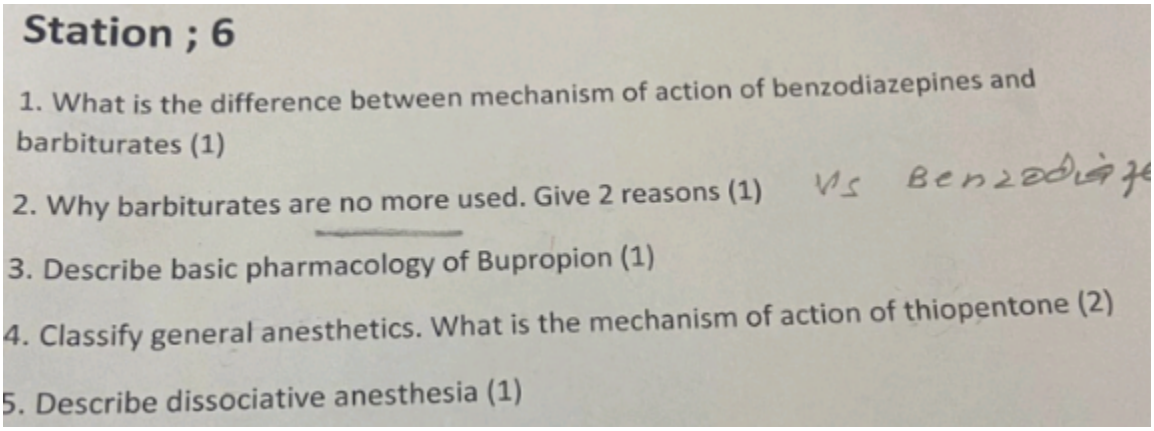
BLOCK J OSPE COMPILED

OSPE STATIONS

- 2 patho viva (one will be from tumours and 2nd one will be pther CNS topics)
- 2 community viva(1 from biostats and 1 from epidemiology)
- 2 forensics viva(one from poisons and other from other topics)
- 2 pharma(1 from epilepsy and other from other pharma topics)
- 1 psychiatric counselling
- 1 from epilepsy diagram
- 1 patho cns tumor identification
- 2 prescription drugs
- 2 biostat formulae
- 1 patho station of meningitis
- 2 forensic station of poison
- 1 examination which the college doesn't give and will be replaced by a viva

PHARMACOLOGY


Station ; 6

1. What is the difference between mechanism of action of benzodiazepines and barbiturates (1)
2. Why barbiturates are no more used. Give 2 reasons (1)
3. Describe basic pharmacology of Bupropion (1)
4. Classify general anesthetics. What is the mechanism of action of thiopentone (2)
5. Describe dissociative anesthesia (1)

Barbiturates vs Benzodiazepines

Feature	Barbiturates	Benzodiazepines
Mechanism of Action	Increases **duration** of GABA-A receptor chloride channel opening	Increases **frequency** of GABA-A receptor chloride channel opening
Uses	Seizures, anesthesia, insomnia	Anxiety, seizures, insomnia, muscle spasms
Examples	Phenobarbital, Thiopental	Diazepam, Lorazepam, Midazolam
Onset & Duration	Longer onset, longer duration	Rapid onset, shorter duration
Risk of Dependence	High	Moderate
Overdose Risk	High (respiratory depression, coma)	Lower than barbiturates but still possible
Reversal Agent	None	**Flumazenil**

Side Effects	Sedation, respiratory depression, tolerance	Sedation, dizziness, anterograde amnesia

Key reasons why Barbiturates are no longer used:

1. **Narrow therapeutic index** – High risk of toxicity and overdose.
2. **Severe CNS and respiratory depression** – Can be fatal in overdose.
3. **High potential for dependence and abuse** – Leading to addiction and withdrawal symptoms.
4. **Development of tolerance** – Reduces efficacy over time, requiring higher doses.
5. **Drug interactions** – Induces cytochrome P450 enzymes, affecting the metabolism of other drugs.
6. **Safer alternatives available** – Benzodiazepines and newer drugs for anxiety, sleep disorders, and seizures.

BUPROPION

Class	Mechanism	Uses	Effects	Adverse Effects	Contraindications	Advantages	Disadvantages
Atypical Antidepressant	Inhibits NE & DA reuptake	Depression, Smoking cessation	Energizing, Reduces cravings	Insomnia, Seizures, Weight loss	Seizure & Eating disorders	No sexual dysfunction, Weight loss	Seizure risk, Anxiety

Thiopentone MOA: Enhances **GABA-A**, increases **Cl^- influx**, causing **CNS depression** and **rapid unconsciousness**. Acts on **reticular activating system (RAS)**. **Ultra-short acting**: Onset in 10–20 sec, duration 5–10 min (due to redistribution).

Dissociative Anesthesia: Induced by **Ketamine**, patient appears **awake but unresponsive** with **analgesia, amnesia, and sedation**. Preserved **airway reflexes** and **spontaneous breathing**. Signs: **Open eyes, nystagmus, increased BP and HR**.

It's commonly used in **emergency settings** and **pediatric anesthesia**.

Treatment of Alcoholism

1. **Detoxification**
   - Benzodiazepines (e.g., **Diazepam**, **Lorazepam**) for withdrawal symptoms
   - Thiamine (Vitamin B1) to prevent **Wernicke's encephalopathy**
2. **Maintenance Therapy**
   - **Disulfiram**: Causes unpleasant reaction with alcohol
   - **Naltrexone**: Reduces cravings
   - **Acamprosate**: Restores neurotransmitter balance, prevents relapse

3. **Psychological Support**
    - **Counseling and Cognitive Behavioral Therapy (CBT)**
    - **Alcoholics Anonymous (AA)**
4. **Lifestyle Changes**
    - Healthy diet, regular exercise, and social support

Mechanism of Myocardial Infarction (MI) with Cocaine Use:

1. **Coronary Vasoconstriction**: Cocaine blocks **reuptake of norepinephrine**, causing prolonged **vasospasm** and reduced blood flow to the heart.
2. **Increased Platelet Aggregation**: Enhances clot formation, increasing the risk of **thrombosis**.
3. **Increased Oxygen Demand**: Cocaine stimulates **sympathetic activity**, raising **heart rate** and **blood pressure**, which increases oxygen demand while reducing supply.
4. **Accelerated Atherosclerosis**: Chronic use promotes **vascular inflammation** and **plaque formation**.

Drug Interactions of Levodopa

1. **Contraindicated Drugs**
    - **Non-selective MAO inhibitors** (e.g., phenelzine) → Risk of **hypertensive crisis**
2. **Reduced Effectiveness**
    - **Antipsychotics** (e.g., haloperidol) → Block dopamine receptors
    - **Pyridoxine (Vitamin B6)** → Increases peripheral metabolism of levodopa
3. **Increased Side Effects**
    - **Antihypertensives** → Enhanced hypotensive effect
    - **Entacapone or Carbidopa** → Potentiates levodopa's action
4. **High-Protein Diet** → Reduces absorption of levodopa in the gut

Migraine Prophylaxis:

- **Beta-blockers**: Propranolol, Metoprolol
- **Antidepressants**: Amitriptyline, Venlafaxine
- **Anticonvulsants**: Topiramate, Valproate
- **Calcium Channel Blockers**: Flunarizine
- **CGRP Inhibitors**: Erenumab
- **Botulinum toxin A** (Chronic migraine)

Adverse Effects of Triptans (e.g., Sumatriptan, Rizatriptan)

1. **Cardiovascular**
    - Chest pain, coronary vasospasm, hypertension
2. **Central Nervous System**
    - Dizziness, fatigue, drowsiness
3. **Gastrointestinal**
    - Nausea, vomiting, dry mouth

4. **Injection Site Reactions** (for subcutaneous forms)
    - Pain, redness, swelling
5. **Rare but Serious**
    - Serotonin syndrome (if combined with SSRIs or SNRIs)

Anti-psychotics

Class	Examples	Mechanism of Action	Key Features/Tricky MCQ Points	Mnemonic
1. Typical (First-Generation) Antipsychotics				
High Potency	Haloperidol, Fluphenazine, Trifluoperazine	Strong dopamine D2 receptor blockade.	- **High risk of extrapyramidal symptoms (EPS)**: Dystonia, akathisia, tardive dyskinesia. - Used in **acute psychosis** and **schizophrenia**. - Hyperprolactinemia.	**"Try to Fly High"**:- Trifluoperazine, Fluphenazine, Haloperidol are high potency.
Low Potency	Chlorpromazine, Thioridazine	Weak D2 receptor blockade; strong anticholinergic and antihistaminic effects.	- **Low EPS risk** but causes **sedation**, weight gain, orthostatic hypotension. - **Thioridazine**: Retinal deposits (MCQ!). - **Chlorpromazine**: Corneal deposits (MCQ!).	**"Cheating Thieves are Low"**:- Chlorpromazine and Thioridazine are low potency.
2. Atypical (Second-Generation) Antipsychotics				
Dopamine + 5-HT2 Blockers	Risperidone, Olanzapine, Quetiapine, Aripiprazole, Ziprasidone	Block dopamine (D2) and serotonin (5-HT2) receptors.	- **First-line drugs** due to fewer EPS. - Risperidone: High prolactin (MCQ!). - Olanzapine: Weight gain and metabolic syndrome. - Quetiapine: Sedation, low EPS.	**"ROQAZ"**: Risperidone, Olanzapine, Quetiapine, Aripiprazole, Ziprasidone.
D2 Partial Agonists	Aripiprazole	Partial agonist at D2 and	- Minimal EPS and prolactin increase.	**"Aripiprazole is Atypical for partial agonism."**

		antagonist at 5-HT2A.	- Used in schizophrenia, bipolar disorder, and depression adjunctively. - Low sedation, low metabolic risk.	
Dopamine Stabilizer	Brexpiprazole	Similar to aripiprazole.	- Better metabolic profile than aripiprazole. - Adjunct for major depressive disorder (MDD).	
Clozapine	Clozapine	Blocks D4, weak D2, and 5-HT2 receptors.	- Drug of choice for **treatment-resistant schizophrenia**. - Side effects: Agranulocytosis (MCQ!), myocarditis, seizures, weight gain. - Requires weekly CBC.	**"Watch CLOZeLY"**:- CLOzapine causes agranulocytosis, monitor CBC.
3. Third-Generation Antipsychotics				
Glutamate Modulators	Lumateperone	Modulates glutamate and dopamine pathways.	- New drug with lower side effects. - Used in schizophrenia.	

Q. Drug Classification and Indication

1. **A. Midazolam**
   - **Class**: Benzodiazepine (Short-acting)
   - **Indication**: Sedation, premedication for anesthesia, seizures
2. **B. Propofol**
   - **Class**: General Anesthetic (Intravenous)
   - **Indication**: Induction and maintenance of anesthesia, sedation in ICU
3. **C. Tramadol**
   - **Class**: Opioid Analgesic (with dual action: μ-opioid agonist & serotonin-norepinephrine reuptake inhibitor)
   - **Indication**: Moderate to severe pain, neuropathic pain

Drugs for alcohol aversion

1. **Disulfiram** – It inhibits aldehyde dehydrogenase, causing the accumulation of acetaldehyde when alcohol is consumed, leading to unpleasant symptoms such as nausea, vomiting, flushing, and headache. This reaction creates an aversion to alcohol.
2. **Naltrexone** – An opioid antagonist that reduces alcohol craving and helps maintain abstinence.
3. **Acamprosate** – Activates GABA receptors, reducing relapse rates.
4. **Ondansetron** – A 5-HT3 antagonist that decreases alcohol consumption.
5. **Topiramate** – Reduces alcohol craving.

Anesthesia classification

1. **General Anesthetics**
    - **Inhalational Anesthetics**:
        - **Volatile Liquids**: Ether, Halothane, Isoflurane, Desflurane, Sevoflurane
        - **Gas**: Nitrous oxide
    - **Parenteral Anesthetics**:
        - **Inducing Drugs**: Propofol, Etomidate, Thiopentone, Methohexitone
        - **Slow-acting Drugs**:
            - **Benzodiazepines**: Diazepam, Lorazepam, Midazolam
            - **Ketamine**
            - **Opioids**: Fentanyl, Alfentanil, Sufentanil, Remifentanil.
2. **Local Anesthetics**
    - Classified based on clinical use and structure:
        - **Clinical Use**:
            - Surface anesthetics (e.g., Cocaine, Lignocaine, Benzocaine)
            - Injectable anesthetics (Short, Intermediate, and Long-acting)
        - **Structure**:
            - **Esters** (Cocaine, Procaine)
            - **Amides** (Lignocaine, Bupivacaine).

Why is Nitrous Oxide (NO) used in Anesthesia?

1. **Potent Analgesic** – Excellent pain relief (strong analgesic effect).
2. **Rapid Induction & Recovery** – Low blood solubility → fast onset and quick recovery.
3. **Minimal Cardiovascular Effects** – Safe for patients with heart issues.
4. **Reduces Dosage of Other Anesthetics** – Decreases side effects of stronger anesthetic agents.
5. **No Muscle Relaxant Needed** – Does not cause muscle rigidity.

Why it's not used alone:

- Weak anesthetic → Cannot induce full unconsciousness or muscle relaxation.

drug causing cortisol imbalance

the drugs causing cortisol imbalance are primarily **glucocorticoids**, such as:

1. **Hydrocortisone and Cortisone** – Prolonged use leads to cortisol excess, causing **Cushing's syndrome** (moon face, buffalo hump, thin limbs).

2. **Prednisolone, Dexamethasone, and Betamethasone** – Suppress the hypothalamic-pituitary-adrenal (HPA) axis, leading to decreased endogenous cortisol production and potential adrenal insufficiency when stopped abruptly

and in which surgery it is used

Surgical Use of These Drugs

1. **Glucocorticoids** are used in surgeries to:
    - **Prevent adrenal insufficiency** in patients on long-term steroids.
    - **Reduce brain swelling** in neurosurgery (e.g., Dexamethasone).
    - **Suppress inflammation** in organ transplants or major surgeries.
2. **Etomidate** is used for **rapid sequence intubation** and sedation during short procedures, but it may cause temporary suppression of cortisol synthesis.

tramadol unique action

Unique Action of Tramadol (for OSPE)

1. **Dual Mechanism of Action**
    - **Opioid Agonist**: Binds to μ-opioid receptors (weak agonist).
    - **Inhibits Reuptake of Serotonin and Norepinephrine**, enhancing pain inhibition in the descending pathway.
2. **Less Respiratory Depression** – Compared to other opioids, safer for respiratory function.
3. **Low Potential for Addiction** – Though it can still cause dependence, it is less addictive than morphine.
4. **Useful for Neuropathic Pain** – Due to its effect on serotonin and norepinephrine pathways.

lithium MOA

Mechanism of Action of Lithium (for OSPE)

Lithium's exact mechanism is not fully understood, but it works by:

1. **Inhibiting Inositol Monophosphatase**
    - Reduces **inositol triphosphate (IP3)** and **diacylglycerol (DAG)**, which are crucial in neurotransmission.
    - This stabilizes mood and reduces manic episodes.
2. **Modulates Neurotransmitters**
    - Increases **serotonin** and decreases **dopamine** activity.
    - Balances **glutamate** levels, preventing excitotoxicity.
3. **Inhibits Glycogen Synthase Kinase-3β (GSK-3β)**
    - Promotes neuroprotection and synaptic plasticity.

CLINICAL USES

- Diazepam: anxiolytic, status epilepticus

- Quetiapine: schizophrenia, acute manic disorder
- Ecsitalopram: depression, GAD

🦋: Depression prescription

1 Parkinson disease drugs 2 status epilepticus management 3 phenytoin adverse effects 4 Levodopa and carbidopa are used in combination why 5 ???????

1. Parkinson's Disease Drugs

Classes & Examples:

1. **Dopamine Precursors**: Levodopa + Carbidopa
2. **Dopamine Agonists**: Pramipexole, Ropinirole
3. **MAO-B Inhibitors**: Selegiline, Rasagiline
4. **COMT Inhibitors**: Entacapone, Tolcapone
5. **Anticholinergics**: Trihexyphenidyl, Benztropine
6. **Amantadine**: For dyskinesia control

2. Status Epilepticus Management

Stepwise Approach:

1. **First-line (Benzodiazepines):** IV Lorazepam or Diazepam
2. **Second-line (Antiepileptics):** IV Phenytoin or Fosphenytoin
3. **Refractory Cases:** IV Phenobarbital or Midazolam infusion

3. Phenytoin Adverse Effects

Chronic Use:

1. **CNS**: Nystagmus, ataxia, sedation
2. **Gingival Hyperplasia**
3. **Hirsutism**
4. **Osteomalacia**
5. **Megaloblastic Anemia**
6. **Teratogenicity**: Fetal hydantoin syndrome

Acute Toxicity:

1. **Cardiac Arrhythmias** (with IV use)
2. **Hypotension**

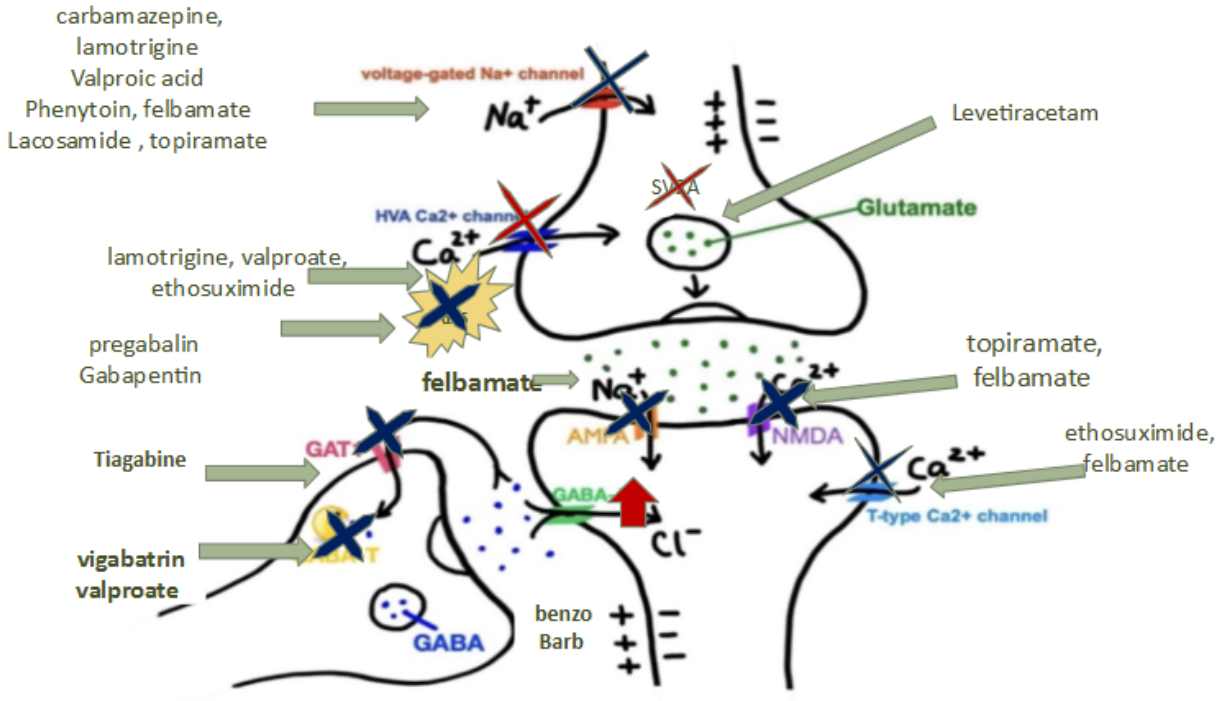
4. Why Levodopa and Carbidopa Are Used in Combination

1. **Levodopa** converts to dopamine in the brain to relieve symptoms.

2. **Carbidopa** inhibits **DOPA decarboxylase** in the periphery, preventing Levodopa breakdown before it reaches the brain.
    - **Benefits:** Reduces peripheral side effects (nausea, vomiting) and allows lower doses of Levodopa.

ANTI EPILEPTIC DRUGS CLASSIFICATION:

- Hyadantoin: Phenytoin
- Barbiturates: Phenobarbitone
- Iminostilbenes: Carbamezapine & Oxcarbazepine
- Succinamides: Ethosuximide
- Benzodiazepines: Diazepam, Lorazepam,clonazepam.
- Carboxylic Acid Derivatives: Valproic Aid
- Others: Vigabatrin, Lamotrigine, Gabapentin,lacosamide, Levitiracetam,felbamate, Topiramate.



Antiepileptic Drugs Mechanism of Action (Table Form)

Drug Class	Mechanism of Action	Examples
Na+ Channel Blockers	Inhibit voltage-gated sodium channels	Carbamazepine, Lamotrigine, Phenytoin, Valproate, Topiramate, Lacosamide
Ca2+ Channel Blockers	Inhibit T-type or HVA (high voltage-activated) calcium channels	Ethosuximide (T-type), Valproate, Lamotrigine
GABA Enhancers	Increase GABA activity by various mechanisms	Benzodiazepines, Barbiturates, Vigabatrin (inhibits GABA transaminase), Tiagabine (inhibits GABA reuptake), Valproate

Glutamate Inhibitors	Reduce excitatory neurotransmitter release (glutamate)	Felbamate, Levetiracetam, Topiramate
Ca2+ Channel Modulators	Block presynaptic calcium channels, reducing neurotransmitter release	Pregabalin, Gabapentin

MECHANISM OF ACTION OF ANTIEPILEPTIC DRUGS

Drugs that are effective in seizure reduction accomplish this by a variety of mechanisms, including

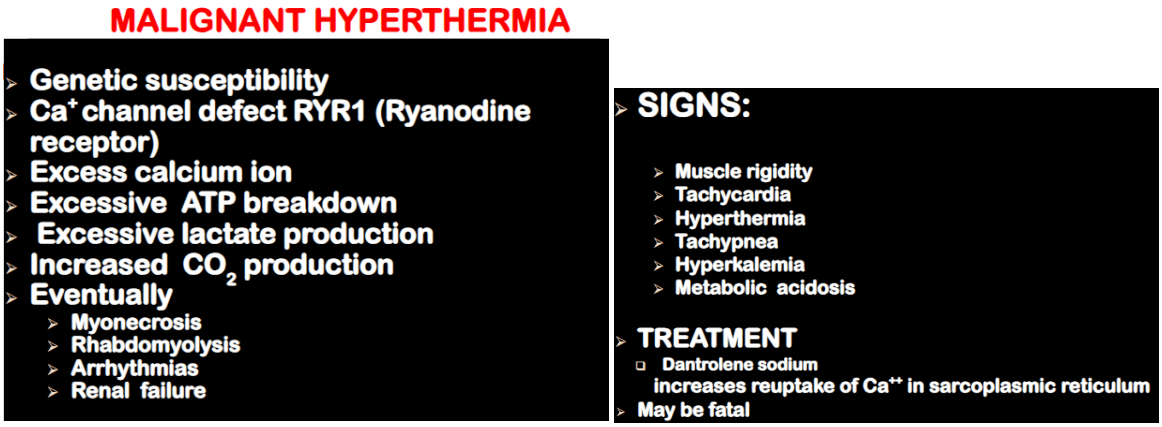
- Blockade of voltage-gated channels (Na+ or Ca2+ (**Valproate, Ethoxusimide**)),
- Enhancement of inhibitory GABAergic impulses,
- Or interference with excitatory glutamate transmission.
- Some antiepileptic drugs appear to have multiple targets.

Types of Seizures: Short Chart

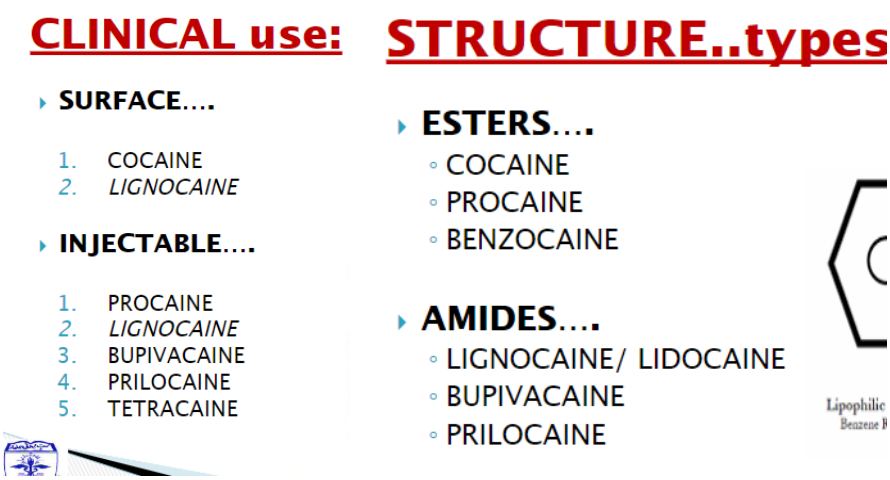
Type	**Key Features**	**Consciousness**
Focal Seizures (Partial)		
Focal Aware (Simple Partial)	Jerking, sensory symptoms, no confusion	Preserved
Focal Impaired Awareness (Complex)	Automatisms (lip-smacking), confusion	Impaired
Focal to Bilateral Tonic-Clonic	Progresses to convulsions	Impaired
Generalized Seizures		
Tonic-Clonic (Grand Mal)	Stiffening (tonic) + jerking (clonic)	Impaired
Absence (Petit Mal)	Staring, brief, no postictal confusion	Impaired briefly
Myoclonic	Sudden muscle jerks	Preserved
Atonic	Sudden loss of muscle tone (drop attack)	Impaired briefly
Tonic	Sustained muscle stiffening	Impaired
Clonic	Repeated jerking movements	Impaired

Cocaine causing MI (Cocaine induces acute myocardial infarction through various mechanisms, including coronary artery vasoconstriction, platelet activation, and thrombus formation, leading to coronary artery occlusion)

DUE TO HALOTHANE



LOCAL ANESTHETICS



- **Obstetric** analgesia.
  - Bupivacaine
- **C-Section**.
  - Bupivacaine(single shot)
- **Episiotomy**.
  - lignocaine
- **Mucosal surfaces**.
  - Benzocaine
- **Nerve block**.
  - most of LA

Which local anesthetic is an useful antiarrhythmic agent?? Lidocaine is a class Ib antiarrhythmic that reduces the permeability of the neuron membrane to sodium, which inhibits depolarization and blocks conduction. This action decreases the rate of depolarization of cardiac tissue, making lidocaine useful in treating ventricular arrhythmias

Which local anesthetic is also known as universal anesthetic?? LIDOCAINE

L-DOPA DRUG INTERACTIONS AND SIDE EFFECTS:

Adverse Effects of ldopa in

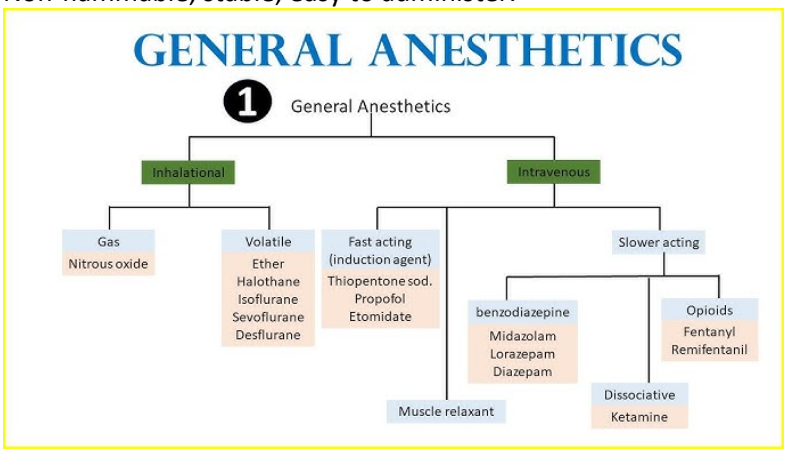
Nauseaa Vomiting 4) Postural Hypotension 131 Arrhythmias 141 Hypertension (5) Mydriasis (6) Dyskinesia 171 Psychotic symptoms

🦋: Epilepsy prescription

- classify gen anesthetics

1. **Stages of General Anesthesia**:
    - **Stage 1: Analgesia** – Loss of pain sensation but consciousness preserved.
    - **Stage 2: Excitement** – Delirium with irregular breathing and possible involuntary movements. (most dangerous)
    - **Stage 3: Surgical Anesthesia** – Regular respiration with muscle relaxation; divided into four planes. (surgery is performed)
    - **Stage 4: Medullary Paralysis** – Severe depression of medulla leading to respiratory and cardiovascular failure.
2. **MAC50 (Minimum Alveolar Concentration)**:
    - The concentration required to prevent movement in 50% of patients during surgery.
    - Lower MAC = higher potency.
3. **Significance of MAC50**:
    - Assesses anesthetic potency and guides dosing.
    - Age, temperature, and comorbidities influence MAC.
4. **Second Gas Effect**:
    - Rapid uptake of one gas (e.g., N2O) enhances uptake of a second gas.
5. **Diffusion Hypoxia**:
    - Rapid diffusion of N2O into alveoli dilutes oxygen, causing transient hypoxia post-operatively.
6. **Malignant Hyperthermia**:
    - Triggered by agents like Halothane.
    - Symptoms: Hypermetabolism, rigidity, hyperthermia.
    - Management: Immediate cessation, **Dantrolene**, cooling measures.
7. **Properties of Ideal Inhaled Anesthetic**:
    - Low solubility for fast induction/recovery.
    - Minimal side effects.

- Non-flammable, stable, easy to administer.



- procedure of CSF taking

The procedure for Cerebrospinal Fluid (CSF) collection is called **Lumbar Puncture (LP)**.

Preparation

1. **Consent:** Take informed consent from the patient (or guardian if necessary).
2. **Positioning:**
   - Patient in a lateral decubitus position (lying on their side with knees drawn up to the chest).
   - Alternatively, they may sit on the edge of the bed with the back arched forward.
3. **Identify Site:** The puncture is usually done between **L3-L4 or L4-L5** vertebral spaces. Use the iliac crest as a landmark for the L4 level.
4. **Sterilization:** Clean the area with an antiseptic solution (chlorhexidine) and drape sterile sheets around the puncture site.
5. **Anesthesia:** Use local anesthesia (lidocaine) to numb the area.

Procedure

1. **Insert the Needle:** Use a sterile spinal needle with a stylet.
   - Direct the needle slightly upward toward the umbilicus.
   - Advance it slowly until a "pop" is felt, indicating the needle has passed through the dura mater.
2. **Check for CSF Flow:** Remove the stylet to check for CSF flow.
   - Normal CSF is clear and colorless.
3. **Collect CSF:** Collect **3–4 mL** of CSF in sterile tubes for laboratory analysis. Label the tubes properly.
   - **Tube 1:** Biochemistry (protein, glucose)
   - **Tube 2:** Microbiology (Gram stain, culture)
   - **Tube 3:** Cytology/Cell count
   - **Tube 4:** Special tests (if required, e.g., PCR for TB or viral infections)

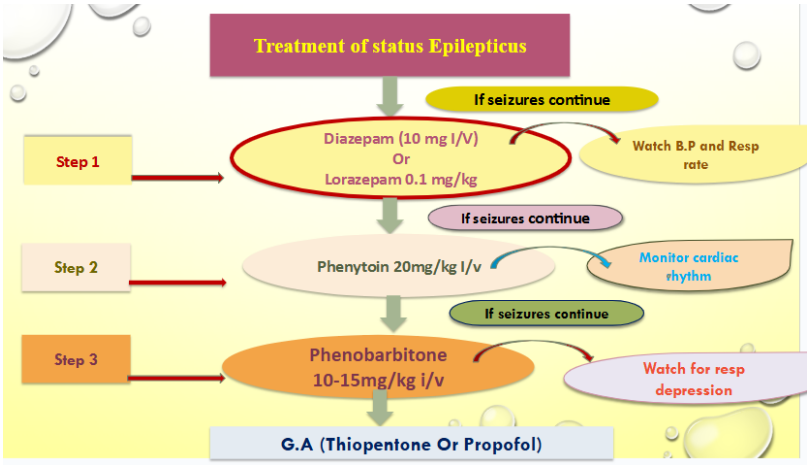
Post-procedure Care

1. **Needle Removal:** Reinsert the stylet and remove the needle.
2. **Apply Pressure:** Place a sterile bandage over the site.
3. **Observation:** Keep the patient lying flat for at least 1–2 hours to reduce the risk of a post-lumbar puncture headache.
4. **Monitor for Complications:**
    - Headache
    - Infection
    - Bleeding
    - Neurological symptoms (e.g., numbness, weakness)

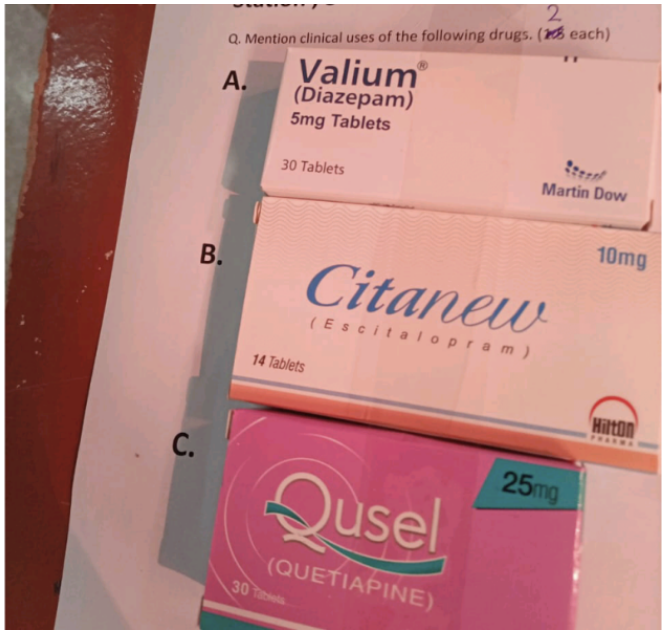
- wet preparation

- why tramadol is dif from other opioids

- management of status epilepticus



- prescription migraine and depression



FORENSICS

🦋: Skull fracture identify??? Names of skull fractures

HEAD INJURY:

Head injury is defined as "*a morbid state resulting from gross or subtle structural changes in the scalp, skull and/ or the contents of the skull, produced by mechanical force*"

Classification

Depending on the state of dura

1. Closed head injury (DURA INTACT) even if there is a fracture
2. Open head injury (DURA TORN)

Depending on duration of unconsciousness and Glasgow coma scale

a. Minor or mild head injury
b. Moderate head injury
c. Severe head injury

Table 11.1: Classification of head injury

Type	*Duration of un-consciousness*	*Glasgow coma scale*
Minor or mild head injury	< 30 minutes	13-15
Moderate head injury	> 30 min and < 6 hours	9-12
Severe head injury	> 6 hours	8 or less

SCALP:

Contusion of Scalp

- Bruise of scalp may be **mobile**
- A bruise in the **anterior scalp** may shift **downward** to appear **around the eye**, thus causing "**black eye" or spectacle hematoma (**Hemorrhage in the soft tissue around the eyes in eyelids of both eyes is called spectacle hematoma or raccoon eyes i.e. in other words black eye on both side is a spectacle hematoma. It usually suggests fracture of base of skull.**).**
- A contusion in **temporal scalp** may shift do**wnward** and appear **behind the ear** – similar to **battle sign.**
- These **shifting bruises** are also called as **ectopic contusion**, percolated bruises or migratory contusions.
- Hematoma may occur **beneath the galea aponeurotica** and called as **under-scalp hematoma** or sub-galeal hemorrhage or sub-galeal hematoma

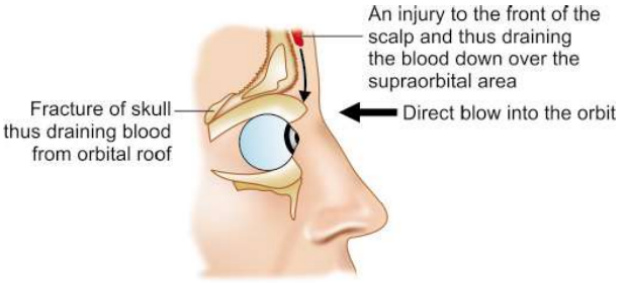


FIG. 11.3: Mechanism of production of black eye

Mechanism of Skull Fracture

As per Rowbotham's hypothesis, fracture of skull is caused by:

1. **Direct** application of force to skull – for example blow over head with iron rod.
2. **Indirect** violence – for example fall from height on feet or buttock: Fracture due to general deformation results in **fissured type** and occur in part of the skull distant from the site of application of force

Puppe's Rule

• When two or more separate fracture occurs from successive impacts and meet each other, the later fracture (second fracture) will terminate in the earlier fracture (first fracture)

Skull Fracture Due to Indirect Violence

- Force applied to chin
- Force applied to feet or buttock

Types of skull fractures are

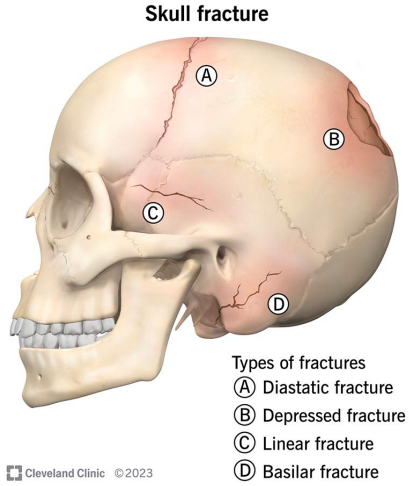
A) Fracture of vault of skull

1. Linear or fissured

2. Depressed (signature)-- as the pattern resembles that of causative weapon
3. Comminuted [Mosaic (spider web)]-- fragmentation of bones occurs
4. Pond or indented --occurs only in skull of infants
5. Gutter --when part of the thickness of skull bone is removed so as to form a gutter or furrow in the bone.
6. Diastatic or sutural --along the line of sutures of skull
7. Perforating
8. Cut fracture

B) Fracture of base of skull (basilar fracture)

1. Linear or fissured
2. Ring --This is a fissured fracture that occurs round the foramen magnum in posterior cranial fossa
3. Hinge --linear fracture that passes across the floor of middle cranial fossa, often following the petrous temporal or greater wing of sphenoid bone into pituitary fossa on both sides thus separating the base of skull into two halves--- motorcyclist's fracture
4. Longitudinal
5. Secondary



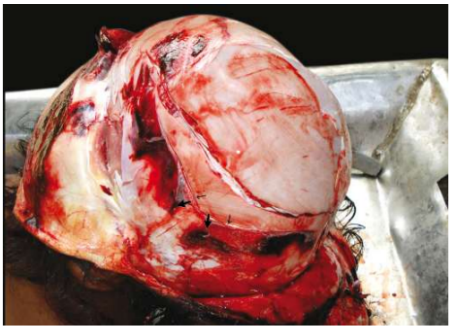


FIG. 11.9: Linear fracture (black arrows)

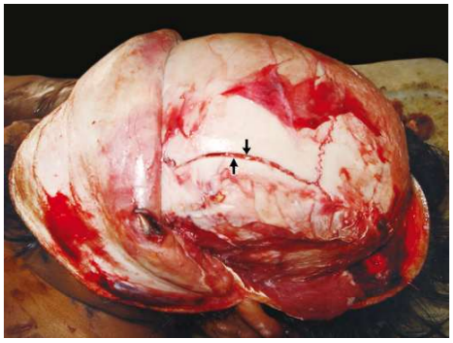
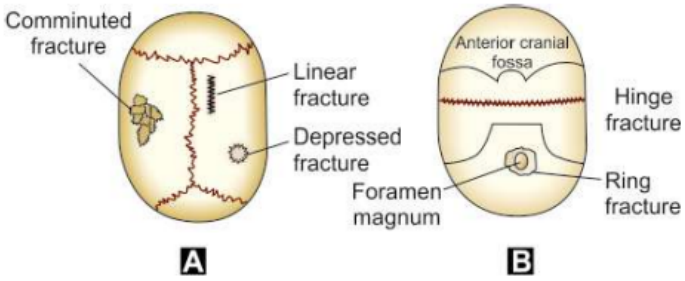


FIG. 11.10: Linear fracture (black arrows)



FIGS 11.20A and B: Different types of fracture

Complication of Skull Fracture

1. Injury to brain
2. Intracranial hemorrhage
3. Fracture of anterior cranial fossa may involve frontal, ethmoidal or sphenoidal air sinuses
4. Intracranial infections – meningitis/encephalitis
5. Cranial pneumatocele or pneumocranium
6. Cranial nerve injury
7. Traumatic epilepsy
8. CSF otorrhea
9. Coma
10. Cerebral edema
11. Increased intracranial pressure/tension
12. Death

Classification of brain injury

Primary brain injury

1. Diffuse axonal injury
2. Cerebral concussion
3. Cerebral contusions and lacerations

Secondary brain injury

1. Intracranial hematoma
2. Cerebral edema

3. Cerebral ischemia
4. Cerebral herniation
5. Infection
6. Epilepsy
7. Hydrocephalous

- **Cerebral Concussion** Also called as **commotio cerebri** or stunning brain shock.
  Definition "a transient paralytic state due to head injury which is of ***instantaneous*** onset, does ***not show any evidence of structural cerebral injury*** and is always ***followed by amnesia*** from the actual moment of the accident"

Cause: It occurs due to acceleration/deceleration of head. At low levels of acceleration/deceleration, anatomic changes of neurons do not occur but **physiologic functions are affected**.

Classification: Cerebral concussion are classified into three grades as

1. Grade I: No loss of consciousness
2. Grade II: Loss of consciousness but for **less than 5 minutes**
3. Grade III: Unconsciousness for more than 5 minutes associated with memory loss for more than 24 hours.

Autopsy Findings: at autopsy **no visible structural damages** are noted in brain. Occasionally, **punctate hemorrhages** may be present.

Medicolegal Importance

1. Retrograde amnesia – here patient is unable to recollect the event that leads to accident or injury. The retrograde amnesia may be true or false (feigned) as in **malingering act**.
2. It may be confused with punch drunk or drunkenness.

- **Cerebral Contusion**

- Cerebral contusions are the **circumscribed areas** resulting from **extravasations of blood in traumatized area of brain**. The integrity of cortex is maintained.
- Contusions are produced as a result of **shearing forces** within the brain tissue at the moment of impact.
- In almost all cases there is also some degree of subarachnoid hemorrhage.

Cerebral Edema

Traumatic cerebral edema is an accumulation of fluid in the extracellular space. It may be of following types:

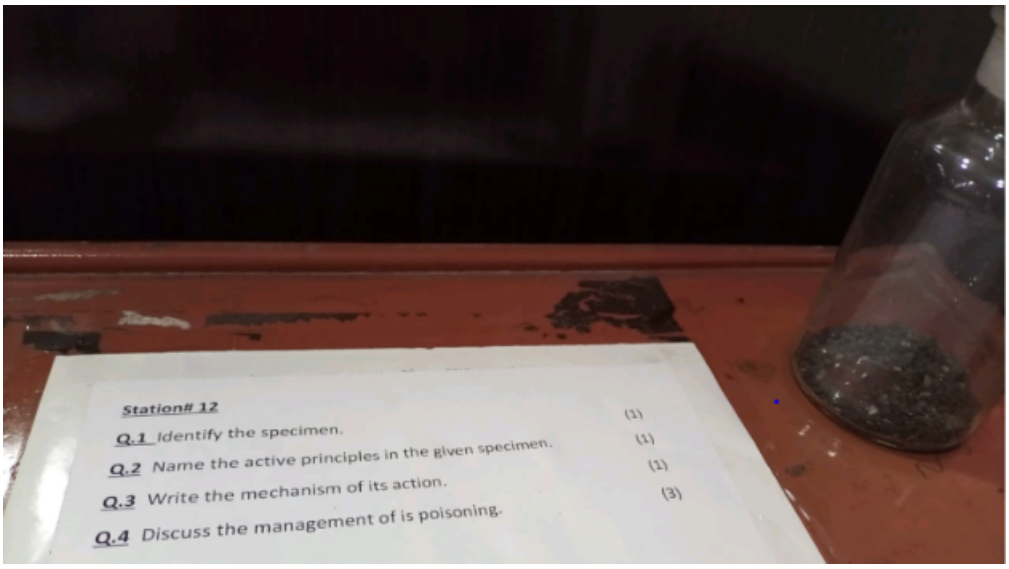
1. Vasogenic cerebral edema
2. Cytotoxic cerebral edema
3. Mixed type

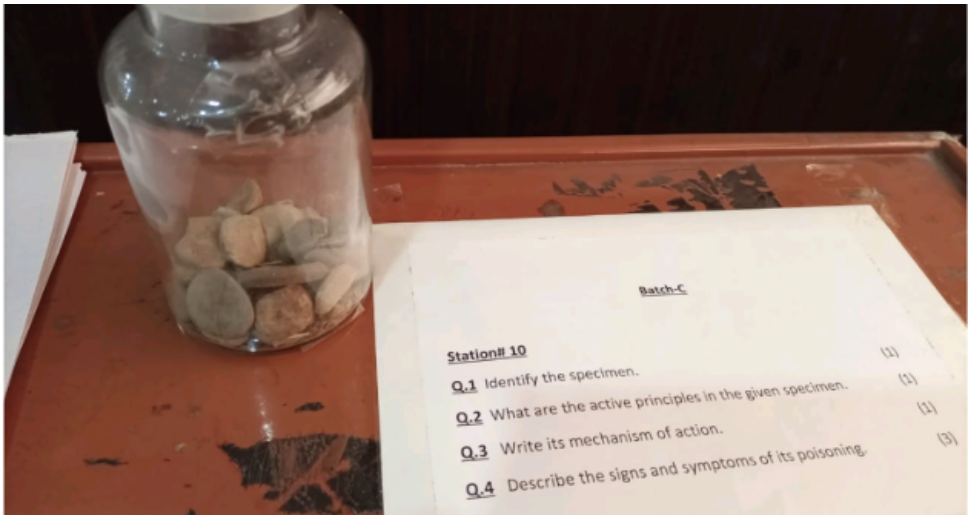
Boxer's Injury (Punch Drunk Syndrome)

- The **most common lesion** in acute episode appears to be **subdural hematoma**. Extradural bleed never occurs as boxing hardly causes skull fracture. Subarachnoid hemorrhage may occur in some cases due to rupture of berry's aneurysm.
- It is the chronic changes induced in the brain that concerns more. Repeated blowing in boxing over head induces **traumatic encephalopathy** known as "punch drunk syndrome". This syndrome is characterized by ***deterioration in speed and coordination, slurred speech, defective memory, slow thoughts, stiff-limbs, ataxia, unsteady gait, parkinsonian like dementia*** etc.
- Brain may show cortical atrophy, hydrocephalous, perforation of septum pellucidum, and loss of neurons from cerebellum and substantia nigra

Alcohol Withdrawal Drugs (Summary)

1. **Benzodiazepines:** Diazepam, Lorazepam – Prevent seizures and DTs.
2. **Anticonvulsants:** Carbamazepine – Alternative to benzodiazepines.
3. **Adrenergic Agents:** Clonidine, Propranolol – Control tremors and tachycardia.
4. **Thiamine (Vitamin B1):** Prevent Wernicke's Encephalopathy.
5. **Antipsychotics:** Haloperidol – For agitation and hallucinations (use cautiously).





Trip and bad trip, flash back ,opioid specific antidote

1. Trip and Bad Trip

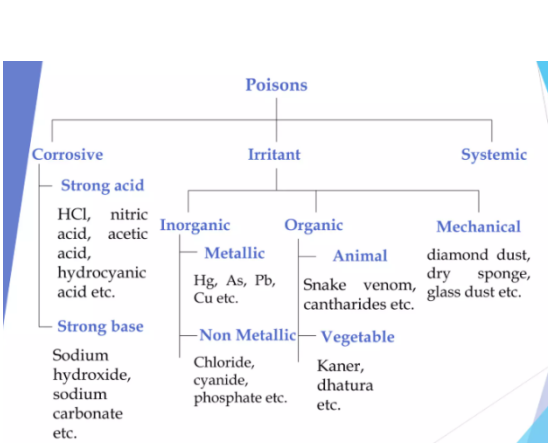
- **Trip:** A **psychedelic experience** caused by hallucinogenic drugs (LSD, psilocybin). It can involve sensory distortions, altered thoughts, and hallucinations.
- **Bad Trip:** An **unpleasant or terrifying experience** during a hallucinogenic trip, characterized by anxiety, paranoia, confusion, and panic attacks.

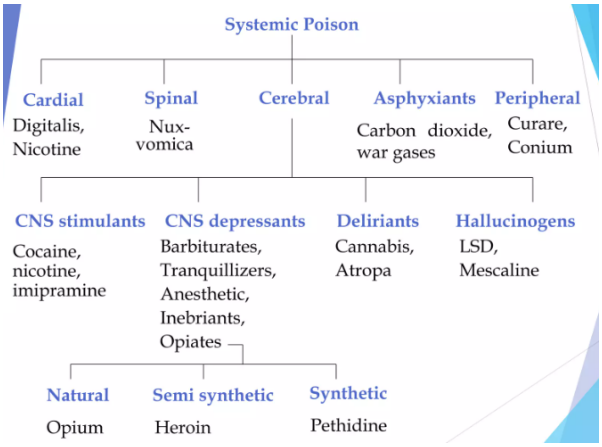
2. Flashback

- A **sudden, intense re-experience** of a past hallucinogenic trip without drug use. It may occur weeks, months, or even years after the original experience. Common in **LSD users**.

3. Opioid-Specific Antidote

- **Naloxone** (Narcan)
  - **Mechanism:** Competitive antagonist at **opioid receptors (mu, kappa, delta)**.
  - **Route:** IV, IM, SC, or intranasal.
  - **Indication:** Used for the **reversal of opioid overdose** symptoms such as respiratory depression, sedation, and miosis.





Nux vomica seeds identify ?? Active Principle?? Management?

Nux Vomica Seeds

1. **Identification:**
    - **Shape:** Flat, circular, and disc-like.
    - **Size:** About **2 cm in diameter**, with a hard, smooth, and glossy surface.
    - **Color:** Ash-gray or greenish-gray.
    - **Taste:** Extremely bitter.

Active Principles:

- **Strychnine** and **Brucine** (both are alkaloids).
- **Strychnine** is the primary toxic component, causing severe convulsions by blocking glycine receptors in the spinal cord.

Management of Nux Vomica Poisoning:

1. **Immediate Treatment:**
    - **Gastric lavage** with **potassium permanganate** (1:5000) to oxidize the alkaloids.
2. **Anticonvulsants:**
    - **Diazepam or Thiopental Sodium** to control convulsions.
3. **Supportive Therapy:**

- Ensure **adequate oxygenation** and **cardiovascular support**.

4. **Specific Antidote:** There is no specific antidote; treatment is **symptomatic and supportive**.

- **chronic alcohol poisoning** (delirium tremens, korsakoffs psychosis, acute hallucinosis)
- **Delirium tremens**: It is defined as the unsoundness of mind, due to long continued action of alcohol in chronic alcoholics.

Causes:

- An unusual bout of drinking
- Sudden withdrawal of alcohol
- Acute infection
- Shock from injury
- Exposure to cold

Signs and Symptoms of Dhatura Poisoning (9 Ds)

1. **Delirium** – Confusion, hallucinations, and disorientation
2. **Dryness** – Dry mouth and throat due to inhibition of secretions
3. **Dysphagia** – Difficulty in swallowing
4. **Dilated Pupils (Mydriasis)** – Pupils widely dilated, causing photophobia and blurred vision
5. **Diplopia** – Double vision
6. **Dysarthria** – Slurred speech
7. **Dizziness** – Loss of balance and vertigo
8. **Drowsiness** – Lethargy and reduced alertness
9. **Decreased Sweating** – Hot, dry skin with hyperthermia

Additional Symptoms:

- Rapid heart rate (tachycardia)
- Urinary retention
- Seizures in severe cases

Korsakoffs psychosis: It is a syndrome characterized by hallucinations, disorientation and multiple neuritis. The patient's memory for recent events is lost and he fills the gap by confabulation.

Cause:

- Severe, untreated thiamine deficiency, secondary to chronic alcohol abuse

Acute Hallucinosisa rare psychotic disorder that involves auditory hallucinations, delusions, and affective symptoms.

Treatment of CHRONIC ALCOHOL POISONING

1. ANTABUSE(DISULFURUM) 0.5gm
2. Citrated calcium carbamide
3. Psychotherapy

Treatment of METHANOL POISONING

- Gastric lavage with 5% Sod.Bic solution.
- Oral administration of sodium bicarbonate in a dose of 2 grams in 250ml of water every two hours to maintain neutral or slightly alkaline urine.
- Oral administration of Ethyl Alcohol 50%, 0.75 to 1ml/kg every 2 hours for 3 to 4 days.
- Eye care--- protect from bright light.
- Indications for hemodialysis include any ocular findings, metabolic acidosis, renal failure and a blood methanol level over 50 mg%.
- **Antidote** : **4-methyl pyrazole** is a specific alcohol dehydrogenase inhibitor. It blocks the formation of formaldehyde and formic acid.

why is ethanol used as an antidote for methanol poisoning

Ethanol, the active ingredient in alcoholic beverages, acts as a competitive inhibitor by more effectively binding and saturating the alcohol dehydrogenase enzyme in the liver, thus blocking the binding of methanol.

- Active principle of poppy plant: 1. Phenanthrene 2. Isoquinoline

ANTIDOTE= NALOXONE

STAGES=

- Excitement
- Stupor
- Narcosis

- DIAGNOSTIC SIGNS AND SYMPTOMS= **Diagnosis**
  - Classic triad
  - Smell
  - Sweating
  - Cyanosis
  - Marquis test

- **McNaughton's rules**: Every man is to be presumed to be sane and to possess a sufficient degree of reason to be responsible for his crimes, until the contrary be proved.
- **The Durham rule**, also known as the product test, is a criminal law rule that states that a defendant is not guilty by reason of insanity if their criminal act was the result of a mental disease or defect.
- A cerebral **concussion**, also known as a concussion, is a mild traumatic brain injury (TBI) that affects brain function
- GBS= campulobacter, ascending weakness, nerve conduction test, plasmaphoresis

Forensic 2 run amok ..somnambulism.. dhatura active Principle ..strychinine complications ..Korsakoff psychosis ..dhatura fatal period remaining????

1. Run Amok

- **Definition:** A condition often associated with the **continued use of cannabis**, characterized by a **frenzied homicidal tendency** where the person kills several individuals, starting with those perceived as enemies and continuing until they surrender or commit suicide.

2. Somnambulism (Sleepwalking)

- **Definition:** A state of **dissociated consciousness** during sleep.
- **Features:** The person may walk, perform complex tasks, or even commit crimes without memory of the event. Electroencephalographic studies confirm that the person is not awake.

3. Dhatura

- **Active Principle: Laevohyoscyamine**, **Hyoscine (Scopolamine)**, and traces of **Atropine**.
- **Fatal Period:** Death may occur within **24 hours**, typically due to **respiratory failure**.

4. Strychnine Complications

- **Mechanism:** Stimulates all parts of the **central nervous system**, especially the **anterior horn cells** of the spinal cord, leading to **increased reflex excitability** and **severe muscle spasms**.
- **Complications:**
    - **Opisthotonos:** Arching of the body.
    - **Asphyxia:** Due to respiratory muscle spasms.
    - **Exhaustion and Death:** From repeated convulsions.

5. Korsakoff's Psychosis

- **Definition:** A chronic memory disorder associated with **alcoholism**.
- **Symptoms:**
    - Severe **anterograde amnesia** (inability to form new memories).
    - **Confabulation:** Fabrication of information to fill memory gaps.
    - **Extreme disorientation**.

Classification of Neurotoxins

Kerosene Antidote

1. Classification of Neurotoxins

- **Snake Venoms:** Neurotoxic venom in **elapids** (cobra, krait) causes **muscle paralysis**.
- **Heavy Metals: Lead, mercury**—affect the **central nervous system**, causing cognitive deficits and peripheral neuropathy.
- **Organophosphates:** Inhibit **acetylcholinesterase**, causing excess acetylcholine and resulting in paralysis.
- **Cyanotoxins:** Affect ion channels and respiration at the cellular level.

2. Kerosene Antidote and Management

Treatment Steps:

- **Gastric lavage** with **5% sodium bicarbonate** solution.
- Administer **250 ml of liquid paraffin** followed by a saline cathartic to slow absorption.
- If poison is inhaled, ensure fresh air and administer **artificial respiration**.
- **Symptomatic management**: Treat respiratory failure and prevent lung complications.

- features of alcohol poisoning
- stages of alcohol poison
- snake bites classification

Features of Alcohol Poisoning

- **Stage 1: Excitement (30–100 mg%)**
  - Feeling of well-being, increased talkativeness, laughter, or anger.
  - Impaired judgment and inappropriate behavior.
- **Stage 2: Incoordination (100–300 mg%)**
  - Slurred speech, confusion, unsteady gait.
  - Vomiting, dilated pupils, staggering.
- **Stage 3: Narcosis (>300 mg%)**
  - Deep coma, muscle relaxation, and abolished reflexes.
  - Slow breathing, pinpoint pupils, cold, clammy skin, and cyanosis.
  - Death occurs from respiratory failure.

Classification of Snake Bites

1. **Elapids (Neurotoxic Venom)**
   - Examples: **Cobra, King Cobra, Common Krait**
   - Effects: Paralysis of respiratory muscles, muscle weakness.
2. **Vipers (Vasculotoxic Venom)**
   - Examples: **Russell's Viper, Saw-scaled Viper**
   - Effects: Severe bleeding, swelling, tissue damage.
3. **Sea Snakes (Myotoxic Venom)**
   - Effects: Generalized muscle pain, myoglobinuria, respiratory failure.

Snake Bite Management (Parikh's Textbook)

1. **Immediate First Aid:**
   - **Immobilization:** Keep the bitten limb below heart level.
   - **Tourniquet:** Apply it 5 cm above the bite site to prevent lymphatic flow, but not too tight to cut off blood supply.
   - **Cleansing:** Wash the bite site with plain water or saline.
2. **Hospital Management:**
   - **Antivenin Therapy:**
     - **Specific Antivenin**: For a particular species (e.g., cobra).

        - **Polyvalent Antivenin**: Effective for cobra, krait, Russell's viper, and saw-scaled viper bites.
        - Dosage: Typically 60 ml, given subcutaneously, intramuscularly, and intravenously. Repeat IV dose if symptoms persist.
    - **Supportive Care:**
        - Neostigmine-atropine for **neurotoxic bites** (elapids).
        - Heparin and fibrinogen transfusion for **vasculotoxic bites** (vipers).
    - **Tetanus Prophylaxis and Antibiotics** to prevent secondary infections.
3. **General Measures:**
    - **Artificial Respiration** if needed.
    - **Corticosteroids** for allergic reactions to antivenin.
    - **Blood Transfusion** in hemorrhagic cases.

Forensic Psychiatry Definition

Forensic Psychiatry is a specialized branch of psychiatry that focuses on the intersection of **mental health and the law**. It involves the evaluation and treatment of individuals involved in legal cases, addressing issues such as:

- **Competency to stand trial**
- **Criminal responsibility (insanity defense)**
- **Risk assessment for violence**
- **Assessment of mental health in civil cases** (e.g., guardianship, disability claims)

Fact (Definition)

A **fact** is something that is **objectively true**, based on **actual events, evidence, or reality**, and can be **verified** or **proven**. In law and forensic contexts, a fact refers to a **piece of information presented as evidence**, such as an event or statement that is **accepted as true**.

C. MED

PH is a multidisciplinary field whose goal is to promote the health of the population through organized community efforts.

Public Health activities include

- Assessing the health status of the population
- Diagnosing its problems
- Searching for the causes for those problems
- Designing solutions for them.

Epidemiology: *The study of the distribution and determinants of disease frequency in human populations and the application of this study to control health problems.*

- **DIESEASE FREQUENCY**

Counting, a key activity of epidemiologists, includes 3 steps

1. **Developing a definition of disease**
2. **Instituting a mechanism for counting cases of disease within a specified population, &**

3. **Determining the size of that population.**

A hypothesis is defined as "a tentative explanation for an observation, phenomenon, or scientific problem that can be tested by further investigation". E.g. "children who take vitamin C are less likely to become ill during flu season than those who do not.

- **Rate:** = No. of events in a specified period x K
  Pop. at risk in a specified period

A rate comprises a numerator, denominator, time specification & multiplier. The time dimension is usually a calendar year. Rate is expressed per 1000, 10,000 or 100,000 selected according to convenience to avoid fractions

Rate is used to estimate probability or risk of occurrence of a disease or to assess the accessibility or coverage of healthcare system.

Example

Crude death rate= Number of deaths in one year X1000

Mid – year Population

- **Ratio:**
  - Relationship b/w 2 numbers expressed as
  - x:y or x/y e.g ratio of males to females 2:3.
  - The numerator is not a component of the denominator.
- **Proportion**:
  - Specific type of ratio in which numerator is included in the denominator and the resultant value is expressed as %age. E.g 1: If there are 1000 boys and 800 girls in a school, the proportion of boys:
  - Boys / Boys+ Girls= 1000 x 100 = 55%
  - 1000+800
- All proportions are ratios, but not all ratios are proportions
-

Attack rate= # of people at risk in whom a certain illness develops

total # of people at risk

- A person who acquires the disease from that exposure (from a contaminated food) is called a *primary case*.

- A person who acquires the disease from exposure to a primary case is called a *secondary case*.

The *secondary attack rate* is therefore defined as the attack rate in susceptible people who have been exposed to a *primary case*.

Secondary attack rate= No. of cases among contacts

of primary cases X 10n

total No. of susceptible contacts

Quiz: Of 75 persons who attended a church picnic, 46 subsequently developed gastroenteritis. The attack rate of gastroenteritis is:

46/75 *100= 61%

Incidence : The no. of **new cases** of a disease that occur during a specified period of time in a population at risk for developing the disease.

Incidence= No. of new cases of a specific disease during a given time period X1000

pop. at risk during that period

=16.7per 1000 per year.

NOTE: the incidence rate must include the unit of time used in the final expression.

Prevalence is the number of all current cases **(old & new**) of a disease at one point in time in relation to a defined population.

Prevalence = # of all current cases (old & new) of a specified disease existing at a given point in time X 100

estimated population at the same point in time

P = I X D (incidence x mean duration)

Example (for a stable condition)

- Incidence = 10 cases/1000 pop./year
- Mean duration of disease = 5 years
- Prevalence = 10 x 5= 50/1000 population
- Conversely
  I = P/D D = P/I

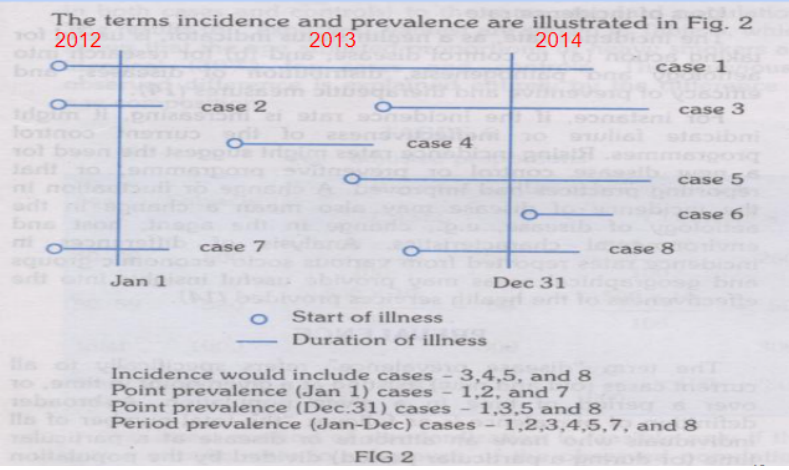
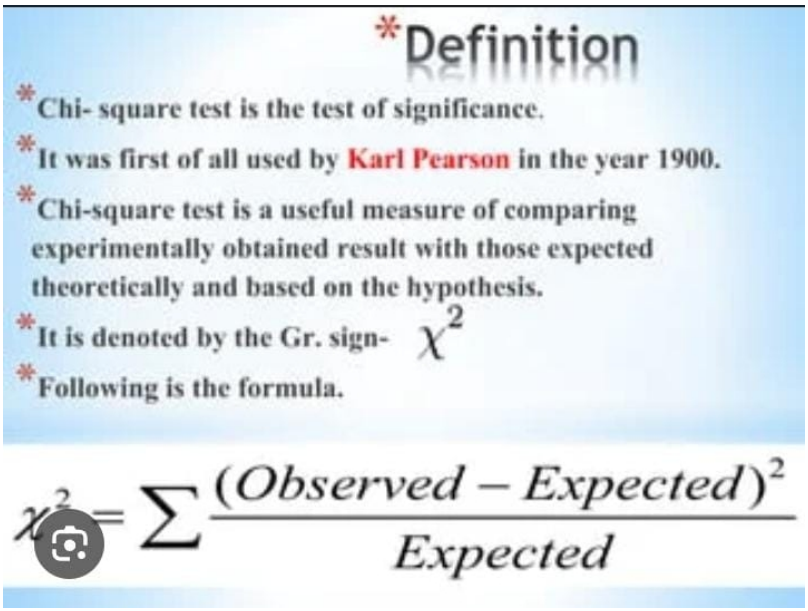


FIG 2

- I= Measure of risk
- P= Generally preferred for chronic diseases without clear date of onset
- P= Affected by duration of illness



Mortality

- **Crude Death rate:**

CDR= No. of deaths occurring in a specified 12 months' period X 1000

No of persons in the pop. at the mid-point of the 12-month period (mid-year pop)

- **Cause Specific Death Rate:**

CSDR = No of deaths from a specific cause during a calendar year X 1000

No of persons in the mid-point of that period

- **Age Specific Death Rate:**

ASDR = No. of deaths of a specific age group

No of persons in the pop. of that age

- IMR is a special age specific death rate

Formula: No of infants dying during 1st year x1000

No of live births during the same period

- **Case Fatality Rate** (ratio) =

Total # of deaths due to a particular disease X 100

Total # of cases due to the same disease.

a. Proportional mortality from a specific disease =

# of deaths from a specific disease in a year x 100

total deaths from all causes in that year

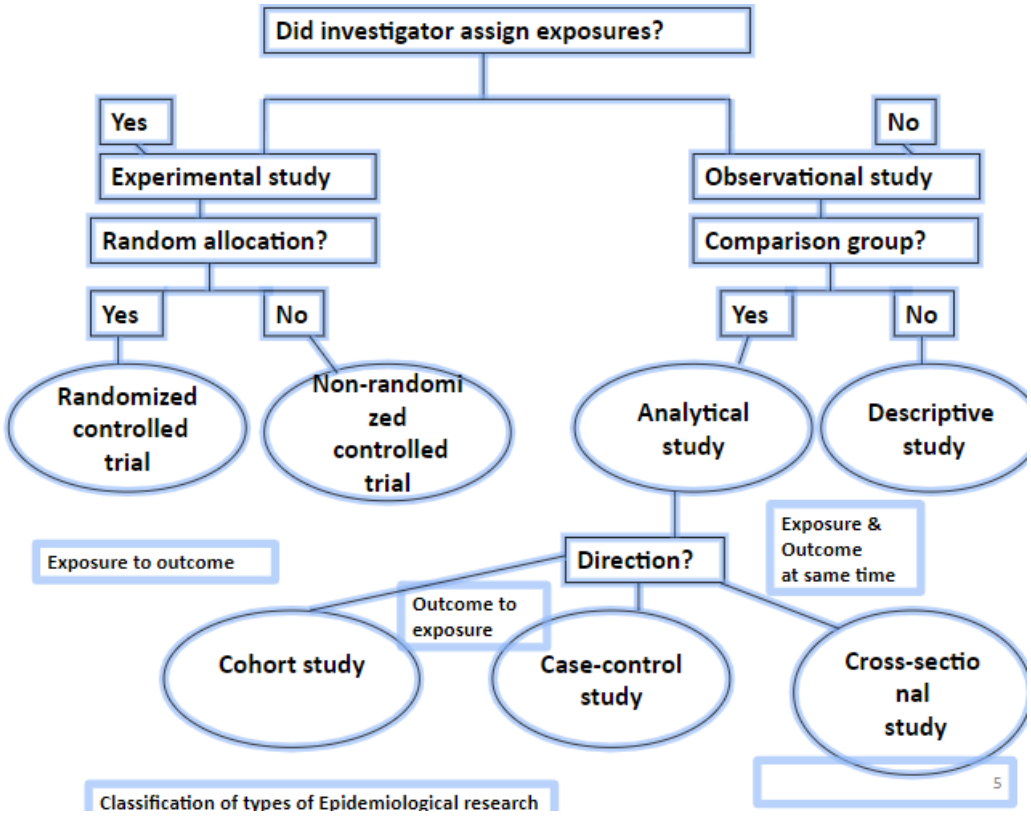
b) Under – 5 proportionate mortality rate=

$$\frac{\text{\# of deaths under 5 years of age in the given year}}{\text{total \# of deaths during the same period}} \times 100$$

c. Proportional mortality rate for aged 50 yrs & above =

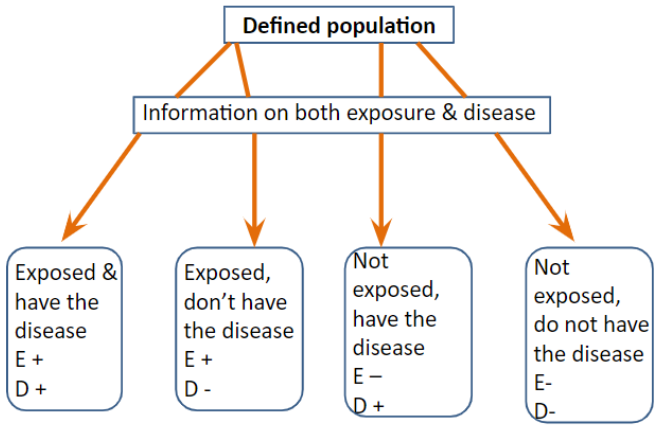
$$\frac{\text{\# of deaths of persons aged 50 yrs \& above}}{\text{total deaths of all age groups in that year}} \times 100$$

Evidence-based medicine (EBM) is the process of systematically reviewing, appraising and using clinical research findings to aid the delivery of optimum clinical care to patients.



- In *observational* studies, the researcher observes and systematically collects information, but does not try to change the people (or animals, or reagents) being observed.
- In an *experiment*, by contrast, the researcher intervenes to change something (e.g., gives some patients a drug) and then observes what happens.

In an observational study there is ***NO*** intervention.



Sketch of a cross- sectional study design

22

Cross-sectional design is referred to as non-directional or one point in time survey, where data is collected on both outcome and exposure status of the individuals under study (Provide snap shot picture). Exposure & disease assessed simultaneously

Main outcome is Prevalence. Using a 2 x2 table:

	Disease	Non-diseased	
Exposed	a	b	a+b
Non-exposed	c	d	c+d
	a+c	b+d	

	Disease	Non-diseased
Exposed	a b	
Non-exposed	c	d
	a+c	b+d

Disease status in exposed

	Disease	Non-diseased
Exposed	a	b
Non-exposed	c	d
	a+c	b+d

Exposure status in diseased

	Disease	Non-diseased
Exposed	a b	
Non-exposed	c	d
	a+c	b+d

Prevalence of disease compared in exposed & non-exposed

$\frac{a}{a+b}$ Vs. $\frac{c}{c+d}$

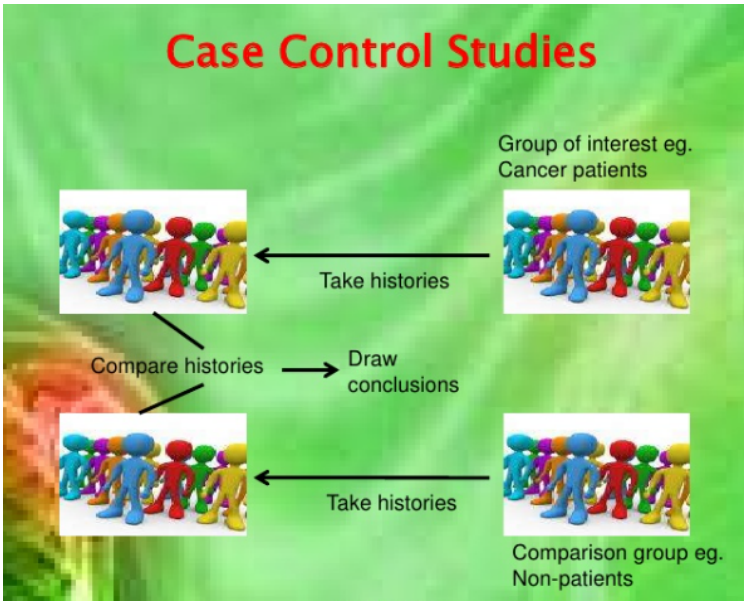
	Disease	Non-diseased
Exposed	a	b
Non-exposed	c	d
	a+c	b+d

Prevalence of exposure compared in diseased & non-diseased

$\frac{a}{a+c}$ Vs. $\frac{b}{b+d}$

	Disease Status		
	Yes	**No**	**Total**
Exposed	**A**	**B**	**A + B**
Not Exposed	**C**	**D**	**C + D**
	A + C	**B + D**	**N**

Prevalence Ratio (PR) = $\frac{a/(a+b)}{c/(c+d)}$



Odds Ratio, which is defined as the ratio of the odds that the cases were exposed to the odds that the controls were exposed

Cohort study: **Calculate**

Then, follow to see whether **& compare**

First, identify

	Disease develops	Disease does not develop	Totals	Incidence of Disease
Exposed	a	b	a + b	a/a + b
Not exposed	c	d	c + d	c/c + d

Incidence in exposed = a/a + b

Incidence in not exposed = c/c + d

Risk among exposed = a / a + b = 0.028 = 1.61

Risk among non-exposed c /c + d 0.0174

Interpretation:

- If RR is **more than 1 exposure** is causative
- If **less than 1 exposure** is protective.
- If **equal to 1 means** that the two incidence rates are equal so the factor has no effect.

RELATIVE RISK *is the ratio of the risk of disease (or death) among people who are exposed to the risk factor, to the risk among people who are unexposed.*

Attributable risk= the amount of disease incidence that can be attributed to a specific exposure

Difference in incidence of disease between exposed and non-exposed individuals

= a/a+b – c/c+d = 0.028 – 0.0174 = 0.0106

RCT=" An epidemiological experiment in which subjects in a population are **randomly allocated** into groups, usually called study and control groups to receive and not receive an experimental preventive or therapeutic procedure, maneuver, or intervention". Gold standard" in epidemiological research

BIAS= 'a tendency of an estimate to deviate in one direction from a true value.'

A **sham** procedure, also known as placebo surgery, is a simulation of an invasive procedure that mimics the active intervention as closely as possible without harming the patient

DRUG ABUSE= HABITUAL USE OF DRUGS NOT NEEDED FOR THERAPEUTIC PURPOSES TO ELEVATE THE MOOD.

> **Frequency polygon**

Continuous Quantitative data

According to the data ,Men ages 25-34 has cholesterol levels 139.5 mh/100ml at relative frequency of 40 %

And Men with the ages 55_64 have cholesterol levels 180 mg/100ml at relative frequency of 35%

> Interpret what data is presenting walay question me sab ko confusion hau but this is what is think is right

So Idk

> How is data collected

- Polulation surveys
- Epidemiological surveillance
- Census
- Notification of diseases
- Registeration of vital events
- Hospital records
- Disease registers
- Manpower health statistics
- Other routine health related statistics

A **p-value**, or probability value, is a number that measures the likelihood of an observed difference between groups being due to chance.

A p-value measures the probability of obtaining the observed results, assuming that the null hypothesis is true. The lower the p-value, the greater the statistical significance of the observed difference. A p-value of 0.05 or lower is generally considered statistically significant.

A **chi-square test** is a statistical hypothesis test that compares observed results with expected results

Q. Null hypothesis.

Ans. –

Null Hypothesis

- *Hypothesis (H):* Is an assumption about the status of a phenomenon
- *Null Hypothesis (H_0):* In Biostatistics, when we have to prove a particular hypothesis about difference between 2 regimens, we make Null Hypothesis (For examples, If we have to prove that new treatment is better than older treatment, H_0 = new treatment is not better than older treatment)[Q].

1. Community Attack Rate Example & Formula

Formula:

$$\text{Attack Rate} = \left(\frac{\text{New cases during an outbreak}}{\text{Population at risk}}\right) \times 100$$

Example:

In a population of 1,000, 100 people develop cholera during a flood outbreak.

$$\text{Attack Rate} = \left(\frac{100}{1{,}000}\right) \times 100 = 10\%$$

Significance of Attack Rate

1. **Measures Spread in Outbreaks** – Helps identify how quickly a disease spreads in a specific population.
2. **Evaluates Risk** – Determines the proportion of people at risk who become ill.
3. **Helps in Outbreak Control** – Useful in identifying high-risk groups and planning interventions.
4. **Comparison of Epidemics** – Allows comparison between different outbreaks or geographic areas.

2. Cross-Sectional Study Drawback

- **No Causal Relationship:** Cannot determine cause and effect, only association.
- **Snapshot in Time:** Provides data at one point, missing changes over time.
- **Recall Bias:** Relies on participants' memory, leading to inaccurate data

3. How Prevalence is Affected by Incidence and Duration

Relationship Formula:

Prevalence=Incidence×Duration

Explanation:

- **High Incidence + Long Duration = High Prevalence** (e.g., Diabetes)
- **High Incidence + Short Duration = Low Prevalence** (e.g., Common Cold)

- **Low Incidence + Long Duration = Moderate Prevalence**

🦋: Sensitivity and specificity calculation

🦋 1. Sensitivity and Specificity Calculation

Formulas:

- **Sensitivity** = $\frac{\text{True Positives}}{\text{True Positives} + \text{False Negatives}} \times 100$
  → Ability to detect those with the disease.
- **Specificity** = $\frac{\text{True Negatives}}{\text{True Negatives} + \text{False Positives}} \times 100$
  → Ability to detect those without the disease.

Example:

If 80 out of 100 diseased people test positive (True Positives), and 90 out of 100 healthy people test negative (True Negatives):

- **Sensitivity** = $\frac{80}{80+20} \times 100 = 80\%$
- **Specificity** = $\frac{90}{90+10} \times 100 = 90\%$

Z score

Z-Score Definition

A **Z-score** (or standard score) indicates how many **standard deviations** a value is away from the **mean**. It helps compare individual data points to a standard distribution.

Key Points:

- **Positive Z-score**: Value is **above the mean**.
- **Negative Z-score**: Value is **below the mean**.
- **Z = 0**: Value is **exactly at the mean**.

Example:
If the mean height in a population is 170 cm with a standard deviation of 10 cm, a person with a height of 180 cm has a Z-score of:
Z=180−170/10=+1Z = **Interpretation:** The person is 1 standard deviation above the mean height

2. Z-Score Calculation

Formula:

$Z = \frac{X - \mu}{\sigma}$

Where:

- X = Observed value
- μ = Mean
- σ = Standard deviation

Example:

For a score of 85, with $\mu = 70$ and $\sigma = 10$:

$Z = \frac{85 - 70}{10} = 1.5$

This means the score is **1.5 standard deviations above the mean**.

Standardization

3. Standardization (Direct Method)

Used to **remove the effect of differences** in population age structures.

Steps:

1. **Calculate Age-Specific Rates** for the population.
2. Multiply each rate by the standard population.
3. Sum the expected cases and divide by the total standard population to get the standardized rate.

Example: Age-standardized mortality rates.

Conventinal probabilty

4. Conventional Probability

Formula:

$$P(E) = \frac{\text{Number of favorable outcomes}}{\text{Total number of outcomes}}$$

Example:

In tossing a die, the probability of rolling a 4 is:

$P(4) = \frac{1}{6}$

- ratio and proportion definition

Difference Between Ratio and Proportion

Aspect	Ratio	Proportion
Definition	Compares two quantities; numerator is not part of the denominator.	Compares a part to the whole; numerator is part of the denominator.

Expression	Expressed as a fraction, colon (e.g., 2:1), or decimal.	Expressed as a fraction, percentage, or decimal.
Example	Male-to-female ratio in a class (20 males, 10 females) = 2:1.	Proportion of females in a class of 30 students = 1030×100=33.3%\frac{10}{30} \times 100 = 33.3\%.
Use	Used to compare two separate quantities.	Used to show how much of the whole is represented by a part.

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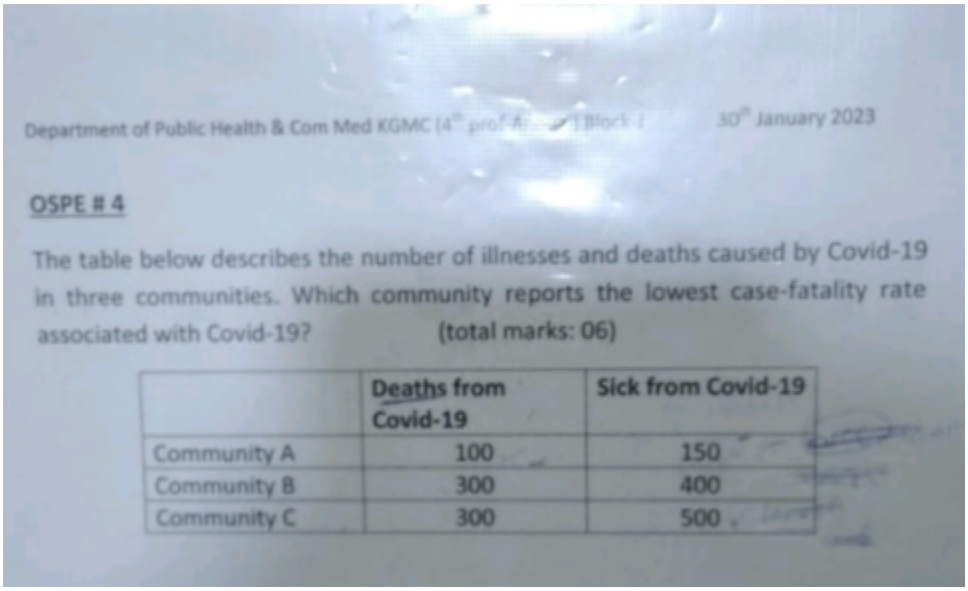


Department of Public Health & Com Med KGMC (4th prof A... Block) 30th January 2023

OSPE # 4

The table below describes the number of illnesses and deaths caused by Covid-19 in three communities. Which community reports the lowest case-fatality rate associated with Covid-19? (total marks: 06)

	Deaths from Covid-19	Sick from Covid-19
Community A	100	150
Community B	300	400
Community C	300	500

MEDICINE

- cranial nerve examination

Examinations

1. Cranial Nerve Examination

- Full cranial nerve assessment (see above for detailed steps).
- Common pathologies: Stroke, Multiple Sclerosis (MS), Brainstem lesions.

Cranial Nerve	Function	How to Test	Abnormal Findings
I - Olfactory	Smell	Ask patient to identify a familiar smell (e.g., coffee, peppermint) with each nostril separately	Loss of smell (anosmia) – common in head trauma, Parkinson's disease
II - Optic	Vision	- Test visual acuity (Snellen chart) - Visual fields by confrontation - Fundoscopy to inspect the optic disc	Visual field defects (hemianopia), optic atrophy, papilledema
III - Oculomotor	Eye movement (superior, inferior, medial), pupil constriction	- Check for ptosis - Test eye movements (H-pattern) - Pupillary light reflex and accommodation	Ptosis, dilated pupil, impaired eye movement (down and out)
IV - Trochlear	Eye movement (superior oblique – down and in)	Test eye movements (ask the patient to follow your finger downward and inward)	Diplopia (difficulty looking down), head tilt to compensate
V - Trigeminal	Facial sensation, mastication	- Test sensation in 3 areas: ophthalmic (V1), maxillary (V2), mandibular (V3) - Test corneal reflex - Ask patient to clench jaw and palpate masseter and temporalis muscles	Loss of sensation, absent corneal reflex, jaw deviation
VI - Abducens	Eye movement (lateral rectus – lateral gaze)	Test lateral eye movement (ask the patient to follow your finger laterally)	Diplopia, inability to move eye laterally
VII - Facial	Facial expression, taste (anterior 2/3 of tongue)	- Ask patient to raise eyebrows, close eyes tightly, smile, puff cheeks - Taste test (sweet, salty) on anterior tongue	Facial asymmetry, loss of taste, Bell's palsy (LMN), central facial palsy (UMN)
VIII - Vestibulocochlear	Hearing, balance	- Whisper test or tuning fork (Rinne and Weber tests) - Test balance (Romberg test)	Hearing loss (sensorineural vs conductive), vertigo, nystagmus
IX - Glossopharyngeal	Taste (posterior 1/3), swallowing, gag reflex	- Test gag reflex - Ask patient to say "Ah" and observe the uvula	Absent gag reflex, uvula deviates away from the lesion
X - Vagus	Swallowing, speech	- Test gag reflex - Ask patient to speak and check for hoarseness	Dysphagia, dysphonia, absent gag reflex
XI - Accessory	Shoulder shrug, head turn	- Ask patient to shrug shoulders and turn head against resistance	Weakness in shrugging shoulders or turning head
XII - Hypoglossal	Tongue movement	Ask patient to protrude tongue and move it side to side	Tongue deviates toward the lesion (LMN), fasciculations

Tips for OSCE/OSPE

- Always follow a **systematic approach** (cranial nerves in order).
- Explain each step to the patient.
- Document any abnormalities and correlate with possible causes.

2. Upper and Lower Limb Neurological Examination

- **Inspection**: Muscle wasting, fasciculations, tremors.
- **Tone**: Assess for spasticity or rigidity.
- **Power**: Test muscle strength (0-5 scale).
- **Reflexes**: Biceps, triceps, knee, ankle, Babinski's sign.
- **Sensation**: Light touch, pinprick, vibration, proprioception.
- **Coordination**: Finger-nose test, heel-shin test, rapid alternating movements.
- Common conditions: Stroke, peripheral neuropathy, Parkinson's, spinal cord lesion.

3. Cerebellar Examination

- **Signs**: Dysdiadochokinesia, past pointing, nystagmus, intention tremor, ataxic gait.
- Common causes: Stroke, alcohol intoxication, cerebellar tumor.

4. Gait Assessment

- **Observe**: Normal, hemiparetic (stroke), spastic (cerebral palsy), ataxic (cerebellar), shuffling (Parkinson's).

5. Mental Status Examination (MSE)

- Assess **orientation, memory, attention, mood**, and **thought processes**.
- Used for: Dementia, delirium, psychiatric disorders.

The **Mini-Mental State Examination (MMSE)** is a widely used tool for screening **cognitive impairment**. It assesses functions such as **orientation, memory, attention**, and **language**. The maximum score is **30 points**

MMSE Structure and Scoring

1. Orientation (10 points)

- **Time (5 points)**: Ask for the year, season, date, day, and month. (1 point each)

  Time Orientation (5 points)

  - *"What is today's date?"*
  - *"Can you tell me what season it is?"*
  - *"What day of the week is it?"*
  - *"Which month are we in?"*
  - *"What year is it?"*

- **Place (5 points)**: Ask for the country, city, hospital/clinic, floor, and specific location. (1 point each)

    "Where are we right now?" (Country, City, Hospital/Clinic, Floor, and Room)

2. Registration (3 points)

- Name **3 unrelated objects** (e.g., "apple, table, penny").
- Ask the patient to repeat them. (1 point per correct repetition)

    You: "I will say three words. Please repeat them after me: apple, table, penny."
    (Score 1 point for each correct repetition. Repeat up to five times if needed.)

3. Attention and Calculation (5 points)

- **Serial 7s**: Ask the patient to subtract 7 from 100 five times (93, 86, 79, 72, 65). (1 point per correct answer)
  OR
- **Spell "WORLD" backward** (DLROW). (1 point per correct letter in sequence)

You: *"Now, subtract 7 from 100 and keep subtracting 7 five times."* (93, 86, 79, 72, 65)

4. Recall (3 points)

- Ask the patient to recall the 3 objects mentioned earlier. (1 point per correct answer)

You: *"Can you tell me the three words I mentioned earlier?"* (apple, table, penny)

5. Language (8 points)

- **Naming (2 points)**: Show a pen and a watch; ask the patient to name them.
- **Repetition (1 point)**: Ask the patient to repeat, "No ifs, ands, or buts."
- **3-Stage Command (3 points)**: Ask the patient to follow this: "Take this paper, fold it in half, and place it on the floor."
- **Reading (1 point)**: Show a written command: "Close your eyes." The patient must read and obey.
- **Writing (1 point)**: Ask the patient to write a sentence.

Naming (2 points)*: "What is this?" (Show a pen and a watch)*
Repetition (1 point)*: "Please repeat after me: No ifs, ands, or buts."*
3-Stage Command (3 points)*: "Take this paper, fold it in half, and place it on the floor."*
Reading (1 point)*: "Please read this and do what it says: 'Close your eyes.'"*
Writing (1 point)*: "Please write a complete sentence on this paper."*

6. Visuospatial (1 point)

Ask the patient to copy a drawing of two overlapping pentagons. (1 point if correctly done)

"Please copy this drawing of two overlapping pentagons."

Scoring Interpretation

- **25–30**: Normal
- **20–24**: Mild cognitive impairment
- **10–19**: Moderate cognitive impairment
- **<10**: Severe cognitive impairment

- COUNSELLING

STEPS OF COUSELLING

- **Active Listening**: Attentive listening ensures understanding and builds rapport with clients, vital for effective counseling.
- **Empathy**: Demonstrating understanding and compassion helps clients feel validated and supported.
- **Nonjudgmental Attitude**: Creating a safe, accepting environment encourages clients to share openly.
- **Paraphrasing**: Restating the client's words shows understanding and encourages clarification.
- **Funneling**: Navigating from broad to specific topics helps explore issues systematically and thoroughly.

Counselling

Definition: A process where a trained professional helps individuals explore and manage personal, emotional, or psychological challenges to improve well-being.

Types of Counselling

1. **Psychological Counselling**: Mental health issues like anxiety or depression.
2. **Career Counselling**: Guidance for career decisions.
3. **Family/Marriage Counselling**: Resolving family conflicts.
4. **Rehabilitation Counselling**: For addiction recovery or chronic illness support.
5. **Grief Counselling**: Coping with loss.

Counselling Techniques

1. **Active Listening** – Fully focus on what the client says without judgment.
2. **Empathy** – Understanding and sharing the client's feelings.
3. **Open-ended Questions** – Encourage the client to express thoughts and feelings.
    - Example: *"Can you tell me more about what's troubling you?"*
4. **Reflection** – Repeat or paraphrase what the client says to clarify feelings.
5. **Cognitive Behavioral Therapy (CBT)** – Help clients identify and change negative thought patterns.
6. **Solution-Focused Approach** – Focus on finding solutions rather than dwelling on problems.

- **Sympathy**: Feeling *for* someone's pain (pity).
- **Empathy**: Feeling *with* someone's pain (understanding).

- **Open-ended questions**: Encourage detailed responses.
  Example: "How do you manage stress?"
- **Closed-ended questions**: Require brief, specific answers (Yes/No or short).
  Example: "Do you exercise daily?"

Rapport building is creating a connection with someone through trust, understanding, and empathy. It helps patients feel comfortable and open in clinical settings.

Steps for Effective Rapport Building

1. **Greet warmly**: *"Hello, how are you today?"*
2. **Introduce yourself**: *"I'm Dr. Fatima. I'll be taking care of you."*
3. **Active listening**: Nod, maintain eye contact, and don't interrupt.
4. **Show empathy**: *"I understand this must be difficult."*
5. **Ask open-ended questions**: *"Can you tell me more about how you're feeling?"*
6. **Be non-judgmental** and respectful.

Nutritional Neuropathies are nerve disorders caused by vitamin deficiencies.

Common Causes & Associated Deficiencies

- **Vitamin B1 (Thiamine)**: Beriberi → Peripheral neuropathy, muscle weakness
- **Vitamin B6 (Pyridoxine)**: Sensory neuropathy (high doses), irritability, seizures
- **Vitamin B12 (Cobalamin)**: Subacute combined degeneration → Ataxia, numbness, weakness
- **Vitamin E**: Ataxia, loss of proprioception, hyporeflexia
- **Copper**: Myelopathy, spastic gait (resembles B12 deficiency)
- **Folate**: Similar to B12 but without neurological signs

Rabies prophylaxis

Rabies Prophylaxis Treatment Regimen

Category	Details
Post-Exposure Prophylaxis (PEP)	
1. Wound Cleaning	Immediately clean the bite or scratch with **soap and water for at least 15 minutes**; apply **iodine** or alcohol.
2. Rabies Vaccine	**Rabies Vaccine Human Diploid Cell Vaccine [HDCV]**
	4 doses (**1 m**l) given on days **0, 3, 7, 14, 28 and sometimes 90** (can be adjusted for immunocompromised individuals). Intramusclar/ Deltoid Muscle
	CAN BE GIVEN TO PREGNANT/ BREAST FEEDING.

3. Rabies Immune Globulin (RIG)- antibody serum	**RIG** should be administered on day 0.
	- 20 IU/kg body weight (given in and around the wound site, and the remainder intramuscular).
	- If the patient has previously received PrEP, RIG is not required, but the vaccine schedule remains.
Pre-Exposure Prophylaxis (PrEP)	
1. Rabies Vaccine	Administered in **3** doses (1 ml each) on days 0, 7, and 21 or 28.
2. Booster Dose	A booster dose is required every 2-3 years for high-risk individuals, depending on exposure.

Psychiatry

Delusion vs illusion

- Dellusion are fixed, **false beliefs** in absence of external stimulus
- Illusions are **misinterpretation** of real stimuli

Types of delusions:

1. **Grandiose Delusion**
   - Belief of having exceptional power, wealth, or fame.
   - Example: "I am a famous inventor who created the internet."
2. **Erotomanic Delusion**
   - Belief that someone (usually of higher status) is in love with them.
   - Example: "That celebrity is secretly sending me messages."
3. **Delusion of Infidelity (Jealous Delusion)**
   - Belief that a partner is unfaithful without any proof.
   - Example: "My spouse is cheating every time they leave the house."
4. **Delusion of Control**
   - Belief that thoughts, actions, or feelings are controlled by an external force.
   - Example: "Aliens are controlling my movements."
5. **Persecutory Delusion**
   - Belief that one is being targeted, harassed, or conspired against.
   - Example: "My coworkers are plotting to harm me."
6. **Somatic Delusion**
   - Belief of having a physical illness or defect despite no medical evidence.

Alkaloid poisoning is an illness formed from the excess consumption of leafy greens or animal products containing alkaloids. Alkaloids are chemical substances that contain at least one nitrogen atom.

- Atropine
- Strychnine
- Nicotine
- Caffeine
- Morphine

- Cocaine
- Aconitine

Here's a **concise overview** of each compound:

1. Atropine

- **Source:** Belladonna (Atropa belladonna)
- **Mechanism:** Anticholinergic (blocks muscarinic receptors)
- **Effect:** Dilated pupils, dry mouth, tachycardia, hallucinations
- **Use:** Antidote for organophosphate poisoning

2. Strychnine

- **Source:** Nux Vomica seeds
- **Mechanism:** Glycine receptor antagonist in the spinal cord
- **Effect:** Severe convulsions, opisthotonos, respiratory failure
- **Fatal Dose:** 30–60 mg

3. Nicotine

- **Source:** Tobacco leaves (Nicotiana tabacum)
- **Mechanism:** Stimulates nicotinic receptors; biphasic action (stimulation → paralysis)
- **Effect:** Increased heart rate, tremors, seizures, respiratory depression
- **Use:** Smoking cessation therapies

4. Caffeine

- **Source:** Coffee, tea, cola nuts
- **Mechanism:** Adenosine receptor antagonist; increases dopamine release
- **Effect:** Stimulation, increased alertness, tachycardia, insomnia
- **Toxic Dose:** >500 mg (can cause seizures and arrhythmias)

5. Morphine

- **Source:** Opium poppy (Papaver somniferum)
- **Mechanism:** Opioid receptor agonist (mu-receptor)
- **Effect:** Analgesia, respiratory depression, miosis, sedation
- **Use:** Pain relief in severe conditions

6. Cocaine

- **Source:** Coca plant (Erythroxylum coca)
- **Mechanism:** Blocks dopamine and norepinephrine reuptake
- **Effect:** Euphoria, tachycardia, hypertension, hyperthermia
- **Use:** Local anesthetic (limited use), recreational drug

7. Aconitine

- **Source:** Monkshood (Aconitum species)
- **Mechanism:** Opens sodium channels, causing persistent depolarization
- **Effect:** Tingling, vomiting, arrhythmias, respiratory paralysis
- **Fatal Dose:** 2–5 mg

"insanity" refers to a mental state in which an individual is unable to understand the nature and consequences of their actions or to differentiate between right and wrong due to a severe mental disorder or illness.

Testimonial capacity refers to an individual's ability to provide sworn testimony in a legal or civil proceeding

Testimonial Capacity and Its Rules

1. **Definition**
   Testimonial capacity refers to the **ability of a witness to provide evidence** in a legal proceeding. The witness must understand the **nature and significance of the questions** asked and provide **rational and relevant answers**.
2. **Legal Criteria for Testimonial Capacity**
   - **Section 118 of the Indian Evidence Act:** A witness must have the ability to comprehend the nature of the questions and respond rationally.
   - **Exceptions:** A person may not be competent to testify if they suffer from:
     - **Mental illness** that prevents rational understanding.
     - **Intoxication or unsoundness of mind** at the time of testimonyes and Guidelines**
   - **Competence to Testify:**
     - Mentally ill persons can testify during **lucid intervals**.
     - Minors may testify if they understand the nature of the questions.
   - **Cross-examination:**
     - Designed to **challenge the credibility** of the witness by exposing contradictions.
     - Leading questions are allowed during cross-examination.
   - **Privilege:**
     - Professional secrets may be revealed only if **ordered by the court** .

PATHOLOGY

4 major classes of brain tumors

- The **Gliomas** (Astrocytomas {Fibrillary astrocytoma, Glioblastoma, Pilocytic astrocytoma, and Pleomorphic xanthoastrocytoma}, Oligodendrogliomas, and Ependymoma)
- **Neuronal tumors**
- **Poorly differentiated neoplasms**
- **Meningiomas**

1. A 54 year old male presented with one month history of headache and blurred vision. One day before he came to hospital, he had a seizure as well. On investigations he was found to have a CNS tumor. The surgeon resected it completely and sent it for biopsy. Following are the gross and microscopic appearance of the tumor. Considering these diagrams answer the following questions.

1) What is your diagnosis? (1)
2) Briefly explain the grades of this tumor?(WHO grading system) (3)
3) Write one gross and one microscopic feature of the tumor? (2)

> **Meningioma**

- Grade1-meningioma
- Grade2-atypical meningioma
- Grade3-anaplastic

- Microscopic: psamomma bodies
- Gross: firm, well circumscribed, grey/pink/white

Tumor Type	Age Group	Location	Imaging Features	Histological Features	Key Symptoms
Glioblastoma	Adults	Supratentorial	Ring-enhancing, **butterfly** lesion	Pseudopalisading **necrosis**, vascular proliferation	Headache, seizures, focal deficits
Meningioma	Adults	Extra-axial	Well-circumscribed, dural attachment	Whorled pattern, **psammoma bodies**	Often asymptomatic or compressive
Oligodendroglioma	Adults	Frontal lobe	Calcifications, well-demarcated lesion	**Fried egg appearance**, chicken wire vasculature	Seizures
Ependymoma	Children/Young Adults	Ventricles (4th)	Well-demarcated, intraventricular	Perivascular pseudorosettes	Hydrocephalus, ataxia
Medulloblastoma	Children	Cerebellum	Hyperdense on CT, midline mass	Small round blue cells, **Homer-Wright rosettes**	Ataxia, raised ICP
Pilocytic Astrocytoma	Children	Cerebellum	Cystic lesion with **mural nodule**	**Rosenthal fibers**, eosinophilic corkscrew fibers	Ataxia, raised ICP
Craniopharyngioma	Children	Suprasellar	Cystic lesion with calcifications	Cystic spaces, cholesterol crystals	**Endocrine symptoms**, visual loss

Astrocytoma is a **type of brain tumor** that arises from **astrocytes**, the star-shaped glial cells in the brain and spinal cord. These tumors can be **benign or malignant** and are classified based on their **histological grade** and **degree of malignancy**.

Classification (WHO Grades):

- **Grade I**: Pilocytic Astrocytoma (low-grade, mostly in children)
- **Grade II**: Diffuse Astrocytoma (low-grade, slow-growing)
- **Grade III**: Anaplastic Astrocytoma (high-grade, more aggressive)
- **Grade IV**: Glioblastoma (most aggressive, poor prognosis)

Symptoms:

Depends on the location but can include:

- Headache
- Seizures

- Cognitive changes
- Neurological deficits

LOCATION OF ASTROCYTOMA

Astrocytomas can occur in various parts of the brain, depending on the **grade and type**. Here's a breakdown of common locations:

1. Low-Grade Astrocytomas (Grade I & II)

- **Cerebellum** – Pilocytic Astrocytoma (common in children)
- **Cerebral Hemispheres** – Diffuse Astrocytoma
- **Optic Pathway/Hypothalamus** – Seen in children, often associated with NF1

2. High-Grade Astrocytomas (Grade III & IV)

- **Cerebral Hemispheres** – Most common for Anaplastic Astrocytoma and Glioblastoma
- **Brainstem** – Particularly in diffuse intrinsic pontine gliomas (DIPG)
- **Spinal Cord** – Rare, but can occur

Schwannoma vs **Neurofibroma**:

Feature	Schwannoma	Neurofibroma
Cell of Origin	Schwann cells	Schwann cells + fibroblasts
Encapsulation	Encapsulated, well-circumscribed	Non-encapsulated, diffuse
Location	Peripheral nerves, commonly CN VIII (vestibular nerve)	Peripheral nerves, skin, plexiform in NF1
Association with NF1	Rarely associated with NF1	Strong association with NF1
Histology	Antoni A (cellular) and Antoni B (myxoid) areas	Mixed cell types, wavy nuclei
Malignant Potential	Rare	Can transform into MPNST (in NF1)
Symptoms	Tinnitus, hearing loss (if vestibular)	Soft, painless mass, neuro deficits
Immunohistochemistry	S-100 positive	S-100 positive but less intense
Treatment	Surgical resection	Surgery (depending on size/location)

Antoni A and Antoni B areas are histological patterns seen in **schwannomas**. Here's what they mean:

Antoni A Areas

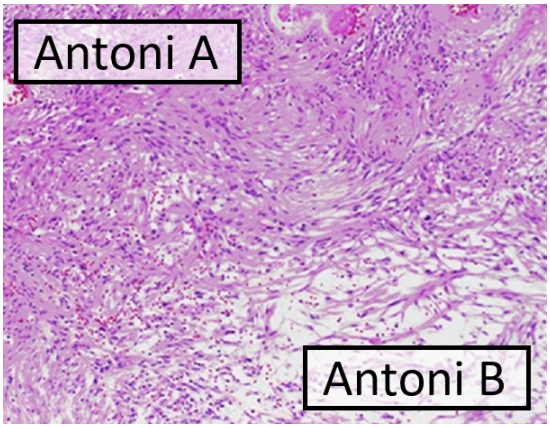
- **Dense, cellular areas**
- Spindle-shaped Schwann cells arranged in **palisading patterns** (Verocay bodies)

- High nuclear content and compact appearance

Antoni B Areas

- **Loose, myxoid areas** with fewer cells
- Contains **microcysts**, edema, and disorganized cells
- More vacuolated and less structured

These patterns help distinguish **schwannomas** from other nerve sheath tumors like **neurofibromas**, which lack these distinctive Antoni areas.



> Epilepsy

Type of Seizure	Category	Key Clinical Features	EEG Findings	Common Causes
Focal (Partial) Seizures	Localized			
- Focal Aware Seizures	Simple Partial	- No loss of consciousness.	Focal spikes or sharp waves in the affected region.	Structural brain lesions (tumor, stroke), cortical dysplasia.
- Focal Impaired Awareness Seizures	Complex Partial	- Altered consciousness, automatisms (lip-smacking, picking movements), postictal confusion.	Focal slowing or spikes in **temporal/frontal lobe**.	Temporal lobe epilepsy, mesial temporal sclerosis.
Generalized Seizures	Bilateral			
- Tonic-Clonic Seizures	Grand Mal	- Sudden loss of consciousness, tonic (stiffening) phase, followed by clonic (jerking) movements, postictal state.	Generalized polyspike-and-slow wave discharges.	Hypoxia, metabolic disturbances, withdrawal (alcohol, benzodiazepines).

- Absence Seizures	Petit Mal	- Brief **staring** episodes, unresponsive, no postictal state; common in children.	3 Hz generalized spike-and-wave pattern.	Idiopathic (childhood epilepsy syndromes).
- Myoclonic Seizures		- Sudden, brief jerks of muscles, often bilateral, no loss of consciousness.	Generalized spike-and-wave or polyspike discharges.	Juvenile myoclonic epilepsy, metabolic disorders.
- Atonic Seizures	Drop Attacks	- Sudden loss of muscle tone, leading to falls; brief, no postictal confusion.	Generalized slow spike-and-wave activity.	Lennox-Gastaut syndrome, brain injury.
- Tonic Seizures		- Sudden stiffening of muscles, often during sleep; can lead to falls.	Generalized fast activity or electrodecrement.	Structural brain lesions, Lennox-Gastaut syndrome.
Febrile Seizures	Pediatric	- Occur with fever in children (6 months–5 years); generalized tonic-clonic, no underlying CNS infection.	Normal or nonspecific EEG.	Fever, genetic predisposition.
Status Epilepticus	Emergency	- **Continuous seizure activity (>5 minutes) or repeated seizures without recovery between them**.	Generalized or focal epileptiform activity.	Poorly controlled epilepsy, acute CNS insult, metabolic disturbances.

Depression

Migraine

Stroke

Type of Stroke	Vascular Territory	Key Clinical Features	Imaging Findings	Common Causes
Ischemic Stroke	**Middle Cerebral Artery (MCA)**	- Contralateral hemiparesis (**face/arm** > leg), hemisensory loss, aphasia (dominant hemisphere), neglect (non-dominant).	CT: Hypodense region; MRI: DWI hyperintensity	Atherosclerosis, embolism (e.g., AF).
	Anterior Cerebral Artery (ACA)	- Contralateral **leg** weakness/sensory loss, abulia, urinary incontinence.	CT: Hypodense in medial frontal region	Atherosclerosis, embolism.

	Posterior Cerebral Artery (PCA)	- Contralateral homonymous hemianopia, thalamic pain syndrome, memory impairment.	CT/MRI: Lesion in occipital lobe or thalamus	Embolism, vertebrobasilar atherosclerosis.
	Lacunar Stroke (small vessels)	- Pure motor (internal capsule), pure sensory (thalamus), or ataxic hemiparesis.	CT: Small hypodense lesion; MRI: DWI hyperintensity	Hypertension, lipohyalinosis.
Hemorrhagic Stroke	**Intracerebral Hemorrhage (ICH)**	- Sudden onset focal neurological deficits, headache, vomiting, reduced consciousness.	CT: Hyperdense region; mass effect.	Hypertension, amyloid angiopathy.
	Subarachnoid Hemorrhage (SAH)	- "Thunderclap" headache, nuchal rigidity, photophobia, vomiting, loss of consciousness.	CT: Blood in cisterns/sulci; CTA: aneurysm.	Ruptured aneurysm, AVM.
Brainstem Stroke	**Basilar Artery**	- Quadriparesis, **locked-in syndrome** (preserved vertical eye movements), cranial nerve abnormalities.	MRI: Brainstem infarction	Thrombus, vertebrobasilar occlusion.
	Vertebral Artery/PICA	- Lateral medullary (**Wallenberg) syndrome**: Dysphagia, ataxia, ipsilateral Horner's syndrome, contralateral sensory loss.	MRI: Infarction in medulla/cerebellum.	Dissection, atherosclerosis.
Venous Stroke	**Cerebral Venous Sinus Thrombosis (CVST)**	- Headache, seizures, focal deficits, papilledema.	MRI/MRV: Empty delta sign, sinus thrombosis.	Hypercoagulable states (pregnancy, OCPs).

Causes of stroke

- Ischemic
- Thrombus
- Emboli
- Hypertension
- Hemorhagic
- Aneurysm
- Av malformation
- Anticoagulation drug

Types of Stroke

1. **Ischemic Stroke**: Caused by blocked blood flow to the brain.
2. **Hemorrhagic Stroke**: Caused by bleeding in or around the brain.

Symptoms of Stroke

- Sudden **weakness** of limbs
- Loss of **sensation**
- **Loss of coordination**
- **Headache** (thunderclap headache in hemorrhagic stroke)
- **Loss of consciousness**
- **Dysphagia** (difficulty swallowing)
- Sudden **loss of vision**
- **Bell's Palsy** (facial paralysis)

Types of Paralysis

1. **Quadriplegia**: Paralysis of all four limbs, sparing the face
2. **Hemiplegia**: Paralysis of one side of the body
3. **Paraplegia**: Paralysis of both lower limbs

Diagnosing Hemorrhagic vs. Ischemic Stroke

1. **Clinical History**: Sudden onset, "worst headache of my life" suggests hemorrhagic stroke.

    - **Ischemic stroke**: Sudden weakness, numbness, loss of coordination.
    - **Hemorrhagic stroke**: Severe headache (thunderclap), vomiting, altered consciousness.

2. **Examination**: Check for signs of raised intracranial pressure.
3. **Non-contrast CT Scan**: Key to differentiate hemorrhagic from ischemic stroke.
4. **Response to Thrombolytic Therapy**: Worsening symptoms may indicate hemorrhage.

6. Decision-Making & Management

- **Ischemic Stroke**: Thrombolysis with tPA if within 4.5 hours of onset.
- **Hemorrhagic Stroke**: Control blood pressure, manage intracranial pressure, and refer to neurosurgery if needed.

Cope and Counter-Cope Injuries refer to specific patterns of brain injury caused by trauma.

Cope Injury

- **Definition**: The **primary impact** injury where the brain hits the inner surface of the skull.
- **Location**: Occurs at the **site of impact**.
- **Example**: Frontal lobe contusion if the forehead hits a hard surface.

Counter-Cope (Countercoup) Injury

- **Definition**: The **secondary impact** injury where the brain recoils and hits the opposite side of the skull.
- **Location**: **Opposite the site of impact**.
- **Example**: Occipital lobe injury after a frontal head impact.

Common Causes

- Falls
- Motor vehicle accidents
- Assaults

MENINGITIS

- ➢ 1 bacterial meningitis
- 2 in viral normal glucose levels
- While in bacterial low glucose
- Bacterial neutrophils
- Viral lymphocytes

Type of Meningitis	Etiology	Key Clinical Features	Investigations	Treatment
Bacterial Meningitis	- *Neonates (birth-28days)*: Group B Streptococcus, ***E. coli***, *Listeria*.	- Fever, headache, neck stiffness, altered mental status.	- CSF: ↑WBC (**neutrophilic**), **↓glucose**, ↑protein.	- Empiric antibiotics (e.g., ceftriaxone + vancomycin ± ampicillin for *Listeria*).
	- *Children/Adults*: *S. pneumoniae*, *N. meningitidis*.	- Photophobia, vomiting, seizures.	- Gram stain and culture.	- Corticosteroids (dexamethasone) for pneumococcal meningitis.
	- *Elderly/Immunosuppressed*: *S. pneumoniae*, *Listeria*.	- Kernig and Brudzinski signs positive.	- Blood cultures.	
Viral Meningitis	- *Enteroviruses* (e.g., coxsackievirus), HSV-2, VZV, HIV.	- Fever, headache, photophobia, meningismus.	- CSF: ↑WBC (**lymphocytic**), **normal glucose**, mild ↑ protein.	- Supportive care (analgesics, antipyretics).
		- Typically milder than bacterial meningitis.	- PCR for viral DNA/RNA (e.g., HSV, enterovirus).	- Acyclovir for suspected HSV meningitis.

Fungal Meningitis	- *Cryptococcus neoformans* (common in HIV/AIDS), *Candida*.	- Subacute onset; fever, headache, confusion.	- CSF: ↑WBC (lymphocytic), ↓ glucose, ↑ protein.	- Amphotericin B + flucytosine followed by fluconazole (for *Cryptococcus*).
Tuberculous Meningitis	- *Mycobacterium tuberculosis*.	- Gradual onset; headache, fever, weight loss, cranial nerve palsies.	- CSF: ↑WBC (lymphocytic), ↓ glucose, ↑ protein; AFB stain and culture.	- RIPE therapy (rifampin, isoniazid, pyrazinamide, ethambutol) + corticosteroids.
Parasitic Meningitis	- *Naegleria fowleri* (primary amoebic meningoencephalitis), *Toxoplasma gondii* (immunosuppressed).	- Rapidly progressive headache, fever, altered mental status (PAM).	- CSF: ↑WBC (eosinophilic/lymphocytic), wet mount for amoebae.	- Amphotericin B (PAM); pyrimethamine + sulfadiazine for toxoplasmosis.
Carcinomatous Meningitis	- Metastasis of solid tumors (e.g., breast, lung cancer) to meninges.	- Subacute onset; headache, cranial nerve deficits, back pain.	- CSF: ↑ protein, ↓ glucose, malignant cells on cytology.	- Intrathecal chemotherapy (e.g., methotrexate).

ziehl nelson stain for **TB**

Wet Preparation in CSF Examination is primarily used to detect the presence of cells, microorganisms, and certain abnormal elements in cerebrospinal fluid (CSF). It's a **rapid diagnostic method** that involves observing a drop of fresh CSF under a microscope without staining.

Steps:

1. **Collection**: CSF is collected via lumbar puncture.
2. **Preparation**: Place a drop of CSF on a glass slide and cover it with a coverslip.
3. **Microscopy**: Examine under low and high power magnification using a light microscope.

Uses and Findings:

Component	Possible Findings

Cells	Leukocytes (indicative of infection or inflammation), RBCs (trauma or hemorrhage)
Microorganisms	Fungal elements (Cryptococcus), protozoa (Naegleria fowleri), bacteria (rarely visible directly)
Parasites	Naegleria trophozoites in primary amoebic meningoencephalitis
Fungi	Cryptococcus (seen as refractile, round yeasts)

MOST IMPORTANT VIRUSES CAUSING ENCEPHALITIS:

1. **Herpes Simplex Virus (HSV-1, HSV-2)**
2. **Varicella-Zoster Virus (VZV)**
3. **Japanese Encephalitis Virus (JEV)**
4. **West Nile Virus**
5. **Enterovirus 71**
6. **Measles Virus**
7. **Rabies Virus**
8. **Cytomegalovirus (CMV)** (in immunocompromised patients)
9. **Nipah Virus**

- Epilepsy
- Depression
- Migraine

Fluoxetine is not right , wo to serotonin ki reuptake inhibit krta hai na ..

Qn mai they have asked ke konsi drug serotonin reuptake increase karti ha

Mirtazipine belongs to TCAs.Its mechanism of action is a bit unique it blocks the presynaptic adrenrgic receptors which causes increase release of NE and Serotonin

Tumor in NF1

Grading of tumor

Pilocytic astrocytoma involve which area of brain

St: Pic → Schwannoma ① Diagnosis ② Diff btw neurofibroma and schwannoma

St: ↑glial cells nuclei, nuclear pleomorphism, GFAP+ve
Fibrillary appearance, invading brain
Diagnosis
Histological features.
lesion which is common in which site?

St: CSF → technique used → LP.
Normal volume: 90-150ml
findings in CSF in bacterial meningitis
26 years old boy → bacterial meningitis
most common bacteria?
which blood cells are found in CSF in b.M?

Pharmacology

St: viva Contraindication of Tryptans
local anaesthetic?
Alcohols?
Why L-dopa is given with Carbidopa?

St
- Anti-seizure drugs
- Na+ch blocking 3d
- NMDA receptor antagonist
- GAT-1 block
- Ca+ch bl.ng

viva TCA → mechanism of Action
Which drugs doesn't cause EPS → Atypical Antidep
Malignant Neuroleptic syndrome → Bromocriptine
All of the Rest → anticholinergics
Diff in MoA of barbiturates and BZP.

St: Prescription of migraine

St: Diazepam → which class of drug
Three clinical indication. Antagonist → Flumenazil (block which recep tor?)
Drugs → Inverse agonist? β-carboline

St: Forensic Med: Nux vomica seeds
Signs & symptoms? Active principle? Medicolegal

OSPE (Neuro) → How is data collected
→ Chi-square test

Community viva (1) → How is rabies spread?
→ WHO mental illness classification → Management? → Highest risk factor for stroke
name → which Neurological disease Is No1 and No2 of the neurological diseases burden?

Viva (2) Case control study? Cohort? with examples
Breast cancer test? Prescription drug abuse (opium analgesic)

Station → stress among drivers and office workers by checking their b.p
Type of study → Cross-sectional
Why? → outcome and exposure at the same time is studied.

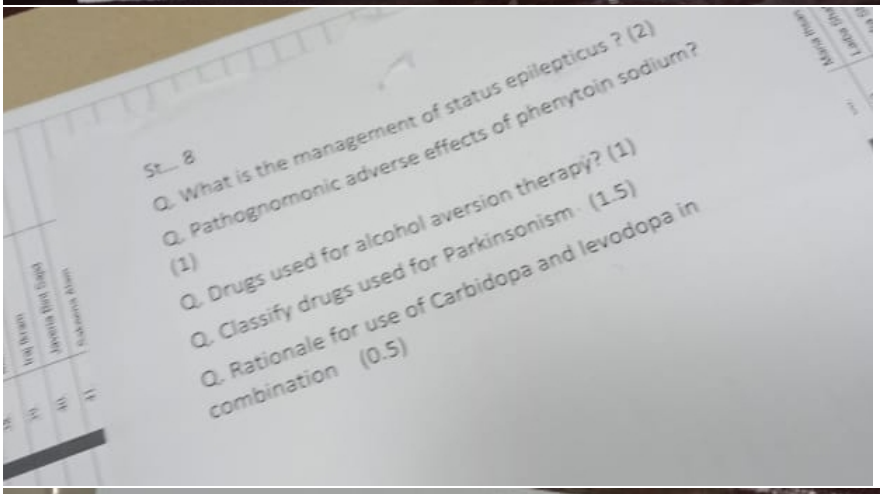
Station → team member depressed → what will you do to improve mental health
→ Seek professional help → Avoid self-isolation
→ Engage in physical activity → Engage in healthy relationships with family and friends → Self-help books
→ Motivational speeches

Student
St → Counselling → supple in final exam

Pathology
St → females 50yr, unilateral eye impairement, No tumour
Diagnosis → Multiple sclerosis
Which gene affected?

St.No 9

Write prescription for 35 yrs old salman .he suffered from depression after sad demise of his parents 6 months back in an accident.



St_ 8

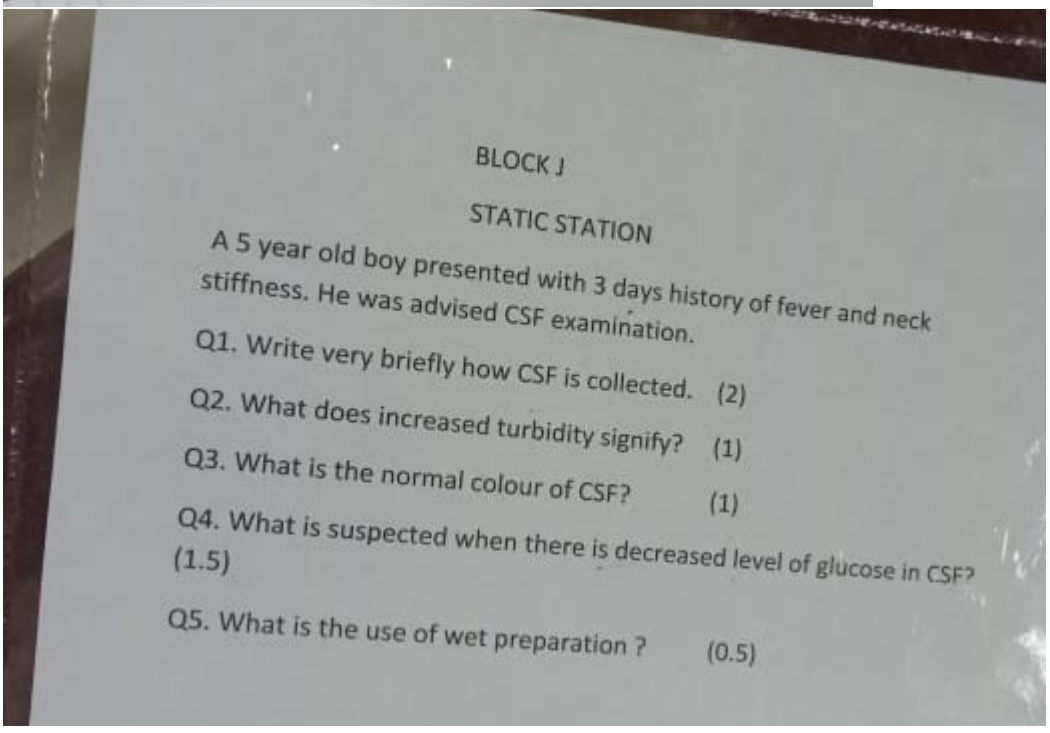
Q. What is the management of status epilepticus ? (2)

Q. Pathognomonic adverse effects of phenytoin sodium? (1)

Q. Drugs used for alcohol aversion therapy? (1)

Q. Classify drugs used for Parkinsonism (1.5)

Q. Rationale for use of Carbidopa and levodopa in combination (0.5)



BLOCK J

STATIC STATION

A 5 year old boy presented with 3 days history of fever and neck stiffness. He was advised CSF examination.

Q1. Write very briefly how CSF is collected. (2)

Q2. What does increased turbidity signify? (1)

Q3. What is the normal colour of CSF? (1)

Q4. What is suspected when there is decreased level of glucose in CSF? (1.5)

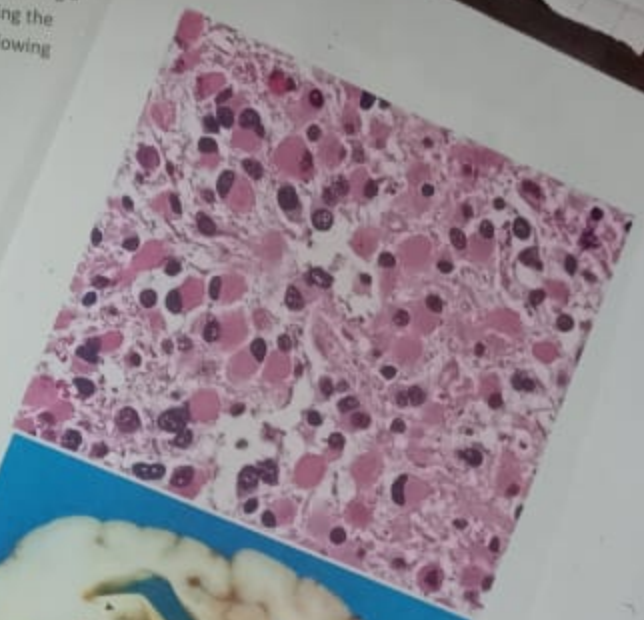
Q5. What is the use of wet preparation ? (0.5)

A 50 year old man presented with complaints of headaches since a month and he has a seizure one day back. He was diagnosed as having a tumor of brain paranchyma that was poorly defined and invading the brain tissue. Examine the photomicrograph and answer the following questions.

1 Comment on the gross morphology of the tumor. (2.5)

2 Describe the microscopic appearance of the tumor. (2.5)

3 Enlist different grades of the tumors. (1)

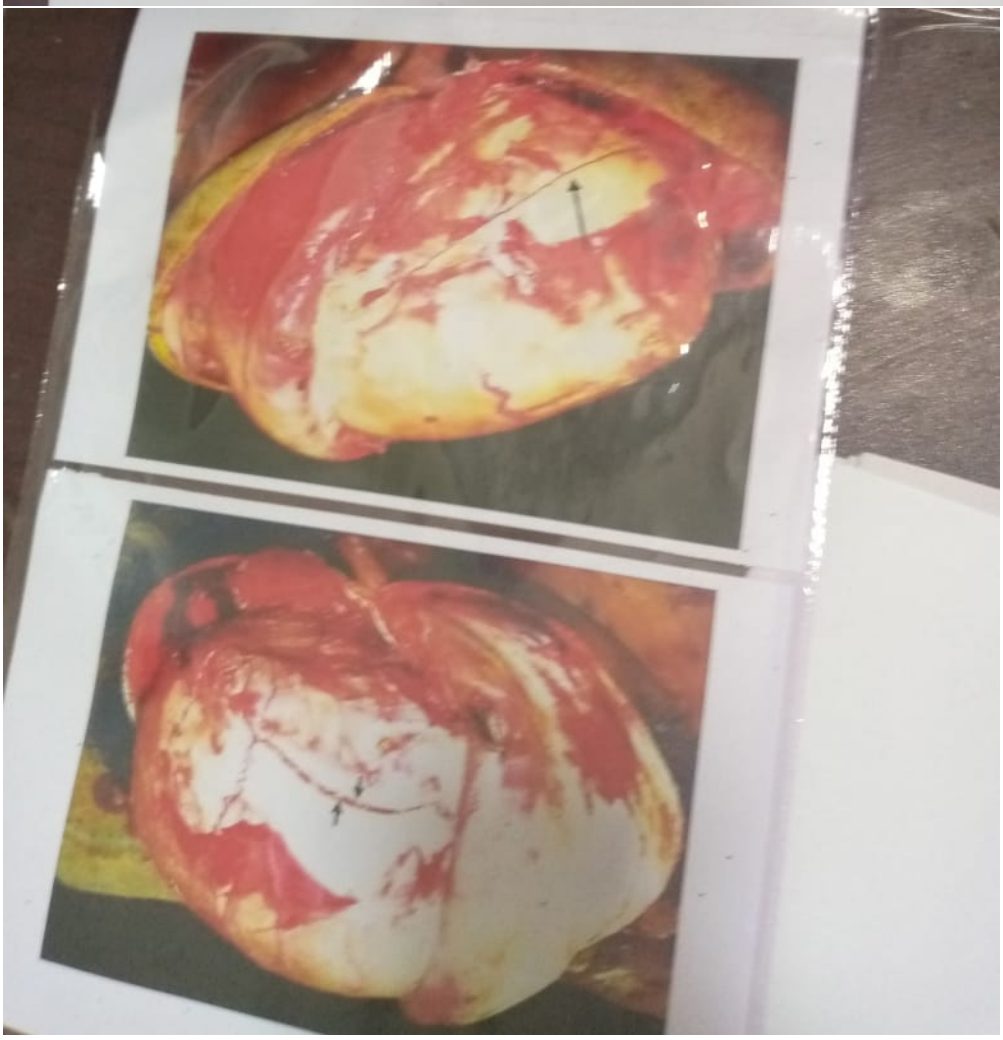


Station No. 10

1. Identify the fracture (0.5)

2. Enlist complications of Skull Fracture (3)

3. Enumerate the Fractures of skull (2.5)

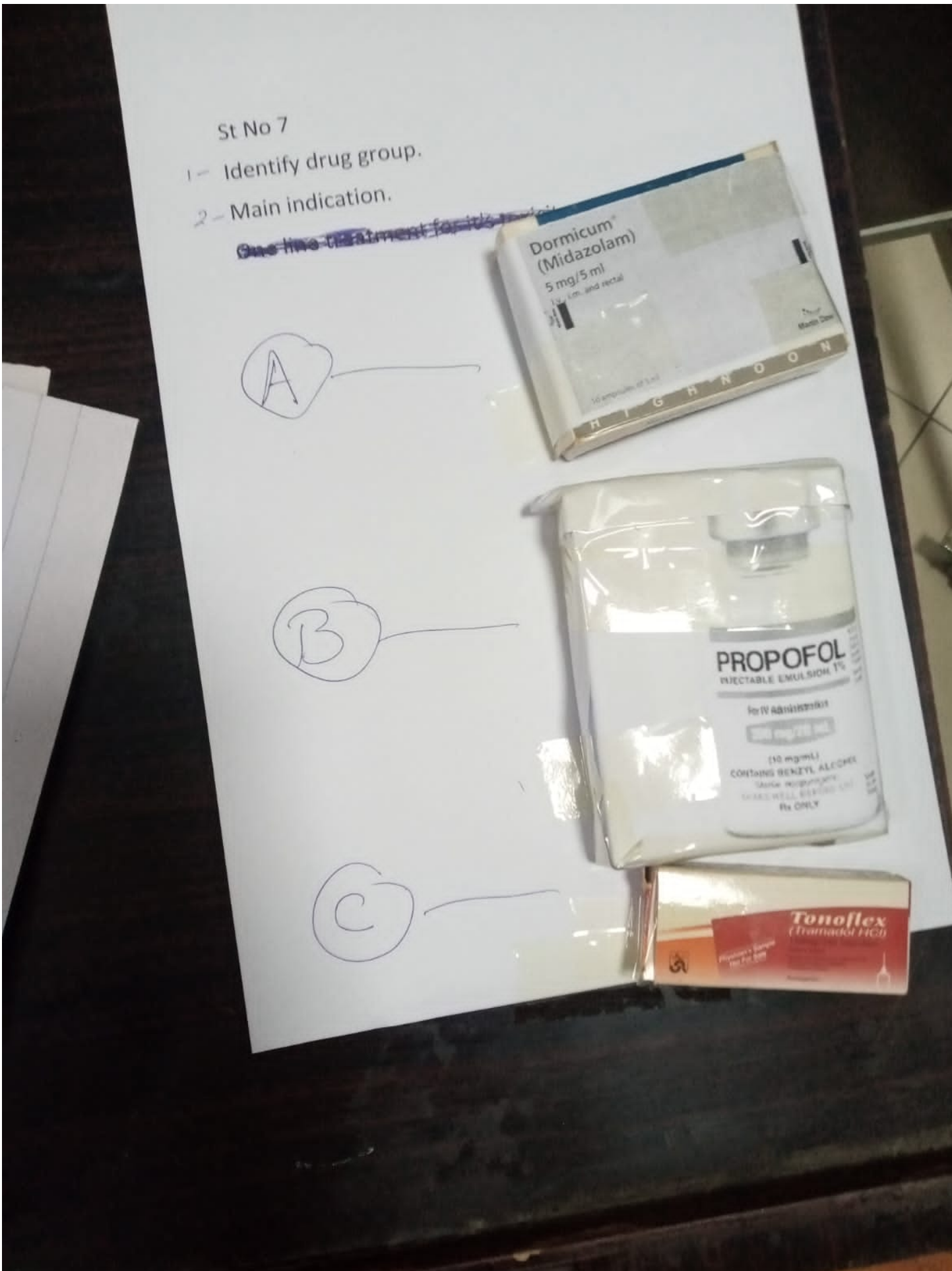


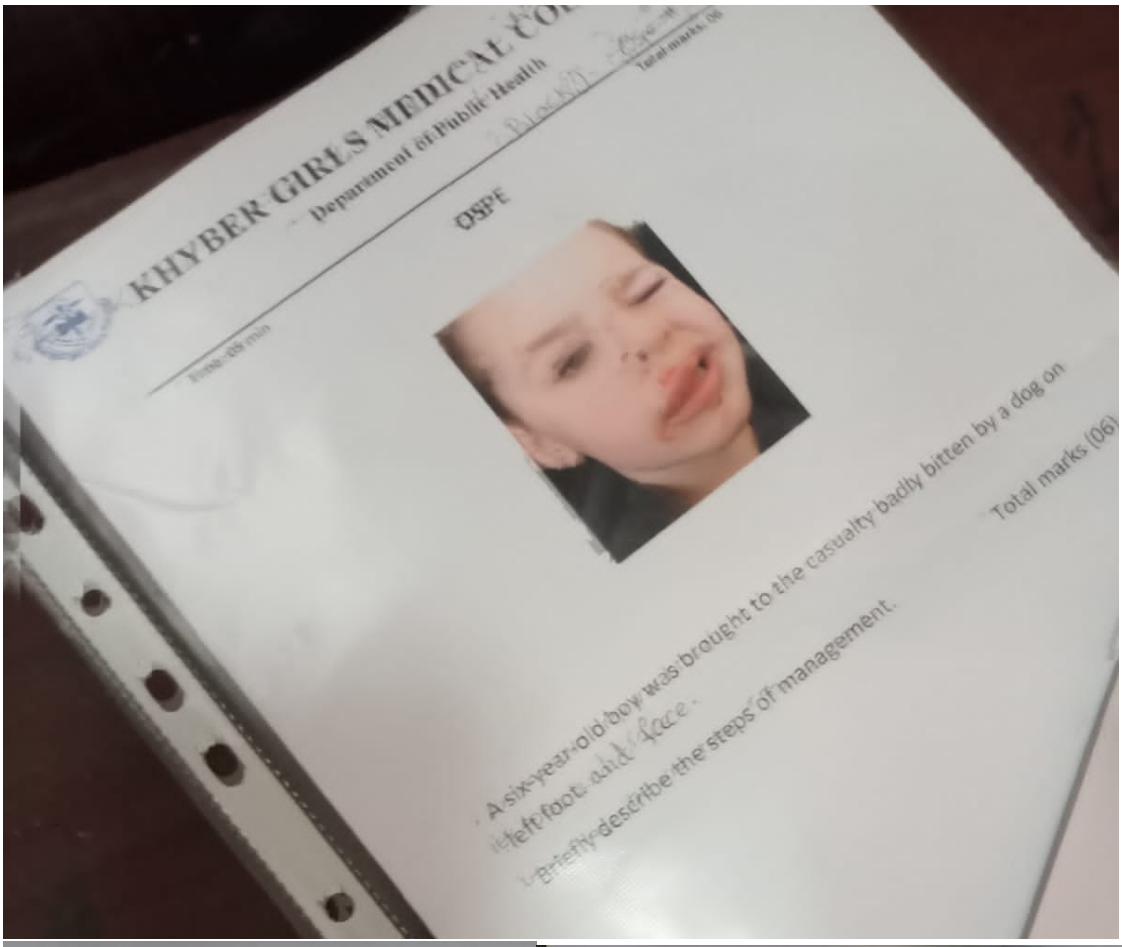
St No 7

1 – Identify drug group.

2 – Main indication.

~~One line treatment for it's~~





KHYBER GIRLS MEDICAL CO

Department of Public Health

OSPE

Total marks: 06

A six-year-old boy was brought to the casualty badly bitten by a dog on left foot, and face.

Briefly describe the steps of management.

Total marks (06)

Prophylaxis Migraine

Dr. ABC
KMC Pesh.
MBBS.

Name : XYZ Age : 40yrs Gender : Female
Cont : 0345--- Add : Pesh. Date : 7-3-22

Dx : Prophylaxis of migraine.

Rx

- Tab Propanolol (40mg)
  1 گولی روزانہ 6 ماہ کے لیے

احتیاطی تدابیر: • دوائی کا استعمال باقاعدگی سے کریں۔
• تکلیف کی صورت میں OPD تشریف لائیں۔

Sign : B—

Acute Attack Migraine

Dr. XYZ
KMC, Pesh.
MBBS.

Name : XYZ Age : 40 yrs Gender : ♀
Contact : 034... Add : Pesh. Date : 7-3-22

Dx : Acute Migraine Attack.

Rx :

- Tab Sumatriptan (30mg)
  1 گولی بوقتِ ضرورت
- Tab Metoclopramide (10mg)
  1 گولی استعمال کریں۔

احتیاطی تدابیر: • دوائی کا استعمال باقاعدگی سے کریں۔
• تکلیف کی صورت میں OPD تشریف لائیں۔

Scenario:
A 20 year old lady presented to OPD with complaints of depression for the last 6 months due to the death of her parents because of COVID-19. write the prescription for her?

Dr Zarmish
MBBS HMC
Phone No ------
Address: ------

Patient's Name: Gul-e-Rana Gender: Female Age: 20yrs
Address: Peshawar Date: 4th. Apr. 2022

Diagnosis:
"Depression"

R_x

1) Tab Escitalopram 10mg
1 گولی رات کے وقت 6 ماہ کیلئے

2) Tab Alprazolam 0.5mg
1 گولی رات کو 1 ماہ کیلئے

3) ~~Tab Propanolol 10mg~~
~~1 گولی صبح نہار 1 ماہ کیلئے~~

✓

ہدایات:
1) دوائی باقاعدگی سے استعمال کریں۔
2) مضر اثرات ثابت ہونے پہ دوبارہ معائنہ کرائیں۔
3) مریض کو اکیلا نہ چھوڑے

Dr. Zarmish
MBBS HMC
Phone No. ------
Peshawar

Patient's Name: Ahmad . Age: 25 . Gender: Male
Address: Peshawar . Date: 4th April-2022

Diagnosis : Epilepsy - Admit to ICU. ABC management & secure airway and oxygen inhalation

Rx

(1) Inj. Lopazepam 1mg i-v stat
or
Inj. Diazepam 10mg i-v stat

(2) Inj. Fosphenytoin 10mg/kg i-v stat

(3) Tab Carbamezapine 200mg
ایک گولی صبح و شام (1+1) جاری

(4) Tab Sodium valproate 200mg
1 گولی صبح و شام جاری
کم از کم دو سال

ہدایات:
① دوائیاں باقاعدگی سے استعمال کریں۔
② دوبارہ جھٹکے آنے کی صورت میں فوری ڈاکٹر سے رجوع کریں۔
③ مریض کو گاڑی چلانے منع دے اور آگ کے قریب نہ جانے دیں۔

Sign:

Scenario:-
A 25 years old male came to emergency with generalized seizure (Grand mal epilepsy). He has a history of recurrent seizures. He is a known case of epilepsy. He is having fits for half an hour interval. CT-Scan is normal.

General Anaesthesia

- General anaesthetics are the drugs that produce reversible loss of all sensation & consciousness.

Classification:

1. **Inhalational**

A. Gas: N_2O, Xenon

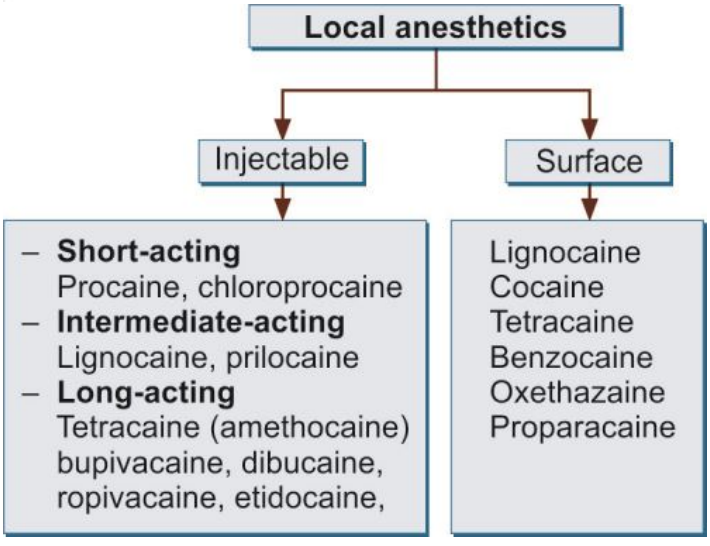
B. Liquids: Ether, Halothane, En/Iso/Des/Sevo-flurane

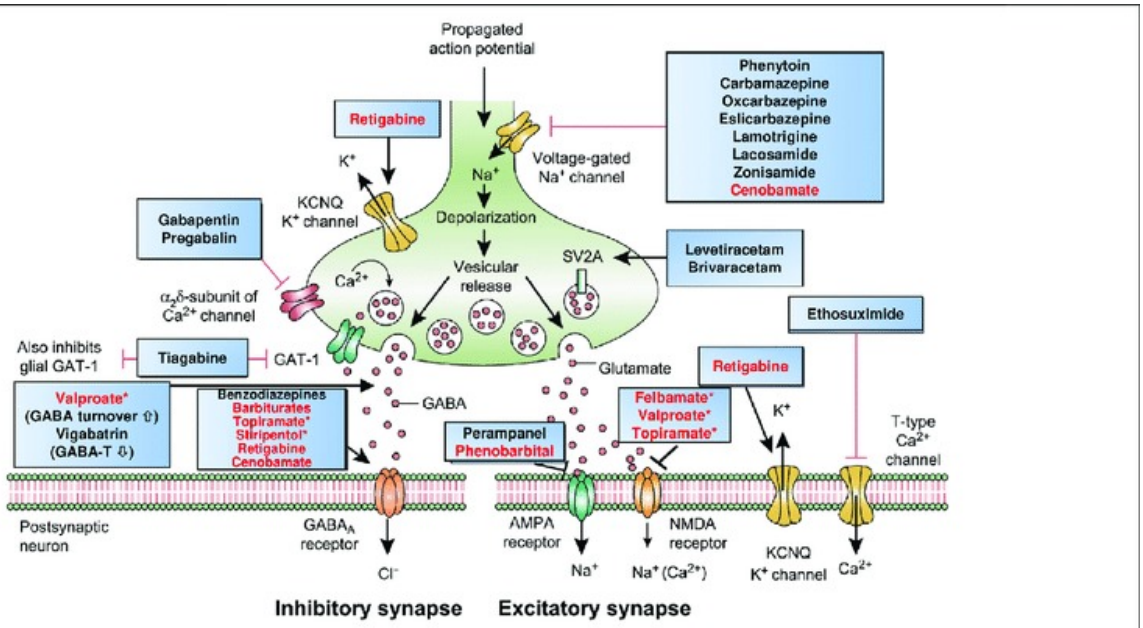
2. **Intravenous**

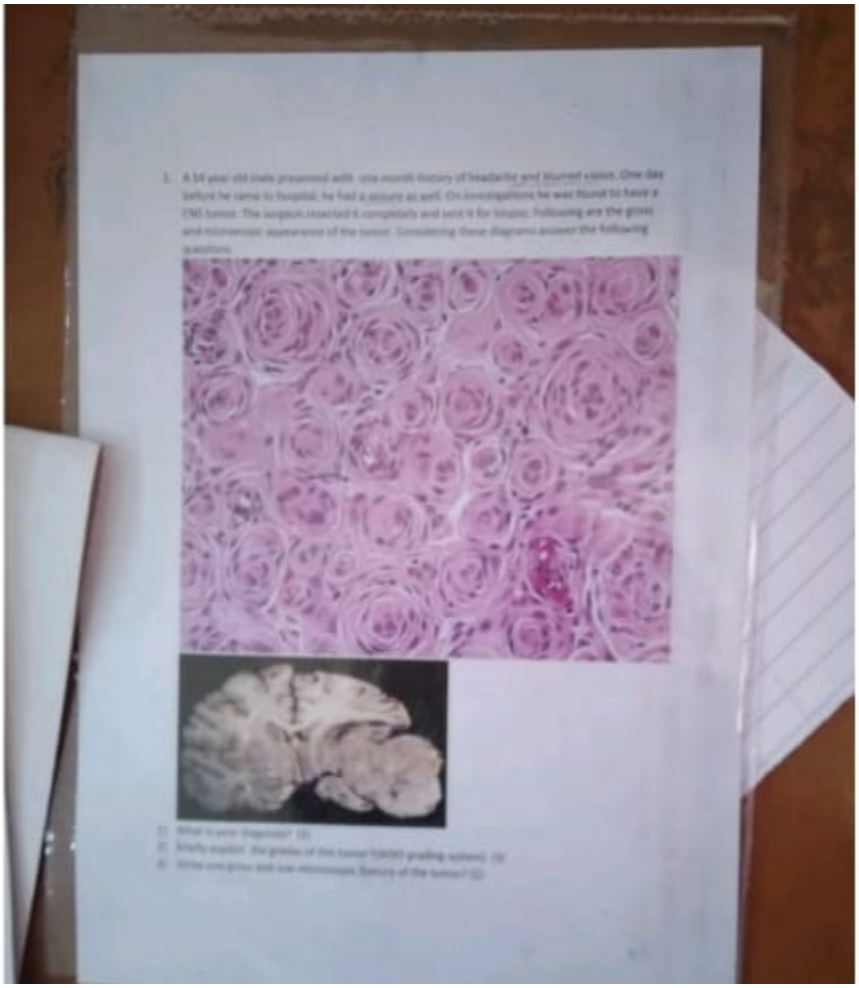
A. Inducing agents: Thiopentone sodium, Propofol, Methohexitone, Etomidate

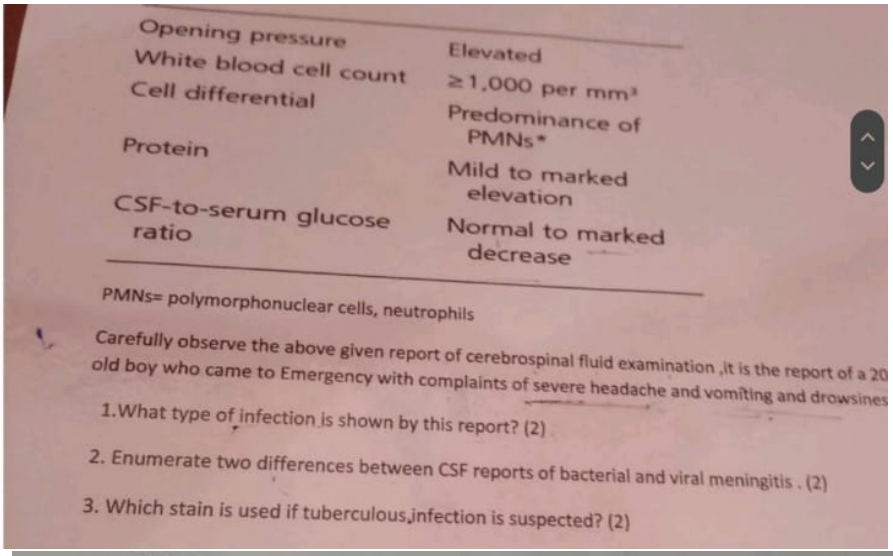
B. Slow acting

- **Dissociative anesthesia: Ketamine**
- **Benzodiazepines: Diazepam, Lorazepam, Midazolam**
- **Opioid analgesics: Fentanyl**




Propagated action potential
Phenytoin
Carbamazepine
Oxcarbazepine
Eslicarbazepine
Lamotrigine
Lacosamide
Zonisamide
Cenobamate
Retigabine
Voltage-gated Na+ channel
K+
Na+
KCNQ K+ channel
Depolarization
Gabapentin
Pregabalin
Levetiracetam
Brivaracetam
SV2A
Ca2+
Vesicular release
α2δ-subunit of Ca2+ channel
Ethosuximide
Also inhibits glial GAT-1
Tiagabine
GAT-1
Glutamate
Retigabine
Valproate*
(GABA turnover ⇧)
Vigabatrin
(GABA-T ⇩)
Benzodiazepines
Barbiturates
Topiramate*
Stiripentol*
Retigabine
Cenobamate
GABA
Felbamate*
Valproate*
Topiramate*
K+
T-type Ca2+ channel
Perampanel
Phenobarbital
Postsynaptic neuron
GABAA receptor
Cl-
AMPA receptor
Na+
NMDA receptor
Na+ (Ca2+)
KCNQ K+ channel
Ca2+
Inhibitory synapse
Excitatory synapse





Opening pressure	Elevated
White blood cell count	≥1,000 per mm³
Cell differential	Predominance of PMNs*
Protein	Mild to marked elevation
CSF-to-serum glucose ratio	Normal to marked decrease

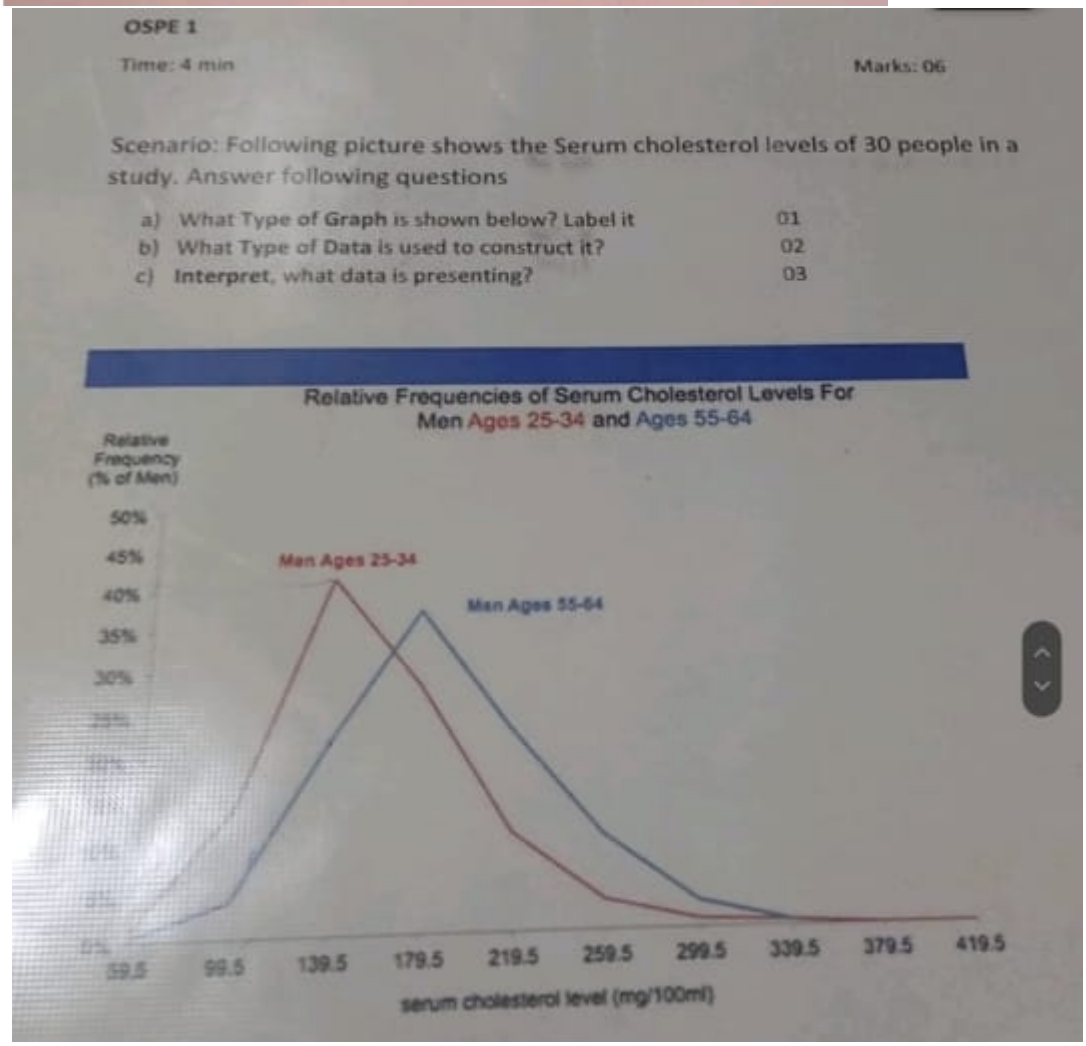
PMNs= polymorphonuclear cells, neutrophils

Carefully observe the above given report of cerebrospinal fluid examination ,it is the report of a 20 old boy who came to Emergency with complaints of severe headache and vomiting and drowsines

1.What type of infection is shown by this report? (2)

2. Enumerate two differences between CSF reports of bacterial and viral meningitis . (2)

3. Which stain is used if tuberculous,infection is suspected? (2)



- Frequency polygon: A **frequency polygon** is a graphical representation of a frequency distribution. It uses **line segments** to connect points corresponding to class frequencies, making it easier to visualize patterns or trends in the data.

- Continuous Quantitative data
- According to the data, Men ages 25-34 has cholesterol levels 139.5 mh/100ml at relative frequency of 40 % And Men with the ages 55_64 have cholesterol levels 180 mg/100ml at relative frequency of 35%

Station# 12

Q.1 Identify the specimen. (1)

Q.2 Name the active principles in the given specimen. (1)

Q.3 Write the mechanism of its action. (1)

Q.4 Discuss the management of is poisoning. (3)

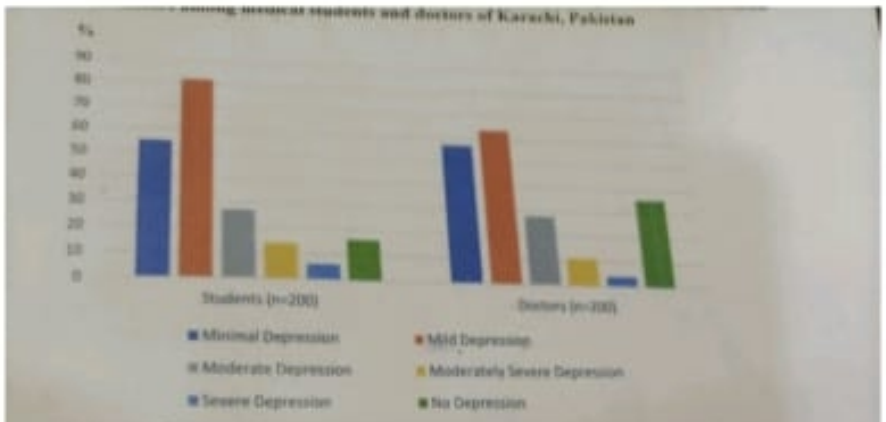


Fig: Comparison of depression scores among doctors and students

Qureshi MFH, Mohammad D, Sadiq S, Abubaker ZJ, Kanzari U, Devnani I, et al. A comparative cross-sectional analysis on prevalence of depression and associated risk factors among medical students and doctors of Karachi, Pakistan. Middle East Curr Psychiatry. 2020 Oct 30;27(1):39.

tructions:

he above graph is an "Extract from a paper". Attempt all questions. Each question carries one mark

1. Which study design is used for the above research?
2. Name the two groups that are being compared?
3. Which type of graph is used to present the data?
4. What is the total number of study population (N=?)?
5. In which group "mild depression" is higher & write the percentage?
6. In which group "no depression" is higher & write the percentage?

St : Poppy plant — phenanthrene isoquinoline Active principle? — Naloxone Antidote?

Which Stages? — Diagnostic signs & symptoms?

St viva → Insanity? Types? Snake venom? types

St viva → hemorrhages types? , Cerebral concussion? , cephalic cyst.

tetanus — this position name.

Subarachnoid hemorrhage causes? Non-traumatic cause

St : Medicine — 4days → lower limb weakness preceeded by Diarrhea . Now upper limb weakness ; LMN (Peripheral N) → Signs? Guillaine barre synd due electrolyte depletion due to diarrhea caused by Campylobacter

Test : Nerve conduction test
electrolyte depletion test

Na+ channel

A → 1- phenytion
2- carbamazapine
3- Valproic acid
4- Lamotrigine.

B → Voltage Gated Ca+ (HVG Ca+).
1 → Lamotrigine
2 → Ethosuxamide (Mam slide but L type)
↘ Topiramati

C → α2δ1 (GABA Release)
1 → Gabapentin
2 → pregabalin.

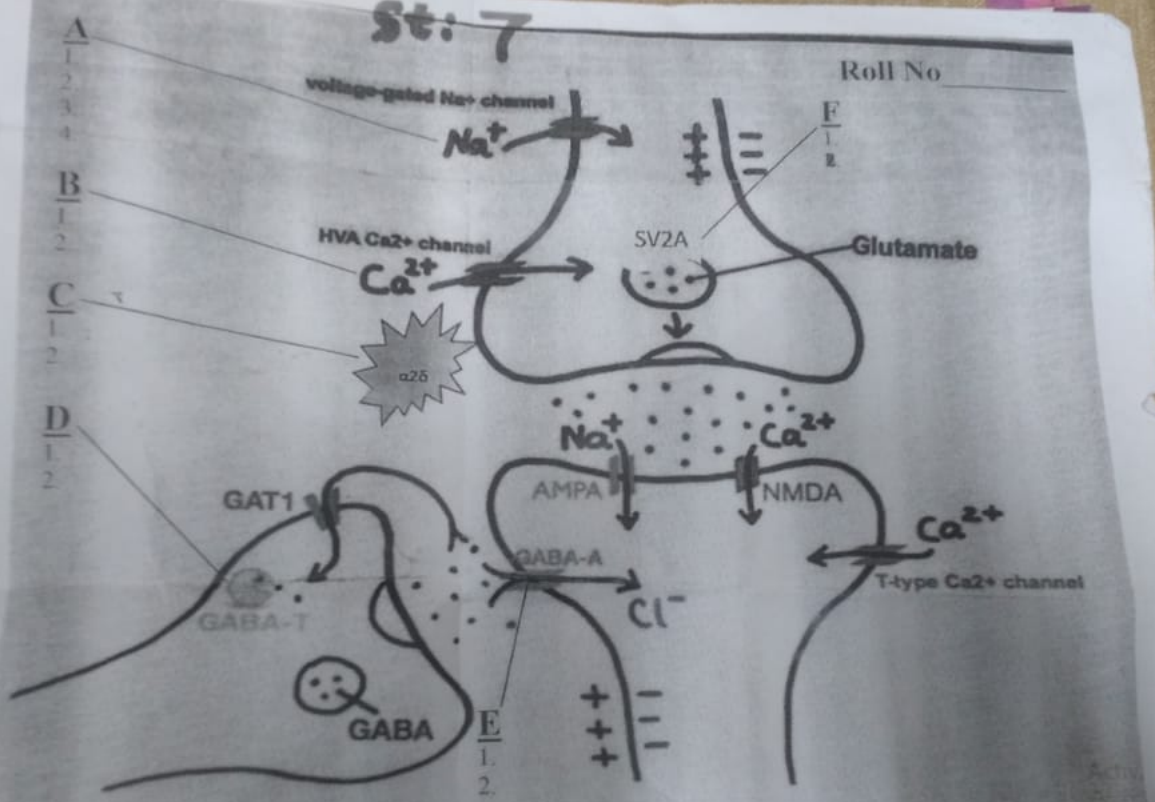
D → GABA T (Enzyme That prevent degradation of GABA)
1 → Vigabatrin
2 → Valproic acid.

E → GABA A
1 → Benzodiazapine
2 → Barbiturates.

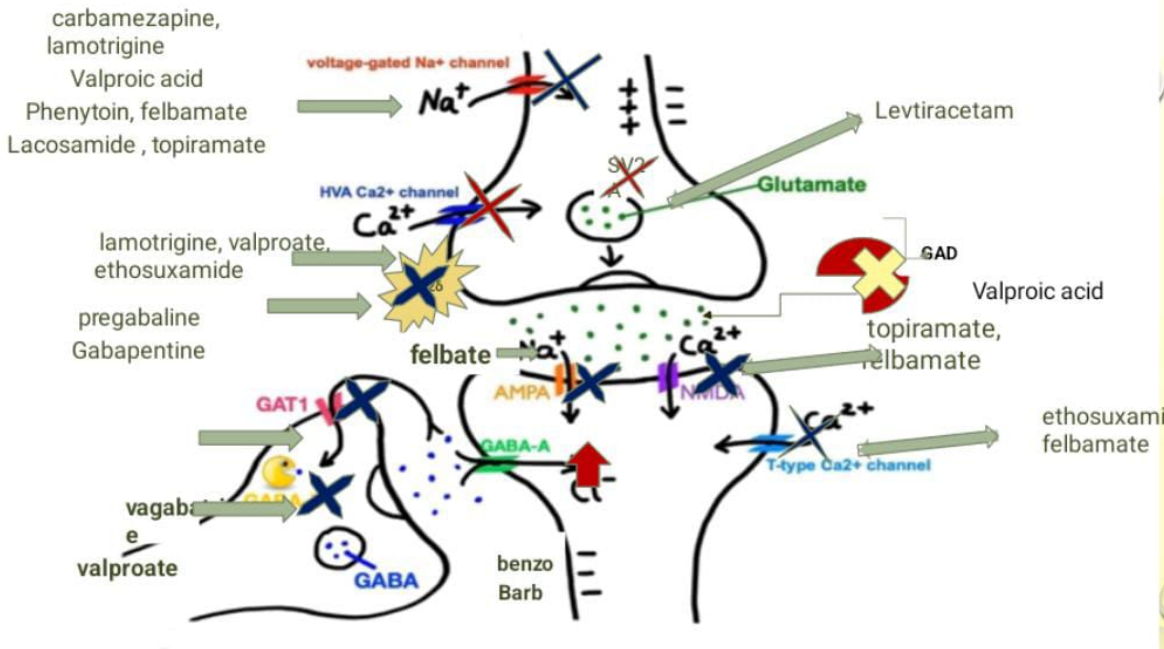
F → SV2A (Release of Neurotransmitter: Glutamati)
1 → Levitiracetam
2 → Brivaracetam

St: 7

Roll No



Q: Which anti-Epileptic drugs act on above lablled sites


carbamezapine,
lamotrigine
Valproic acid
Phenytoin, felbamate
Lacosamide , topiramate
voltage-gated Na+ channel
Na+
HVA Ca2+ channel
Ca2+
SV2A
Levtiracetam
Glutamate
lamotrigine, valproate,
ethosuxamide
pregabaline
Gabapentine
GAD
Valproic acid
topiramate,
felbamate
felbate
Na+
Ca2+
AMPA
NMDA
GAT1
GABA-A
Ca2+
T-type Ca2+ channel
ethosuxamide,
felbamate
vagaba
e
valproate
GABA
benzo
Barb