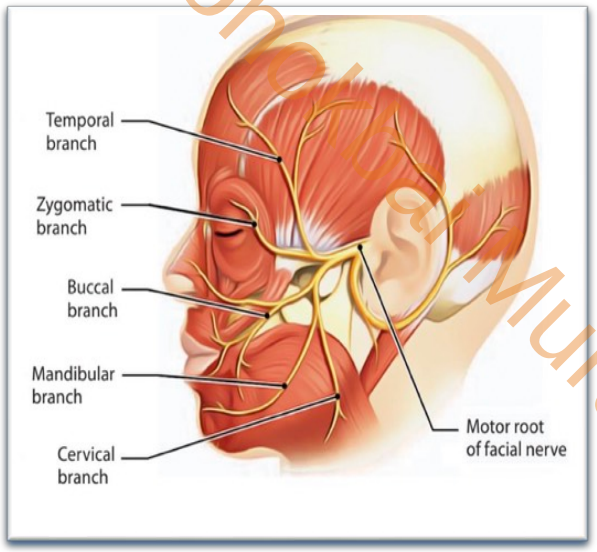


- The facial nerve consists of motor, sensory, and parasympathetic fibers.
- It travels within the temporal bone in a structure called the **fallopian canal** (canalis nervi facialis).
- The facial nerve exits the skull base through the **stylomastoid foramen**.
- Within this canal, it branches into four main divisions, from proximal to distal:

Branches of the Facial Nerve:

1. **Nervus Petrosus Major (Superficial Petrosal Nerve):**
   - Originates from the geniculate ganglion.
   - Innervates the lacrimal gland.
2. **Nervus Stapedius:**
   - Innervates the stapedius muscle.
   - Dysfunction of the stapedius muscle causes **hyperacusis** (increased sensitivity to sound).
3. **Ramus Communicans Plexus Tympani**
4. **Chorda Tympani Nerve:**
   - Carries parasympathetic fibers to the **sublingual** and **submandibular glands**.
   - Transmits taste sensation from the anterior two-thirds of the tongue.
   - Travels closely along the tympanic membrane.

After exiting the temporal bone, the facial nerve divides within the parotid gland into five branches that supply the mimetic (facial expression) muscles:



- **Temporal branch**
- **Zygomatic branch**
- **Buccal branch**
- **Mandibular branch**
- **Cervical branch**

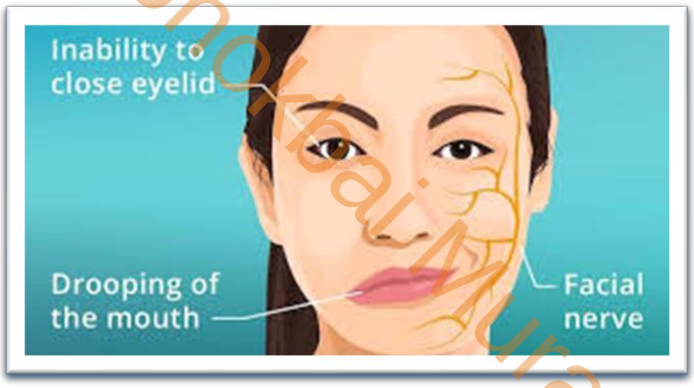
Types of Facial Paralysis:

- Divided into **peripheral** and **central** facial paralysis:
    - **Central facial paralysis:** Lesions at the supranuclear level. Only the **contralateral lower half of the face** is affected.
    - **Peripheral facial paralysis:** The entire half of the face on the **same side** is affected.

Facial Paralysis Assessment:

- The most commonly used grading system is the **House-Brackmann system**.
- Functional tests include:
    - **Lacrimal function** (Schirmer's test)
    - **Stapedius reflex**
    - **Taste sensation**
    - **Saliva quantity** (submandibular gland)
    - **Saliva pH**

Bell's Palsy (Idiopathic Peripheral Facial Paralysis):



- A facial paralysis of unknown exact cause.
- Most commonly linked to viral etiology, especially **HSV (Herpes Simplex Virus)**.
- In more than 50% of cases, involvement of other cranial nerves occurs (e.g., trigeminal nerve, often causing numbness and tingling before facial paralysis).
- **Best treatment:** Steroids, often combined with antiviral therapy.
- **VZV (Varicella-Zoster Virus)** is **not a cause**.

Ramsay-Hunt Syndrome (Herpes Zoster Oticus):

- Distinguished from Bell's palsy by its association with **VZV**.
- Features include painful vesicles in the external ear canal, auricle, face, and mouth.
- Similar cranial nerve findings as Bell's palsy may occur.
- Treatment includes:
  - **Systemic steroids**
  - **Antiviral drugs**
  - **Topical steroids** (to reduce postherpetic neuralgia). *Note: Topical steroids are not used in Bell's palsy.*

Bilateral Facial Paralysis:

- Most common cause: **Guillain-Barré Syndrome**.
- Other causes include:
  - **Lyme disease**
  - **Syphilis**
  - **Sarcoidosis**
  - **Bell's palsy**
  - **Brainstem encephalitis**
  - **Bacterial meningitis**
  - **Benign intracranial hypertension**

Progressive Paralysis:

- Gradual onset and progression exclude Bell's palsy.
- Often associated with:
  - Tumors of the facial nerve.
  - Other tumors affecting the facial nerve (e.g., acoustic neuroma).

Traumatic Facial Paralysis:

- Most commonly injured at the **labyrinthine segment** of the facial nerve or at the **geniculate ganglion** level.

Facial Paralysis in Pregnancy:

- Bell's palsy is three times more common in pregnant women than in non-pregnant women of the same age.

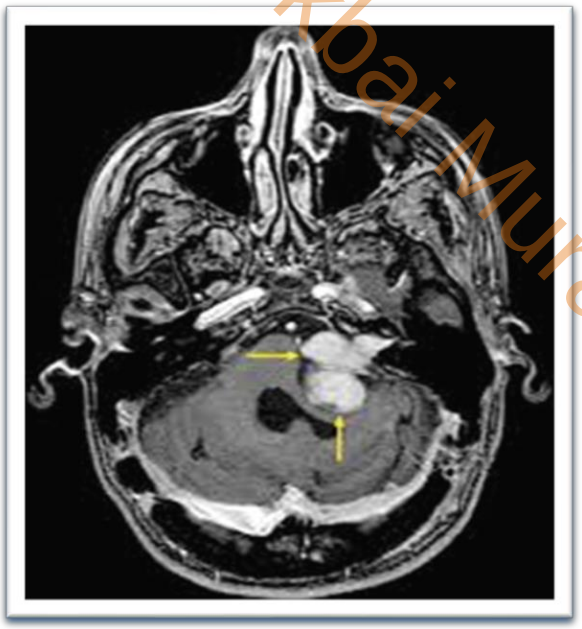
Melkersson-Rosenthal Syndrome:

- Key feature: Recurrent, asymmetric swelling of the face and upper lip.
- Cause is unknown.
- Characterized by a triad:

1. **Recurrent orofacial edema**
2. **Recurrent facial paralysis**

3. **Fissured tongue (lingua plicata)**

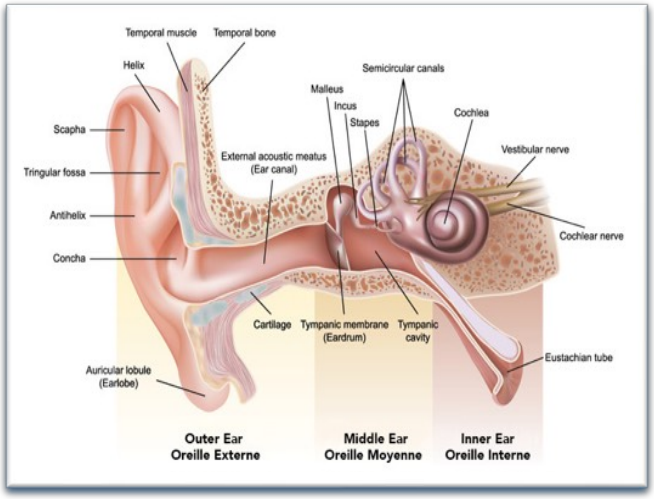
CEREBELLOPONTINE ANGLE TUMORS



- Approximately **90%** of cerebellopontine angle (CPA) tumors are **acoustic neuromas** (vestibular schwannomas) and **meningiomas**.
- Symptoms arise due to the involvement of the **neurovascular structures** located in the CPA.
- Cranial nerves **5, 6, 7, 8, 9, 10, and 11** are affected due to compression.
  - **CN 12** is not affected.

Vestibular Schwannoma (Acoustic Neuroma)

- Originates from the **Schwann cells** of the vestibular nerve.
- **Most common** CPA tumor.
- **95%** of cases are sporadic, while **5%** are associated with **neurofibromatosis type 2 (NF2)**.
  - NF2 patients often have **bilateral** vestibular schwannomas.



- Histological features include **Antoni A** and **Antoni B** areas.
- Well-encapsulated and exhibits a slow growth pattern over years.

Clinical Features

- **Unilateral hearing loss** and **tinnitus** are the initial symptoms.
- Due to slow growth, **vertigo** is less commonly observed as the brain compensates.

Diagnosis

- **Gadolinium-enhanced MRI** is the gold standard.

Treatment

- Options include:
    - **Observation** (regular follow-up)
    - **Surgery**
    - **Radiotherapy** (e.g., Gamma Knife).

MAXILLOFACIAL TRAUMAS

Nasal Fractures

- **Most frequently fractured bone** in the body.

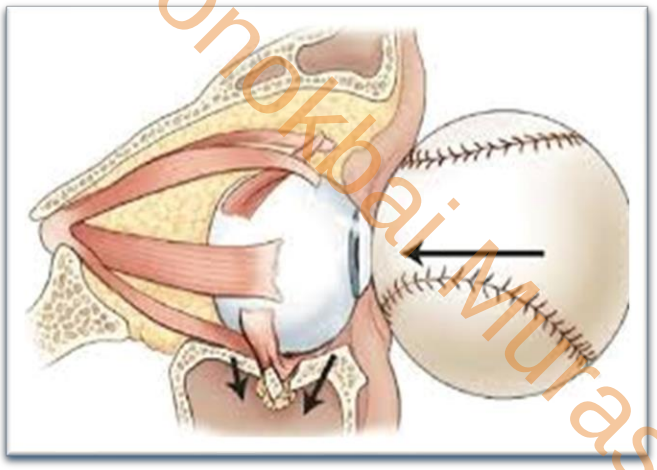
Orbital Fractures

- The **weakest part of the orbit** is its floor.

Nasofrontal – Ethmoid Fractures

- **Intercanthal distance** should be measured.
    - Damage to the medial canthal tendon is referred to as **telecanthus**.
- Symptoms:
    - **Epistaxis**
    - **Loss of sensation** in the infraorbital nerve distribution
    - **Periorbital ecchymosis**
    - **CSF rhinorrhea**
    - **Septal hematoma**
    - **Enophthalmos** and **diplopia**

Blow-Out Fractures



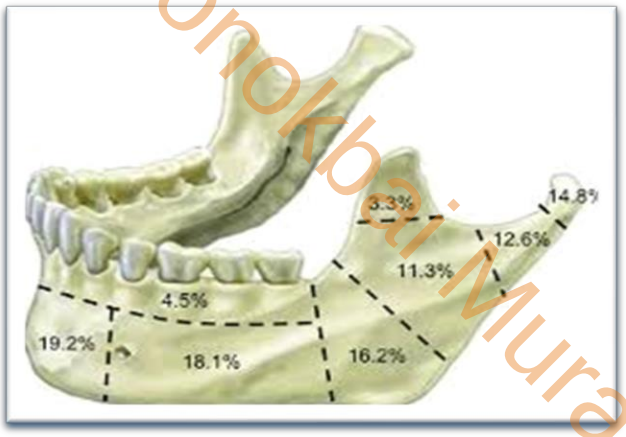
- Energy directed at the orbit causes soft tissues to compress the walls of the orbit, leading to fractures at the weakest points.
- **Enophthalmos** becomes evident within **2-3 days**.
- **Numbness** in the infraorbital nerve distribution may occur.
- **Inferior oblique** and **inferior rectus muscles** are most commonly affected, causing **diplopia**.

Zygoma Fractures

Tripod Fractures (Zygomaticomaxillary Fractures)

- Extensive facial swelling can mask clinical findings.
- Caused by **direct trauma** to the cheek.
- Often involves the **zygomatic arch** and the **anteromedial wall** of the maxillary sinus.
    - May also include fractures of the **frontal bone** and orbit.
- Symptoms:
    - **Pain** and **numbness**
    - **Trismus**
    - **Diplopia**, **hyphema**, **enophthalmos**
    - **Periorbital ecchymosis**
    - **Restricted eye movements**
- **CT scan** is the most effective diagnostic method.

Mandibular Fractures



- The **second most commonly injured bone** after the nasal bone.
- Most fractures occur in the **condylar (subcondylar) region**.

Clinical Findings

- Pain and numbness
- Limited mouth opening
- Excessive salivation
- **Deformity**
- **Mobility** of fractured segments
- **Malocclusion**: Misalignment of teeth when the mouth is closed
- Crepitation
- Possible injury to the **parotid gland** and **duct**
- **Ecchymosis** in the mouth floor
- Loss of sensation in the lower lip
- **Subcutaneous emphysema**
- **Gingival ecchymosis**

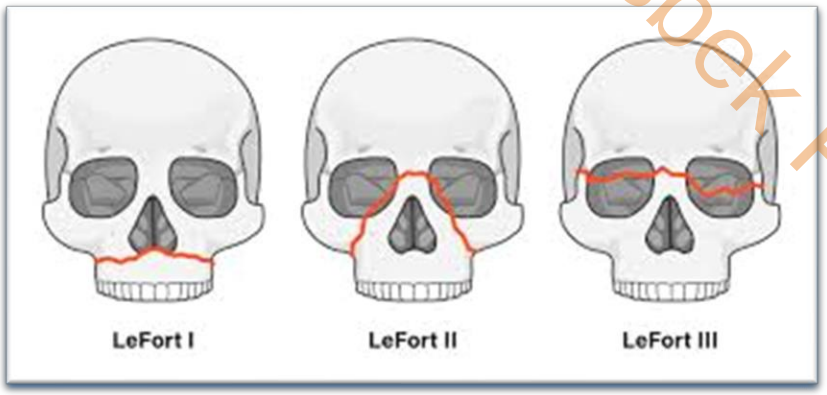
Treatment

- **Archbar method**: Prefabricated archbars are commonly used for intermaxillary fixation.

Maxillary Fractures

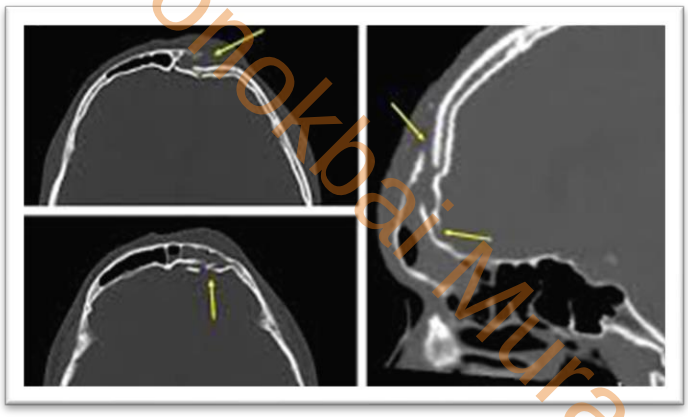
- **Most common complication**: Malocclusion
- Symptoms:
  - **Periorbital hematoma**
  - **Nasopharyngeal bleeding**
  - **Mobility of the maxillary dental arch** is a critical finding.
- **Diagnosis**: CT scan
- **Treatment**: Fixation using the **Archbar method**

Le Fort Fractures



- **Le Fort I Fracture (Guerin's Fracture):**
  - Horizontal fracture line across the lower part of the maxilla, just above the alveolar crest.
  - Involves the **pterygoid plates**.
- **Le Fort II Fracture (Pyramidal Fracture):**
  - **Most common** Le Fort fracture.
  - If the fracture involves the **pterygomaxillary fossa** and **pterygoid plates**, it results in a **"floating maxilla"**.
- **Le Fort III Fracture (Craniofacial Disjunction):**
  - **Most severe** maxillofacial fracture.

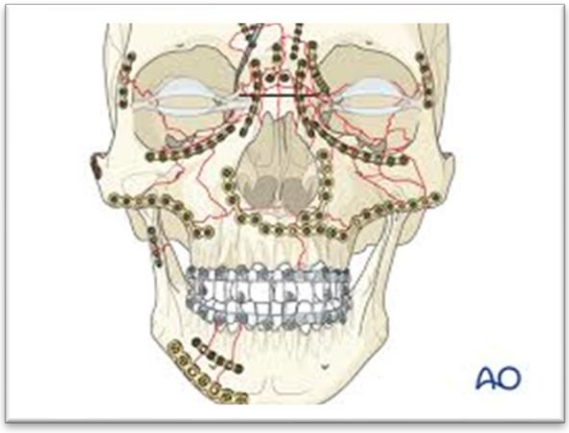
Frontal Bone Fractures



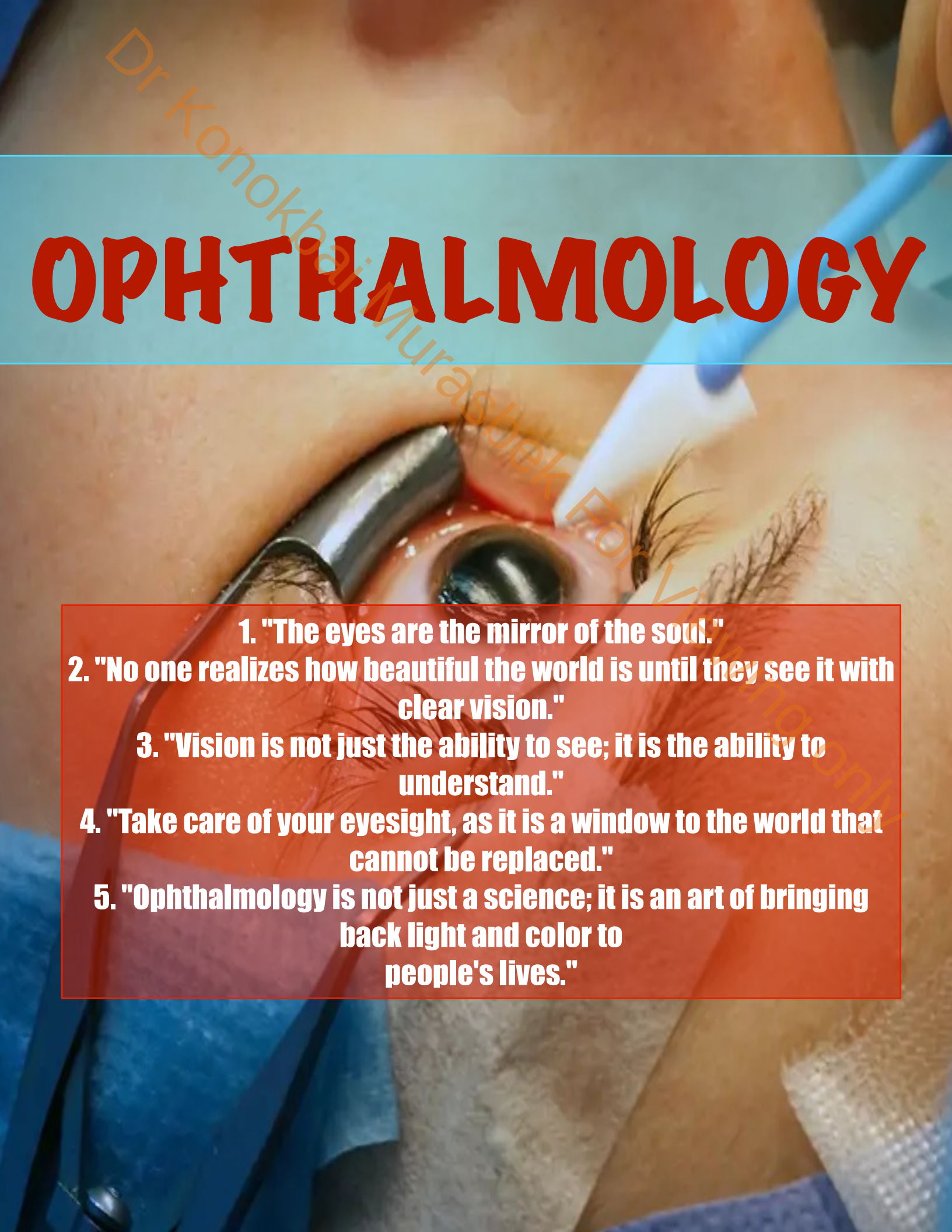
Clinical Findings

- **Pneumocephalus**
- **CSF rhinorrhea**
- **Periorbital hematoma**
- **Globe depression**
- **Ptosis**
- **Superior orbital syndrome**:
    - Involves fractures of the orbital roof, leading to paralysis of **CN 3, 4, 6**, and **ophthalmic branch of CN 5**.
    - Results in a fixed globe.

Panfacial Fractures



- **Treatment goal**: Restoration of facial height.
- Reconstruction begins with the **mandible**.

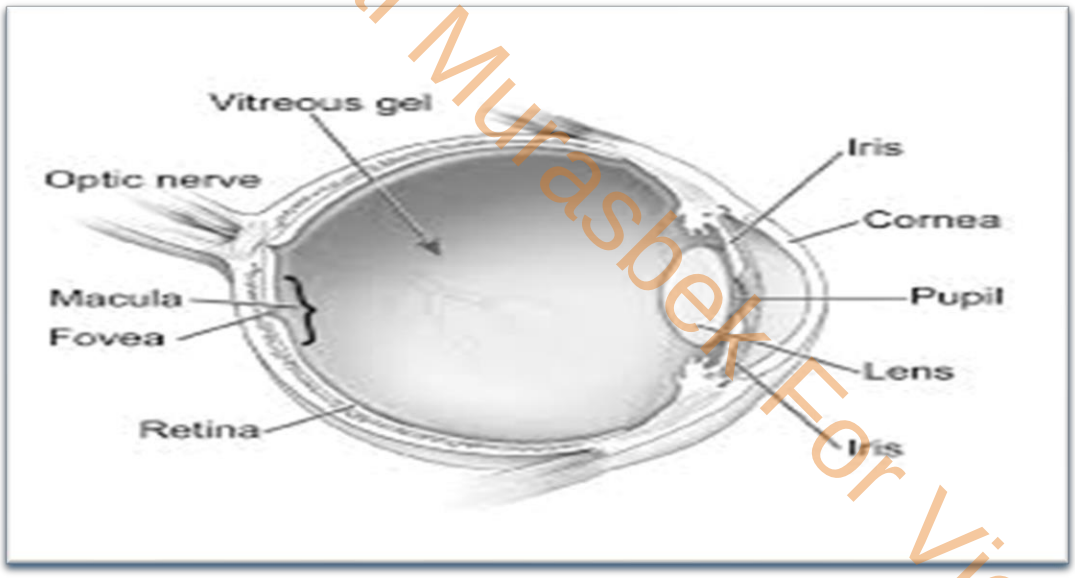


OPHTHALMOLOGY

1. "The eyes are the mirror of the soul."
2. "No one realizes how beautiful the world is until they see it with clear vision."
3. "Vision is not just the ability to see; it is the ability to understand."
4. "Take care of your eyesight, as it is a window to the world that cannot be replaced."
5. "Ophthalmology is not just a science; it is an art of bringing back light and color to people's lives."

Unit : 1

EYES ANATOMY

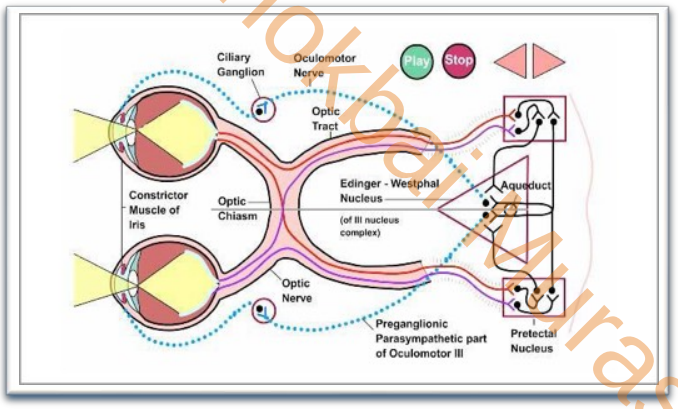


TOPOGRAPHY

- **Anterior pole:** The center of the corneal curvature.
- **Posterior pole:** The center of the posterior curvature of the globe, located slightly temporal to the optic nerve.
- **Geometric (anatomic) axis:** The line connecting the anterior and posterior poles.
- **Visual axis:** The line drawn from the center of the fovea to the fixation point.
- **Optic axis:** The line extending from the anterior pole through the center of the lens to the retina.
- **Equator:** The line encircling the eye at the midpoint between the two poles.
- **Meridian:** A circular line passing perpendicularly through the anterior and posterior poles.
- **Anterior-posterior diameter:** 22-26 mm.
- **Circumference:** 69-81 mm.
- **Average weight:** 7.5 grams; volume is 6.5 mL.

PHYSIOLOGY OF VISION AND REFRACTIVE ERRORS

Physiology of Vision



Binocular vision and depth perception:

- **Stereoscopic vision:** The perception of depth when viewing with both eyes.

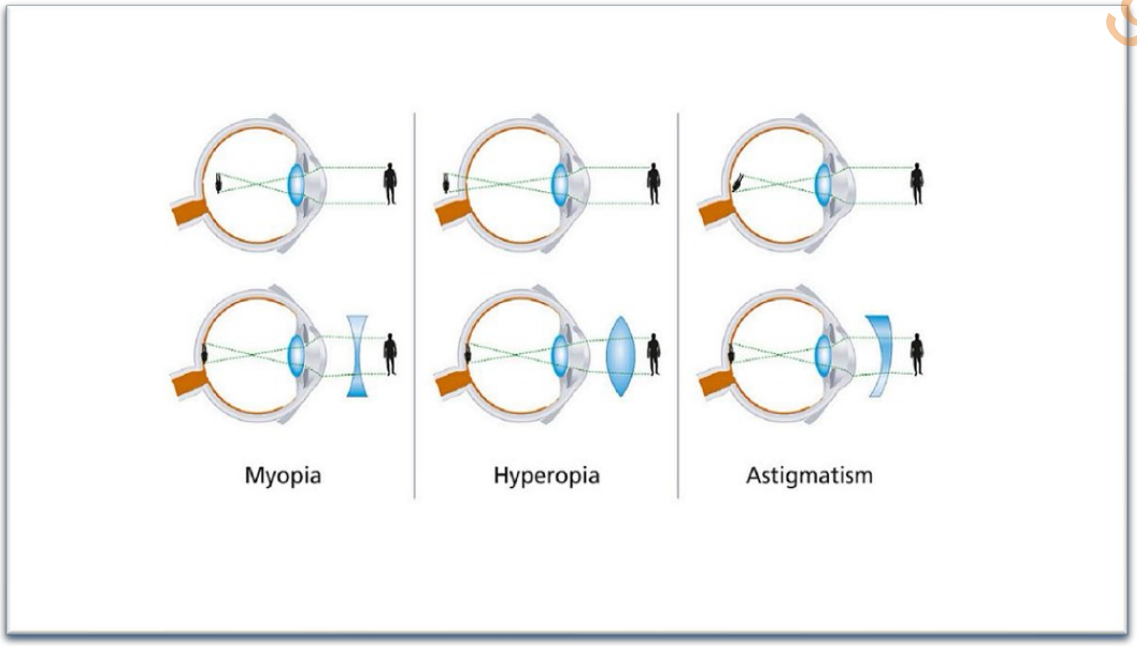
Color Vision:

- **Daylight:** Responsible for color vision and black-and-white perception through cone cells.
- **Twilight:** Responsible for achromatic vision through rod cells.

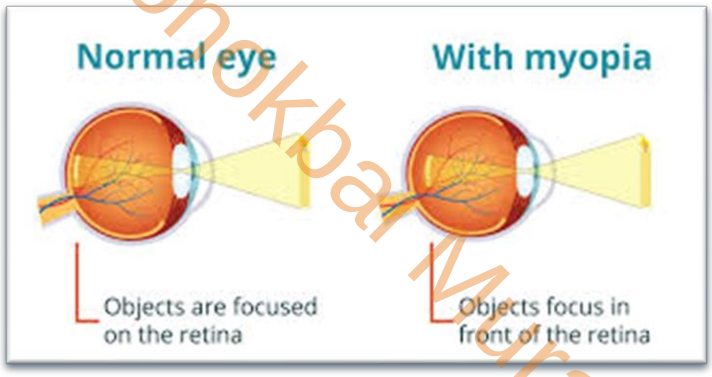
REFRACTIVE ERRORS

- **The refractive power of the cornea** is 43 diopters (the most refractive medium in the eye), and the lens is 20 diopters. The contribution of the aqueous humor and vitreous body to refraction is minimal.
- Eyes without refractive errors are called **emmetropic eyes**.
- Eyes with refractive errors are called **ametropic eyes**.

Types of Ametropia:



1. **Myopia:**



- In resting state, light rays parallel to the optical axis focus in front of the retina.
- **Treatment:** Concave (diverging) lenses, contact lenses, or refractive surgery.

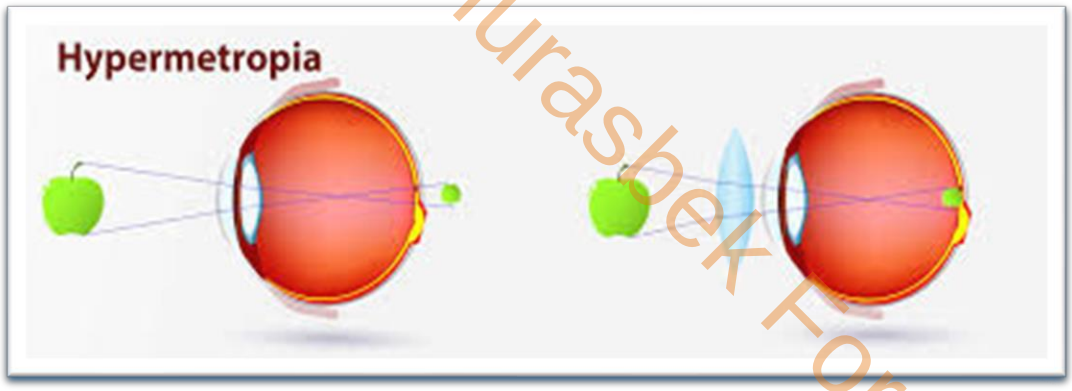
Types of Myopia:

- **Axial Myopia:** Due to increased axial length of the eye, light focuses in front of the retina.
  - Average axial length is 23-24 mm. In myopia, axial length increases.
  - The most common type of myopia.
- **Simple Myopia:**
  - Refractive error up to -6 diopters, often called physiological, school-age, or benign myopia.
  - Axial length is less than 26 mm. Stabilizes by ages 20-25.
  - **Mild type:** Up to -3 diopters; vision fully corrected with lenses.
  - **Moderate type:** -3 to -6 diopters; may show mild myopic crescent and peripheral retinal changes.
  - **Intermediate type:** -6 to -12 diopters; associated with thinned retina and myopic crescent.
- **Degenerative Myopia:**
  - Progresses after age 30, with axial length exceeding 26 mm.
  - Vision often cannot be fully corrected; associated with complications like glaucoma, cataracts, and retinal detachment.
- **Curvature Myopia:**
  - Seen in conditions like keratoconus, where corneal curvature increases.

- **Treatment:** Rigid contact lenses, refractive surgery, or keratoplasty.

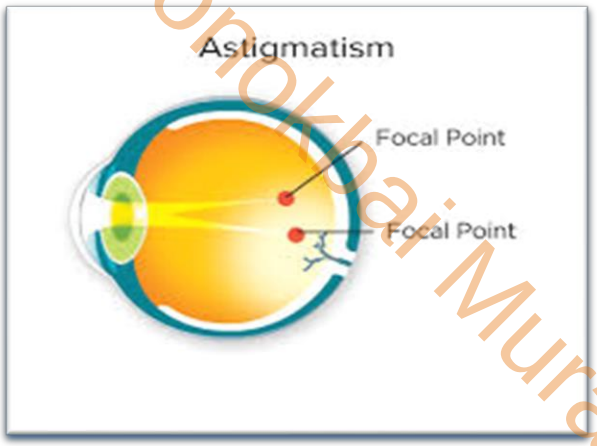
- **Index Myopia:**
  - Increased lens refractive power, as seen in nuclear cataracts.
- **Transient Myopia:**
  - Seen in conditions like diabetic hyperglycemia.

2. **Hypermetropia:**



- In resting state (no accommodation), light focuses behind the retina.
- **Types of Hypermetropia:**
  - **Axial Hypermetropia:** Due to shorter axial length. Treated with convex lenses.
  - **Curvature Hypermetropia:** Reduced corneal curvature leads to light focusing behind the retina.
  - **Index Hypermetropia:** Seen in cases like aphakia (post-cataract surgery without intraocular lens placement).
  - **Transient Hypermetropia:** Seen in hypoglycemic phases of diabetes mellitus.

3. **Astigmatism:**



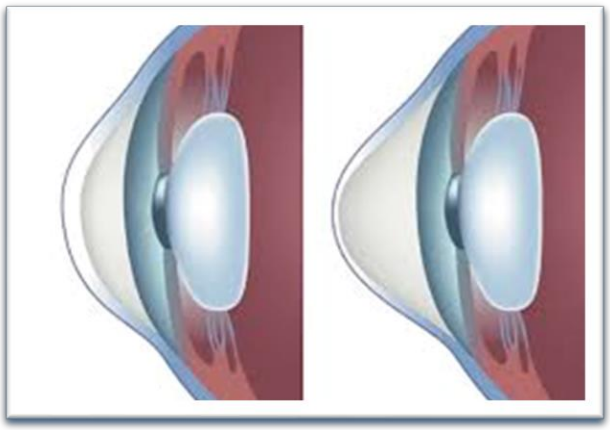
- Due to irregularities in the curvature of the eye's optical system, light refracts differently across meridians, preventing the formation of a single focus.

Types of Astigmatism:

- **Regular Astigmatism:** Uniform dioptric differences between meridians. Corrected with cylindrical lenses or soft contact lenses.
- **Irregular Astigmatism:** Variations across the optical zone. Corrected with rigid contact lenses. Examples include keratoconus.

Treatment:

- **Regular Astigmatism:** Cylindrical lenses or soft contact lenses.
- **Irregular Astigmatism:** Rigid contact lenses, refractive surgery, or keratoplasty. Soft lenses may be tried in mild keratoconus cases.



KERATOCONUS

Keratoconus is defined as the thinning of the center and periphery of the cornea, leading to a conical protrusion. Clinical signs include **Vogt's striae** (ruptures in the Descemet membrane) and **Fleischer's ring** (iron deposits in the epithelium). Patients commonly present with irregular astigmatism and myopia. It is associated with conditions such as **atopic dermatitis** and **vernal conjunctivitis**.

- **Early Stage**: Vision defects can be corrected with glasses or contact lenses (rigid gas-permeable lenses are preferred).
- **Progressive Disease**: If these measures are insufficient, refractive surgery may be considered.

- **Advanced Disease**: If progression cannot be controlled, keratoplasty (corneal transplantation) may be necessary.

ACCOMMODATION (FOCUSING MECHANISM)

In an emmetropic (normal) eye, images of objects at 6 meters or further are focused on the retina due to the refractive properties of the eye.
As objects come closer than 6 meters, their images tend to fall behind the retina. The accommodation mechanism increases the lens's refractive power to refocus the image on the retina.

Mechanism of Accommodation:

1. **Ciliary muscle contraction** causes relaxation of the **Zonules of Zinn**, allowing the lens to become more spherical and increase its refractive power.
2. **Convergence**: Both eyes move inward to maintain binocular vision as objects approach.
3. **Miosis**: The pupil constricts.

DISORDERS OF ACCOMMODATION

Presbyopia

- Age-related loss of lens elasticity, reducing accommodation.
- Typically occurs after age 40 and is physiological.
- **Hyperopes** experience presbyopia earlier, while **myopes** develop it later.
- Treated with convex (+) lenses.

Accommodation Paralysis

- Occurs in the ciliary and iris sphincter muscles due to **oculomotor nerve (CN III)** paralysis.
- Symptoms: **Mydriasis** (dilated pupil), inability to focus on near objects.
- Causes: Use of cycloplegic eye drops (e.g., atropine), or **atropine poisoning**.

Accommodation Spasm

- Continuous contraction of the ciliary muscle.
- Often seen in schoolchildren due to psychological factors.

- Symptoms: Induced myopia and miosis.
- Causes: Parasympathomimetic drugs (e.g., pilocarpine), iridocyclitis, or hysteria.
- Treatment: Atropine eye drops for hysteria; addressing the underlying cause for other cases.

AMBLYOPIA (LAZY EYE)

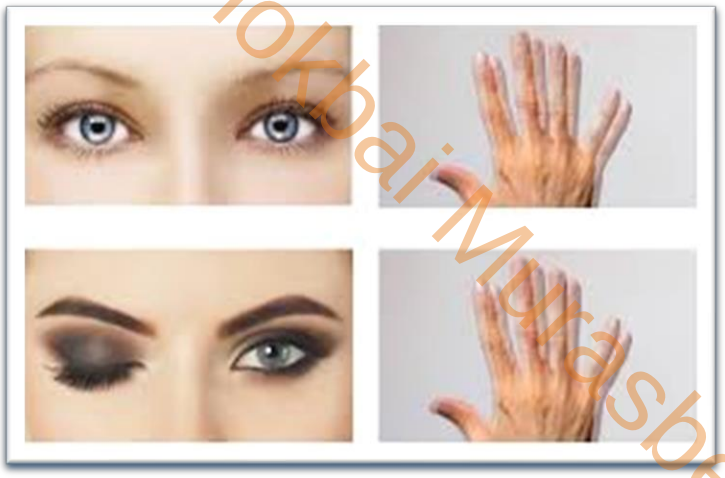
Amblyopia refers to reduced visual acuity in one or both eyes during the first 8–10 years of life when visual maturation occurs.

- The initial 3–6 months are critical for the development of proper projections between the eyes and the visual cortex.
- The earlier the causative factor, the more severe the visual loss.
- Diagnostic Method: **Visual Acuity Test** in newborns.

Classification:

1. **Strabismic Amblyopia**:
   - Most common cause.
   - Commonly seen in preschool children (e.g., **infantile esotropia**).
2. **Anisometropic Amblyopia**:
   - Second most common cause.
   - Occurs when there is a refractive error in one eye or a difference of more than 1.50–2.00 diopters between the eyes.
   - The eye with higher refractive error develops amblyopia.
3. **Bilateral Ametropic Amblyopia**:
   - Occurs in cases of significant refractive errors in both eyes (e.g., hyperopia > +5.00D or myopia > -10.00D).
4. **Deprivation Amblyopia**:
   - Caused by physical obstructions preventing a clear image from reaching the retina (e.g., congenital ptosis, corneal opacities, cataracts, or eyelid tumors).

DIPLOPIA (DOUBLE VISION)



Diplopia arises when the alignment of the eyes is disrupted, resulting in the brain receiving non-overlapping images.

Types:

Binocular Diplopia:

- Disappears when the unaffected eye is closed.
- Causes:

1. Myopathies (e.g., Kearns-Sayre syndrome, myotonic dystrophy).
2. Disorders affecting CN III, IV, or VI (e.g., diabetes, atherosclerosis).
3. Restrictive strabismus (e.g., thyroid ophthalmopathy, trauma).
4. Intracranial masses or aneurysms compressing cranial nerves.
5. Convergence paralysis.
6. Brainstem lesions (e.g., tumors, stroke, multiple sclerosis).

Monocular Diplopia:

- Persists even when the unaffected eye is closed.
- Causes:

1. Lens dislocation.
2. Astigmatism.
3. Cataracts.
4. Retinal issues.
5. Psychogenic causes.

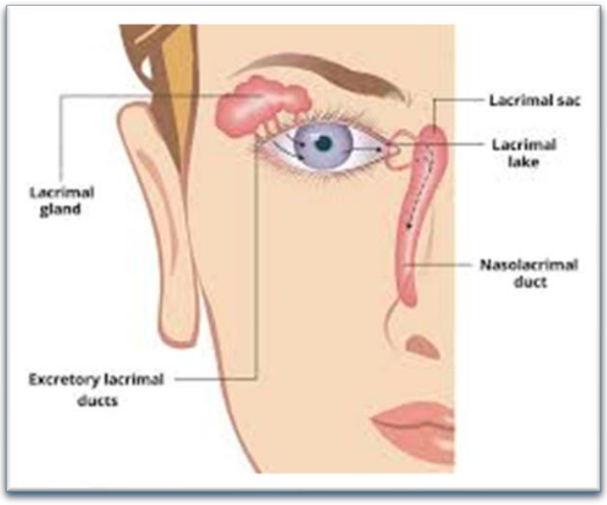
Unit : 2

EYELID AND ORBITAL DISEASES

EYELID ANATOMY

- The outer surface of the eyelids is covered by skin, and the inner surface is lined with conjunctiva.
- Between the skin and conjunctiva are fibrous tarsal plates, orbital septum, upper lid elevators (levator muscle, levator aponeurosis, and Müller's muscle), and lower lid retractors (inferior rectus fascia and inferior tarsal muscle).
- The levator muscle is innervated by the 3rd cranial nerve, while Müller's and inferior tarsal muscles are innervated by sympathetic nerves.
- The palpebral fissure is closed by the orbicularis oculi muscle, innervated by the 7th cranial nerve.

Lacrimal System



Secretory Component:

- **Lacrimal Gland (Glandula Lacrimalis):** Located in a shallow depression in the orbital portion of the frontal bone.
- **Accessory Glands:** Accessory lacrimal glands (Wolfring and Krause glands) are located near the proximal edges of the eyelid, contributing to approximately 10% of the total lacrimal system.

Collecting Component:

Includes puncta, canaliculi, lacrimal sac, and nasolacrimal duct.

CONGENITAL DEVELOPMENTAL AND STRUCTURAL DISORDERS OF THE EYELID

Ablepharon

Absence of the eyelid.

Coloboma

•A defect, often in the medial eyelid. It may be associated with other embryological defects such as colobomas of the iris, lens, choroid, retina, and optic nerve.

Epicanthus

A semilunar skin fold on the medial side of the eye, common in "Mongolian eyes."

Can cause pseudostrabismus (false appearance of inward squinting) in newborns and young children.

Symblepharon

- Adhesions between the palpebral conjunctiva and bulbar conjunctiva.
- Caused by conditions like chemical burns (alkali > acid), Stevens-Johnson syndrome, ocular cicatricial pemphigoid, trachoma, herpes zoster, atopic keratoconjunctivitis, scleroderma, or graft-versus-host disease.

INFLAMMATORY EYELID DISEASES

Hordeolum (Stye)

- **Cause:** Most commonly *Staphylococcus aureus*.
- **Types:**
  - Internal stye: Inflammation of Meibomian glands.
  - External stye: Inflammation of Zeis or Moll glands.
- **Treatment:** Warm compresses, eyelid hygiene, antibiotic drops, and ointments.

Chalazion

- Lipogranuloma of Meibomian glands.
- Presents as swelling in the eyelids.
- Managed surgically through excision.

Blepharitis

- Chronic, bilateral inflammation of the eyelids.
- Types: Seborrheic, staphylococcal, or mixed.
- Symptoms: May cause *madarosis* (loss of eyelashes).
- Treatment targets the underlying cause.

PRESEPTAL CELLULITIS

- Infection of the subcutaneous tissues anterior to the orbital septum.
- **Causes:**
  - *Staphylococcus aureus* or beta-hemolytic streptococci following skin trauma.
  - Spread from local infections like hordeolum, dacryocystitis, conjunctivitis, or sinusitis.
  - Hematogenous spread from systemic infections like upper respiratory or middle ear infections.
- **Symptoms:**
  - Unilateral, tender, red, and swollen periorbital region.
  - No proptosis or impairment of visual acuity and ocular motility.
- **Treatment:** Oral antibiotics such as methicillin or flucloxacillin.

ORBITAL CELLULITIS

- Infection of the soft tissues posterior to the orbital septum.

- **Causes:**

  - Sinus-related infections, often secondary to ethmoid sinusitis (most common).
  - Trauma penetrating the orbital septum.
  - Post-surgical infections.

- **Symptoms:**
  - Swollen, red, and tender eyelids.
  - Proptosis.
  - Restricted and painful eye movements.
  - Possible optic nerve dysfunction and vision loss.
- **Treatment:** Hospitalization and intravenous antibiotics.

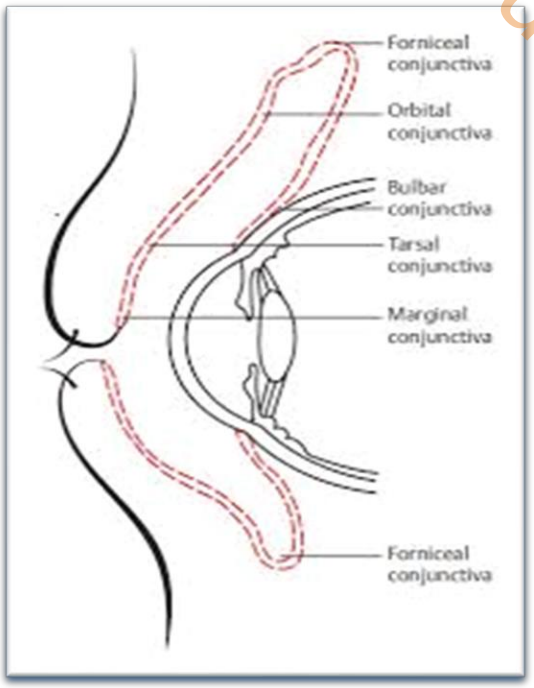
ACUTE BACTERIAL ENDOPHTHALMITIS

- A devastating complication with an incidence of 1 in 1000.
- Despite early treatment, 60% of affected eyes become blind.
- **Common pathogens:** *Staphylococcus epidermidis, Staphylococcus aureus, Pseudomonas, and Proteus species.*

Unit : 3

CONJUNCTIVAL DISEASES

Conjunctiva is divided into palpebral and bulbar conjunctiva.



CONJUNCTIVAL HYPEREMIA (REDNESS):

1. **SuperficialHyperemia**

Superficial hyperemia is the dilation of the superficial blood vessels of the conjunctiva originating from the eyelids.

This type of hyperemia, mostly seen in conjunctival diseases, has the following features:

- Light red color
- Dilated vessels move with the conjunctiva
- Blanching occurs with pressure

2. **DeepHyperemia**

Deep hyperemia is the dilation of the anterior ciliary vessels originating deep in the conjunctiva.

This type of hyperemia is dark red, associated with cornea, uvea, sclera diseases, and acute glaucoma crises.

Features include:

- Vessels do not move with the conjunctiva
- Hyperemia does not blanch with pressure

CONJUNCTIVITIS

- **Acute Conjunctivitis**

  Onset is sudden, initially unilateral, with inflammation in the second eye occurring within a week. The duration is less than 4 weeks.

- **Chronic Conjunctivitis**

  Duration exceeds 3-4 weeks.

Bacterial Conjunctivitis

1. **Acute Bacterial Conjunctivitis**
   - Symptoms: Vessel dilation, mucopurulent discharge, eyelids sticking together.
   - Common pathogens: *Staphylococcus aureus, Streptococcus pneumoniae, Haemophilus influenzae, Moraxella catarrhalis*
2. **Hyperacute Bacterial Conjunctivitis (Acute Purulent Conjunctivitis)**
   - **Cause:** *Neisseria gonorrhoeae*
   - Diagnosis: Gram-negative diplococci in the sample.
   - Leading cause of severe *Ophthalmia Neonatorum* (newborn conjunctivitis).
   - Complications: If untreated, severe corneal complications such as perforation may occur.
   - Prophylaxis:
     - Povidone-iodine 2.5% drops
     - Erythromycin 0.5% or tetracycline 1% drops/ointment
   - In some endemic areas, silver nitrate ($AgNO_3$) 1% solution is still used.
   - Maternal infection requires benzylpenicillin or ceftriaxone.
3. **Chronic Bacterial Conjunctivitis**
   - Most common cause: *Staphylococcus aureus*
   - Symptoms: Crusting at the eyelid margins, often associated with blepharitis.

Neonatal Conjunctivitis (Ophthalmia Neonatorum)

- **Chemical Conjunctivitis ($AgNO_3$):** Appears within hours.
- **Gonococcal Conjunctivitis:** 2nd-4th days.

- **Other Bacteria (e.g., Staphylococcus, Streptococcus, Haemophilus, Pseudomonas):** 4th-5th days.
- **Viral (HSV Type I-II):** 5th-7th days.
- **Chlamydial:** 5th-14th days.

Chlamydial Conjunctivitis

1. **Neonatal Inclusion Conjunctivitis**
   - Most common type of neonatal conjunctivitis.
   - Cause: *C. trachomatis* D-K strains.
   - May be associated with systemic infections like otitis, rhinitis, and pneumonia.
   - Diagnosis: Intracytoplasmic inclusion bodies.
   - Treatment: Topical erythromycin or tetracycline, and oral erythromycin for 2 weeks.
2. **Adult Inclusion Conjunctivitis**
   - Cause: *C. trachomatis* D-K strains.
   - Symptoms: Hyperemic eye, preauricular lymphadenopathy, and follicular conjunctivitis.
   - Transmission: Genital contact, swimming pools, or cosmetics.
   - Treatment:
     - Topical erythromycin or tetracycline
     - Systemic azithromycin (1 g single dose, repeated after a week if needed)
     - Doxycycline 100 mg twice daily for 10 days

3. **Trachoma**
    - Cause: *Chlamydia trachomatis* A, B, Ba, C strains.
    - Leading cause of preventable irreversible anterior segment blindness worldwide.

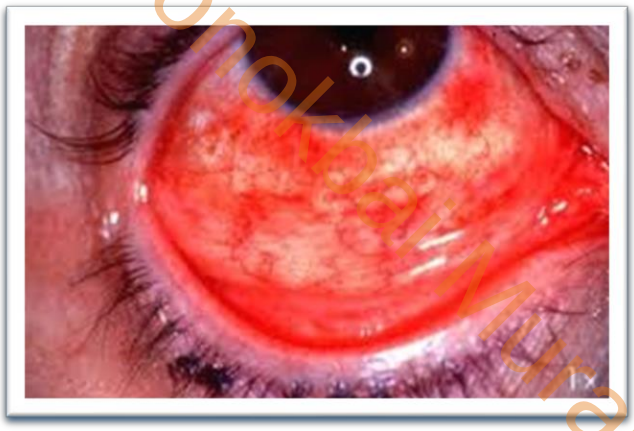
    - Features:
        - Chronic mixed follicular and papillary keratoconjunctivitis
        - Tearing, photophobia, edema, pain
        - Bulbar conjunctival chemosis, papillary hypertrophy, limbal and tarsal follicles, pannus formation, punctate keratitis, epithelial keratitis, and infiltrates on the cornea
        - Herbert's pits (limbal bacterial infiltration and destruction)
        - Arlt's lines (chronic linear scarring on eyelid conjunctiva)
    - Treatment:
        - Topical tetracycline
        - Systemic antibiotics: Azithromycin (1 g single dose) or erythromycin (500 mg for 14 days) or doxycycline (100 mg twice daily for 10 days).

Viral and Allergic Conjunctivitis

A) Pharyngoconjunctival Fever (PCF)

- **The most common ocular adenovirus infection.**
- **Causative agents:** Adenovirus type B (3, 4, 7, and 21).
- **Classic triad:** Pharyngitis, fever, and conjunctivitis.
- Transmitted via droplets.
- Causes follicular conjunctivitis.
- Punctate epithelial keratitis develops in approximately 30% of cases but is rarely severe.
- Preauricular lymph node involvement may occur.
- Resolves spontaneously in about two weeks.

B) Epidemic Keratoconjunctivitis (EKC)



- **Causative agents:** Adenovirus types 8, 19, and 37.
- **The most severe ocular adenovirus infection.**
- Causes **bilateral acute follicular keratoconjunctivitis,** often more severe in the first affected eye.
- Preauricular lymph node involvement may occur.
- Highly contagious, often causing epidemics.
- Unlike pharyngoconjunctival fever, systemic symptoms are absent, but the risk of punctate epithelial keratitis is higher (about 80%).
- Spontaneous resolution within two weeks, though it may take longer in some cases.

Herpes Simplex Conjunctivitis

- Conjunctivitis may occur in cases of primary herpes infection.
- Herpetic vesicles are visible on the eyelids and periorbital skin.
- Follicular conjunctivitis is seen in the affected eye.
- Frequently associated with keratitis featuring **corneal dendrites.**
- Preauricular lymph node involvement is typical.

- **Treatment:**
  - Topical **acyclovir 3%** is used.
  - Corticosteroids are contraindicated in keratitis cases as they exacerbate the infection and may lead to corneal ulceration.

Acute Hemorrhagic Conjunctivitis (Apollo XI Conjunctivitis)

- **Causative agents:** Enterovirus type 70, Coxsackie A24, and Adenovirus.
- Features **diffuse subconjunctival hemorrhages.**

Measles Conjunctivitis

- Conjunctival Koplik spots, similar to those on the oral mucosa, appear alongside skin eruptions.

Allergic Conjunctivitis

Seasonal Allergic Conjunctivitis (Hay Fever Conjunctivitis)

- Occurs in spring and summer due to airborne allergens (e.g., tree and grass pollens).

- Mediated by **type I hypersensitivity reactions.**
- Often associated with allergic rhinitis and asthma.
- Cytology shows **eosinophils** in the conjunctiva.

- **Treatment:** Cold compresses, steroids, and cromolyn sodium.

Vernal Keratoconjunctivitis (VKC)

- Common in warm climates, peaking in spring and summer.
- Affects males aged 5–20 years.
- The second worst prognosis among allergic conjunctivitis types.
-

- **Types:**
  - Palpebral: Upper lid displays a "cobblestone" appearance.
  - Limbal: Horner-Trantas dots (degenerate cells) appear on the conjunctival surface.
- Corneal involvement: **Punctate epithelial erosions, shield ulcers, and subepithelial scarring.**

Atopic Keratoconjunctivitis (AKC)

- Seen in individuals aged 30–50 years.
- Associated with atopic dermatitis and asthma.
- Chronic course with continuous symptoms.
- Corneal and conjunctival scarring are common.
- **Shield-like ulcers** may develop.
- **Worst prognosis** among allergic conjunctivitis types.

Giant Papillary Conjunctivitis (GPC)

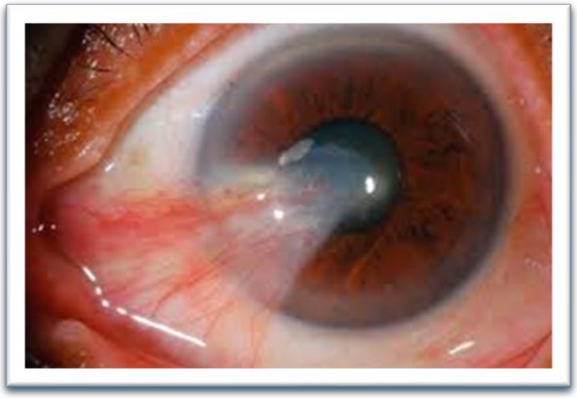
- Typically occurs in patients using foreign objects like **contact lenses** or **prosthetics**.
- Mediated by **type I hypersensitivity reactions.**
- Resolves completely upon lens removal and steroid treatment.
- **Best prognosis** among allergic conjunctivitis types.

Degenerative Diseases of the Conjunctiva

Pinguecula

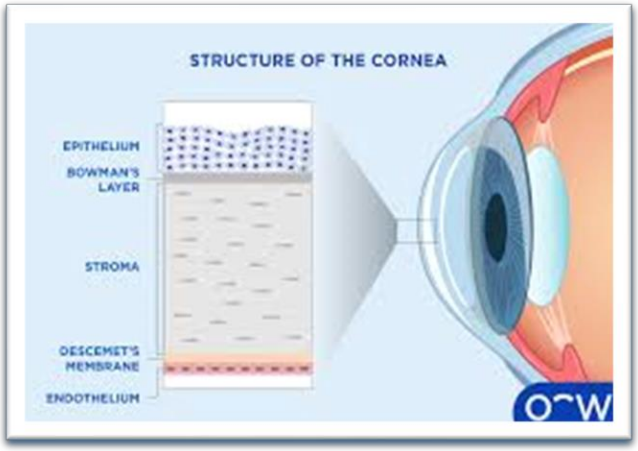
- The most common conjunctival degeneration.
- A harmless gray-yellow thickening (hyaline degeneration) of the epithelium, typically observed in the nasal portion of the bulbar conjunctiva within the palpebral fissure.
- It has a higher prevalence in Gaucher disease.

Pterygium



- A fold of conjunctiva that extends from the bulbar conjunctiva, usually from the nasal side, onto the cornea.
- The yellow-green crescent-shaped iron deposition along the leading edge of the pterygium at the Bowman membrane level is called **Stocker's line**.

CORNEAL DISEASES



•Microscopically, the cornea consists of the following layers from outside to inside:

1. Epithelium
2. Bowman's layer
3. Stroma
4. Descemet's membrane
5. Endothelium

- The transparency of the cornea is maintained by the perfect arrangement and avascular nature of the epithelial layer, the lamellar structure of collagen fibers in the stroma, and the

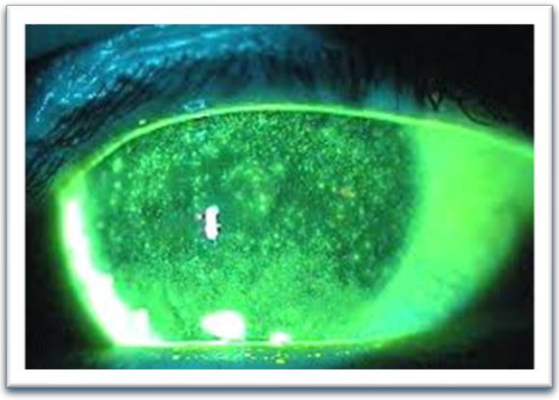
endothelial layer's regulation of fluid dynamics, keeping the intrastromal water level constant (approximately 76%). The endothelium plays the most critical role in maintaining corneal transparency.

- While the epithelium can regenerate after damage, Bowman's layer and the endothelium cannot regenerate.
- The limbus is 1-2 mm thick and contains the trabecular meshwork.

CORNEAL INFECTIONS AND INFLAMMATIONS (KERATITIS)

- Two essential features of the corneal epithelium are its barrier function against microorganisms and its ability to regenerate. However, bacteria such as *Corynebacterium diphtheriae*, *Neisseria*, *Listeria*, and *Haemophilus* species can penetrate an intact epithelium.
- Injuries, infections, or allergies involving the deeper corneal layers can stimulate inflammatory reactions, leading to structural changes (ulcers, necrosis, neovascularization) and permanent vision impairment due to corneal opacities.
- There are three types of corneal opacities: nebula, macula, and leucoma.
    - The first two have minimal effect on vision.
    - Leucoma involves all corneal layers, and if it covers the pupillary area, vision is severely reduced. In such cases, corneal transplantation (keratoplasty) may be necessary.

Superficial Punctate Keratitis



- Characterized by small, pinpoint staining defects in the corneal epithelium.
- The condition is nonspecific and has various causes, including:
    - Dry eye syndrome
    - Blepharitis
    - Hypersensitivity reactions to *Staphylococcus*
    - Infections such as adenovirus, chlamydia, and herpes simplex.
- Treatment is etiology-based, with artificial tears, bandage lenses, and therapeutic contact lenses providing symptomatic relief.

BACTERIAL KERATITIS

- Most bacteria, except for *Neisseria*, *Corynebacterium*, *Listeria*, and *Haemophilus*, cause keratitis only when the epithelial integrity is compromised.
- **Risk factors**:
  - Contact lens use (especially *Pseudomonas*)
  - Trauma (e.g., refractive surgery)
  - Ocular surface damage (e.g., dry eye)
  - Other conditions like immunosuppression, diabetes, or vitamin A deficiency.
- Common causative agents:
  - *Staphylococcus* (*S. aureus*, *S. epidermidis*)
  - *Streptococcus* (*S. pneumoniae*, others)
  - *Pseudomonas aeruginosa*
  - Enterobacteria (*Proteus*, *Enterobacter*, *Serratia*).
- **Symptoms**: Pain, photophobia, blurred vision, mucopurulent or purulent discharge.
- **Clinical findings**: Chemosis (conjunctival edema), deep conjunctival hyperemia, corneal stromal edema, corneal ulceration and perforation, anterior uveitis, hypopyon, posterior synechiae, endophthalmitis, etc.
- **Treatment**: Topical fortified vancomycin-ceftazidime combination and systemic fluoroquinolones.

VIRAL KERATITIS

Herpes Simplex Keratitis (Dendritic Keratoconjunctivitis)

- Caused by HSV Type I.
- Associated with blepharoconjunctivitis and preauricular lymphadenopathy.
- Symptoms include foreign body sensation, photophobia, and decreased corneal sensitivity.
- Corneal anesthesia may lead to neurotrophic keratitis and ulceration, characterized by painless corneal ulcers.
- Exists in three forms: epithelial, disciform (endotheliitis), and stromal (interstitial).

Varicella Zoster Keratitis

- Characterized by dendritic lesions similar to herpes simplex.
- Symptoms and clinical types resemble herpetic keratitis.

FUNGAL KERATITIS (KERATOMYCOSIS)

- Corticosteroid use increases the risk.
- Common causative agents: *Candida* and filamentous fungi (*Aspergillus*, *Fusarium*).
  - *Candida* keratitis is usually associated with chronic corneal disease or immunosuppression.
  - Filamentous fungal keratitis commonly occurs following ocular trauma with organic matter (e.g., tree branch).

PROTOZOAL KERATITIS (ACANTHAMOEBA)

- Most common protozoal keratitis in the community.
- Found in soil, freshwater, and seawater.
- Causes ring-shaped corneal abscesses and ulcerations.
- In developed countries, the most common transmission route is contaminated tap water and contact lens use.

NEUROTROPHIC KERATITIS

- Results from corneal anesthesia due to the loss of innervation (5th cranial nerve).
- The absence of protective sensory stimulation leads to intracellular edema, epithelial defects, and persistent ulceration.
- Causes include herpes simplex, herpes zoster oticus, trigeminal nerve surgery, or radiation therapy.

LAGOPHTHALMIC (EXPOSURE) KERATITIS

- Occurs in cases of 7th cranial nerve paralysis.
- Incomplete eyelid closure leads to dryness and secondary corneal infections.

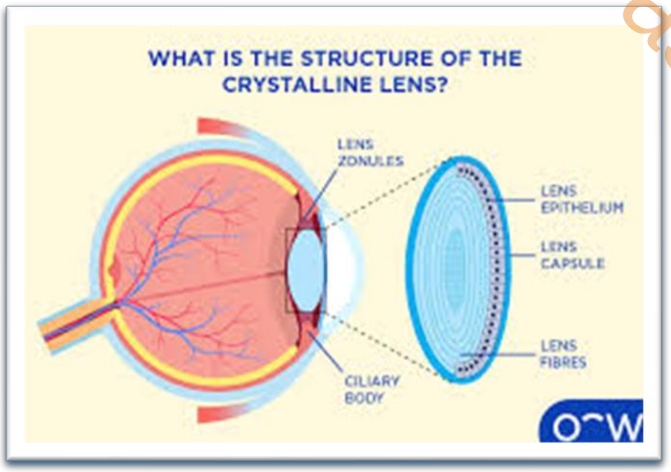
KERATOMALACIA

- Caused by vitamin A deficiency.
- Features dryness and necrosis of the cornea and conjunctiva.
- *Bitot's spots*, foamy plaques, are seen on the conjunctiva.

Unit : 4

LENS DISEASES

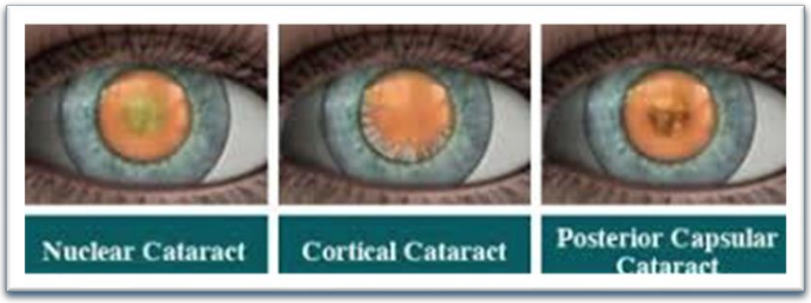
The lens is one of the main refractive structures of the eye, complementing the fixed refractive power of the cornea (approximately 43 diopters) with variable refractive power depending on accommodation (10-20 diopters). It is suspended between the iris anteriorly and the vitreous posteriorly by fine filamentous zonules originating from the ciliary processes.



The loss of transparency and opacification of the crystalline lens is termed **cataract**.

ACQUIRED CATARACT CAUSES

Age-related cataracts



1. **Subcapsular-cataracts**
   (a)Anterior-subcapsular-cataract
   (b) Posterior subcapsular cataract

Posterior subcapsular cataracts

2. **Nuclear cataracts**

- Myopia is observed due to an increased refractive index in the lens nucleus.
- Elderly patients with nuclear cataracts may experience the ability to read close objects without glasses due to this induced myopia ("second sight").

3. **Cortical cataracts**
   - Cortical opacities begin as clefts and vacuoles between lens fibers caused by lens cortex hydration.
   - Patients often complain about light scattering effects.

Presenile cataracts

Presenile cataracts are associated with systemic diseases, including:

1. Diabetes
2. Myotonic dystrophy
3. Atopic dermatitis
4. Neurofibromatosis type 2

Traumatic cataracts

- Trauma is the most common cause of cataracts in young individuals.

1. Direct penetrating injuries to the lens.
2. Blunt trauma leading to iris pigmentation imprinting on the anterior capsule ("Vossius ring") and flower-shaped opacities.
3. Electric shocks and lightning strikes.
4. Exposure to ionizing radiation for ocular tumors.
5. Infrared radiation (glassblower's cataract).

Effects of Blunt Trauma on the Eye

- Corneal erosion and edema
- Hyphema
- Iridodialysis
- Inflammation-induced hypertension (elevated intraocular pressure)
- Hypotony due to ciliary body depression (low intraocular pressure)
- Cataracts
- Globe rupture
- Vitreous hemorrhage
- Retinal edema and detachments
- Traumatic optic neuropathy
- Optic nerve avulsion
- Central retinal artery occlusion (rare)

Toxic cataracts

1. **Steroids**: Both systemic and topical steroid use has cataractogenic properties.
    - Lens opacities typically begin in the posterior subcapsular region, eventually involving the anterior subcapsular region.
2. Chlorpromazine, miotics, busulfan, amiodarone, and gold.

SECONDARY CATARACTS

Secondary or complicated cataracts occur as a result of other ocular conditions, such as:

1. Chronic anterior uveitis (most common cause of secondary cataracts).
2. Acute congestive angle-closure glaucoma.
3. High myopia.
4. Hereditary fundus dystrophies (retinitis pigmentosa, Leber congenital amaurosis, gyrate atrophy, Stickler syndrome).

CONGENITAL CATARACT ETIOLOGY

Systemic metabolic associations

1. Galactosemia
2. Galactokinase deficiency

3. Mannosidosis
4. Lowe oculocerebrorenal syndrome
5. Fabry disease
6. Other metabolic causes: maternal diabetes, hypoparathyroidism, pseudohypoparathyroidism, hypoglycemia, and hyperglycemia.

Associated intrauterine infections

- Congenital rubella
- Toxoplasmosis
- Cytomegalovirus infections
- Syphilis
- Herpes simplex
- Varicella

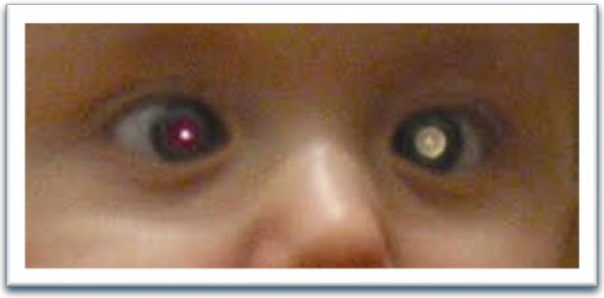
Associated chromosomal abnormalities

- Down syndrome (trisomy 21)
- Patau syndrome (trisomy 13)
- Edward syndrome (trisomy 18)
- Cri-du-chat syndrome (deletion of chromosome 5)
- Turner syndrome

COMPLICATIONS OF CORTICOSTEROID USE ON THE EYE

- Anterior and posterior subcapsular cataracts
- Bacterial and fungal infections
- Glaucoma

LEUKOCORIA AND OCULAR SYNDROMES



Leukocoria refers to the presence of a white reflex from the pupil ("white pupil").

Causes

- **Retinoblastoma** (most critical cause; frequency: 1/20,000)
- **Congenital cataract** (most common cause; frequency: 15/10,000)
- Senile cataract
- Retinopathy of prematurity (ROP)
- Retinal detachment
- Congenital toxoplasmosis
- Persistent hyperplastic primary vitreous (PHPV)
- Retinal dysplasia
- Coats disease

COATS DISEASE

- A congenital retinal telangiectasia associated with vascular anomalies, leading to exudation and eventually exudative retinal detachment.
- Almost always unilateral, commonly affecting boys and young males in the first decade.
- Presents with leukocoria, vision loss, and strabismus.

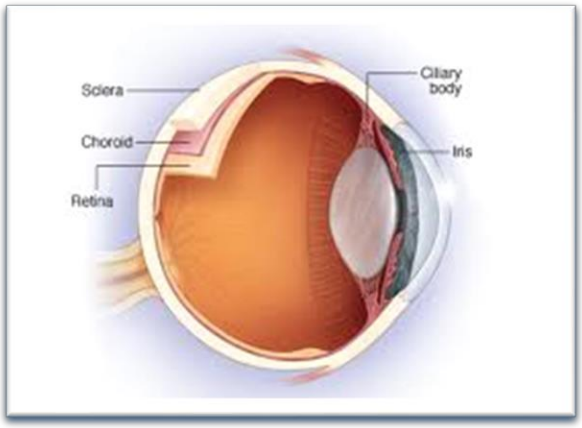
Unit : 5

UVEA AND RETINAL DISEASES

UVEA DISEASES

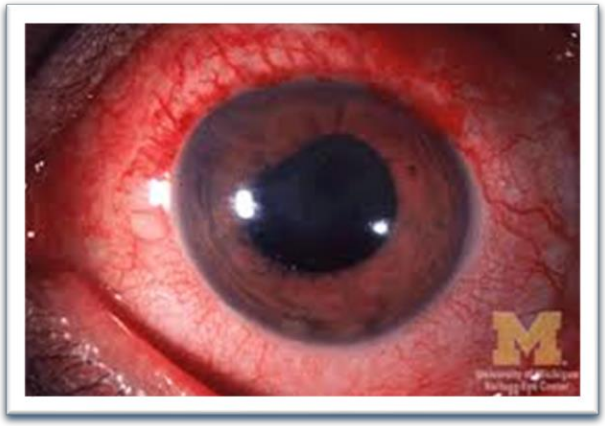
INFLAMMATORY DISEASES OF THE UVEA (UVEITIS)

The uvea consists of three components:



1. **Iris**
2. **Ciliary body**
3. **Choroid**

Inflammation of the iris and ciliary body is called **iridocyclitis** (anterior uveitis). Inflammation of the posterior part of the ciliary body, known as the pars plana, and adjacent areas is called **intermediate uveitis** (pars planitis). **Choroiditis** (posterior uveitis) refers to inflammation of the choroid, and when all components are inflamed, it is referred to as **panuveitis**.



Clinical Classification:

- **Acute**: Symptoms have a sudden onset and persist for 6 weeks or less.
- **Chronic**: Can last months or years, often recurring (chronic recurrent uveitis).

Etiological Classification:

- Frequently of unknown cause (**idiopathic**).
- **Autoimmune**:
  - Behçet's disease
  - Vogt-Koyanagi-Harada syndrome

- Sympathetic ophthalmia
- Juvenile rheumatoid arthritis
- Ankylosing spondylitis
- Reiter's syndrome
- Ulcerative colitis
- Crohn's disease
- Psoriasis

- **Infectious**:
  - **Bacteria** (e.g., tuberculosis, staphylococcus, Brucella, Borrelia)
  - **Viruses** (e.g., CMV, HSV, EBV, HIV)
  - **Fungi** (e.g., Candida, Histoplasma, Aspergillus)
  - **Parasites** (e.g., Toxoplasma, Toxocara)
- **Malignancies**:
  - Retinoblastoma
  - Leukemia
  - Lymphoma
  - Malignant melanoma
- **Others**:
  - Traumatic uveitis
  - Sarcoidosis
  - Retinal detachment

Pathological Classification:

a) Granulomatous Uveitis:

- Insidious onset with mild pain and photophobia.
- Large keratic precipitates on the corneal endothelium ("mutton fat").
- Associated with systemic diseases like sarcoidosis, tuberculosis, Lyme disease, and syphilis.
- **Nodules**:
  - Koeppe nodules: At the pupillary margin.
  - Busacca nodules: On the anterior iris surface.

b) Non-Granulomatous Uveitis:

- Acute, with prominent anterior segment involvement.
- Symptoms include pain, photophobia, and redness.
- Typically associated with **HLA-B27** diseases (e.g., ankylosing spondylitis) and infections.

Specific Conditions:

Acute Iridocyclitis:

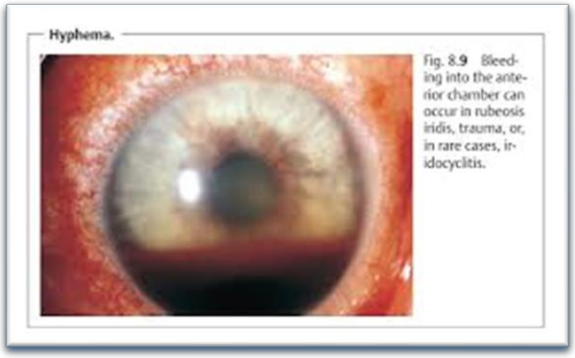


Fig. 8.9 Bleeding into the anterior chamber can occur in rubeosis iridis, trauma, or, in rare cases, iridocyclitis.

- Most common form of uveitis.
- Sudden onset, nongranulomatous.
- Symptoms: Pain, redness, tearing, photophobia, blurred vision.
- Findings:
  - Keratic precipitates on the corneal endothelium.

- Hypopyon (settled inflammatory cells in the anterior chamber).
- Synechiae (adhesions between iris and lens).
- Tyndall effect (protein and cell infiltration in the anterior chamber).

Fuchs' Uveitis Syndrome (Fuchs' Heterochromic Iridocyclitis):

- Chronic, nongranulomatous uveitis.
- Unilateral iris depigmentation, keratic precipitates, secondary cataract.

Sympathetic Ophthalmia:

- Bilateral granulomatous panuveitis following trauma or surgery to one eye.
- Prophylactic enucleation of the injured eye may prevent involvement of the unaffected eye.

- Symptoms: Pain, lacrimation, ciliary injection, nodules (Dalen-Fuchs nodules).
- Complications: Cataracts, secondary glaucoma, phthisis bulbi.
- Treatment: High-dose corticosteroids and periocular steroid injections.

Uveal Tumors:

- Most common is **malignant melanoma**, typically unilateral and located in the choroid.

RETINAL DISEASES

Retina Structure:

The retina consists of multilayered neural tissue and a single-layered pigment epithelium.

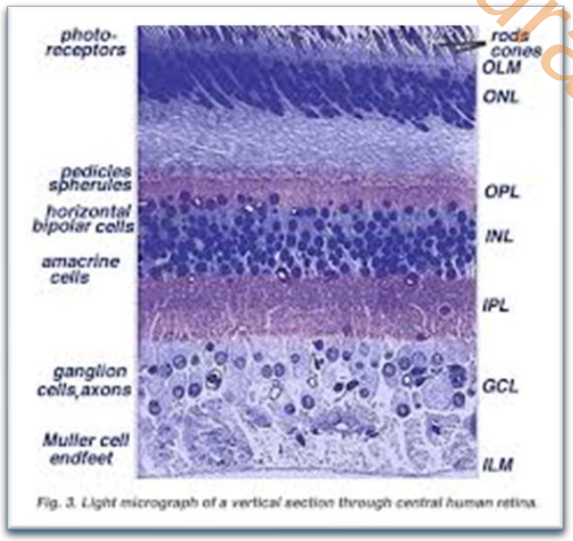


Fig. 3. Light micrograph of a vertical section through central human retina.

- **Retinal pigment epithelium (RPE)**: Single layer of pigmented cells responsible for renewing photoreceptor outer segments and facilitating metabolic exchange.
- **Photoreceptor layer**: Contains ~100 million rods and 6 million cones.
  - Fovea centralis: Photoreceptors are exclusively cones.
  - Peripheral retina: Higher rod density.
- **External limiting membrane (ELM)**: Composed of photoreceptor connections.
- **Outer nuclear layer (ONL)**: Contains photoreceptor nuclei.
- **Outer plexiform layer (OPL)**: Synapses between photoreceptors and bipolar cells.
- **Inner nuclear layer (INL)**: Contains bipolar, horizontal, and Müller cell nuclei.
- **Inner plexiform layer (IPL)**: Synapses between bipolar and ganglion cells.
- **Ganglion cell layer (GCL)**: Contains ganglion cell bodies, multilayered in the macula.
- **Nerve fiber layer (NFL)**: Formed by ganglion cell axons.
- **Inner-Limiting-Membrane-(ILM):**
  The ILM is a membrane formed by the surface modification of the vitreous and the terminal projections of Müller cells. In the fovea, only the inner limiting membrane, outer plexiform layer, and photoreceptor cells are present from the retinal layers. Therefore, the center of the fovea is the thinnest part of the retina.**Only cone cells are present in the fovea** (responsible for vision in bright light and sharp vision).**Rod cells are more abundant in the periphery** (responsible for vision in dim light).

Retinal Vascular Diseases

Central Retinal Artery Occlusion (CRAO):

- Causes sudden, painless, and severe vision loss in elderly patients.
- The most common causes are thrombosis and embolism.
- The fovea centralis, which does not contain nerve fibers, appears cherry-red (resembling the Japanese flag) due to the red color of the underlying choroid.
- Pupils are dilated, and the light reflex is weak or absent.

Central Retinal Vein Occlusion (CRVO):

- Leads to sudden, painless vision loss in elderly patients.
- Since blood reaches the retina via the central retinal artery and drains through the central retinal vein, venous blockage results in blood accumulation in the retina.
- The optic disc and its borders are edematous.
- Intraretinal hemorrhages are present.
- CRVO is also associated with numerous soft exudates.

Hypertensive Retinopathy:

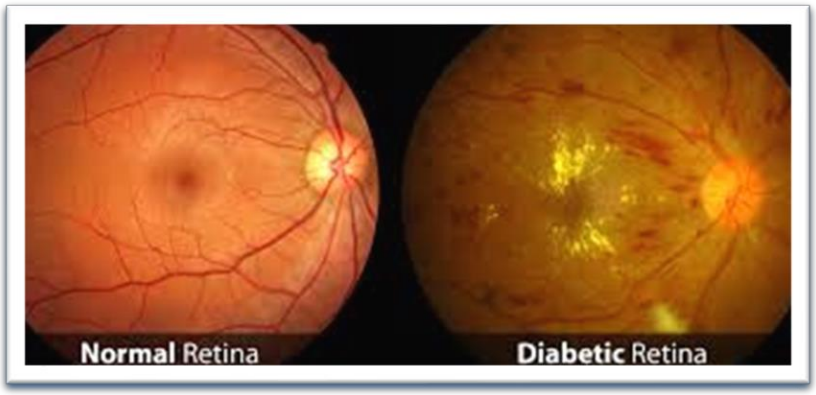
- **Key finding of hypertension in the eye:** narrowing of arterioles.
- Normally, the artery/vein diameter ratio is 2/3, but with hypertension, it decreases to 1/2 or 1/4.

- When hypertension coexists with arteriosclerosis:
  - **Gunn sign**: Vein compression.
  - **Salus sign**: Vein deflection.
  - **Humping**: Bridging at crossings.

Grading:

- **Grade 1:** Mild generalized retinal arteriolar narrowing.
- **Grade 2:** Increased narrowing with focal constrictions. Salus sign (vein deflection) at A-V crossings. Copper wire appearance may begin.
- **Grade 3:** Retinal hemorrhages, hard and soft exudates, and copper wire appearance. Bonnet sign (vein dilation distal to crossing) and Gunn sign (vein disappearance at crossings).
- **Grade 4:** Severe Grade 3 with optic disc edema and silver wire appearance of arteries.

Diabetic Retinopathy:



- Reduced pericytes and increased endothelial cells in affected capillaries.
- Involves arteries, veins, and capillaries.
- The affected vessels become occluded or leaky.
- There is a close relationship between the duration of diabetes and retinopathy.

Pathophysiology:

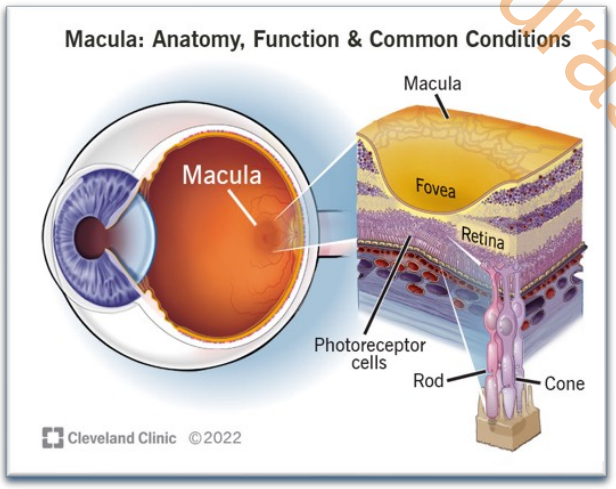
1. **Microaneurysms**: The earliest clinically detectable lesion in diabetic retinopathy.
2. **Retinal Hemorrhages**: May be intraretinal, preretinal, or intravitreal.

3. **Exudates:**
    - **Hard Exudates:** Result from chronic localized retinal edema.
    - **Soft Exudates (Cotton-Wool Spots):** Debris caused by ischemia in the nerve fiber layer.
4. **Venous Changes:**
    - Venous dilation is the earliest clinical finding but difficult to assess.
    - **Venous beading:** Focal narrowing and dilations resembling sausages, indicating widespread ischemia.
5. **Intraretinal Microvascular Abnormalities (IRMA):**
    - Abnormal vascular connections between arteries and veins in ischemic retina areas.
6. **Retinal Thickening and Diabetic Macular Edema (DME):**
    - Caused by the breakdown of the blood-retinal barrier.
7. **Neovascularization (Most Severe Lesion):**
    - Develops in ischemic retinal areas due to the release of cytokines like VEGF to combat hypoxia.
    - If located on or within one disc diameter of the optic disc, it is termed **disc neovascularization (NVD)**.
    - If located elsewhere in the retina, it is termed **retinal neovascularization (NVE)**.

Unit : 6

Macular Diseases

Practical Anatomy



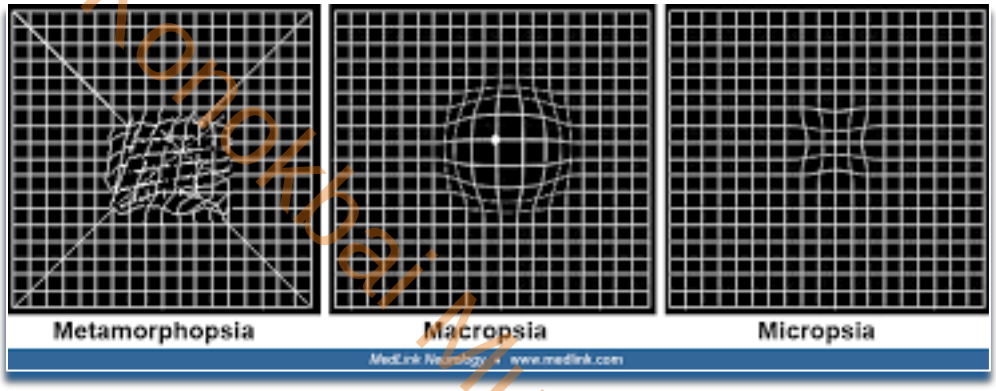
- The macula is an oval area approximately 5 mm in diameter located at the posterior pole of the eye.
- The macula contains points of clinical importance, including the fovea, foveola, and the foveal avascular zone.
- The fovea has a diameter of approximately 1.5 mm.
- The foveola is a region without capillaries.

Symptoms of Macular Disease

- **The primary symptom** of macular disease is the weakening of central vision.
- Typically, patients complain about something obstructing their central vision (positive scotoma).
- In contrast, optic nerve pathologies usually present with negative scotoma, which is unnoticed by the patient but detectable on visual field testing.

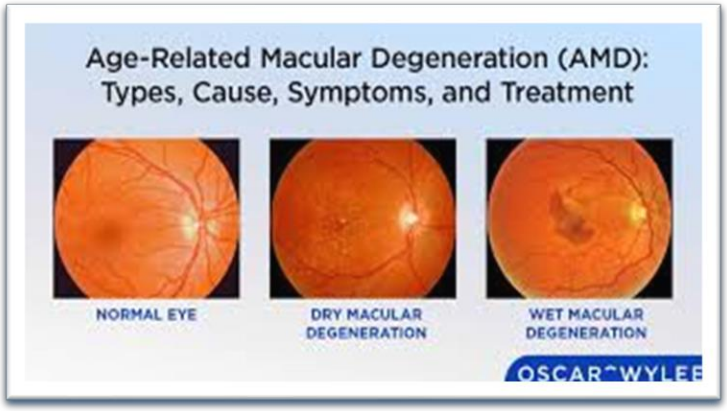
Other symptoms specific to macular disease that are not observed in optic nerve disorders include:

1. **Metamorphopsia**: Distortion in the shape of objects.
2. **Micropsia**: Objects appear smaller than their actual size.
3. **Macropsia**: Objects appear larger than their actual size.



- In optic nerve lesions, **color vision is impaired**, whereas in macular (retinal) disease, color vision is not affected in the early stages.

Age-Related Macular Degeneration (AMD)



1. **Prevalence**:
   - AMD is the leading cause of irreversible blindness in individuals over 70 years of age in the Western world.
2. **Two Main Types of AMD**:
   - **Dry Type (Non-Exudative)**:
     - A slowly progressive atrophic condition responsible for ~90% of cases.
   - **Wet Type (Exudative)**:
     - Though less common, it is more devastating, often leading to blindness in a short time.
3. **Drusen**:
   - Yellow-white spots frequently associated with AMD development are located at the posterior pole of the retina.
4. **Screening Test**:
   - The Amsler grid test is used for screening AMD.

Histopathology

Dry (Atrophic) AMD

- Dry AMD is the most common type, accounting for approximately 90% of cases.

- There is no definitive treatment, but providing low-vision aids can be beneficial for many patients.

Wet (Exudative) AMD

- Wet AMD manifests as sudden-onset **metamorphopsia** and **blurring of central vision**.
- Subretinal areas may show dirty gray or yellowish-gray discoloration.

Treatment Options for Wet AMD:

- Argon laser photocoagulation
- Photodynamic therapy
- Experimental treatments (macular surgery, intravitreal gas injection, transpupillary thermotherapy)
- Anti-VEGF agents (Ranibizumab, Aflibercept, and Bevacizumab)

Uses of Anti-VEGF Agents in Ophthalmology

- Diabetic macular edema
- Proliferative diabetic retinopathy
- Retinal vein occlusion
- Wet AMD
- Retinopathy of prematurity
- Preoperative adjunct therapy in neovascular glaucoma

Inflammatory Retinal Diseases

Toxoplasmosis

- Caused by *Toxoplasma gondii*.
- Leads to **neuroretinitis** and presents with:

- Chorioretinitis
- Intracranial calcifications
- Convulsions
- Hydrocephalus

Viral Retinitis

- **CMV (Cytomegalovirus)**: Causes necrotizing inflammation of the sensory retina and choroid.
- **Rubella**: Leads to inflammation affecting the retinal pigment epithelium.

Retinitis Pigmentosa

- The most common retinal dystrophy.
- Characterized by melanin deposits around retinal vessels ("bone spicule" pigmentation).
- Progresses slowly, beginning around the age of 30 and continuing for years.
- Symptoms include:
  - Annular scotomas and tunnel vision in the visual field
  - Impaired night vision (nyctalopia)

Associated Syndromes:

- Usher syndrome
- Kearns-Sayre syndrome
- Cockayne syndrome
- Refsum disease

Albinism

Eye Findings

- Foveal hypoplasia is observed, along with the absence of vessels in the foveal pit.
- Visual acuity is generally below 6/60 due to foveal hypoplasia.
- Nystagmus is typically pendular and horizontal.
- The iris is translucent, leading to a pink-eye appearance.

Retinal Detachment

- Sensory retina separates from the retinal pigment epithelium due to subretinal fluid.
- There are two main types: rhegmatogenous (tear-related) and non-rhegmatogenous.
    - **Rhegmatogenous Detachment**: The most common type in adults.
    - In children, trauma is the most common cause of retinal detachment.

Rhegmatogenous (Tear-Related) Detachment

- Occurs when liquefied vitreous passes through a retinal tear into the subretinal space.
- The vitreous is the largest volume of the eye and supports its internal structures. It is a clear gel-like substance containing hyaluronic acid and a collagen network.
- It can be bilateral.
- Predisposing peripheral lesions include:
    1. **Lattice Degeneration**:

A peripheral retinal degeneration.

Present in ~8% of the general population and ~40% of eyes with detachment.

Common in Marfan syndrome, Stickler syndrome, Ehlers-Danlos syndrome, and moderate to high myopia.

2.Snail-Track Degeneration

3.Degenerative Retinoschisis:

- ~70% of patients are hypermetropic.
- Detachment is very rare.

4.White Without Pressure:

- Giant retinal tears may occur.

Importance of Myopia

- Myopia is present in ~40% of cases with detachment.
- The higher the refractive error, the greater the risk of detachment.
- Conditions increasing the risk in myopic eyes include lattice degeneration and snail-track degeneration.

Non-Rhegmatogenous Detachments

- No retinal tear is present.

1. **Tractional Retinal Detachment**
    - Caused by vitreoretinal membranes pulling the sensory retina.
    - Main causes:
        1. Proliferative diabetic retinopathy
        2. Retinopathy of prematurity
        3. Proliferative sickle cell retinopathy
        4. Penetrating posterior segment trauma

2. **Exudative (Serous) Retinal Detachment**

- Caused by choroidal-origin subretinal fluid passing into the subretinal space through damaged retinal pigment epithelium.
- Main causes:

1. **Choroidal Tumors**: Melanoma, etc.
2. **Intraocular Inflammation**
3. **Iatrogenic Causes**: Various surgeries
4. **Subretinal Neovascularization**
5. **Systemic Causes**: Hypertension, pregnancy toxemia
6. **Uveal Effusion Syndrome**

Clinical Features

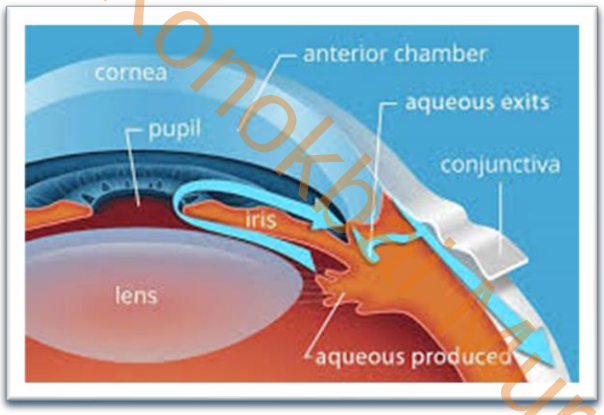
1. **Rhegmatogenous Detachment**
   - ~60% present with photopsia (flashes of light) and floaters.
   - Tobacco dust sign: Pigment cells in the anterior vitreous.
   - Marcus-Gunn pupil may be present.
   - Visual field defects worsen as the day progresses, as subretinal fluid is absorbed in the morning.
2. **Tractional Detachment**
   - Photopsia and floaters are usually absent.
   - Visual field defects progress slowly.
3. **Exudative Detachment**
   - Photopsia is not expected, but floaters may be seen.
   - Visual field defects occur suddenly and progress rapidly.

Retinoblastoma

- The most common malignant intraocular tumor in childhood.
- Most frequently seen in the first two years of life.
- ~30% are bilateral, and ~20% are inherited in an autosomal dominant manner.
- Calcification is observed in ~70% of cases on radiological imaging.
- The most important cause of leukocoria in newborns.
- Characteristic **Flexner-Wintersteiner rosettes** are seen histologically.

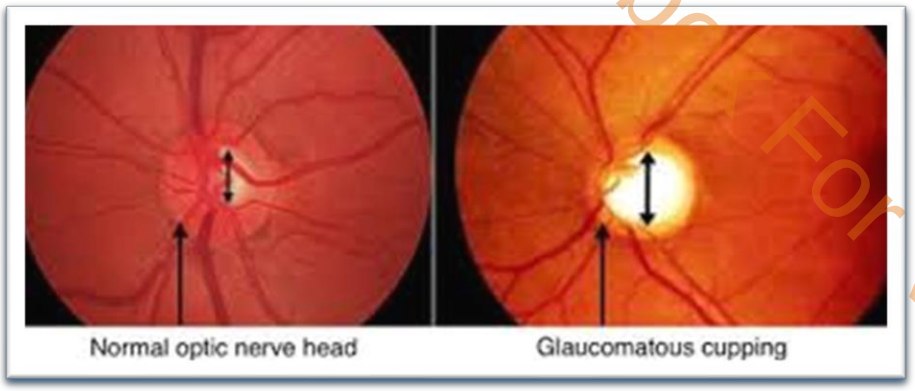
Glaucoma and Optic Nerve Diseases

- The normal intraocular pressure measured by tonometry is **11–21 mmHg**.
- Intraocular pressure tends to increase with age.
- **Aqueous humor** plays a crucial role in the formation and regulation of intraocular pressure.
- Aqueous humor is secreted by the **non-pigmented ciliary epithelium** of the **ciliary processes**.



Flow of aqueous humor: Ciliary body → Posterior chamber → Pupil opening → Anterior chamber → Trabecular meshwork → Schlemm's canal → Venous system

Fundoscopic Examination

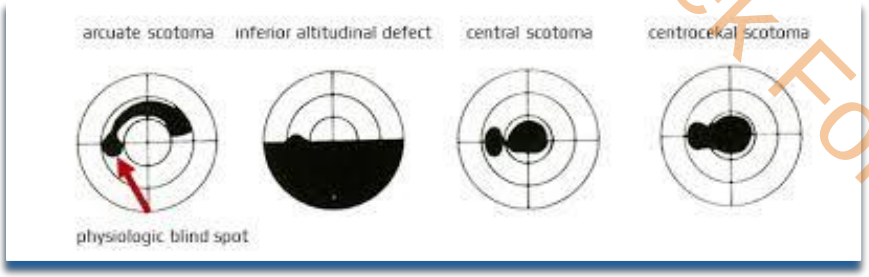


Optic Disc (Papilla)

- Nerve fibers form thick bundles as they approach the optic disc.
- **Optic atrophy** leads to **excavation** (enlargement of the optic cup).

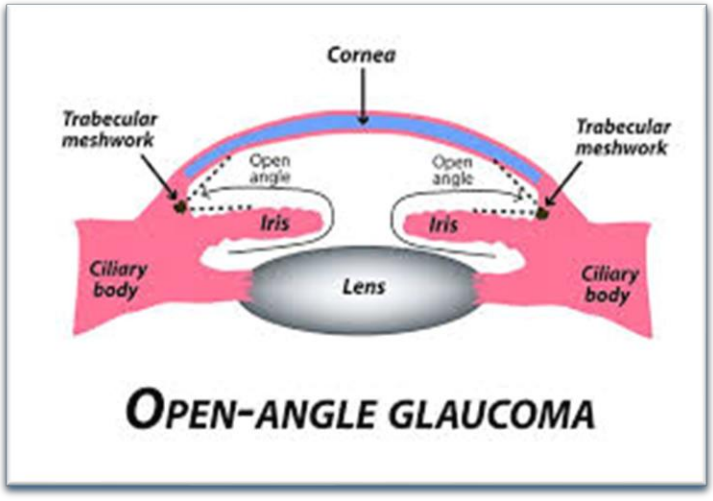
Peripapillary Findings

- **Flame-shaped hemorrhages**
- **Glaucoma halo** around the optic disc
- **Retinal nerve atrophy** → Enlargement of the **blind spot**
- **Paracentral scotoma**
- **Nasal step**
- **Nerve fiber bundle defects**
    - **Arcuate scotoma**
    - **General depression of isopters**
    - **Concentric narrowing of the visual field**



Classification of Glaucoma

Open-Angle Glaucoma



1. Primary Open-Angle Glaucoma (POAG)

- **Most common type of glaucoma**
- **Disease of older adults** (over 40 years old)
- **Usually bilateral**

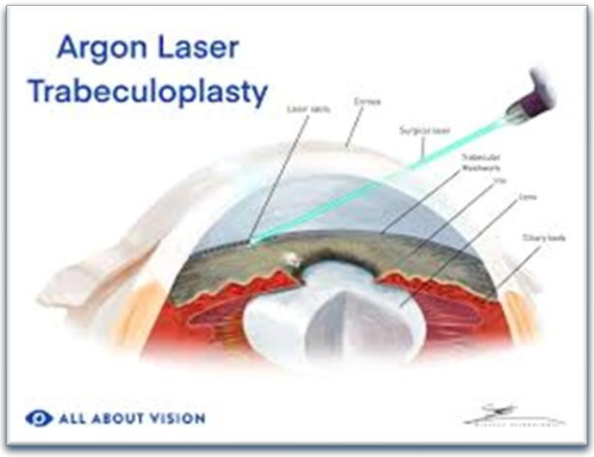
Medical Treatment

a) **Beta-blockers** (Timolol, Betaxolol): Reduce aqueous humor production. Side effects include **burning,redness,and-punctate-keratitis**.

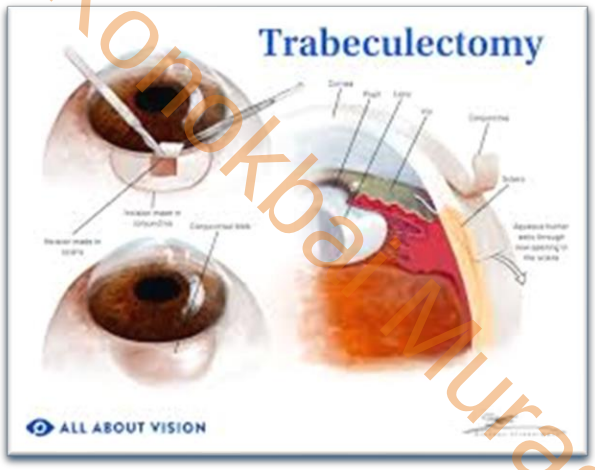
b) **Adrenergic agents** (**Alpha-2 agonists**; Brimonidine, Apraclonidine): Reduce intraocular pressure by **decreasing aqueous humor secretion** and **enhancing uveoscleral outflow**.
c) **Topical Carbonic Anhydrase Inhibitor** (Acetazolamide): Reduces aqueous humor production.
d) **Prostaglandins** (Latanoprost, Bimatoprost): Facilitate **uveoscleral outflow**.

- **Most effective first-line treatment** with **fewer systemic side effects**.
- **Latanoprost (PGF2α)** → Can cause **darkening of eye color** and **increase ocular inflammation**.
- **Contraindicated,in-inflammatory-glaucoma**.
  e) **Parasympathomimetics**: Open the angle and **facilitate aqueous humor drainage**.
- **Pilocarpine** → Most commonly used.
  f) **Systemic Carbonic Anhydrase Inhibitor** (Acetazolamide): Reduces aqueous humor production.
- **Mainly used for acute glaucoma attacks**.

Surgical Treatment



a) **Laser Trabeculoplasty** → Enlarges the trabecular meshwork.



b) **Trabeculectomy** → Most successful surgical treatment.

2. Ocular Hypertension

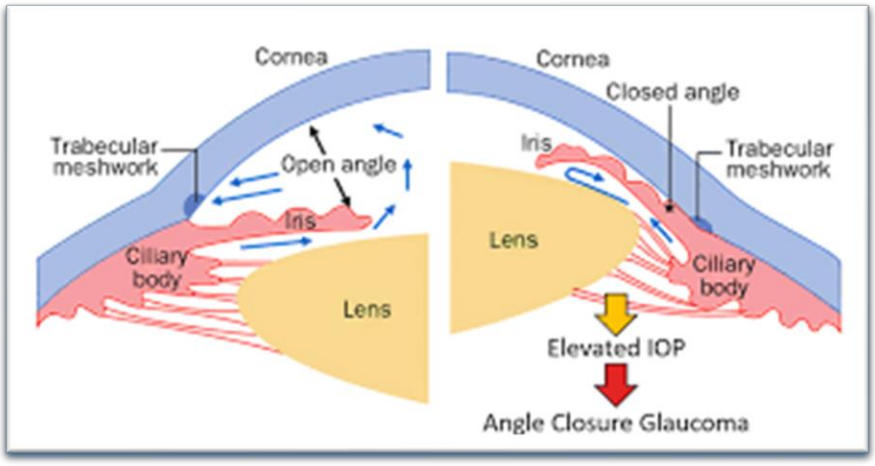
- **Increased intraocular pressure** without **optic nerve damage or visual field loss**.

3. Normal-Tension Glaucoma

- **Normal intraocular pressure** but with **optic atrophy and visual field changes**.

4. Secondary (Acquired) Open-Angle Glaucoma

- **Inflammatory**
- **Pigmentary glaucoma**
- **Pseudoexfoliation glaucoma**
- **Tumor-related glaucoma**
- **Steroid-induced glaucoma**



Angle-Closure Glaucoma

A. Angle Closure Due to Pupil Block

- Aqueous humor **cannot pass through the pupil**, leading to pressure build-up in the **posterior chamber** and pushing the iris forward.
- **Common cause: Uveitis**.

B. Angle Closure Without Pupil Block

- Even though the **pupil remains open**, **peripheral iris** causes angle closure.
- **Common cause: Ciliary melanoma**.

Treatment of Angle-Closure Glaucoma

1. **Oral glycerol or IV mannitol**
2. **Oral or IV acetazolamide**
3. **Topical anti-glaucoma medications** (beta-blockers, alpha-2 agonists, prostaglandins)
4. **Miotic eye drops (pilocarpine)**
5. **Laser iridotomy**

Optic Neuropathy

The term **optic neuropathy** refers to **any condition affecting the optic nerve**.

Types of Optic Neuropathy

1. **Inflammatory** (demyelinating, infectious, autoimmune)
2. **Ischemic optic neuropathy**
3. **Compressive/infiltrative**
4. **Toxic-nutritional**
5. **Traumatic**
6. **Radiation-induced**
7. **Hereditary**

Inflammatory Optic Neuropathies

Optic Neuritis

- **First symptom: Pain with eye movement**
- **After a few days: Decreased vision and color perception**
- **Relative Afferent Pupillary Defect (RAPD)** may be present.

- **Depth perception impairment**
- **Typical in multiple sclerosis** → Vision worsens with **exercise or increased body temperature** (**Uhthoff's phenomenon**).

Visual Field Defects in Optic Neuritis:

- **Central scotoma**
- **Centrocecal scotoma**
- **Nerve fiber bundle defects**

Types of Optic Neuritis

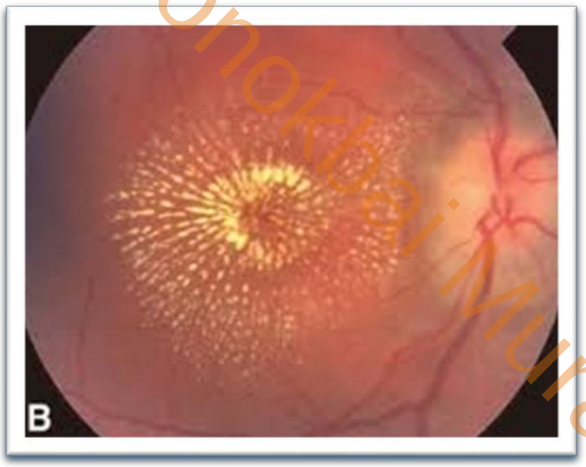
1. **Retrobulbar Neuritis**

- **Inflammation behind the eyeball.**
- **Most commonly associated with multiple sclerosis.**
- **Classic saying:** *"The doctor sees nothing, and the patient sees nothing."*

2. **Papillitis**

- **Most common type of optic neuritis in children.**
- **Usually post-infectious and unilateral.**
- **Findings: Optic disc swelling and flame-shaped hemorrhages.**

3. Neuroretinitis



- **Combination of papillitis and macular star appearance**.
- **90% of cases are due to cat scratch disease**.
- Other causes: **Syphilis, Lyme disease, mumps, leptospirosis**.

Ischemic-Optic-Neuropathies:

A condition characterized by ischemia, edema, and secondary optic atrophy in the chronic phase due to occlusion of the short posterior ciliary arteries at the optic nerve head. Altitudinal visual field defects are observed.

1. Arteritic Anterior Ischemic Neuropathy:

- Optic nerve infarction occurs due to occlusion of short posterior ciliary arteries caused by granulomatous necrotizing arteritis associated with **giant cell arteritis**.
- Seen in **temporal arteritis** and **systemic lupus erythematosus (SLE)**.
- **Treatment: Steroids** are used.

2. Non-Arteritic Anterior Ischemic Neuropathy:

- Optic nerve head infarction caused by occlusion of short posterior ciliary arteries.
- Common in **hypertension** and **diabetes**.
- **Steroids are ineffective**.

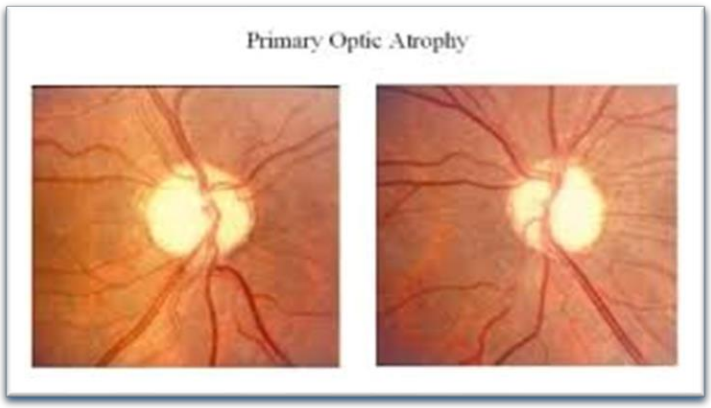
Anterior Toxic Neuropathy:

- Generally presents as **bilateral optic disc swelling** with **scotomas involving the blind spot**.
- **Causes:**
    - **Chloramphenicol**
    - **Ethambutol**
    - **Isoniazid**
    - **Lead poisoning**

Retrobulbar Toxic Optic Neuropathy:

- **Progressive**, **bilateral**, with **scotomas** and **temporal pallor of the optic disc**.
- **Causes:**
  - **Vitamin B deficiency**
  - **Malabsorption syndromes**
  - **Tobacco-alcohol amblyopia** (seen in chronic smokers and alcoholics)
  - **Antipyrine**
  - **Digitalis**
  - **Quinine**
  - **Streptomycin**

Primary Optic Atrophy:



- **Optic atrophy develops without prior swelling of the optic nerve head.**
- **Occurs due to lesions affecting areas from the retrolaminar region to the lateral geniculate body.**
- **Fundoscopic Features:** White, non-swollen disc with sharp margins.

- **Causes:**
  - **Optic neuritis**
  - **Tumor or aneurysmal compression**
  - **Hereditary optic neuropathies**
  - **Trauma**

Secondary Optic Atrophy:

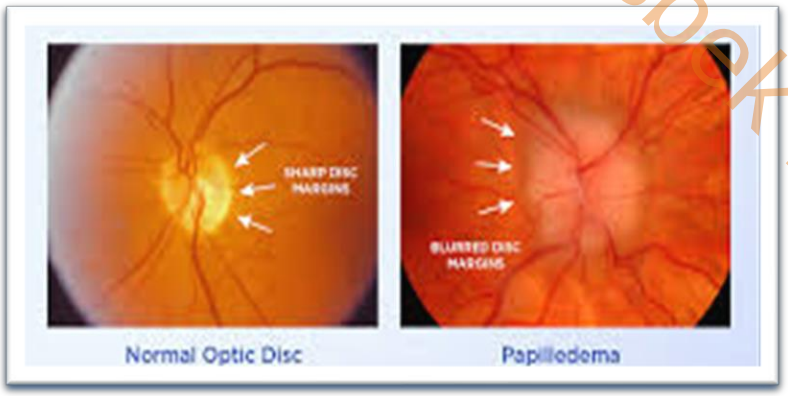
- **Develops after prolonged optic nerve head swelling.**
- **Fundoscopic Features:** White or dirty gray disc, slightly swollen with blurred margins due to gliosis.

- **Causes:**
  - **Chronic papilledema**
  - **Anterior ischemic optic neuropathy**
  - **Papillitis**

Optic Nerve Tumors:

- **The most common primary tumors** are **gliomas** and **optic nerve sheath meningiomas**.
- **Features:** Optic atrophy and enlargement of the optic canal.

Papilledema:



- **Defined as swelling of the optic nerve head due to increased intracranial pressure.**
- **Any patient with papilledema should be evaluated for an intracranial mass or increased pressure until proven otherwise.**
-

Clinical Stages:

1. **Early Papilledema:** Visual acuity remains normal.
2. **Established Papilledema:** Transient vision blackouts begin, and visual acuity is frequently reduced.
3. **Chronic Papilledema:** Hemorrhages in the optic disc with further vision loss.
4. **Atrophic Papilledema:** Severe reduction in visual acuity.

OTHER MAIN OPHTHALMOLOGIC SYMPTOMS

Temporary Vision Loss:

Vision returns within 24 hours, usually within the first hour.

- **Seconds:** Papilledema
- **A few minutes:** Amaurosis fugax (carotid stenosis)
- **10-60 minutes:** Migraine

Sudden Painless Vision Loss:

- **Central retinal artery occlusion**
- **Central retinal vein occlusion**
- **Dense vitreous hemorrhage**
- **Retinal detachment**
- **Cerebrovascular event**
- **Non-arteritic ischemic optic neuropathy**
- **Giant cell arteritis (arteritic ischemic optic neuropathy)**
- **Toxic amblyopia (methanol, quinine, lead)**

Sudden Painful Vision Loss:

- **Acute angle-closure glaucoma**
- **Anterior uveitis**
- **Keratitis**
- **Optic neuritis (papillitis, retrobulbar neuritis)**

Gradual Vision Loss:

- **Refractive errors:** Myopia, hyperopia, astigmatism
- **Corneal changes:** Keratitis (corneal ulcers, leukoma formation), drug intoxications (Chloroquine, Amiodarone, Indomethacin), corneal degenerations
- **Lens changes:** Cataract development
- **Glaucoma:** Primary open-angle (chronic) glaucoma causes vision loss over years
- **Uveitis**
- **Tumors (primary or metastatic)**
- **Retinal involvement:** Vascular diseases (diabetic retinopathy, hypertensive retinopathy), retinal degenerations (age-related macular degeneration)
- **Optic nerve disorders**

Bilateral Sudden Vision Loss:

- **Optic neuritis**
- **Toxic amblyopia (especially methanol intoxication)**
- **Head trauma**

- **Uremic amaurosis**
- **Hysteria**
- **Simulation**
- **Malignancy**
- **Ophthalmic migraine:** Initially unilateral, followed by bilateral pain, flashes of light, and vision loss. Recurrent episodes help in diagnosis.

Floaters (Muscae Volitantes):

- If accompanied by **flashes of light (photopsia), sudden onset, or vitreous hemorrhage**, retinal tear should be suspected.
- **Posterior vitreous detachment**
- **Vitreous hemorrhage**
- **Intermediate or posterior uveitis**
- **Retinitis in immunocompromised patients**

Flashes of Light (Photopsia):

Flashes occur due to **vitreous traction** during retinal tears or posterior vitreous detachment.

- **Early stage of retinal detachment**
- **Migraine aura**
- **Moore's phenomenon:** Seen in darkness when rubbing the eyes or making sudden side-to-side eye movements.
- **Mechanical stimulation of the retina in blunt trauma**

Visual Field Defects (Scotomas):

- **Glaucoma**
- **Vascular lesions in the visual pathways**
- **Space-occupying lesions**
- **Retinal detachment**
- **Vitreous or retinal hemorrhages**

Photophobia (Light Sensitivity):

- **Foreign bodies in the conjunctiva or cornea**
- **Corneal abrasions or keratitis**
- **Acute iritis (uveitis)**

- Acute conjunctivitis
- Albinism
- Congenital glaucoma

Night Vision Impairment:

- Retinitis pigmentosa
- Peripheral retinal degeneration
- High myopia

Halos Around Lights:

- Glaucoma attacks
- Bacterial or allergic conjunctivitis (due to tearing)
- Lens opacities

Double Vision (Diplopia):

(a) Binocular:

- Ocular muscle paralysis
- Orbital diseases (thyroid orbitopathy, orbital pseudotumor, orbital tumor, cavernous sinus/superior orbital fissure syndrome, blow-out fracture; enophthalmos, diplopia, infraorbital nerve anesthesia, subconjunctival hemorrhage)
- Myasthenia gravis (intermittent!!)

(b) Monocular:

- Uncorrected high astigmatism
- Corneal opacity or irregularity
- Cataract and pupil abnormalities (large peripheral iridotomy)

Acute Proptosis:

- Orbital pseudotumor
- Thyroid orbitopathy
- Traumatic orbital hemorrhage
- Orbital cellulitis
- Carotid-cavernous sinus fistula (bilateral)
- Cavernous sinus thrombosis (bilateral)

- **Leukemia or lymphoma**
- **Rhabdomyosarcoma or metastatic neuroblastoma (in children)**

Eye Pain:

Pain in the eyeball can result from **inflammation** of the conjunctiva, cornea, sclera, iris, and eye muscles.

- **Deep, dull pain:** Glaucoma crisis, iris and ciliary body inflammation, orbital tissue and sinus inflammation, orbital periostitis and abscesses, internal carotid artery aneurysms.
- **Fatigue, tenderness, pain on eye pressure:** Inflammatory conditions of the eyelids, sclera, and episclera, fever and headache (flu), and sinusitis.
- **Acute dacryoadenitis (inflammation of the lacrimal gland):** Causes pain, redness, swelling, and an "S"-shaped curve in the outer upper eyelid.

Burning Sensation:

- **Blepharitis**
- **Dry eye syndrome**
- **Conjunctivitis**
- **Corneal problems**
- **Inflamed pterygium and pinguecula**
- **Episcleritis**
- **Superior limbic keratoconjunctivitis**

Tearing (Lacrimation):

Painful: Corneal lesions, anterior uveitis, trichiasis, entropion.
Painless: Dry eye, nasolacrimal duct obstruction, ectropion, conjunctivitis (allergic, toxic), congenital glaucoma.

Acute Red Eye:

- **Conjunctivitis**
- **Iridocyclitis**
- **Corneal abrasion or foreign body**
- **Blepharitis**
- **Endophthalmitis**
- **Episcleritis**
- **Acute glaucoma crisis**

- **Scleritis**
- **Keratitis**

SYSTEMIC DISEASES AND THE EYE

Chromosomal Diseases:

One of the important findings in the retina in chromosomal diseases is coloboma. During the embryological development of the eye, in the 4th week of fetal life, the optic vesicle protrudes from the forebrain to form the eye. To develop into a spherical structure, it invaginates and fuses along the inferonasal axis. Defects in this closure (fusion) process are known as colobomas.

- In **Schmid-Fraccaro syndrome**, choroidal coloboma is seen.
- In **Patau syndrome**, retinal dysplasia and detachment occur.
- In **Trisomy 18**, retinal coloboma and optic atrophy can be observed.

Cardiac Diseases:

- **Cardiac myxoma:** Retinal artery embolism
- **Congenital heart diseases:** Hypoxia-induced changes
- **Endocarditis:** Emboli (thrombus, septic emboli) leading to Roth spots, hemorrhages, and exudates
- **Mitral valve prolapse:** A/V occlusions, amaurosis fugax
- **Hypertension (HT):** Hypertensive retinopathy findings
- **Aortic arch syndrome (Takayasu arteritis):** Retinal neovascularization

Collagen Tissue Diseases:

- **Ankylosing spondylitis:** Uveitis
- **Reiter's syndrome:** Non-specific urethritis, arthritis, conjunctivitis, or iritis
- **Juvenile Rheumatoid Arthritis (JRA):** Uveitis
- **Temporal arteritis:** Ischemic optic neuropathy, amaurosis fugax, central retinal artery occlusion, cranial nerve palsies, bilateral involvement. Symptoms include temporal headache, jaw claudication, high ESR, and diagnosis is confirmed by temporal artery biopsy.
- **Behçet's disease:** Vascular thrombosis, iritis, oral and genital mucocutaneous ulcerations.

Endocrine Diseases:

- **Addison's disease:** Increased MSH → hyperpigmentation (conjunctiva, sclera), papilledema (due to increased intracranial pressure—ICP).

- **Cushing's syndrome:** Increased intraocular pressure (IOP) due to elevated corticosteroids, exophthalmos, hypertensive retinopathy, neuro-ophthalmologic findings due to pituitary tumors.
- **Diabetes Mellitus (DM):** Diabetic retinopathy, papillopathy, cataract formation, oculomotor paralysis.
- **Hyperparathyroidism:** Band keratopathy, conjunctival/scleral calcifications.
- **Hypoparathyroidism:** Cataract in 60% of cases; conjunctivitis, keratitis, papilledema (due to increased ICP).
- **Hyperthyroidism:** Eyelid involvement—retraction, lid lag, reduced blinking; exophthalmos; extraocular muscle involvement; exposure keratopathy; compressive optic neuropathy.
- **Hypothyroidism:** Eyelid edema (due to mucopolysaccharide accumulation); eyelash loss.
- **Pheochromocytoma:** Hypertensive retinopathy findings.

Metabolic Diseases:

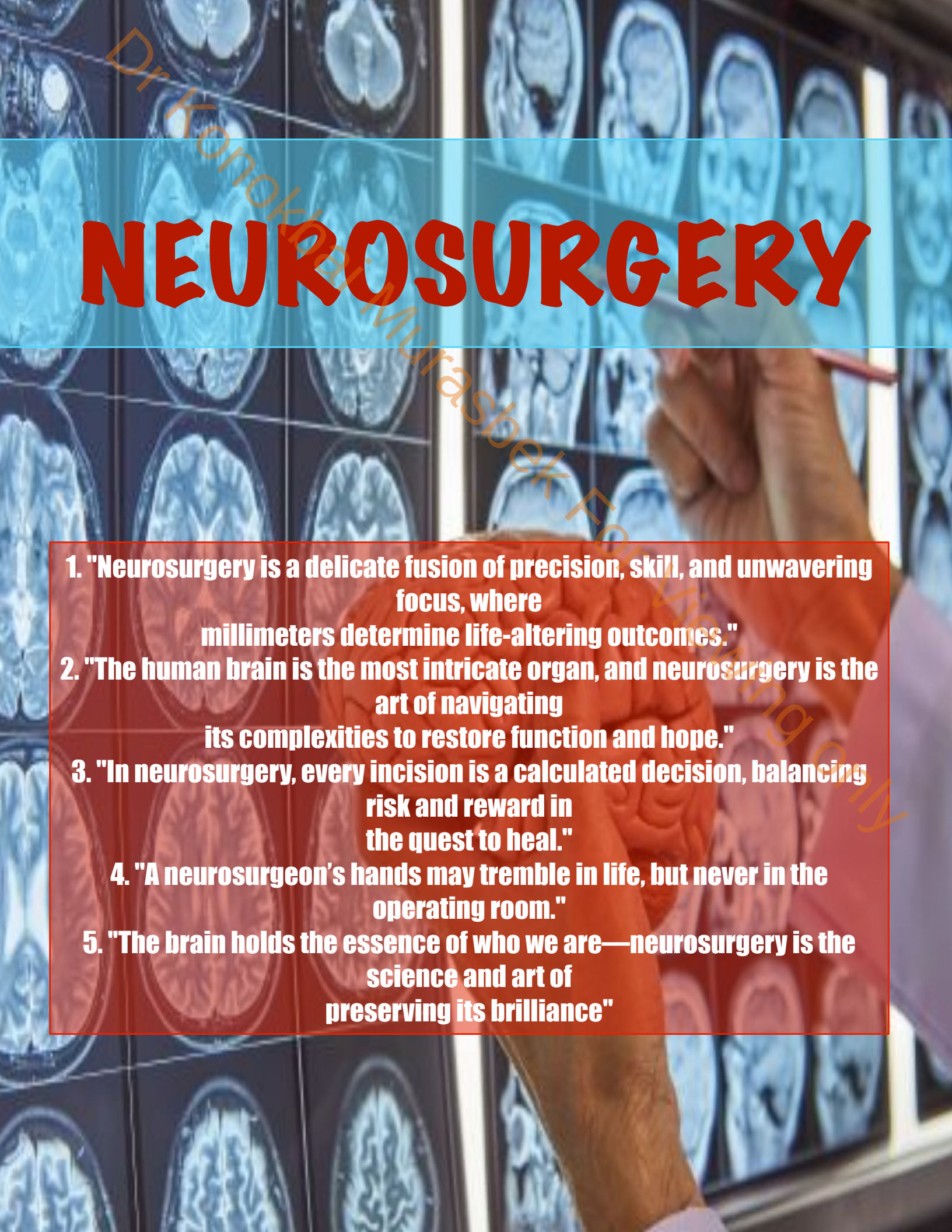
- **Galactosemia:** Causes congenital cataract, which can be prevented with an appropriate diet.
  The corneal endothelium plays the most crucial role in maintaining corneal transparency.
- **Albinism:** Albino fundus, macular hypoplasia, nystagmus, VEP abnormalities.
- **Congenital Lipidoses:** Lipemia retinalis, giving retinal vessels a milky-white appearance.
- **Mucopolysaccharidoses:** Pigmentary retinopathy, optic atrophy.
- **Niemann-Pick disease:** Cherry-red spot in the macula.
- **Roth spots**: Defined as a central white or dirty yellow lesion surrounded by a red hemorrhagic border. These Roth spots are most commonly seen in severe anemia, leukemia, and subacute bacterial endocarditis.

Skin and Mucous Membrane Diseases

- **Angioid Streaks**: These are characteristic cracks in Bruch's membrane, located between the retinal pigment epithelium and the choroid. They appear as streaks radiating in multiple directions from the optic disc, resembling retinal vessels, and suddenly terminate toward the equator. They are commonly seen in **Ehlers-Danlos syndrome, Pseudoxanthoma elasticum, and Paget's disease**.
- **Phakomatoses**: These are hereditary disorders that affect the **central nervous system, skin, and eyes** together.
- **Neurofibromatosis**:

- **NF1 (von Recklinghausen's disease)**: Associated with **plexiform neurofibroma of the eyelid, optic nerve glioma, orbital tumors** (such as schwannoma, plexiform neurofibroma, meningioma), **spheno-orbital encephalocele, bilateral Lisch nodules (hamartomas), congenital ectropion uveae, prominent corneal nerves, glaucoma, choroidal nevus, and choroidal hamartoma**.
- **NF2**: Characterized by **presenile cataracts, epiretinal membrane, and ocular motor defects**.

- **Tuberous Sclerosis (Bourneville's Disease)**: Involves **astrocytic hamartomas** originating from astrocytes in the posterior pole, appearing as **white, mulberry-like benign tumors**. In addition to **fundus astrocytomas, iris hypopigmentation, and atypical iris colobomas** may be observed.
- **Sturge-Weber Syndrome**: Associated with **ipsilateral glaucoma, episcleral hemangioma, iris heterochromia, and diffuse choroidal hemangioma**.
- **Von Hippel-Lindau Disease**: Characterized by **retinal capillary hemangioblastomas**.



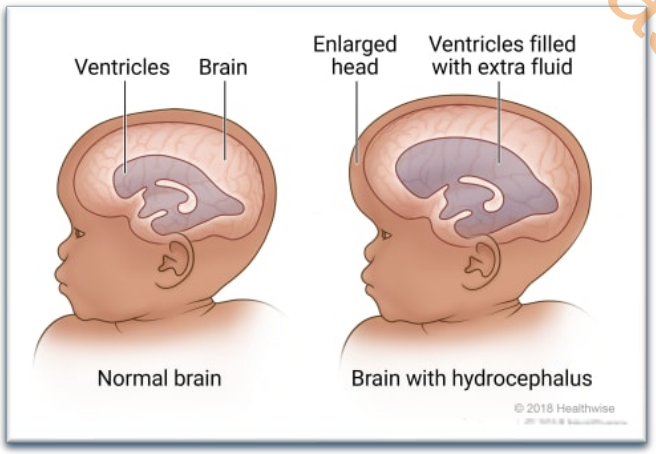
NEUROSURGERY

1. "Neurosurgery is a delicate fusion of precision, skill, and unwavering focus, where millimeters determine life-altering outcomes."
2. "The human brain is the most intricate organ, and neurosurgery is the art of navigating its complexities to restore function and hope."
3. "In neurosurgery, every incision is a calculated decision, balancing risk and reward in the quest to heal."
4. "A neurosurgeon's hands may tremble in life, but never in the operating room."
5. "The brain holds the essence of who we are—neurosurgery is the science and art of preserving its brilliance"

Unit : 1

DISEASES RELATED to CSF (Cerebrospinal Fluid)

HYDROCEPHALUS



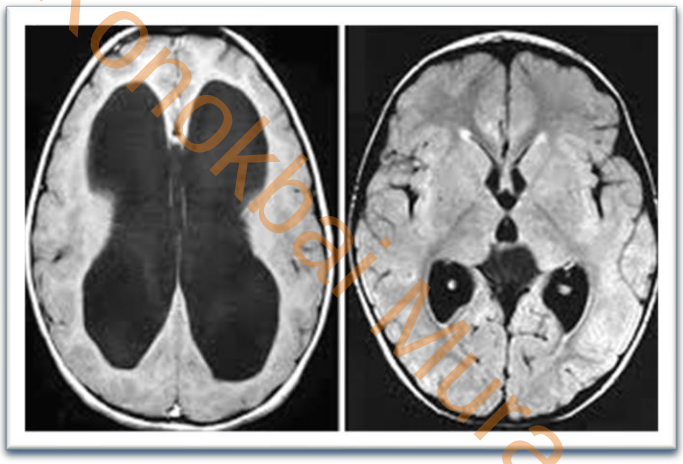
CSF is a physiological third compartment fluid produced at a rate of 20 mL per hour (500 cc/day). It is primarily secreted by the choroid plexuses of the lateral, third, and fourth ventricles. CSF flows from the ventricular system into the subarachnoid space through the Magendie and Luschka foramina.
Absorption: CSF is absorbed into the venous system via the arachnoid granulations located in the superior sagittal sinus.
It is usually caused by an increase in CSF volume and impaired absorption, less commonly by excessive secretion.

Hydrocephalus: Characterized by an increased CSF volume and dilation of cerebral ventricles.

Ex Vacuo Hydrocephalus: Occurs due to cerebral atrophy with an increase in CSF volume in the empty spaces, but intracranial pressure remains normal.

Symptoms and Signs (Adults)

1. Headache, lethargy
2. Incoordination, weakness
3. Cranial nerve palsies (Abducent nerve paralysis, which is more susceptible due to the long intracranial course of this nerve).



4. Corticospinal findings **MRI** is the best diagnostic method. **Treatment**: A shunt is applied. **Shunts** consist of a proximal ventricular catheter, a unidirectional valve, and a distal catheter. The most common cause of shunt obstruction is the coroid.

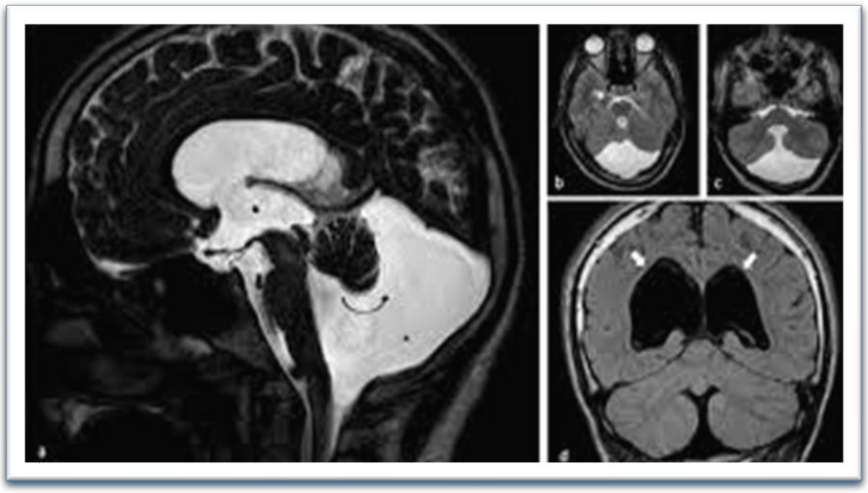
Hydrocephalus Causes

- **Congenital**: Dandy-Walker syndrome, Arnold-Chiari malformation, primary aqueductal stenosis
- **Acquired**: Infectious (the most common cause of communicating hydrocephalus), post-hemorrhagic (the second most common cause), secondary to mass lesions

Hydrocephalus Classification

1. **Obstructive (Non-communicating)**: There is a block at the level of the arachnoid granulations.
2. **Communicating**: Blockage occurs at the level of the arachnoid granulations.

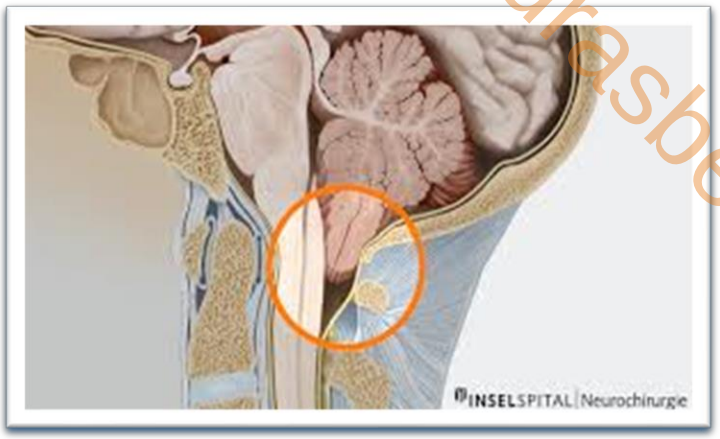
Dandy-Walker Syndrome



- There is atresia of the Foramen Magendie and Luschka.
- Cystic dilation of the fourth ventricle and enlarged posterior fossa with hypoplastic cerebellar hemispheres, a hypoplastic or agenetic vermis.
- Dilation of the lateral and third ventricles.

- Hydrocephalus develops in 90% of cases.
- Agenesis of the corpus callosum and occipital encephalocele are present in 17% and 7% of cases, respectively.
- May be associated with syringomyelia.
- Symptoms include ataxia, spasticity, and weakness in motor control.
- Seizures may accompany.

Arnold-Chiari Malformation



There are four types:

- **Type 1**: The cerebellar tonsils are located below the foramen magnum, while the medulla oblongata does not shift.
  - Often asymptomatic.
  - Syringomyelia and hydromyelia are common in 50% of cases.
  - The most frequent symptom is headache, which may be triggered by neck flexion or Valsalva maneuvers.
  - Lhermitte's sign and bilateral spasticity in the lower extremities can occur.
- **Type 2**: Part of the cerebellar vermis and brainstem extend below the foramen magnum.
  - Associated with syringomyelia, spina bifida, meningomyelocele, and other anomalies.
- **Type 3**: The most severe type, associated with suboccipital encephalomeningocele, where all posterior fossa structures displace below the foramen magnum.
- **Type 4**: Cerebellar hypoplasia.

Klippel-Feil Syndrome

- Caused by fusion of cervical vertebrae.
- The main clinical features are short neck, low hairline, and restricted neck movement.

Childhood Hydrocephalus

- Intraventricular hemorrhages
- Ventriculitis
- Colloid cysts of the third ventricle
- Suprasellar masses (blockage of Foramen Monro)
- Pineal region tumors (pressure on the aqueduct)
- Aqueductal stenosis
- Atresia of Foramen Magendie and Luschka (Dandy-Walker syndrome)
- Tumors, meningitis, subarachnoid hemorrhage
- Arnold-Chiari syndrome

Main Finding: Enlargement of the head circumference.

- Fontanelles become prominent, bulging, and tense.
- Venous distension in the head and face becomes evident.
- Apneic episodes and irregular breathing may occur.
- **Cranial sutures** may separate.

•Increased intracranial pressure leads to a downward gaze (sunset eyes) - also known as the "sunset sign".

- In older children/adults with rigid skulls:
  - Symptoms of increased intracranial pressure: papilledema, headache, nausea, vomiting, and gait disturbances.
  - Spastic gait, increased deep tendon reflexes, and upper motor neuron signs.
  - Endocrine and mental disturbances, including diabetes insipidus, precocious puberty, and hypothalamic signs.
  - Cranial nerve palsies (most commonly involving the 3rd and 6th cranial nerves).
  - Vision loss due to optic nerve compression, nystagmus, drowsiness, seizures, and irritability may develop.
- In patients with closed fontanelles, the clinical picture consists only of increased intracranial pressure syndrome.

Mc-Even Sign: A "cracked pot" sound is heard when tapping on the head.

Adams-Hakim Syndrome (also known as Normal Pressure Hydrocephalus)

- Usually occurs around the age of 60.
- Characterized by three main symptoms:
  1. Gait disturbance/ataxia
  2. Mental changes (dementia)
  3. Urinary incontinence
- Gait disturbance is usually the first symptom and progresses over weeks to months.
- More commonly causes subcortical dementia.
- CT and MRI are used for diagnosis.
- In imaging, ventricular enlargement is seen without cortical atrophy (important to distinguish from Alzheimer's).
- A typical finding is the improvement in gait within hours after removing 25-40 cc of CSF.
- Although there may be some improvement in dementia and incontinence after CSF removal, this is not a definitive finding.

- Treatment: Shunt placement.

Increased Intracranial Pressure (ICP) Syndrome

- The intracranial cavity contains three elements:
    - 80% brain parenchyma,
    - 10% CSF,
    - 10% blood.
- Any change in one of these elements or the addition of foreign masses in the intracranial space is compensated by the displacement of CSF or blood. This principle is known as the **Monro-Kellie-Burrows Doctrine**.

ICP Etiology:

- Increased brain volume (edema and space-occupying lesions)
- Increased brain blood volume (hypertension, sinus thrombosis)
- Increased CSF volume
- Pseudotumor cerebri
- Craniosynostosis

Clinical Signs

1. **Headache**: Most common symptom of increased ICP.
2. **Vomiting**: Often occurs in the morning.
3. **Papilledema**: Swelling of the optic disc due to increased intracranial pressure. If untreated, can lead to secondary optic atrophy and blindness.
4. **Altered Consciousness**: Appears later in the disease course.

Other Findings

- **Diplopia**: Caused by pressure on the cranial nerves III, IV, and VI.
- **Cushing's Triad**: Hypertension, bradycardia, and respiratory slowing occur when ICP increases significantly.
- **Gastrointestinal**: Gastric ulcers and gastrointestinal bleeding.
- **Pulmonary**: Hemorrhagic pulmonary edema.
- **Neurological**: Ischemic changes may occur due to increased ICP.

Radiological Findings

- Erosion of the sella turcica is a common finding in increased ICP.

- In children, widening of cranial sutures can be seen.

Treatment of Increased Intracranial Pressure

- The most effective treatment is to address the underlying etiology.
- Blood pressure should be maintained within normal limits, and normal arterial oxygenation and normothermia should be ensured.
- The patient's head should be slightly elevated (Semi-Fowler's position).
- Normoventilation should be maintained.
- Hypothermia can reduce cerebral metabolism and decrease intracranial pressure.
- Hyperthermia increases intracranial pressure.
- Mannitol infusion: Can be used for 24-48 hours, but may cause fatal increases in blood pressure and acute tubular necrosis.
- **Steroids** (e.g., dexamethasone) can help reduce swelling.
- Sedation is required to prevent excessive movements that can worsen increased intracranial pressure.

Pseudotumor Cerebri (Idiopathic Intracranial Hypertension)

- Increased intracranial pressure without a space-occupying lesion.
- Most often idiopathic.
- More common in women.

Associated Conditions

- Obesity
- Recent significant weight gain
- Systemic hypertension
- Hypervitaminosis A, vitamin A deficiency
- Hypothyroidism, hyperthyroidism
- Pregnancy, oral contraceptive use
- Tetracycline, lithium
- Symptoms mimic those of increased ICP.
- Headache worsens with bending forward and Valsalva maneuvers.

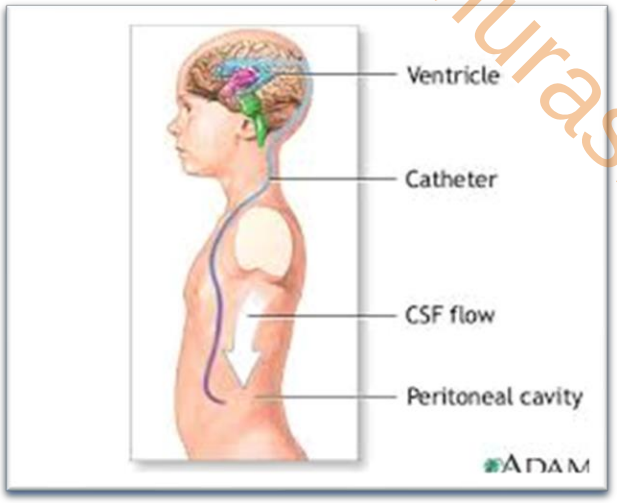
Visual disturbances: Decreased visual acuity is a prominent symptom, and secondary optic atrophy can lead to vision loss.

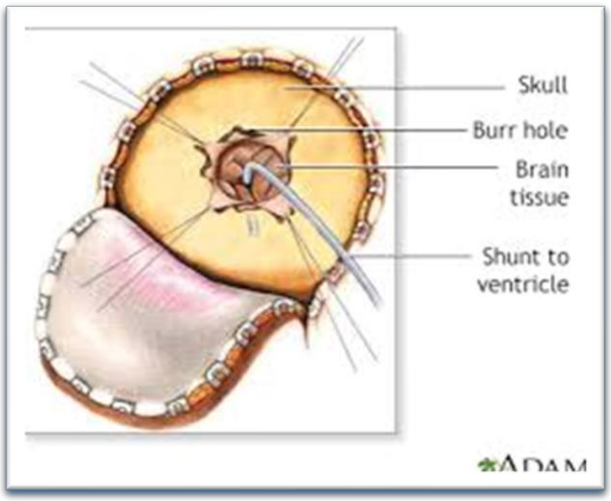
Diagnosis:

- After a visual examination, MRI should be performed.
- The diagnosis is confirmed if CSF pressure exceeds 200 mm H2O during lumbar puncture.

Medical Treatment:

- Fluid and salt restriction, diuretics, and short-term steroid use.
- In resistant cases, a ventriculo peritoneal shunt may be necessary.

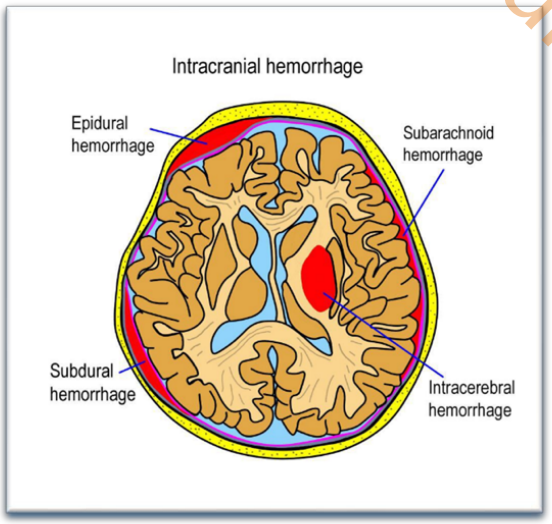




Unit : 2

BRAIN BLEEDING, HEMATOMAS, AND HERNIATIONS

INTRACEREBRAL HEMORRHAGE (ICH)



- **Hemorrhage Locations:**
  - Common sites for hemorrhages include the basal ganglia (especially the putamen), thalamus, pons, cerebellum, and white matter.
- **Etiology:**
  - It is the second most common cause of stroke.
  - Age is one of the most significant risk factors.
  - More common in men.
  - Half of the hemorrhages are located in the basal ganglia (especially the putamen).
  - Hypertension is the most common cause.
  - Other causes include vascular surgery, congenital heart disease in children, physical factors (e.g., physical exertion, cold exposure), vascular anomalies, amyloid angiopathy (particularly in recurrent bleeds), tumors, coagulation disorders, etc.

Diagnosis

1. **CT Scan (non-contrast):**
   - The first diagnostic test.
2. **Magnetic Resonance Imaging (MRI):**
   - Not useful in the early stages.
   - It can be used for follow-up, angiopathic conditions, and vascular malformations.
3. **Angiography:**
   - Used to detect underlying vascular abnormalities.

Treatment

- Medical and surgical interventions are required based on the location and severity.

SUBARACHNOID HEMORRHAGE (SAH)

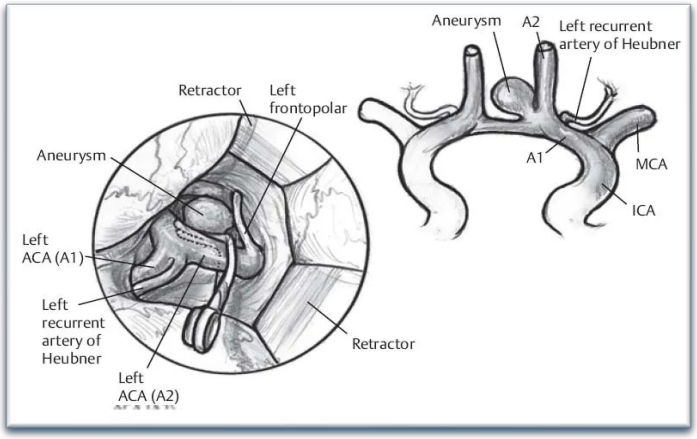
General Information

- The blood vessels in the brain are located in the subarachnoid space, and bleeding from these vessels or their aneurysms leads to subarachnoid hemorrhage (SAH).
- **Most Common Cause:** Traumatic injury to the head. In non-traumatic cases, the most common cause of spontaneous SAH is intracranial aneurysms.

Etiology and Clinical Features

1. **Common Cause:**
    - Most commonly caused by the rupture of an anterior communicating artery aneurysm.
    - The second most common cause is rupture of a posterior communicating artery aneurysm (which may cause pupil dilation).
    - AV malformations, bleeding disorders, tumors, hypertension, and anticoagulants can also lead to SAH.

Anterior communicating artery aneurysm

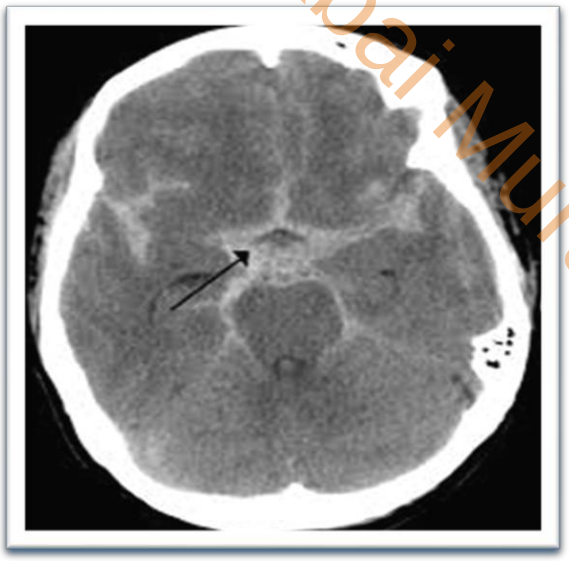


2. **Symptoms:**
    - Sudden onset of severe headache.
    - Temporary loss of consciousness and epileptic seizures may follow the headache.
    - Within 3-12 hours, meningeal irritation signs such as neck stiffness, Kerning's sign, and Brudzinski's sign may develop.
    - Fever is a common finding after bleeding.
3. **Prognostic Factors:**
    - Age, the amount of blood on CT scan, loss of consciousness during the event, clinical condition on hospital admission, and pre-existing hypertension or arterial disease are important prognostic factors.

- Decreasing consciousness within the first 2-3 days after SAH may suggest an intracerebral hematoma and requires emergency surgical intervention.

Diagnosis

1. **CT Scan (non-contrast):**



- This is the first diagnostic test and is highly effective within 72 hours of the event.

2. **Lumbar Puncture (LP):**
   - The most sensitive test for SAH.
   - Initially, erythrochromic CSF is seen, and later, xanthochromic CSF is typically observed.
3. **Angiography:**
   - Should be performed within the first 3 days to confirm the presence of an aneurysm or arteriovenous malformation (gold standard diagnostic test).

Treatment

- **Blood Pressure Management:**
  - Excessive lowering of blood pressure should be avoided as it can cause cerebral vasodilation, leading to increased risk of cerebral ischemia and infarction.
- Treatment is focused on addressing the underlying etiology.
- **Conservative Management:**
  - For idiopathic cases and those awaiting surgery.

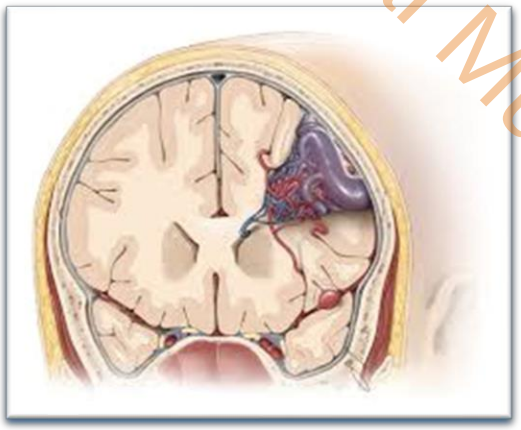
Complications Following SAH

- **Vasospasm:**
  - The leading cause of mortality and morbidity after SAH, typically occurring after the third day.
- **Hyponatremia and Hypovolemia:**
  - Common issues following SAH.
- **Cardiac Arrhythmias:**
  - Observed in about 60% of SAH cases.
- **Recurrent Bleeding:**
  - The risk of rebleeding is high, with 50% of untreated patients experiencing a rebleed within the first 6 months. Factors such as high clinical grade, large aneurysm size, high blood pressure, advanced age, and female gender increase the likelihood of rebleeding.
- **Hydrocephalus:**
  - Acute or chronic hydrocephalus can develop after SAH.

Conservative Treatment Principles:

1. **Rest:**
   - Strict bed rest is essential, with the patient placed in a quiet, dimly lit room, and their vital signs monitored closely.
2. **Pain Management and Sedation:**
   - Paracetamol and/or codeine are used for pain relief (aspirin should be avoided). Sedation with diazepam is often required. Severe symptoms may necessitate IV morphine or midazolam.
3. **IV Fluids:**
   - Early aggressive fluid management is essential to prevent cerebral salt loss.
4. **Antiepileptics:**
   - Phenytoin is often used.
5. **Calcium Channel Blockers:**
   - Nimodipine is given orally or via NG tube every 4 hours for the first 96 hours post-SAH to prevent cerebral vasospasm (although their success in this regard has been limited, they are used for neuroprotection).

6. **Triple H Therapy (3H):**
   - **Hypertension, hypervolemia,** and **hemodilution** to prevent vasospasm. Vasospasm refers to the narrowing of blood vessels, which can reduce blood flow to the brain and lead to ischemia (lack of oxygen), further exacerbating the injury caused by the hemorrhage. The goal of Triple H Therapy is to maintain adequate blood flow to the brain and prevent the development of ischemia .



Arteriovenous Malformation (AVM)

- AVM is the most common cerebral malformation.
- Those in the anterior circulation most commonly occur in the middle cerebral artery distribution.
- As the number of hemorrhages increases, mortality also increases.

Clinical

- Typically presents with hemorrhage, and rarely with epilepsy.
- Hemorrhage can be intracerebral, subarachnoid, or intraventricular.
- Hydrocephalus
- Pseudotumor cerebri
- In neonates, high-output heart failure may occur.

Venous Angiomas

- These are conditions involving only veins or sinuses, without arterial components.

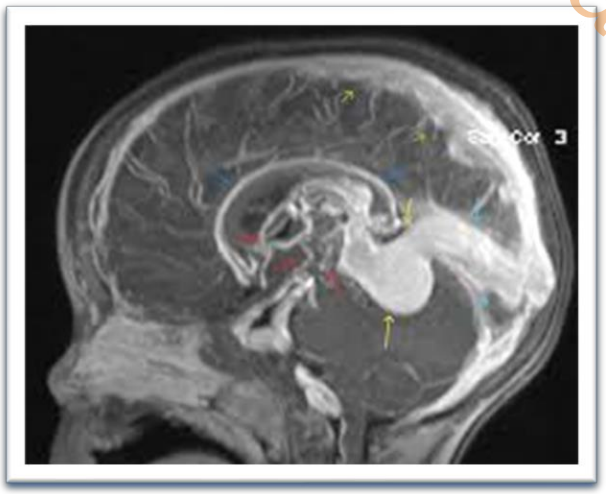
Cavernomas (Capillary Angiomas)

- These are very small, often calcified lesions.
- They are similar to oligodendrogliomas.

Capillary Telangiectasia

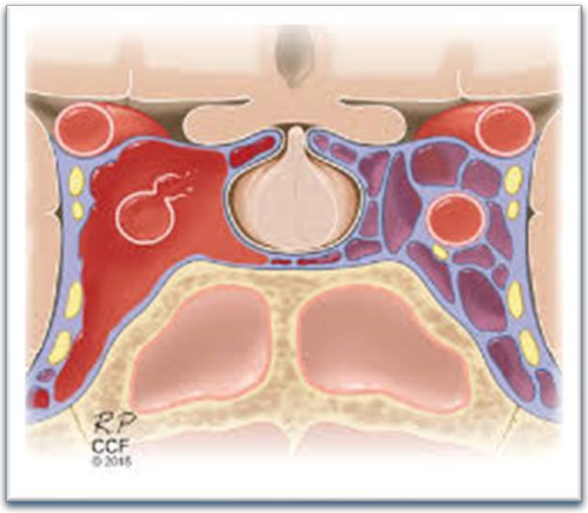
- A very small vascular malformation.
- These lesions are typically located around the fourth ventricle and pons.
- They are the most common cause of pontine hemorrhages.
- Diagnosis is made through histopathological examination of surgically obtained samples.

Galen Vein Aneurysm



- Arteries directly enter the Galen vein, causing aneurysmal dilation.
- This condition typically presents in the neonatal period.
- It may cause high-output cardiac failure, obstructive hydrocephalus, and subarachnoid hemorrhage.

Carotid-Cavernous Fistula



- This occurs after head trauma, when a fistula forms between the internal carotid artery and the cavernous sinus.
- The clinical presentation develops acutely compared to cavernous sinus thrombosis.

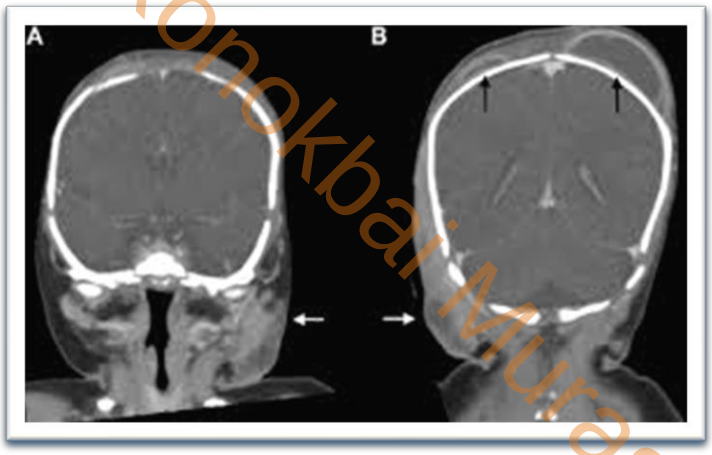
- Symptoms include:
    - Pulsatile exophthalmos and a bruit (a whooshing sound)
    - Prominent facial veins
    - Periorbital edema and conjunctival congestion
    - Papilledema, retinal hemorrhage, opacities in the lens and cornea, optic atrophy, and vision defects
    - Ophthalmoplegia (eye muscle paralysis)

Head Trauma

- **Hemotympanum, rhinorrhea, otorrhea, bilateral orbital ecchymosis (raccoon eyes)** should raise suspicion for anterior skull base fractures.
- In orbital fractures, the preferred diagnostic method is a direct X-ray in the Caldwell position, while the best imaging method is CT.
- **Cerebral concussion** refers to cognitive impairment, headache, memory disturbances, and syncope that occur secondary to head trauma (usually minor). It can be differentiated from contusion by the lack of significant imaging findings.
- **Cerebral contusion** usually involves petechial hemorrhages due to the physical distortion of brain tissue, which may also be accompanied by edema and bleeding.

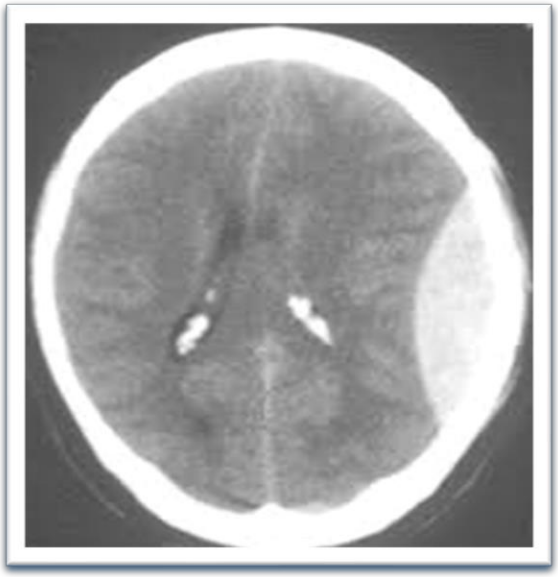
Scalp Hematoma

- **Subperiosteal Hematoma**:
    - Blood accumulation between the cranium and periosteum.
    - Can occur unilaterally or bilaterally, typically in the parietal region.
    - Does not cross suture lines.



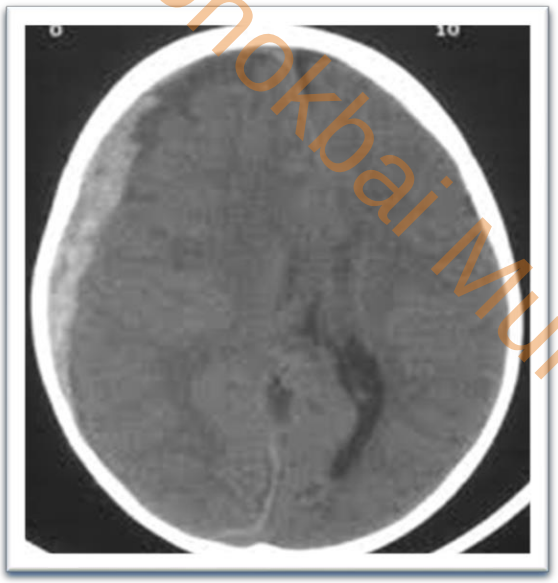
- **Subgaleal Hematoma:**
  - Blood accumulation between the galea aponeurotica and periosteum.
  - Tends to spread beyond suture lines.
  - Up to 250 cc of blood can accumulate, potentially causing hypovolemia in infants.
  - Local cold application is used; it generally resolves in 2-3 weeks.

Epidural Hematoma



- **Epidural Hemorrhage and Associated Fracture**:
  - Blood accumulates between the cranium and dura mater.
  - Fractures are present in 90% of cases.
  - Most commonly occurs due to bleeding from the middle meningeal artery, which originates from the bone fracture edges.
  - Typically occurs with temporal bone fractures.
  - Epidural hematoma is the most urgent type of intraserebral hemorrhage.
  - After trauma, patients may be conscious for a brief period (lucid interval) before losing consciousness.
  - Seizures, increased intracranial pressure (e.g., vomiting, bradycardia, headache) may be observed.
  - If not drained quickly, it may lead to intraserebral herniation.
  - Burr holes are opened, the hematoma is drained, and the bleeding vessel is ligated.

Subdural Hematoma



- Blood accumulates between the dura mater and the arachnoid layer due to rupture of bridging veins.
- Can also develop after cerebral contusion or intraserebral hemorrhage.
- Seen in alcoholics, individuals with drug habits, and rarely in hypertensive patients.
- Venous bleeding is common.
- Most commonly affects the temporal, parietal, and frontal regions.
- One of the most common mass-effect lesions after head trauma.

- **Clinical Features**:
  - If consciousness is preserved, severe headache is present.
  - Focal neurological deficits may occur.
  - Vomiting, seizures, subfebrile fever in children, agitation, and signs of meningeal irritation may be present.
- **Treatment**:
  - Surgical intervention is required for hematomas thicker than 1 cm.
  - **Acute form**: Occurs within 24-72 hours. Mortality rates in acute subdural hematoma are between 90-90%.
  - **Chronic form**: Develops 3-10 days after trauma.
    - Chronic subdural hematomas are more common in infants, the elderly, and alcoholics.
    - They can develop weeks or even months after a minor trauma.
    - A membrane (neomembrane) forms from the dura mater.
    - Symptoms include memory loss, dementia, personality changes, fluctuating consciousness, increased intracranial pressure (Kernig's sign), hemiparesis, and hemiplegia.

Herniation Syndromes

- **Herniation** refers to the displacement of brain tissue between intracranial compartments. The intracranial space is divided into two main areas by the tentorium cerebelli: supratentorial and infratentorial (posterior fossa).
- **Subfalcine Herniation (Singular Herniation)**:
    - The medial portion of the frontal lobe (cingulate gyrus) is displaced across the falx cerebri.
    - The anterior cerebral artery is compressed.
    - There are no distinct clinical signs, but this is the most commonly detected herniation type radiologically.
- **Transtentorial Herniation**:
    - A supratentorial mass can push the uncus and hippocampus through the tentorial notch.
    - This can be divided into axial (central) herniation and uncial herniation.

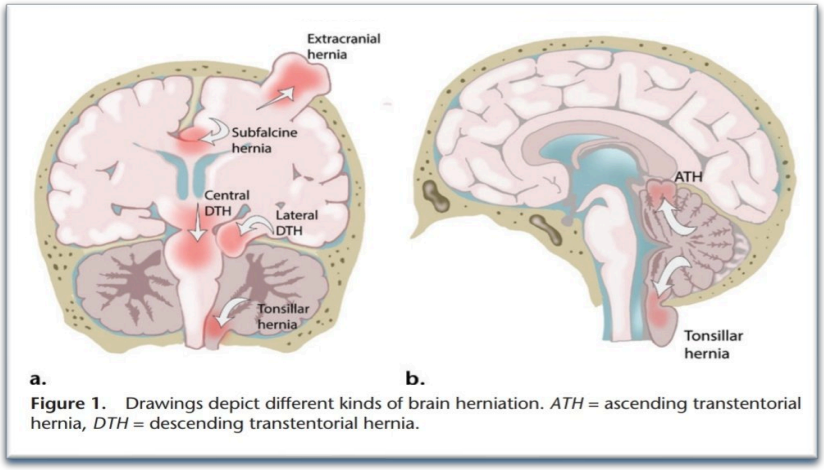
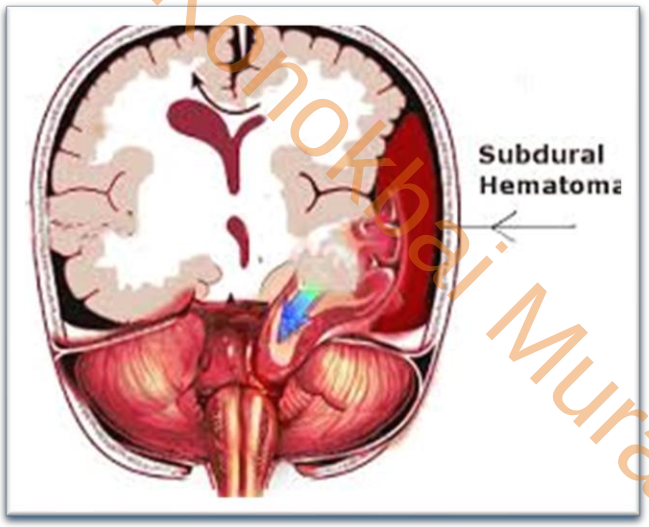


Figure 1. Drawings depict different kinds of brain herniation. *ATH* = ascending transtentorial hernia, *DTH* = descending transtentorial hernia.

1. **Axial (Central) Herniation:**

- Seen in lesions located in the upper parts of the cerebral hemispheres.
- Can occur in bilateral chronic subdural hematomas.
- Clinical features include bilateral mydriasis, limited upward gaze, and Cheyne-Stokes respiration.



2. **Uncal Herniation (Lateral Transtentorial Herniation):**

- The most symptomatic type of herniation.
- Caused by supratentorial masses.
- The posterior cerebral artery and 3rd cranial nerve get compressed.
- Ipsilateral mydriasis (dilated pupil) develops.
- There may be contralateral pyramidal irritation, hemiparesis, and later, altered consciousness.
- As herniation progresses, the brainstem gets compressed at the edge of the tentorium, leading to hemiparesis on the same side as the lesion (Kernohan's sign).
- Transtentorial herniation can be associated with midbrain and upper pons hemorrhages, known as "secondary brainstem hemorrhages" or "Duret hemorrhages."

- **Tonsillar Herniation:**
  - Occurs when the cerebellar tonsils herniate through the foramen magnum.
  - Leads to brainstem compression.
  - Causes sudden loss of consciousness, sudden loss of respiration, and intermittent opisthotonus.
  - Loss of neck stiffness, gag reflex, and cough reflex can occur.
  - This is most commonly seen in cerebellar tumors.

Unit : 3

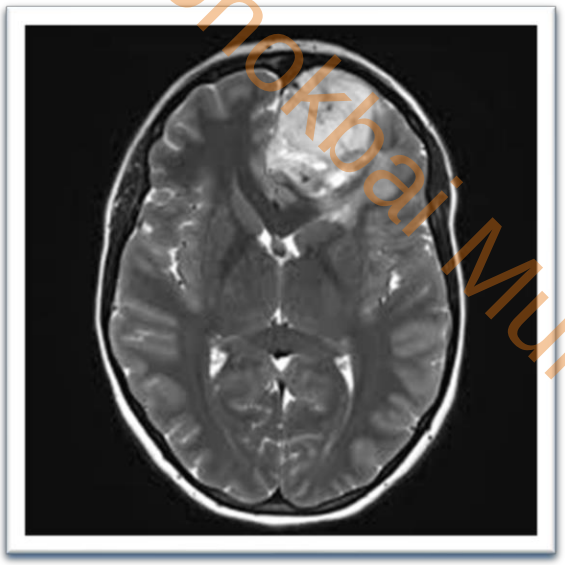
BRAIN TUMOR

ASTROCYTOMAS

Primary brain tumors' most common type is astrocytomas.

- **Pilocytic Astrocytomas (Grade 1)**
  - They are most commonly seen at a younger age compared to other astrocytomas.
  - Benign in nature.
  - Contains Rosenthal fibers and microcysts.
  - The brain tumor with the best prognosis.
  - The most common benign tumor in childhood.
- **Low-Grade Astrocytomas (Grade 1-2)**
  - Well-differentiated.
  - Commonly seen in individuals aged 30-50 years.
  - May undergo malignant transformation.
- **Anaplastic Astrocytoma (Grade 3)**
  - Less malignant than glioblastoma.
  - No necrosis present.
- **Glioblastoma (Grade 4)**
  - Commonly seen in individuals aged 50-60 years.
  - Necrosis, mitotic pleomorphism, and neovascularization are present.
  - The most malignant and most common primary brain tumor.

OLIGODENDROGLIOMA

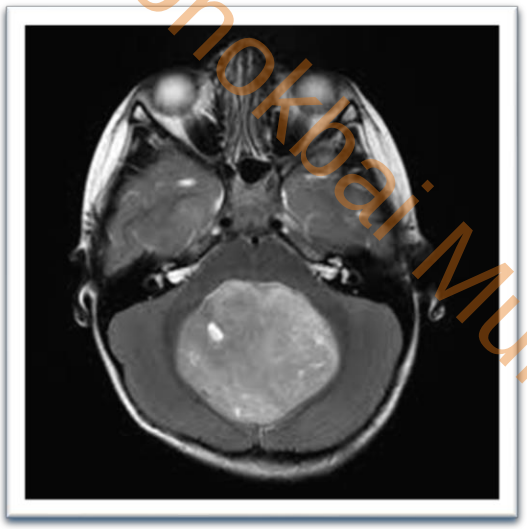


- The average age of occurrence is 40.
- Most commonly located in the frontal lobe.
- Grows slowly, leading to long-lasting focal symptoms (focal seizures).
- 40% show calcification.
- Classic histological findings include "fried egg" (egg-shaped) cells and "chicken wire" vascular structures.
- Most are grade 2, but there are also grade 3 (anaplastic) forms.

EPENDYMOMA

- Most commonly seen in the spinal cord in middle-aged individuals.
- Most commonly seen in the ventricles in younger individuals.
- Usually shows an epithelial appearance with fibrillary structures.
- Rosette and pseudorosette formations are present.
- May spread to the cerebrospinal fluid (CSF) via dissemination.

MEDULLOBLASTOMA (Grade 4)



- Typically arises from the cerebellar vermis.
- **M**: The most common malignant brain tumor in childhood.
- Tends to spread through CSF.
- Highly radiosensitive.

MENINGIOMA (Grade 1)

- Benign tumors originating from the arachnoid granulations that grow slowly.
- Arise around venous sinuses and most commonly located parasagittally.
- May be multiple (as in type 2 neurofibromatosis).
- Causes hyperostosis of the bone above and swelling on the side of the bone.
- Histologically, vortex-like structures and psammoma bodies are present.
- Meningiomas may have progesterone receptors.
- Pregnancy and hormone therapy may cause rapid growth.

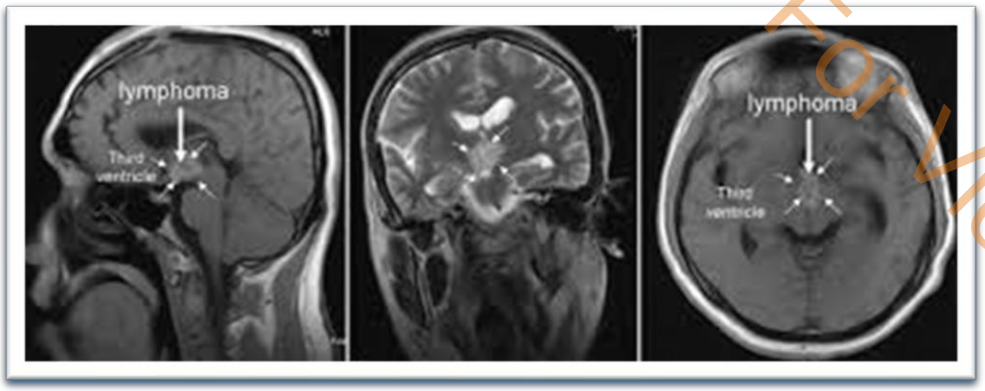
METASTASES

- 50% of all central nervous system tumors are metastases.
- The most common metastasis is from **lung cancer**, followed by **breast cancer**.
- Commonly found in the middle cerebral artery distribution.
- Prostate cancer does not metastasize to the brain but to the dura.

CHOROID PLEXUS PAPILLOMA

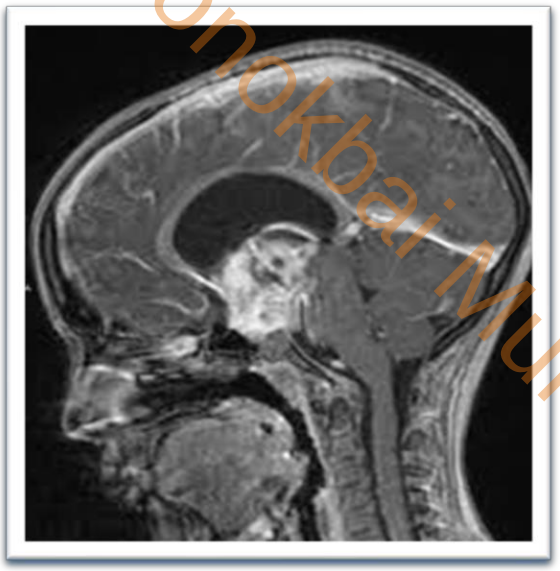
- Always shows a normal choroid plexus structure and produces cerebrospinal fluid, which may cause hydrocephalus.
- Most commonly found in the lateral ventricle in children, and in the 4th ventricle in adults.

PRIMARY BRAIN LYMPHOMA



- Most often seen in immunosuppressed individuals and those with AIDS.
- B-cell origin. Highly malignant and resistant to treatment.
- Epstein-Barr virus is found in about 30-50% of cases, especially in AIDS-related cases.

CRANIOPHARYNGIOMA

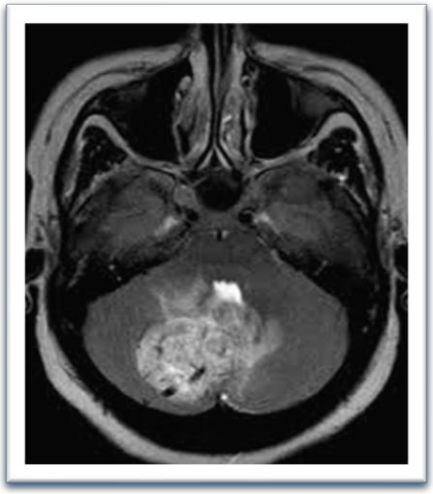


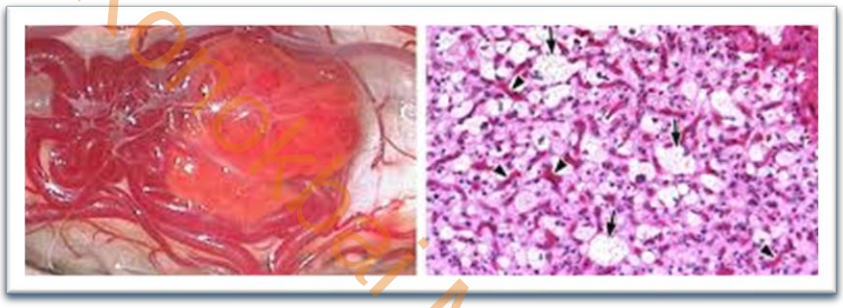
- Tumors located in the sellar-suprasellar region.
- Originates from Rathke's pouch.
- About half of the cases contain calcification, and nearly all contain cystic areas.

GERM CELL TUMORS

- Most commonly seen in the pineal and suprasellar regions.

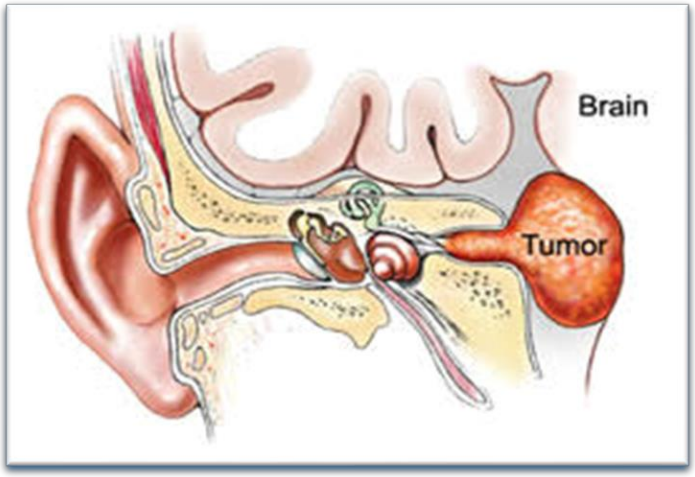
HEMANGIOBLASTOMA





- Causes polycythemia.
- Associated with von Hippel-Lindau syndrome.
- The most common primary cerebellar tumor in adults.

ACOUSTIC SCHWANNOMA

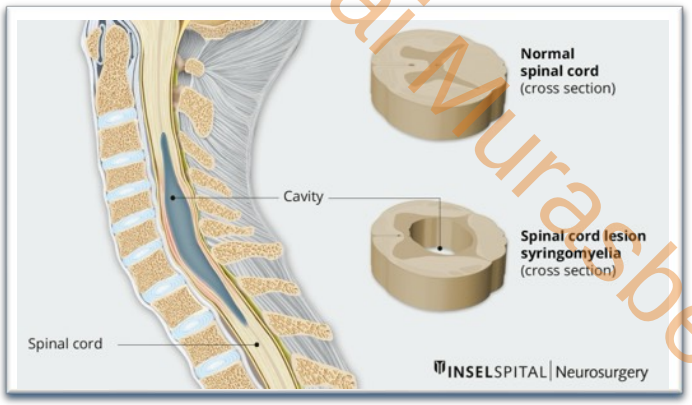


- Benign in nature.
- 90% of cerebellopontine angle tumors are acoustic schwannomas (most common) and meningiomas.
- Symptoms result from pressure on neurovascular structures (CN 5-6-7-8-9-10-11) in the angle.
- Initially presents with 8th cranial nerve findings. Later, there may be facial numbness (5th CN), and then weakness of facial muscles (7th CN).

Unit : 4

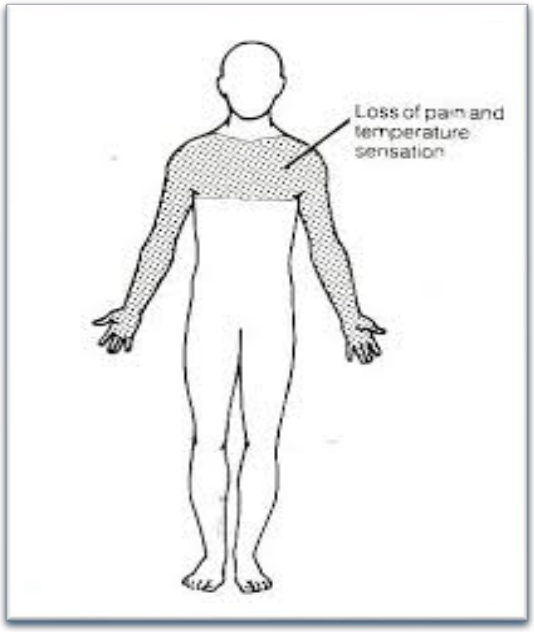
SPINAL CORD AND DISC DISEASES

Syringomyelia



- Typically characterized by cavitation filled with cerebrospinal fluid (CSF) in the cervical region.
- Can develop after spinal cord tumors, spinal cord hemorrhages, or trauma.
- If it progresses towards the brainstem, it is called syringobulbia.

Classic Symptoms:



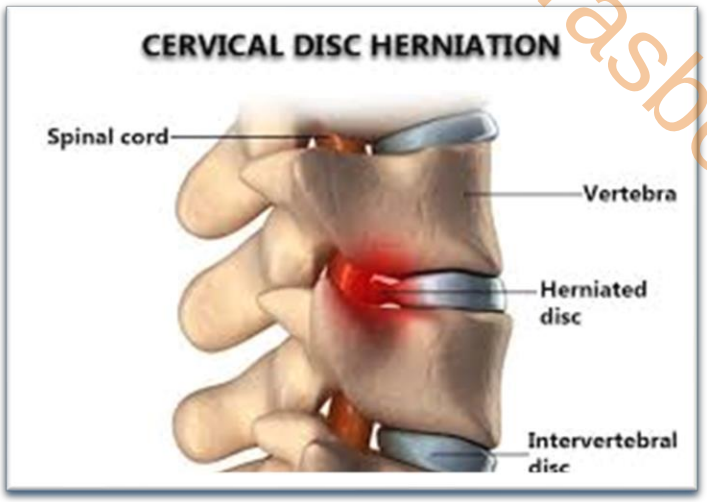
1. Dissociated sensory loss, where pain and temperature sensation are impaired but other sensations remain intact.
2. Segmental weakness and power loss in the hands and arms.
3. Loss of tendon reflexes in the arms.

- Horner syndrome may accompany (in the cervical segment).
- Developmental anomalies of the vertebral column may be present (kyphoscoliosis, Feil syndrome).
- Arnold-Chiari type 1 malformation is the most common syndrome associated with syringomyelia.

Hydromyelia (Central Canal Cavitation):

- Refers to the cavitation of the central canal, and is now used similarly or as a subgroup of syringomyelia.
- Diagnosis: MRI
- Treatment: Syringotomy or shunt placement

CERVICAL DISC HERNIATION



- There is no disc between the C1 and C2 vertebrae.
- The C8 nerve root exits between C7 and T1 vertebrae.
- Most common between C6-7, and less frequently between C5-6.
- Most cases peak in the 3rd and 4th decades and usually herniate laterally or dorsolaterally.
- Symptoms may include radiculopathy, myelopathy, or a combination of both.
- "Brachialgia" (painful syndrome) is often present, with pain radiating from the neck to the shoulder, forearm, and hand.
- Pain worsens with neck movements.
- Reduced neck lordosis.
- Limited head and neck movements.
- Muscle weakness corresponding to the affected root.
- Muscle atrophy in the same direction.
- Cervical disc herniation often compresses the foramen nerve root at the herniation level.

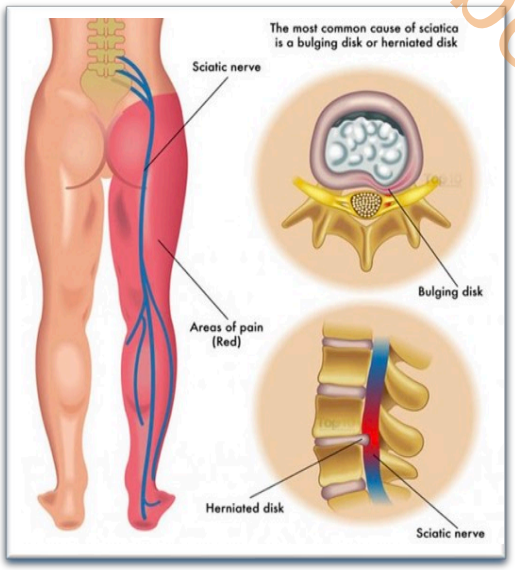
LUMBAR DISC HERNIATION

- The vertebral column consists of 33 vertebrae, with 23 discs between C2 and S1.
- Disc herniation involves the rupture of the annulus fibrosus and the protrusion of the nucleus pulposus.

- Most commonly seen in middle age (40-50 years).
- Most common at L5-S1, followed by L4-L5.
- The anterior and posterior longitudinal ligaments firmly attach to the disc, providing support.
- The anterior longitudinal ligament is very strong, while the posterior one is stronger in the middle and weaker on the sides.
- Therefore, herniation most commonly occurs posterolaterally.

Symptoms:

1. **Lower back and leg pain**: Initial localized back pain lasting 2-3 weeks. Pain worsens with prolonged positions, coughing, sneezing, defecation, or straining.
    - Flexing the knee and hip reduces pain.

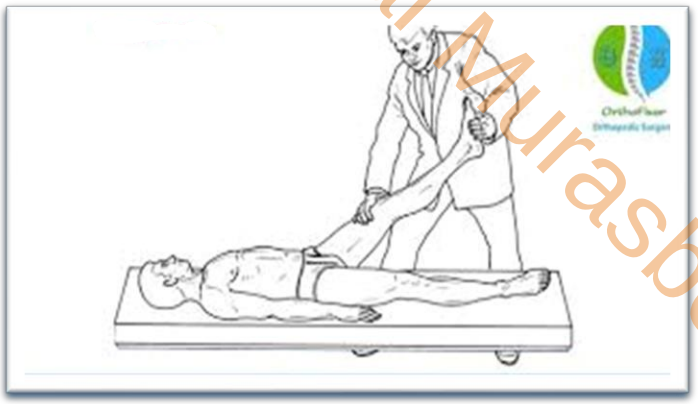


    - L5-S1 (sciatic nerve): Pain radiates from the lower back to the hip, posterior thigh.
    - L2-L4 (femoral nerve): Pain radiates from the lower back to the inguinal region and anterior thigh.
2. **Motor and reflex changes**:
3. **Sensory changes and numbness**:
    - L3: Round area over the medial knee.
    - L4: Below the knee, medial side.
    - L5: Below the knee, lateral side, dorsum of the foot.
    - S1: Sole and lateral foot.
    - S2: Back of the leg.
4. **Neurogenic claudication**:
    - Most common after the 5th decade.
    - Pain, tingling, and numbness increase with walking, often unilateral or bilateral, in the hip, thigh, or leg. Symptoms improve with sitting, squatting, or lying down.
5. **Bladder symptoms**:
    - Decreased bladder sensation is an early sign.
    - Irritative symptoms, including residual urine after micturition.

- Radiculopathy can also lead to less common symptoms like enuresis and dribbling incontinence.

Nerve Tension Signs:

- **Lasegue's test (Straight Leg Raise - SLR)**:



- In supine position, the affected leg is raised with the knee extended, and the hip is flexed.
- If pain or paresthesia occurs between 30°-70°, the test is positive, stretching the L5 and S1 nerve roots.
- No clinical significance if pain occurs beyond 70°.

- **Contralasegue Test**:
  - Lifting the unaffected leg in the same manner as the Lasegue test causes pain in the opposite leg.

- **Femoral Nerve Tension Test (Reverse Straight Leg Raise)**:
  - The patient lies on their side, with the leg flexed, and the hip is extended.
  - Pain in the thigh indicates a positive test, most commonly seen with L2-L3 or L4 nerve root compression.

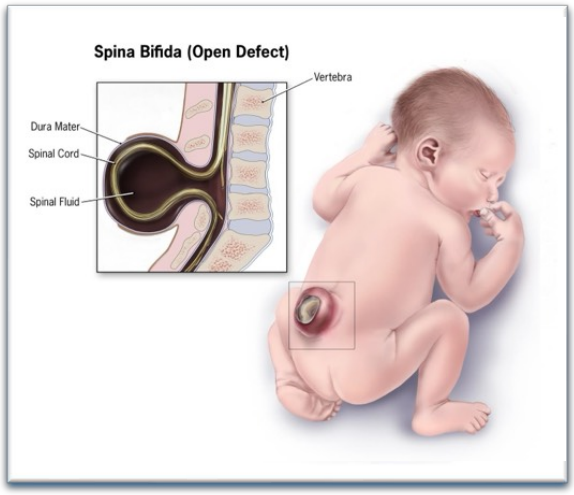
Surgical Indications:

1. **Cauda Equina Syndrome (Emergency Surgery Indication)**:
   - Can result from massive ruptured disc compression.
   - Often occurs in the midline, commonly at L4-5, and usually overlaid on a pre-existing condition (e.g., spinal stenosis, tethered cord).
   - May develop in the context of spinal stenosis or tethered cord syndrome.
   - Possible signs:
     - **Sphincter Dysfunction**: Urinary retention (most stable sign), urinary or fecal incontinence, decreased anal sphincter tone.

- **Saddle Anesthesia**: Seen in the upper hips, posterior-superior thighs, and perineal region.
- **Significant Motor Weakness**: Often affects multiple nerve roots.
- **Bilateral Loss of Achilles or Patellar Reflexes**.
- **Sexual Dysfunction**: Usually detected later.

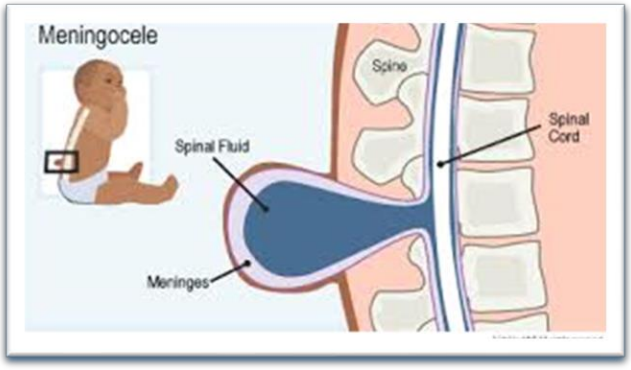
2. **Progressive or Acute Severe Motor Loss (Emergency Surgery Indication)**
3. **Failure to Respond to Medical Treatment**
4. **Recurrent Disc Herniations**

SPINA BIFIDA OCCULTA



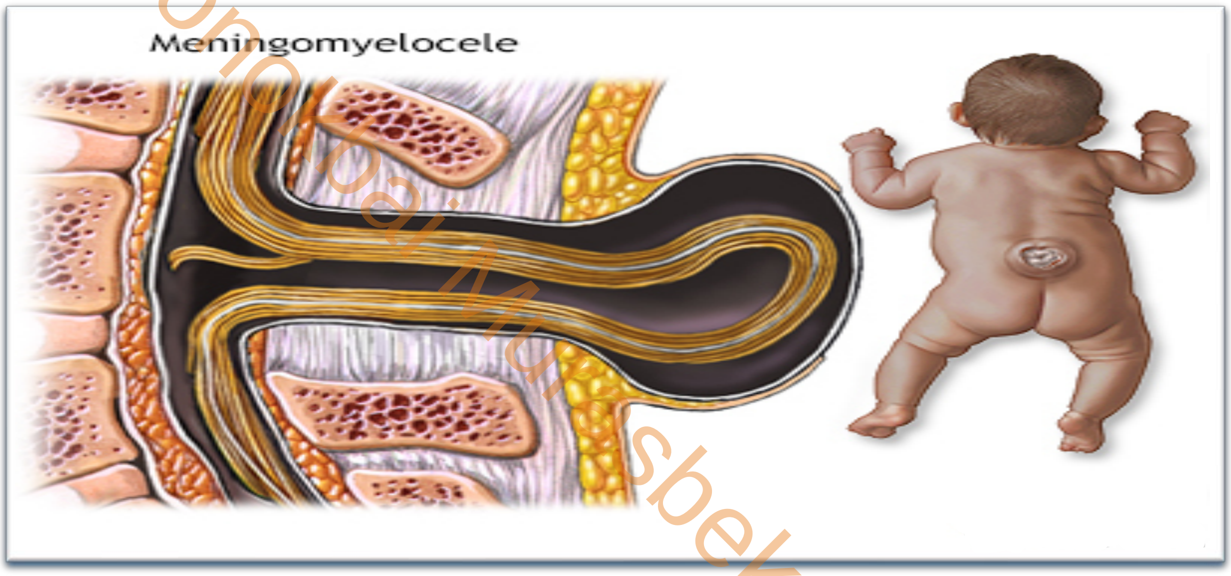
- A defect in the fusion of the spinous processes and laminae without involvement of the meninges or neural tissue.
- Approximately 80% are located in the lumbosacral region.

MENINGOCELE

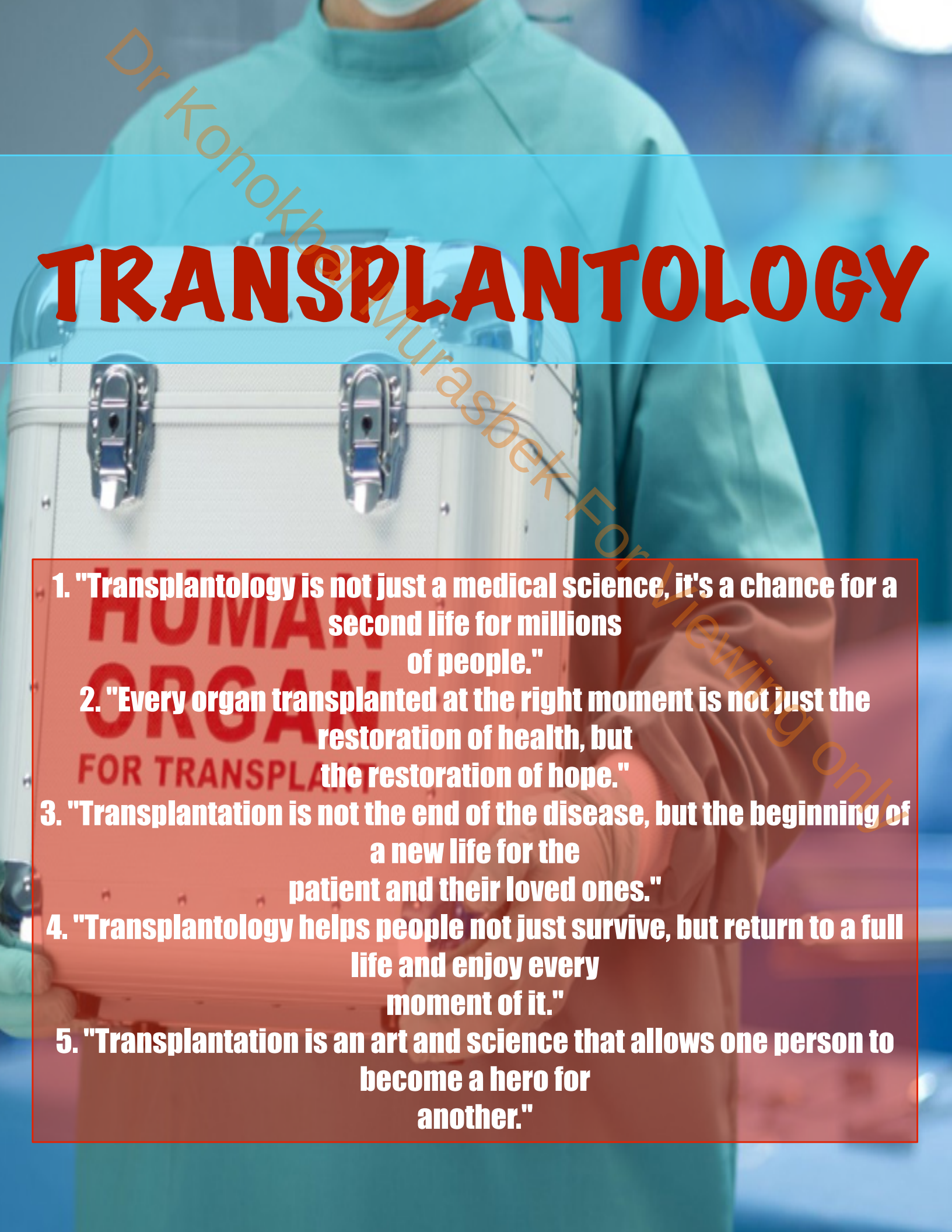


- A herniation of the dura and arachnoid membrane through a localized spina bifida defect in the spinal canal.
- The sac is filled with CSF, and no neurological deficit is present.
- Hydrocephalus may occasionally be present.

MENINGOMYELOCELE



- A condition where neural elements are contained within the meningocele sac.
- Commonly localized in the lumbar or lumbosacral regions.
- Congenital anomalies associated with meningomyelocele include:
    - **Chiari Type II Malformation**
    - **Aguaductus bifurcation**
    - **Hydromyelia**
    - **Hydrocephalus**



TRANSPLANTOLOGY

1. "Transplantology is not just a medical science, it's a chance for a second life for millions of people."
2. "Every organ transplanted at the right moment is not just the restoration of health, but the restoration of hope."
3. "Transplantation is not the end of the disease, but the beginning of a new life for the patient and their loved ones."
4. "Transplantology helps people not just survive, but return to a full life and enjoy every moment of it."
5. "Transplantation is an art and science that allows one person to become a hero for another."

Unit : 1

Nomenclature

- **Orthotopic transplantation**: When the organ is placed in its normal anatomical position (liver, heart).
- **Heterotopic transplantation**: When the organ is placed in a different location (kidney or pancreas).
- **Autotransplantation**: The transfer of tissue or an organ from one part of a person's body to another part.
- **Allotransplantation**: Transplants between genetically different members of the same species.
- **Xenotransplantation**: Transplants between different species.

History • The first kidney transplant research in humans was started in the 1940s by Yu Yu Voronoy, and the first long-term surviving graft transplant was performed by Joseph Murray in 1954. • The most important issue in transplantation is immune rejection, which results in necrosis of the transplanted tissue. This event is related to the recipient's white blood cells. • In transplant rejection, **lymphocytes** play the most important role. Various methods have been tried to prevent rejection and prolong graft survival, including the use of radiotherapy and 6-mercaptopurine. Later, the use of corticosteroids extended graft survival. • In the early 1960s, the immunosuppressive effect of anti-lymphocyte serum was discovered.

Tissue Compatibility Antigens (Major Histocompatibility Complex)

• Allograft rejection is caused by foreign histocompatibility (tissue compatibility) antigens present on the cell surfaces of the transplanted graft tissue. The strongest transplantation antigens are found in a single chromosomal region called the **"major histocompatibility complex (MHC)"**. MHC is a large gene complex that controls the properties influencing the entire immune response. • In humans, it is localized on chromosome 6. Since MHC gene products were first studied in leukocytes, they are referred to as **"human leukocyte antigens" (HLA)**.

CLINICAL REJECTION SYNDROMES

• Hyperacute, acute, and chronic...

• Only acute rejection is treatable.

• Hyperacute rejection is an event that cannot be treated but can be prevented.

• Chronic rejection is resistant to treatment.

HYPERACUTE REJECTION

• Occurs in recipients who have been previously sensitized to donor antigens.

• Develops within minutes to hours following graft reperfusion.

• Triggered by preformed antibodies against the donor's HLA and ABO antigens.

• The antibodies initiate a series of events leading to disseminated intravascular coagulation and ischemic necrosis of the graft.

• Hyperacute rejection is untreatable. However, it can be prevented by pre-transplantation tests.

• These tests include lymphocytotoxic crossmatch and ABO typing. Crossmatch is based on comparing the recipient's serum with the donor's lymphocytes in the presence of complement.

• Thanks to these tests, hyperacute rejection is no longer an issue.

ACUTE REJECTION

• The most common type of rejection; however, with modern immunosuppression, it is becoming increasingly rare.

• Typically develops within a few days or weeks.

• Classified as cellular (T-cell mediated), humoral (antibody-mediated), or combined.

• **Diagnosis:**

- Biopsy, special immunohistochemical stains
- Laboratory tests:
    - Kidney: Elevated creatinine levels
    - Liver: Elevated liver function tests (ALT,AST)
    - Pancreas: Elevated glucose, amylase, lipase

Treatment :

• Mild rejections are usually treated with high-dose methylprednisolone.

• Moderate or severe rejections are treated with antithymocyte globulin or monoclonal antibody OKT3.

• If these are unsuccessful, alemtuzumab, rituximab, or IVIG (immunoglobulin) may be tried.

CHRONIC REJECTION

• Develops slowly.

• Can be seen within the first year; however, it generally occurs after several years.

• Biopsy is useful for differentiating it from acute rejection. Histologically, it is characterized by less lymphocyte infiltration, atrophy, parenchymal fibrosis, and atherosclerosis.

IMMUNOSUPPRESSION

• **Induction Immunosuppression**: Very intensive immunosuppression applied in the early postoperative period. • **Maintenance Immunosuppression**: Long-term immunosuppression used to prevent acute rejection throughout life.

- When doses are well adjusted, these agents are well tolerated.
- However, they also increase the risk of infections and cancer.

• **Rescue Therapy**: Agents used to treat an acute rejection episode. They are the same as those used in induction.

Immunosuppressive Drugs by Group and Mechanism of Action

- **Calcineurin Inhibitors**
  - Cyclosporine A
  - Tacrolimus
- **Non-Calcineurin Inhibitors**
  - Sirolimus
- **Antimetabolites**
  - **Purine synthesis inhibitors**:
    - Azathioprine
    - Mycophenolate mofetil
  - **Pyrimidine synthesis inhibitors**:
    - Leflunomide
- **Biological Immunosuppression**
  - **Polyclonal Antibodies**:
    - ATGAM (antithymocyte globulin)
    - Anti-thymocyte immunoglobulin
  - **Monoclonal Antibodies**:
    - Muromonab-CD3
    - IL-2R (humanized)
    - Basiliximab (Anti-CD25 antibody, blocks IL-2 effect)

    - Belatacept
    - Alemtuzumab (Long-term lymphocyte depletion)
    - Rituximab (Anti-CD20, anti-B cell)
    - Bortezomib
    - Eculizumab (For humoral rejection, pre-transplant desensitization, N. meningitidis prophylaxis)

- **Others**:
    - Corticosteroids
    - JAK-3 inhibitors
    - Protein kinase C inhibitors (e.g., AEB)
    - Bortezomib (Against plasma cells, for humoral rejection)

• **Early Post-Immunosuppression Infections**: The most common surgical infections observed in the early period after immunosuppression. • **Severe Surgical Infections**: These are most commonly seen following pancreas and liver transplants.

Unit : 2

KIDNEY TRANSPLANTATION

• The most common solid organ transplantation.

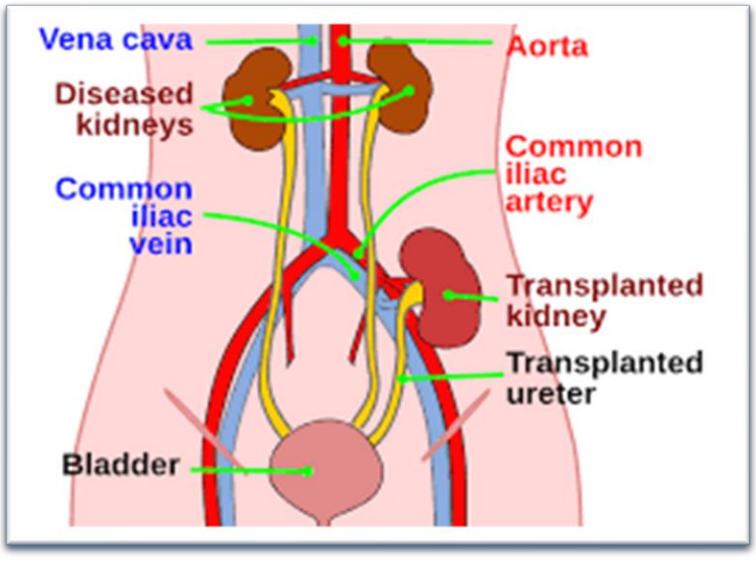
• Preferred treatment for chronic kidney failure.

Indications :End-stage kidney disease

Absolute Contraindications for Kidney Transplantation:

- Active cancer
- Active infection
- Severe peripheral vascular disease
- Severe cardiac or pulmonary disease
- Active intravenous drug abuse
- Severe psychiatric disorders preventing the patient from adhering to complex medical treatments

KIDNEY DONORS



Living Donors

• Relatives and non-relatives.

• In the USA, 25% of all kidney transplants come from living relatives.

• More successful than cadaveric transplants.

• In living donor transplants, compatibility is not the only important condition. The donor's health status must also be excellent.

Advantages of Living Donor Transplantation

- Better results.
- No long wait times or dialysis needs.
- Better early graft function.
- Desensitization can be performed on the recipient if ABO mismatch or HLA crossmatch is positive.
- No serious long-term morbidity for the donor.
- Minor risk factors like mild hypertension, proteinuria, and the potential for trauma to the remaining kidney.

Living Donor Contraindications

- BMI > 40 kg/m^2
- Diabetes
- Active cancer
- HIV positivity
- GFR < 70 mL/min
- Significant albuminuria
- Hypertension requiring multiple medications
- Horseshoe kidney or pelvic kidney
- Severe psychiatric disorder
- High risk of recurrent kidney stones (e.g., cysteine, struvite)

Cadaveric Donors

• If a living donor cannot be found, cadaveric transplantation is an appropriate alternative.

• Represents about 75% of kidney transplants.

ORGAN PRESERVATION

• The purpose of hypothermic organ preservation is to maintain organ viability during transportation, tissue typing, and recipient preparation.

• Hypothermia is the most critical stage of organ preservation.

• During hypothermia, metabolic functions, such as the Na-K pump, slow down, leading to cell swelling. Preservation solutions are designed to prevent swelling and improve preservation quality.

Methods of Kidney Preservation

- Cold storage and machine perfusion are the two main methods.
- Until 1987, most centers used **Collins** solution for cold storage. After 1987, the Wisconsin University (**UW**) solution, developed by Belzer, became commonly used for liver and pancreas, as well as kidney preservation.
- As 85% of donors provide multiple organs, **UW** solution became more common and gradually replaced Collins solution.

COMPLICATIONS

Technical Complications

- Rare (5-10%).
- Often manifest as decreased urine output.
- Some grafts may show delayed function, so decreased urine output is not always a reliable indicator of complications.
- Daily creatinine and hemoglobin tests are crucial in the first few days.
- **Beta-2-microglobulin** can be used to distinguish rejection from surgical complications.

Bleeding

- Presentation: Acute flank pain, a mass at the incision site, rapid drop in hematocrit and hemoglobin levels, hypertension due to renal parenchymal compression.
- Often, tachycardia is absent (most patients are on beta blockers).
- **Ultrasound (USG)** may help detect.
- Surgery: Usually, bleeding source is not found, and hematoma is drained.

Renal Vein Thrombosis

- 0.5-4% incidence.
- Usually occurs within the first week post-op.
- Presentation: Sudden hematuria and decreased urine output.
- **Ultrasound (USG)**: Diagnostic.
- Usually, the graft cannot be salvaged, requiring **nephrectomy**.

Renal Artery Thrombosis

- <1% incidence.
- Presentation: Sudden drop in urine output, lack of decrease in beta-2-microglobulin levels post-transplant period.

- **Ultrasound (USG)**: Diagnostic.
- Typically, the graft cannot be salvaged, and **nephrectomy** is performed.

Renal Artery Stenosis

- A late technical complication (1-23% incidence).
- Presentation: Generally asymptomatic with elevated creatinine, sometimes bilateral lower extremity edema, worsening hypertension.
- **MRI/CT angiography**: Diagnostic.
- **Balloon angioplasty**: Success rate 20-80%.
- ABO compatible cadaveric iliac artery graft for bypass.

Urological Complications

- Ureteral leakage, obstruction, or stenosis.
- The use of stents during implantation reduces these complications, but infection rates may rise.
- **Stenosis**: 2-15% incidence, caused by lymphocele pressure or ischemia in early stenosis, and by polyomavirus (BK virus) in late stenosis.
- **Ultrasound (USG)**: Hydronephrosis may be observed.
- **Leakage**: Severe pelvic pain.

Long-term Complications

• The most common causes of death after kidney transplantation are infections, cancer, and cardiovascular diseases.

• In the early period, infections are the most common cause of death.

• In the long term, cardiovascular diseases are the leading cause of death.

• The most common cause of graft loss after renal transplantation is the recipient's death from non-kidney-related causes (frequently cardiovascular diseases).

Infection

- Post-transplant infections are primarily opportunistic, including herpesvirus, CMV, and EBV infections.
- CMV infections are more frequent after immunosuppressive treatments using monoclonal (OKT3) or polyclonal (antilymphocyte globulin) antibodies.

Cardiovascular Disease

- The risk of ischemic heart disease increases 3-4 times after kidney transplantation.
- More frequent in diabetic, hypertensive, hypercholesterolemic, and smoking patients.

Malignancy

- The most common cancers are skin cancers (especially squamous cell carcinoma).
- Increased risk of Kaposi's sarcoma, vulvar, vaginal, and cervical carcinomas.
- Post-transplant lymphoproliferative disorder (PTLD): A group of B-cell abnormalities caused by EBV infection.

IMMUNOSUPPRESSION

- After cadaveric kidney transplants, immunosuppression is commonly managed with a triple regimen of azathioprine, cyclosporine, and prednisone. However, mycophenolate mofetil has replaced azathioprine in many protocols.

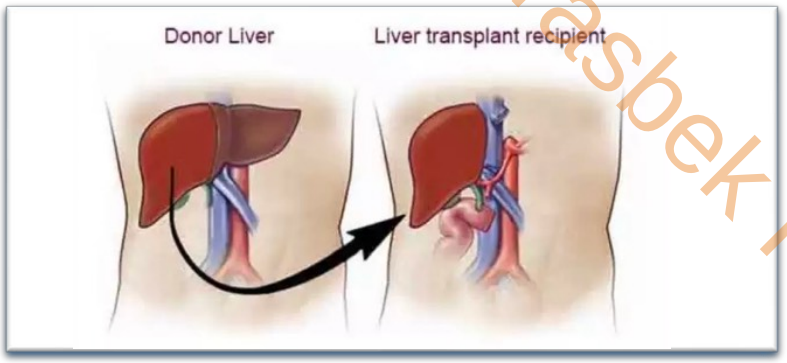
REJECTION TREATMENT

- High-dose steroids, typically methylprednisolone, are the first-line treatment for acute rejection.
- Rejection occurs in 40-50% of kidney recipients.
- Mycophenolate mofetil, cyclosporine, and prednisone reduce the incidence of rejection to 20-25%.
- If rejection does not respond to steroids within 1-2 days, OKT3 is effective in 90% of cases.
- In cases of steroid and OKT3-resistant rejection, tacrolimus may be used.

Unit : 3

LIVER TRANSPLANTATION

• Orthotopic or heterotopic transplantation. • Orthotopic liver transplantation is more commonly performed.



Indications

• Theoretically, all diseases leading to liver failure.

• The most common causes are chronic active hepatitis, primary biliary cirrhosis, sclerosing cholangitis, biliary atresia, and alcoholic cirrhosis.

• In adults, the most common indication is viral hepatitis.

• In children, the most common indication is extrahepatic biliary atresia.

• Chronic hepatitis C virus infection is currently the most common indication for liver transplantation in the Western world.

• Liver transplantation in patients with fulminant hepatic failure generally has worse outcomes compared to transplantation in chronic liver disease.

• In patients with post-hepatic cirrhosis due to hepatitis B or C, there is a risk of recurrence of cirrhosis in the transplanted liver.

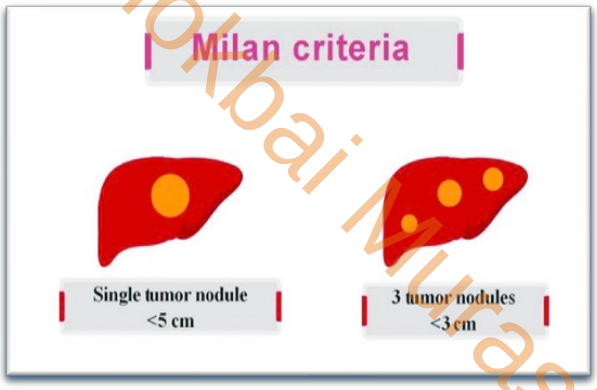
• Liver transplantation in cancer patients is controversial, and the outcomes are worse compared to benign diseases.

• In patients with hepatocellular carcinoma (HCC), resection is generally the first choice.

• However, these patients often have advanced cirrhosis, so resection may not be possible.

• If the Milan criteria are met, transplantation should be performed.

Milan Criteria (Transplantation prerequisites for HCC):



- A single tumor <5 cm.
- At most 3 tumors, each ≤3 cm.
- No vascular invasion.

Common Indications for Liver Transplantation

Adults:

- Viral hepatitis (HBV/C, acute and chronic non-ABV/C)
- Alcoholic cirrhosis
- Primary biliary cirrhosis
- Secondary biliary cirrhosis
- Secondary cholangitis
- α-1 antitrypsin deficiency
- Hemochromatosis
- Budd-Chiari syndrome
- Acute hepatitis B

Children:

- Biliary atresia
- Chronic active hepatitis
- Hepatoma
- Neonatal hepatitis
- Hepatic fibrosis
- Secondary biliary cirrhosis

CONTRAINDICATIONS FOR LIVER TRANSPLANTATION

- Insufficient cardiopulmonary reserve

- Uncontrolled cancer and infections
- **Alcoholism**
- Advanced age (>70 years) and HIV infection are not absolute contraindications
- ABO compatibility is required for liver transplantation, but it is not an absolute condition.
- ABO-incompatible liver transplantation can be successfully performed, but there is a higher risk of graft failure.

POSTOPERATIVE TREATMENT AND COMPLICATIONS

- Elevation in liver enzymes (especially GGT and alkaline phosphatase) and bilirubin levels are suggestive of rejection.
- The best method for detecting rejection is percutaneous liver biopsy.
- Rejection typically responds to bolus steroid therapy, but steroid-resistant rejections can be treated with antithymocyte globulin and OKT3.
- Maintenance immunosuppression mainly involves cyclosporine or tacrolimus.
- Standard surgical complications: bleeding, organ injury, wound infection.

Primary Non-Function

- Failure of the graft to function without a detectable cause.
- Occurs in 3-4% of cases.
- The only solution is retransplantation.
- Peak AST levels greater than 5000 IU/L are predictive of primary non-function.
- Factors associated with primary non-function:
    - Donor macrosteatosis
    - Prolonged cold and warm ischemia times
    - Prolonged donor hospital stay

Vascular Complications

- Occur in 8-12% of cases.
    - **Hepatic Artery Thrombosis**:
        - The most common vascular complication (1.6-4%).
        - Mortality rate is 50% (despite treatment).
        - Dramatic presentation: fulminant hepatic necrosis, transaminitis, fever.

        - Late presentation: asymptomatic or silent, cholangitis, bile leakage, liver abscess.
        - Diagnosis: Ultrasound (sensitivity and specificity >90%).
        - Emergency re-exploration may be required.
    - **Portal Vein Thrombosis**:
        - Very rare.
        - Symptoms: liver dysfunction, ascites, variceal bleeding.
        - Emergency thrombectomy may be needed.

Biliary Complications

- Occurs in 10-35% of cases.
- Clinical signs: fever, abdominal pain, biliary drainage from drains.
- Diagnosis: Cholangiography.
- Bile leaks or strictures:
    - Bile leak: reoperation and repair.
    - Stricture: radiological or endoscopic procedures.

Infectious Complications

- Intra-abdominal infections (consider bile leak).
- Fungal infections (can lead to graft dysfunction).
- Opportunistic infections (similar to other solid organ transplants) — these develop due to T-cell suppression.

Acute Rejection

- Seen in up to 20% of cases.
- Initial treatment is high-dose corticosteroids, which are usually effective. If ineffective, antithymocyte therapy may be required.

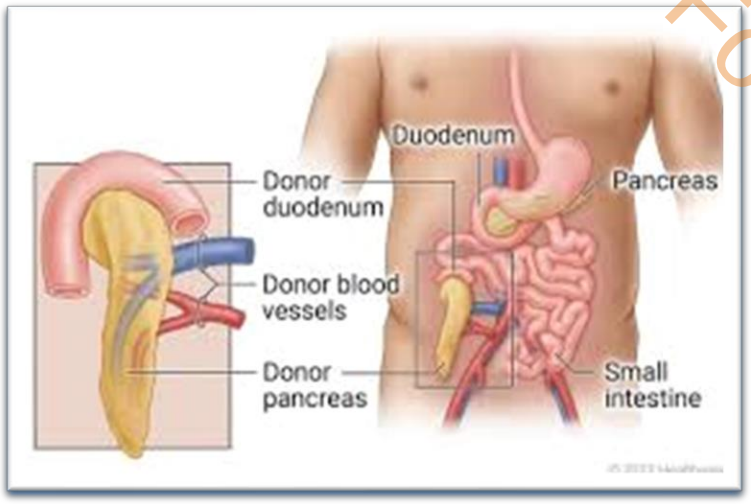
Unit : 4

PANCREAS TRANSPLANTATION

- The most successful method for restoring normal glucose homeostasis in Type 1 diabetes is pancreas transplantation.

Indications:

- Patients under the age of 45 with Type 1 insulin-dependent diabetes are potential candidates for pancreas transplantation.
- Long-term immunosuppression is required after transplantation, therefore untreated malignancy, active infection, and HIV positivity in recipient are contraindications.



Rejection:

- The most common non-surgical complication (30%).
- Diagnosis is made by elevated serum amylase and lipase levels.
- In cases where the pancreas is drained into the bladder, a decrease in urinary amylase levels confirms the diagnosis.
- The definitive diagnosis is made via percutaneous pancreatic biopsy.

Technical Complications:

- The risk of complications is higher in pancreas transplantation than in all other solid organ transplants.

- The most frequent complication is thrombosis, which occurs in 5-15% of cases.
- Islet cell transplants are increasing and are considered complementary rather than alternative.

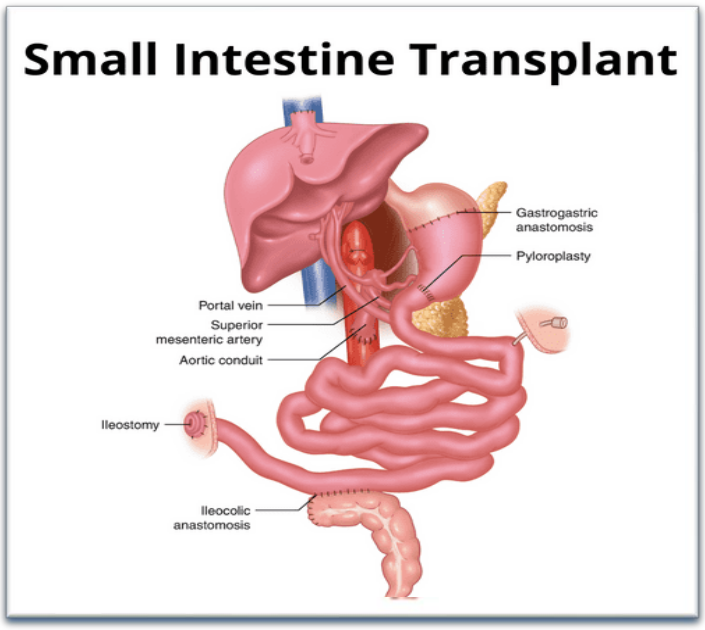
Challenges:

- A major challenge is that 2-4 donor pancreases may be required for one recipient.

Unit : 5

SMALL INTESTINE TRANSPLANTATION

- After the introduction of tacrolimus (in the late 1980s), the graft and patient survival rates in small intestine (SI) transplantation significantly increased.
- Nevertheless, SI transplantation remains the rarest of all transplants and has the lowest graft survival rate.
- The small intestine contains a significant amount of lymphoid tissue.
- High levels of immunosuppression are required.
- Despite this, the rejection rate remains high.
- Until a few years ago, patients receiving long-term TPN (total parenteral nutrition) had higher survival rates than those undergoing SI transplantation.
- Today, survival rates are either equal or SI transplantation survival is higher.



Indications:

- Intestinal and TPN (total parenteral nutrition) failure.
- TPN failure:
  - Liver damage (with significant biochemical or pathological findings).
  - Inability to find a central venous access (at least two central veins are thrombosed).
  - Frequent catheter infections.
  - Severe dehydration despite IV fluid support.

- Causes of small intestine (SI) transplantation failure:
  - Mesenteric ischemia due to SMA (superior mesenteric artery) or SMV (superior mesenteric vein) thrombosis.

- Crohn's disease.
- Trauma.
- Mesenteric desmoid tumors.
- Radiation enteritis.
- Extensive resections due to tumors.
- Chronic intestinal pseudo-obstruction.
- Autoimmune enteropathy.

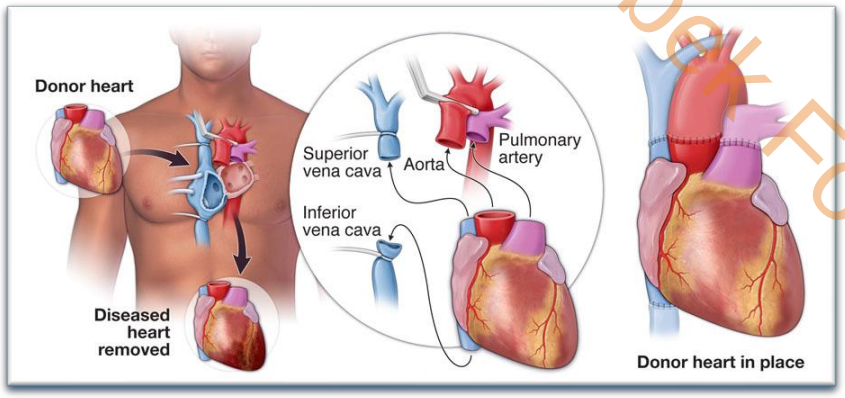
Postoperative Treatment and Complications:

- Small intestine transplantation has the highest rejection rate among all transplantations.
- There are no serologic markers for rejection, so biopsy and histological examination of the intestinal mucosa are very important.
- Complications:
  - Intra-abdominal abscess.
  - Small bowel leakage.
  - Intra-abdominal sepsis.
  - Graft thrombosis.
  - Life-threatening bleeding.
  - Post-transplant lymphoproliferative disease.
  - Graft versus host disease.

Unit : 6

HEART TRANSPLANTATION

It is often the last resort for patients with end-stage heart failure when other treatments, such as medication, surgery, or devices like pacemakers or ventricular assist devices (VADs), are no longer effective.



Indications:

- End-stage heart failure: The most common indication for heart transplantation. Patients with severe heart failure symptoms that are not manageable with medical therapy or devices.
- Coronary artery disease (CAD):
- Dilated cardiomyopathy.
- Restrictive cardiomyopathy.
- Hypertrophic cardiomyopathy.
- Congenital heart defects.
- Refractory arrhythmias: Life-threatening arrhythmias that cannot be controlled by other means.

Contraindications:

- **Active cancer**: A history of or current active cancer is a contraindication because the immunosuppressive therapy post-transplantation increases the risk of cancer recurrence.

- **Infection**.
- **Severe pulmonary disease**: Patients with severe lung disease may not survive the post-transplantation period.
- **Severe renal or liver dysfunction**: End-stage renal or liver failure may make transplantation inappropriate.
- **Obesity**: Extreme obesity (BMI > 35) can lead to poor surgical outcomes.
- **Age**: While heart transplantation is not absolutely contraindicated for older patients, individuals over the age of 70 may have reduced survival rates post-transplantation.
- **Psychological factors**: Individuals who cannot comply with the post-transplant medication regimen or have severe psychiatric disorders may not be suitable candidates.

Complications:

- **Acute rejection**.
- **Chronic rejection**.
- **Infection**: Due to the immunosuppressive drugs, infections, especially viral and fungal infections, are a risk.
- **Coronary artery disease**: Patients with heart transplants are at risk for transplant coronary artery disease (TCAD), which is a type of atherosclerosis affecting the transplanted heart's arteries.
- **Graft failure**: In some cases, the transplanted heart may not function properly, leading to graft failure.
- **Post-transplant lymphoproliferative disorder (PTLD)**: A condition related to the Epstein-Barr virus (EBV), resulting in abnormal growth of lymphocytes.

Survival Rates:

- The survival rate for heart transplant patients has improved over the years due to advances in surgical techniques, immunosuppressive therapy, and post-operative care.
- The **one-year survival rate** is typically around 85-90%.
- The **five-year survival rate** can range from 70-80%, depending on various factors such as the patient's age, overall health, and any complications that arise.
- Many patients can live for 10 years or more with a heart transplant, although the risk of graft failure and rejection increases over time.

Organ Preservation for Transplantation

- The **warm ischemia** period is the first phase, which occurs between the removal of the organ from the body and the washing of the vessels with hypothermic preservation fluid.
- **Cold ischemia** is the second phase, during which the organ is preserved in preservation fluids until transplantation.
- The warm ischemia time should be kept as short as possible, and the organ should be rapidly cooled.
- During cold storage, the organ is cooled to 4°C by washing the vascular system with an appropriate preservation solution.
- Washing helps remove as much blood as possible from the organ.
- The liver is typically washed with 2-3 liters of preservation solution, and the kidney with 200-500 ml.
- After washing, the organ is placed in a sterile container and preserved at 4-6°C.
- The heart should be transplanted within 5 hours after donor heart removal.
- The kidney can be safely preserved for 40-50 hours, but earlier transplantation is preferred.
- The pancreas can be preserved for up to 30 hours, though most pancreas transplants occur within 10-20 hours.
- Liver transplantation is typically performed within 6-12 hours following donor hepatectomy.
- It is recommended that small intestine transplantation be done within 12 hours.

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Other Books by the Author

Master's Today: Medical Surgical Disease
Focused Review of Surgical Diseases: A Guide for Exam Takers