LECTURE 3

# DISTURBED CONSCIOUS LEVEL IN ER

## Disturbed conscious level (Altered mental status)

### Introduction

**Disturbed Conscious Level (DCL)** is one of the most common and critical presentations encountered in emergency medicine and casualty departments.

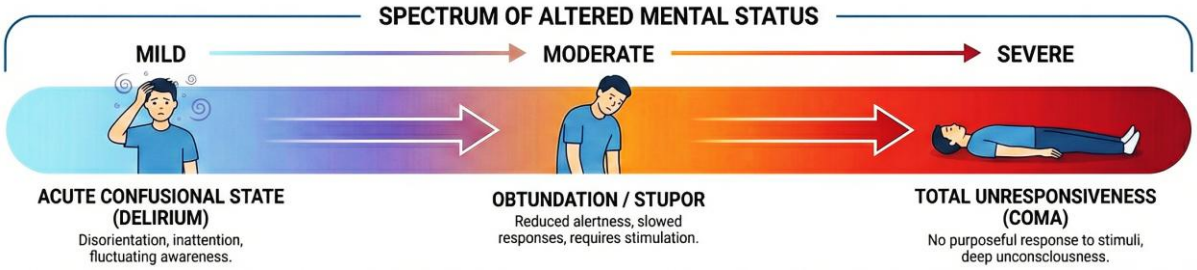
**Clinical Importance:**

- It's a medical emergency that can indicate serious underlying pathology, that Requires rapid assessment, classification, and management.

**Disturbed Conscious Level** is a common term used to describe patients with any change in responsiveness or awareness.

- But the scientific term is Altered Mental Status
- SO, any disturbance in consciousness, cognition, or behavior (ranging from mild confusion to deep coma) called Altered Mental Status (AMS).

Disturbance of consciousness is not one level, but a spectrum → ranging from acute confusional state (delirium) to marked degree of unresponsiveness (coma)




## A COMA (SEVERE DISTURBANCE IN CONSCIOUS LEVEL)

### DEFINITION:

- Coma is a state of complete loss of consciousness and unresponsiveness to external stimuli.

### CLUES:

- Glasgow Coma Scale (GCS) → ≤ 8.
- Unarousable unconsciousness: patient can`t be awakened by any stimulus

- Marked reduction of motor, verbal, or visual response.
- Patient is comatosed.

## B DELIRIUM → ACUTE CONFUSIONAL STATE

### DEFINITION:

- A partial disturbance in consciousness where the patient is awake but confused, disoriented and/or agitated with acute fluctuating course.

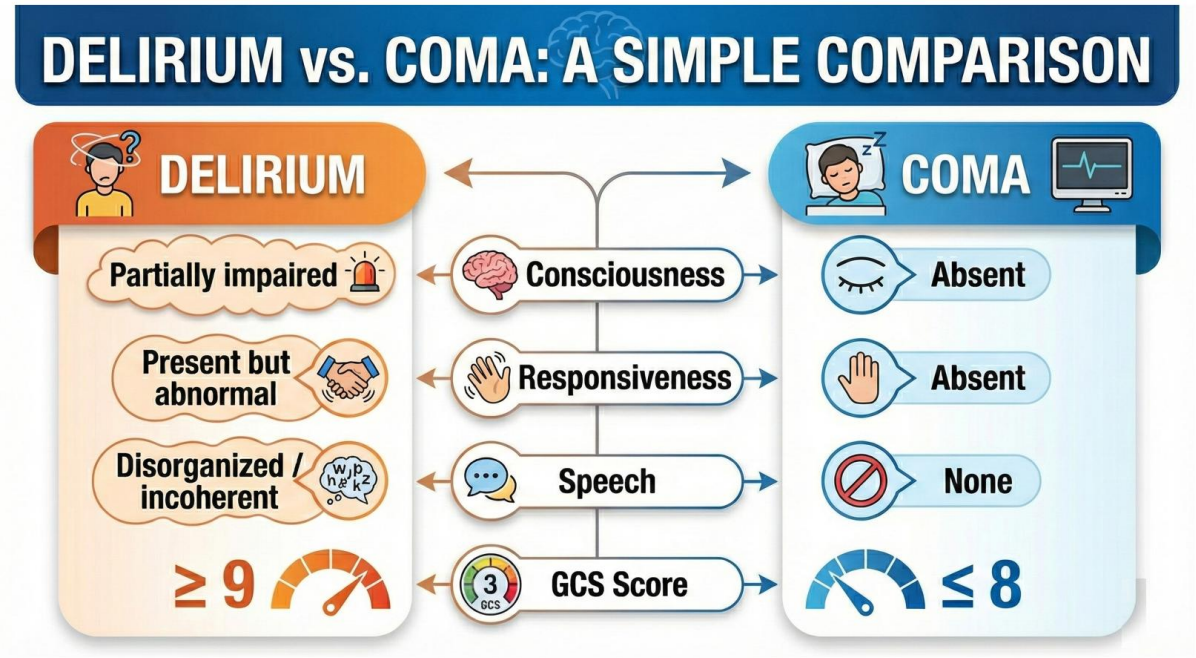
### CLUES:

- Glasgow Coma Scale (GCS) → ranging from 9 – 14
- Eyes may be open but awareness is impaired
- Speech is disorganized or incoherent
- Patient may appear restless, agitated, or irritable
- Fluctuating levels of attention and orientation

**NOTE**

Delirium represents a mild degree of disturbed consciousness.

- Normal Alertness (GCS= 15) → Fully conscious
- Delirium / Confusion (GCS= 9–14) → Partial impairment
- Coma (GCS ≤ 8) → Deep unconsciousness



**NOTE**

- Delirium often fluctuates during the day and worse at night

There are some conditions that mimic coma but are not true comas.

These are known as **Coma Mimics** !!

**Most Common Coma Mimics:**

1. Locked-in Syndrome
2. Vegetative State (Unresponsive Wakefulness Syndrome) [UWS]
3. Psychogenic Unresponsiveness

Understanding these conditions is crucial for accurate diagnosis. They may appear similar to coma, but the underlying mechanisms and awareness levels are different.

## 1 LOCKED-IN SYNDROME

**DEFINITION:**

- A rare neurological condition where the patient is awake and aware but completely paralyzed, except for vertical eye movements.

**CAUSED BY:** lesion of the brainstem, specifically the pons

**CLUES:**

- Intact Consciousness (patient is awake and aware)
- Motor Response (Motor GCS = 1) → No movement
- Verbal Response (Verbal GCS= 1) → No verbal output
- Eye Response (Eye GCS= 4) → Eyes open, respond with vertical movement.
- Communication → Only through eye blinking or vertical eye motion
- Behavior → Appears mute and motionless but conscious

**Key Concept:** The patient sees you and understands everything, but cannot move or speak but communicates only through eyes.

"The patient is conscious but trapped inside the body."

## 2 VEGETATIVE STATE (UNRESPONSIVE WAKEFULNESS SYNDROME)

### DEFINITION:

- A condition in which the patient appears awake (eyes open), but is not conscious, there is no meaningful interaction or response.
- Vegetative State occurs due to end-stage brain damage (extensive cortical damage with preserved brainstem activity).

### POTENTIAL CAUSES:

- Severe traumatic brain injury
- Massive intracranial hemorrhage
- Severe infection (e.g., encephalitis) and advanced brain tumor

### CLUES:

- Absent Consciousness
- Absent Awareness
- Eyes open spontaneously or partially
- Absent Eye Movement (eyes do not follow stimuli)
- No Motor Response
- No Verbal Response
- Arousal (Wakefulness) → (eyes open)

**Comparison between Vegetative State and Coma**

| VEGETATIVE STATE | CRITERIA | COMA |
|---|---|---|
| Open / partially open | **Eye Opening** | Closed |
| Present | **Arousal** | Absent |
| Absent of awareness | **Awareness** | Absent of awareness |
| No Motor/ Verbal Response | **Motor / Verbal Response** | No Motor/ Verbal Response |
| Not conscious (but wakefulness present) | **Consciousness Level** | Not conscious |

**NOTE**

**Vegetative state** often occurs after prolonged coma, representing severe and usually irreversible brain injury.

**Comparison between Locked-in Syndrome vs. Vegetative State**

| LOCKED-IN SYNDROME | CRITERIA | VEGETATIVE STATE (UWS) |
|---|---|---|
| Present | CONSCIOUSNESS | Absent |
| Present | AROUSAL (Eyes Open) | Present |
| Intact | AWARENESS | Absent |
| Vertical movement preserved | EYE MOVEMENT | No eye tracking or movement |
| Rare but may communicate | PROGNOSIS | Poor; end-stage brain injury |

**Key Distinction:**

In Locked-in Syndrome, the patient can move eyes vertically and is aware.

In Vegetative State, there is no awareness and no eye movement response.

## 3 PSYCHOGENIC UNRESPONSIVENESS

### DEFINITION:

- A non-organic condition in which the patient appears comatose, but the cause is psychological or psychiatric, not structural or metabolic.

### GENERAL DESCRIPTION

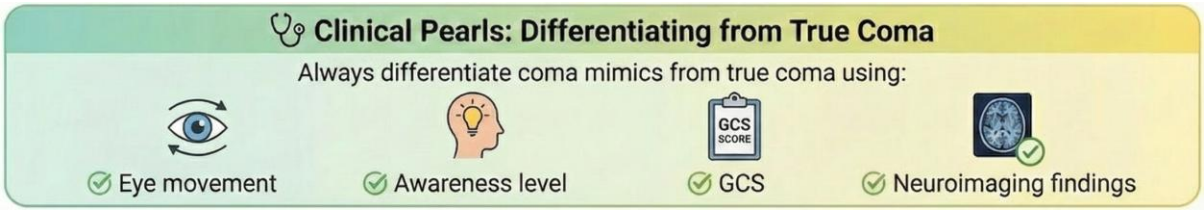
- The patient appears to be in coma, but there is no organic brain disease.
- The condition results from psychological stress or psychiatric disorders (functional causes).
- Consciousness is partially preserved, but the patient chooses or subconsciously fails to respond.

## CLUES:

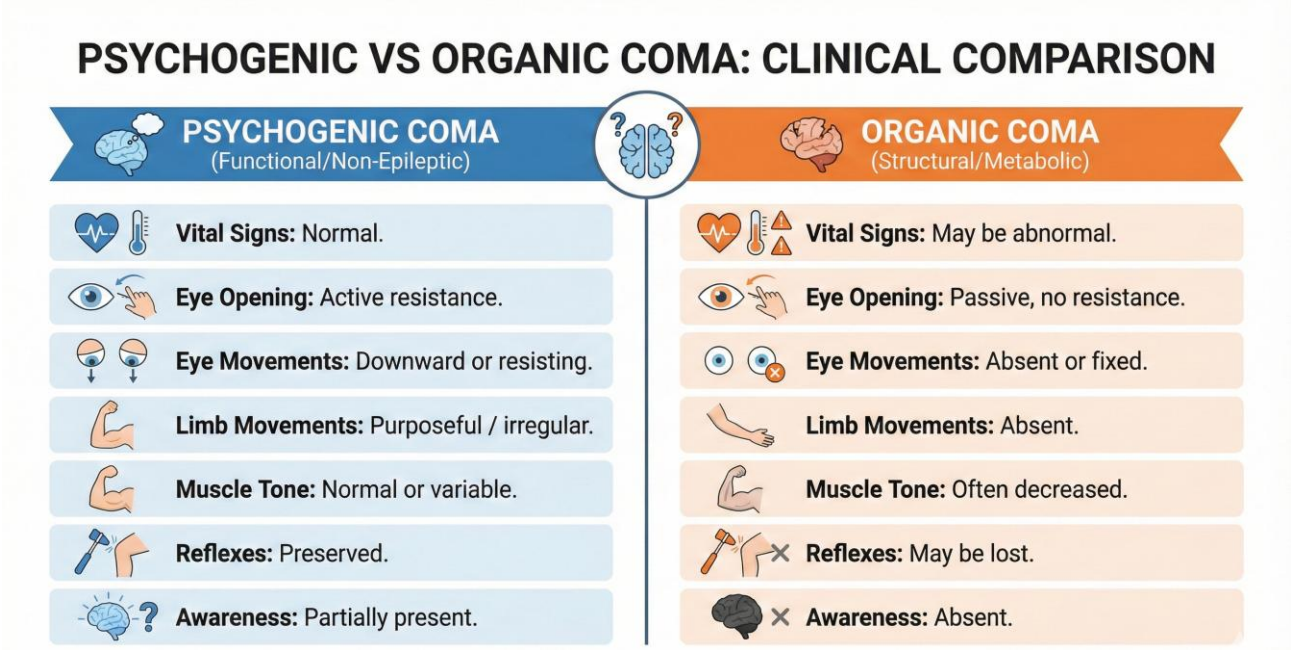
- The patient's vital signs and systemic condition are usually normal, no structural lesion on imaging or laboratory tests.
- When applying painful or sensory stimuli (e.g., pressing on the chest or opening the eyes): you observe inconsistent or unusual reactions not seen in true coma.
- **Patient resists eye opening** → Indicates awareness.
- **Resisting eye movement** is characteristic of psychogenic coma.
- **Purposeful or semi-purposeful limb movement** suggests partial awareness
- **Blinking when eyelashes are touched** → Indicates conscious response
- **Strong eye closure during exam** → voluntary behavior not seen in true coma
- Movements may appear purposeful, irregular, or exaggerated, unlike true coma, muscle tone and reflexes are preserved.
- Some patients may exhibit Waxy Flexibility, where limbs remain in the same position after being moved, Seen in → Catatonia, a psychiatric condition under the spectrum of psychotic or affective disorders.

**Clinical Tip:** Always exclude organic causes first before diagnosing psychogenic unresponsiveness.

| SUMMARY OF COMA MIMICS | | |
|---|---|---|
| **Locked-in Syndrome** | **Vegetative State (UWS)** | **Psychogenic Unresponsiveness** |
| Conscious but paralyzed → communicates with eyes. | Awake but unaware → eyes open, no tracking. | No structural lesion → psychological cause. |




- Recognition prevents misdiagnosis and inappropriate management.

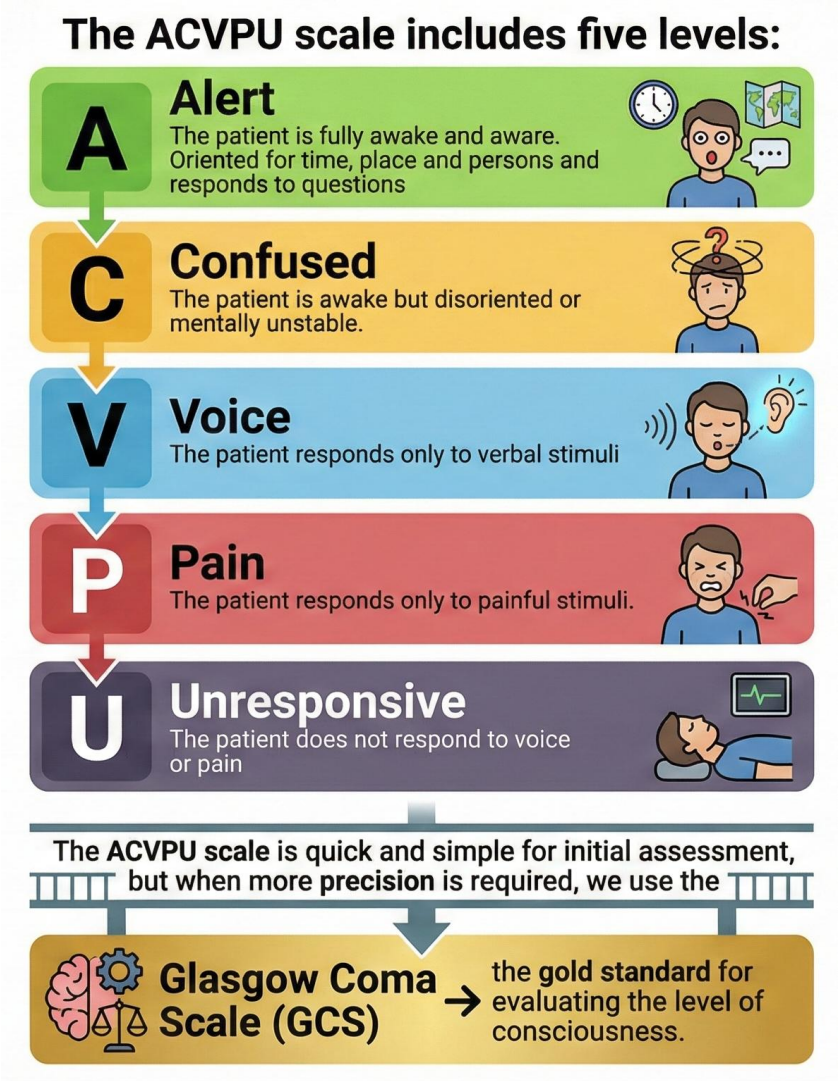



## Coma and Assessment of Consciousness

- Before we discuss coma itself, remember that the entire concept is built on assessing the degree of consciousness.
- Evaluating the level of consciousness is the foundation for managing any patient with a Disturbed Conscious Level.
- This evaluation starts with basic tools like the ACVPU Scale and extends to the more accurate and widely accepted Glasgow Coma Scale (GCS).

### Initial Assessment of Consciousness

- Initially, we discussed the **National Early Warning Score (NEWS)**, which includes a component called level of consciousness.
- This can be assessed using a simple bedside method known as the ACVPU Scale.




## GLASGOW COMA SCALE (GCS)

The GCS is the most accurate and universally accepted tool for assessing a patient's level of consciousness.

It is used globally in emergency departments and intensive care units.

**It is composed of three main components:**

1. Eye Response (E)
2. Motor Response (M)
3. Verbal Response (V)

The total score is the sum of these three components, ranging from 3 to 15.

## 1 EYE RESPONSE (E)

- If the patient opens their eyes spontaneously without any stimulation → Score 4 (Spontaneous).
- If the patient keeps their eyes closed but opens them when you speak to him → Score 3 (To Speech).
- If the patient only opens their eyes in response to a painful stimulus → Score 2 (To Pain).
- If the patient does not open their eyes at all, even to pain → Score 1 (None).

The higher the eye response score, the better the arousal and brainstem function.

## 2 MOTOR RESPONSE (M)

- If the patient follows commands such as "raise your hand" or "move your leg," → score 6 (Obeys Commands).
- If the patient doesn't not follow commands but moves purposefully toward the site of pain, → score 5 (Localizes Pain).
- If the patient withdraws the limb away from the pain but not toward it → score 4 (Withdraws from Pain).
- If the patient shows abnormal flexion (arms bent inward) → score 3 (Flexion to Pain - Decorticate Posture).
- If they show abnormal extension (arms stretched outward) → score 2 (Extension to Pain – Decerebrate Posture).
- If there is no motor response at all → score 1 (None).

**NOTE**

A decorticate posture suggests a lesion above the brainstem (supratentorial), while a decerebrate posture indicates brainstem involvement — a more serious finding.

## 3 VERBAL RESPONSE (V)

- If the patient speaks normally with coherent, appropriate sentences → Score 5 (Oriented Speech).
- If the patient speaks but the conversation is disorganized or incoherent → Score 4 (Confused Speech).
- If the patient produces words but not sentences → Score 3 (Inappropriate Words).
- If the patient makes only incomprehensible sounds → Score 2 (Incomprehensible Sounds).
- If there is no verbal response at all → Score 1 (None).

### TOTAL GCS SCORE AND INTERPRETATION

- The total GCS = E + M + V, with possible values ranging from 3 to 15.
- A total score between 3 and 8 indicates Coma (deep unconsciousness).
- A score between 9 and 14 indicates Confusion or Delirium (partial impairment).
- A score of 15 indicates that the patient is Fully Conscious and alert.

### EXAMPLE CALCULATION

A patient opens their eyes only to pain (E = 2), also the patient withdraws the hand away from pain (M = 4), and produces incomprehensible sounds (V = 2).

**The total GCS = 2 + 4 + 2 = 8.**

This means the patient falls within the **Comatose Range.**

**Clinical Interpretation:**

- Any patient with a GCS score of 8 or less is considered to be in coma. so the maximum score for coma is ≤ 8
- This is the cutoff for defining coma in clinical practice.
- If the score is 9 or higher, the patient has an altered level of consciousness but is not comatose.

- The lower the total GCS, the deeper the level of unconsciousness.
- It's important to document each component separately, for example:
- E2 M4 V2 = GCS 8 — rather than writing only the total score.

# Step 1 – Causes / Clues of Coma

## Causes

- The Ascending Reticular Activating System (ARAS), that responsible for arousal and awareness, if this system is damaged irrespective to the cause → this leads to Coma

| Causes of coma are divided into two main categories | |
|---|---|
| 1 Structural Causes (central coma) | 2 Encephalopathy (extracranial causes) |

## 1 STRUCTURAL CAUSES OF COMA

Structural coma results from direct damage to brain tissue, either within the cerebral hemispheres or the brainstem.

### COMMON STRUCTURAL CAUSES:

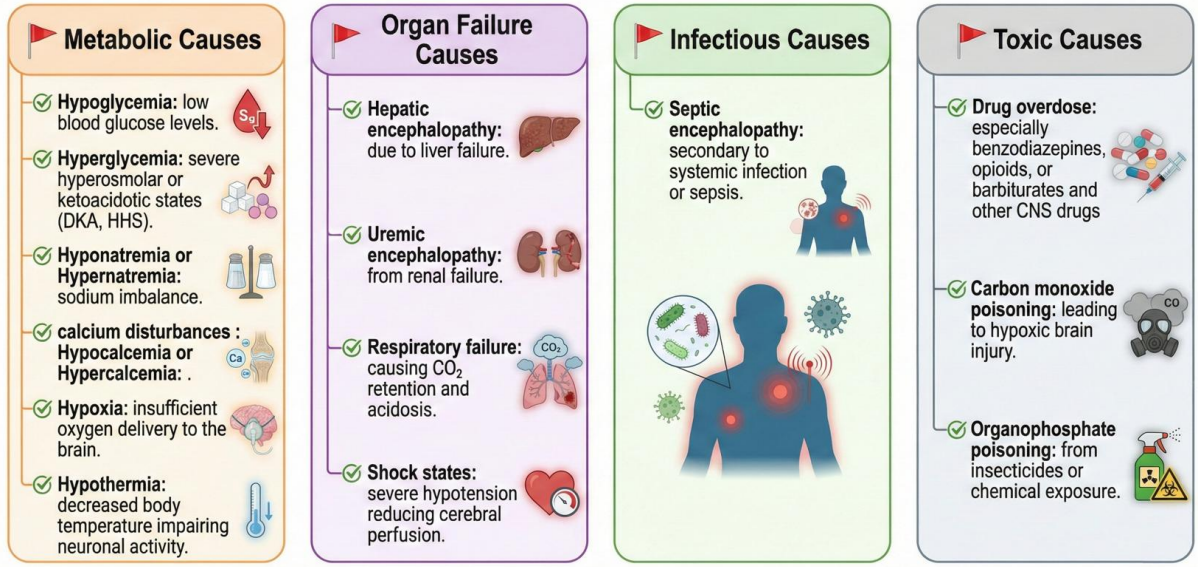
1. **Cerebrovascular accidents (Stroke)**
2. **Vascular injury**
3. **CNS infections**: such as meningitis or encephalitis.
4. **Postictal state**: transient coma following a seizure.
5. **Brain tumors**: space-occupying lesions compressing brain structures.
6. Intracranial hge: subarachnoid, intracerebral, subdural, or epidural bleeding.

## 2 ENCEPHALOPATHY

### CAUSES OF ENCEPHALOPATHY:

- In encephalopathy, the brain structure itself is intact, but its function is disrupted by systemic, biochemical, or toxic abnormalities.
- These are extrinsic causes, arising from conditions outside the brain.

## Examples include:




## DIFFERENTIATION BETWEEN COMA DUE TO STRUCTURAL CAUSES (INSIDE THE BRAIN)

After understanding that coma causes can be either **Structural causes or encephalopathy**, the next essential step is learning how to differentiate between them clinically.

### 1. LATERALIZATION

Lateralization means that the rt. side of the body is not the same as the left side.

| IN COMA DUE TO STRUCTURAL CAUSES | IN ENCEPHALOPATHY |
|---|---|
| - Neurological findings are often asymmetrical.<br>- You may observe weakness or paralysis on one side of the body (hemiparesis or hemiplegia) or unequal eye movements or gaze deviation toward one side<br>- This occurs because the lesion typically affects one cerebral hemisphere or one side of the brainstem. | - The findings are usually symmetrical.<br>- Both sides of the body are affected equally, and there is no lateralized deficit. |

## 2. PUPILLARY CHANGES

| IN STRUCTURAL LESIONS<br>Particularly when there is brainstem compression | IN ENCEPHALOPATHY |
|---|---|
| **The pupils may show characteristic abnormalities:**<br>- Unequal pupils → suggestive of unilateral brainstem compression led to transtentorial herniation as in cerebral hemorrhage<br>- Bilateral Pinpoint pupils → often due to a pontine hemorrhage | - The pupils are typically equal and reactive to light, because the optic and brainstem pathways remain intact. |

**Important note:**

- In toxic comas such as opioid overdose and organophosphate poisoning → there are bilateral pinpoint pupils, but the diagnosis is supported by history and toxicologic features.

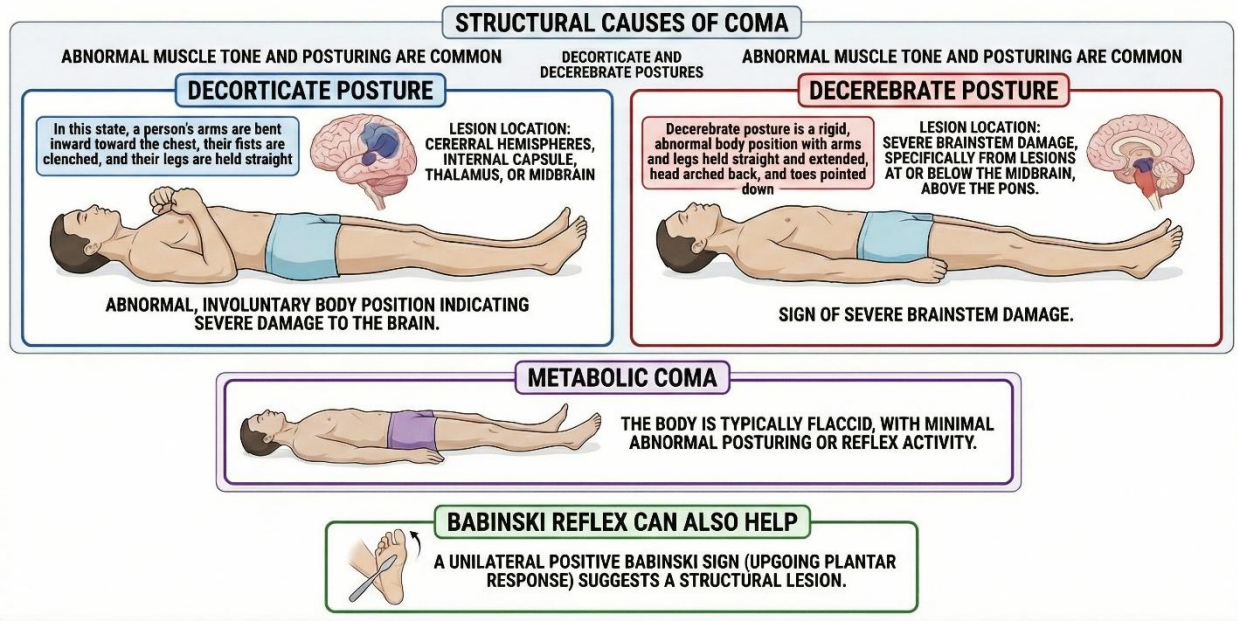
## 3. EYE MOVEMENTS

| IN COMA DUE TO STRUCTURAL CAUSES<br>e.g. Stroke | IN ENCEPHALOPATHY<br>particularly metabolic causes |
|---|---|
| - The eyes often move abnormally.<br>- They may deviate to one side (gaze deviation) or move independently (dysconjugate gaze or conjugate gaze palsy). | - eye movements are usually normal and symmetrical. |

**A critical bedside test here is the oculocephalic Reflex (Doll's Eye Movement) when the head is turned side to side:**

- If the eyes move in the opposite direction, the brainstem is intact.
- If the eyes remain fixed (Absent reflex) it indicates brainstem dysfunction, usually structural in origin.

## 4. MUSCLE TONE AND REFLEXES




### CLINICAL NOTE

In practice, differentiating between Structural and Metabolic Coma is the first diagnostic step in evaluating an unconscious patient.

It guides both the investigation pathway (neuroimaging vs metabolic workup) and the immediate management plan.

### Clues for each cause

## 1 STRUCTURAL CAUSES OF COMA

### 1 CEREBROVASCULAR ACCIDENTS (STROKE)

#### A. hemorrhagic stroke (intracerebral Hge.):

**Clues are:**

- Blood pressure is extremely high (usually >180/110).
- Often presents with sudden severe headache, vomiting then rapid progressive coma.
- Lateralization (one-sided weakness) can also occur.
- Unequal pupils may occur if transtentorial herniation.

- On CT brain, you'll see a hyperdense area (bright region indicating bleeding).

## B. Ischemic stroke :

Ischemic infarction usually doesn't lead to coma but if large area is affected as in occlusion of middle cerebral artery → malignant MCA occlusion with Midline shift → coma.

**Clues:**

- Blood pressure may be elevated.
- Headache is usually absent.
- Focal neurological signs such as hemiplegia, speech disturbance
- Lateralization still occurs (due to vessel occlusion on one side).
- On CT brain, it may look normal initially, and later become hypodense.

## C. Cerebral venous thrombosis:

**Risk factors:** Pregnancy, puerperium, Pills (OCPs), Prothrombotic disorders

**Clinical clues:** "HEAD" Mnemonic:

- Headache – most common (gradual or thunderclap).
- Edema → papilledema (due to ↑ ICP).
- Altered consciousness (coma) / seizures.
- Deficits (focal signs – weakness, speech problems, vision loss).

**Important note:** Stroke symptoms that don't fit the distribution of a single arterial territory → think cerebral venous thrombosis (CVT).
- MRV is diagnostic.

## D. Posterior Reversible Encephalopathy Syndrome (PRES):

**Clues:**

- Acute severe hypertension
- Visual loss / blurring (cortical blindness)
- Altered mental status
- MRI → Posterior (occipital-parietal) involvement

- Headache + Seizures

### 2 MENINGITIS / ENCEPHALITIS

**Clues:**

**Classic Triad:** Fever + Neck stiffness + disturbed conscious level

(any patient with these 3 → suspect meningitis immediately)

| Do CSF examination to differentiate between meningitis and encephalitis | |
|---|---|
| **CSF in Meningitis** | **CSF in Encephalitis** |
| ↑ WBC (neutrophils), ↑ protein, ↓ glucose | ↑ lymphocytes, mild ↑ protein, normal glucose |

### 3 BRAIN TUMORS

Brain tumors can cause coma due to Raised intracranial pressure by tumor mass or surrounding edema.

**Clues:**

- Chronic Recurrent headaches, often worse in the morning.
- Projectile vomiting without nausea.
- Focal neurological deficits.
- Gradual progression from confusion → stupor → coma.

**- CT or MRI Brain to identify and locate the tumor.**

## 2 CLUES FOR THE CAUSES OF ENCEPHALOPATHY

## ORGAN FAILURE CAUSES

### 1 HEPATIC ENCEPHALOPATHY

**Hepatic encephalopathy (HE)** is a serious neurological complication of advanced liver failure, caused by the liver's inability to clear toxins, especially ammonia, leading to brain dysfunction.

**Clues:**

- Patients show signs of advanced liver disease such as jaundice, ascites, lower limb edema, spider nevai.

- Neurological signs include asterixis, slurred speech, impaired cognition, and altered consciousness (Delirium & coma).
- Often triggered by factors worsening liver function or increasing neurotoxic load, such as: Upper GI bleeding, Infections (e.g., SBP), Constipation.

## 2 UREMIC ENCEPHALOPATHY

- Is a recognized neurological complication of advanced renal failure.
- It arises from the buildup of uremic toxins due to impaired kidney function.
- It typically occurs in patients with advanced chronic kidney disease (CKD), especially in end-stage renal disease (ESRD), or in severe acute kidney injury (AKI)

**Clues:**

- Myoclonus (hallmark sign) → sudden, involuntary muscle jerks.
- Generalized seizures.
- Altered mental status → from mild confusion to deep coma, depending on renal failure severity and toxin levels.
- Diagnosis is mainly clinical, based on neurological symptoms and evidence of kidney failure including: High serum urea, High serum creatinine, hyperkalemia or metabolic acidosis.

## 3 RESPIRATORY FAILURE

Is a critical non-neurological cause of coma, mainly due to $CO_2$ retention, resulting in hypercapnic encephalopathy

**Clues:**

**Features of respiratory failure with $CO_2$ retention include:**

- Headache, drowsiness, Flushed skin, Rapid breathing, confusion and Coma (in severe cases) ✚ Cyanosis.

**Diagnosis** relies on clinical history and arterial blood gas (ABG) analysis, which typically shows high $PaCO_2$ and low pH, pointing to respiratory acidosis.

### 4 SHOCK

Shock must be considered in any comatose patient.

**Clues:**

- Cold extremities, poor peripheral perfusion, delayed capillary refill,
- Low or unrecordable blood pressure.
- These findings suggest multiorgan failure, where cerebral hypoperfusion from shock may cause coma.

## METABOLIC CAUSES

### 1 ELECTROLYTE RELATED COMA (NA, CA)

#### A. Sodium ($Na^+$) disorders

- Both hyponatremia and hypernatremia can lead to altered mental status and potentially coma.

**Clues:**

- Clinical manifestations are often non-specific, ranging from seizures and confusion to deep coma.
- These changes are typically not distinguishable on clinical grounds alone; therefore, serum sodium measurement is essential.

#### B. Calcium ($Ca^{2+}$) disorders

- Severe hypercalcemia, particularly when serum calcium exceeds 14 mg/dL, can similarly lead to neurological depression and coma.
- **Clinical signs** are often vague and unreliable for diagnosis.
- Laboratory confirmation via serum calcium testing is required for accurate identification.
- also severe hypocalcemia may lead to coma along with tetany.

## C. Hypoglycemia

- Hypoglycemia remains one of the most important metabolic causes of coma.
- It must be excluded immediately in any patient presenting with impaired consciousness, whether transient or sustained.
- **Random blood glucose measurement** is a fundamental component of the initial assessment of any unresponsive patient.
- A glucose level below 50 mg/dL, particularly < 45 mg/dL, may result in neuroglycopenic symptoms, indicating insufficient glucose supply to brain tissue.

## D. DKA

- DKA is one of the most common causes of metabolic coma, particularly in patients with Type 1 Diabetes Mellitus.

**Clues:**

- Marked hyperglycemia
- Arterial pH < 7.3
- Serum bicarbonate < 18 mmol/L
- Positive serum or urinary ketones
- Severe dehydration
- Often accompanied by Kussmaul respiration, reflecting compensatory hyperventilation

## E. Hyperosmolar Hyperglycemic State (HHS)

- HHS is a less frequent but severe complication, more common in elderly patients with Type 2 Diabetes.

**Clues:**

- Plasma glucose > 600 mg/dL
- Effective serum osmolality > 320 mOsm/kg
- Arterial pH > 7.3

- Bicarbonate > 18 mmol/L
- Absent ketonemia and ketonuria
- Severe dehydration
- Often accompanied by altered mental status and possibly coma

## F. Myxedema Coma

- A rare, life-threatening complication of severe hypothyroidism, primarily affecting older women with untreated or poorly managed disease.

**Clues:**

-
- Profound hypothermia (<35°C)
- Bradycardia, Hypotension, Hypoventilation
- Coma or near-coma states
- Facial puffiness, dry skin, non-pitting edema

**Diagnosis** is supported by elevated TSH and low Free T4, with early recognition crucial due to high mortality.

## G. Thyrotoxic Crisis (Thyroid Storm)

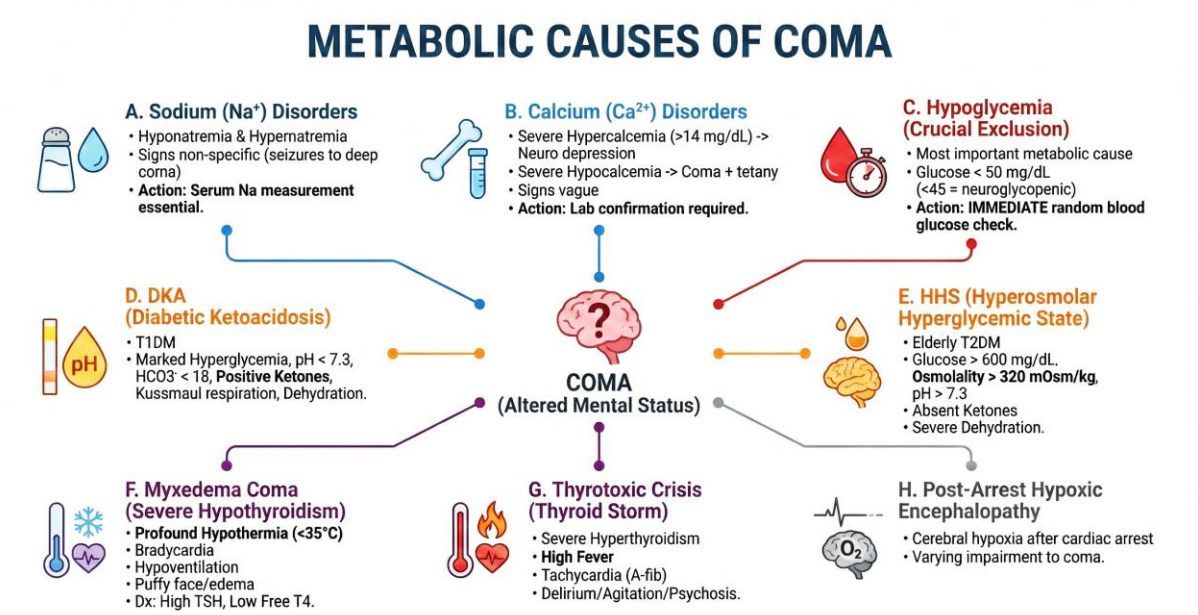
- This condition represents severe, life-threatening hyperthyroidism, frequently seen in patients with Graves' disease.

**Clues:**

- High fever
- Tachycardia, atrial fibrillation may occur
- Neurologic symptoms (delirium, agitation, psychosis)

## H. Post-Arrest Hypoxic Encephalopathy

- This condition is typically caused by cerebral hypoxia, In patients who have experienced a cardiac arrest, which may result in varying degrees of consciousness impairment, including coma.




# SEPTIC ENCEPHALOPATHY

Septic encephalopathy associated with severe systemic infection and inflammation. It can develop early in the course of sepsis.

**Clues:**

- The condition typically begins with an identifiable septic focus (pneumonia, urinary tract infection)

**Common early signs include:**

- Fever (or sometimes hypothermia)
- Tachypnea
- Chills, rigors
- Progressive altered mental status, ranging from confusion to coma

**THE QSOFA SCORE IS USED AS A RAPID BEDSIDE ASSESSMENT TO IDENTIFY HIGH-RISK PATIENTS.**

**It includes:**

- Altered mental status (Glasgow Coma Scale <15).
- Respiratory rate ≥ 22 breaths/min.
- Systolic blood pressure ≤ 100 mmHg.

If qSOFA score ≥ 2 → high risk of poor outcome and possibly septic shock.

# TOXIC ENCEPHALOPATHY

## INTOXICATION AS A CAUSE OF COMA

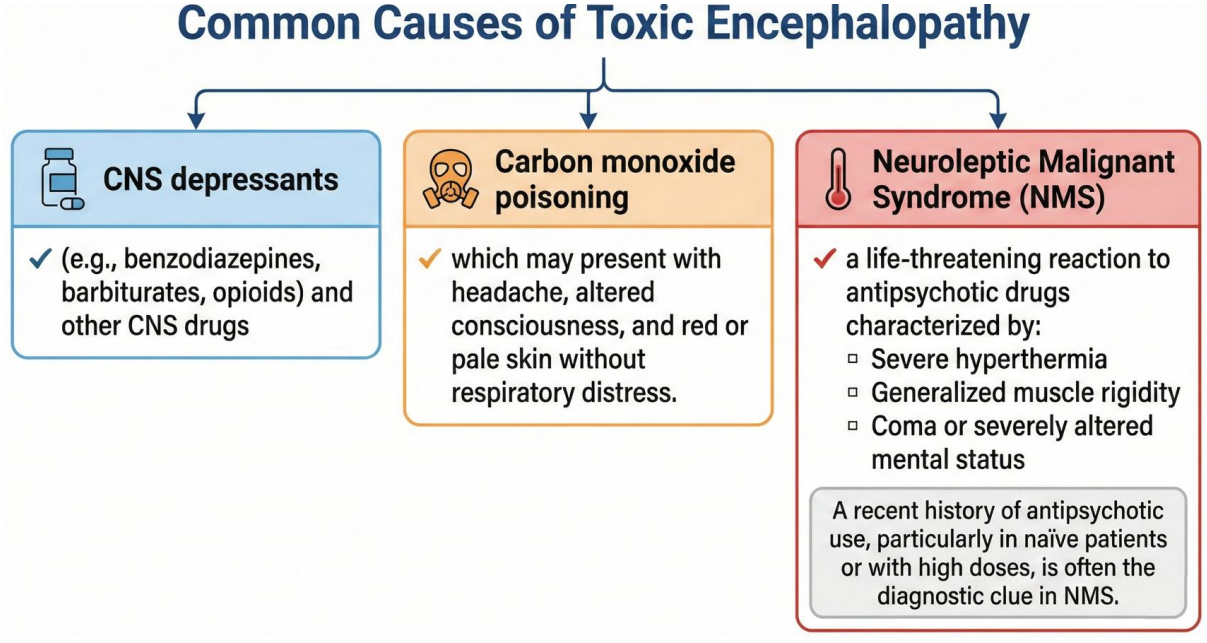
**Intoxication** is a major differential diagnosis in cases of non-focal coma, especially when no clear structural, metabolic, or infectious cause is identified. It is often a diagnosis of exclusion.

### WHEN TO SUSPECT IT?

Toxic coma should be considered in patients who present with:

- Normal pupils
- No lateralizing neurological signs
- No evident organ failure
- Normal ABG and electrolyte profiles
- No hypoglycemia
- Adequate oxygen saturation
- No signs of psychogenic or functional coma

In the absence of a clear etiology, intoxication becomes a leading suspect, particularly when history is limited or unavailable.
Collateral history from family members is essential, as the patient is often unresponsive and unable to provide any details.



# Step 2 – History Taking / Examination

## A HISTORY TAKING

In a coma case, the patient cannot communicate, so history-taking depends entirely on collateral history from (relatives, friends, witnesses).

**A structured and professional history helps determine:**

- The cause of coma (Structural vs Metabolic and other causes).
- The onset and progression of illness.
- Possible reversible or life-threatening conditions.

### Presenting Complaint and Onset

Ask ⤵

- When was the patient last seen normal?
- When and how did the coma start? (Sudden or gradual?)

| If Sudden onset | If acute onset |
|---|---|
| Think about Stroke, seizure, head trauma, hypoglycemia. | Think about Metabolic, hepatic, renal, infectious, or toxic causes. |

- Also ask about progression:
  - ➢ Was the loss of consciousness sudden and complete, or preceded by confusion, headache, or weakness?"

### Events Before Loss of Consciousness

Try to reconstruct what happened just before the coma:

- **Headache or vomiting?** → Raised intracranial pressure or hemorrhage.
- **Seizure or convulsion?** → Post-ictal coma or status epilepticus.
- **Trauma or fall?** → Head injury.
- **Fever or infection symptoms?** → Meningitis, encephalitis, sepsis.
- **Drug ingestion, alcohol, or toxins?** → Poisoning or overdose.
- **Fasting or insulin use?** → Hypoglycemia.

## Associated Symptoms:

Identify other features that may guide the diagnosis:

- **Headache / Neck stiffness / Photophobia** → Meningitis or subarachnoid hemorrhage.
- **Focal neurological deficits** particularly motor deficit (e.g. hemiplegia) points towards stroke
- **Fever / Chills** → Infection or sepsis.
- **Shortness of breath** → Hypoxia, metabolic acidosis, pulmonary embolism.
- **Jaundice** → Hepatic failure.
- **Oliguria with uremic features** → uremic encephalopathy
- **Polyuria / Polydipsia / Weight loss** → Diabetic ketoacidosis.

## Past Medical History

It is essential to identify pre-existing diseases or risk factors:

- **Diabetes mellitus**: hypoglycemia, DKA, HHS.
- **Hypertension**: intracerebral hemorrhage, stroke.
- **Epilepsy**: post-ictal states, status epilepticus.
- **Chronic liver disease**: hepatic encephalopathy.
- **Chronic kidney disease**: uremic encephalopathy.
- **Cardiac disease & AF** may point toward embolic stroke.
- **Thyroid disorders**: myxedema coma or thyroid storm.
- **Ask about** recent infections, hospitalizations, or surgical procedures.
- **Ask about**: Current medications: insulin, oral hypoglycemics, antihypertensives, sedatives, antidepressants, antipsychotics.
- **Ask about** Recent medication changes or overdoses
- Alcohol intake?, Illicit drug use?, Exposure to carbon monoxide?, pesticides?, or chemicals?
- Always check for empty medication bottles, needle marks, or odors (alcohol, acetone, kerosene, etc.).

## B EXAMINATION

Clinical examination in a comatose patient is one of the most critical skills in ER

| "STABILIZE FIRST, THEN EXAMINE SYSTEMATICALLY." |
|---|
| Always ensure Airway, Breathing, and Circulation (ABC) are secured before detailed examination. |

**1. Vital Signs:** BP, HR, RR, Temperature, SPO2

| Marked hypertension | Severely low blood pressure |
|---|---|
| Commonly associated with Intracerebral hemorrhage, Hypertensive encephalopathy, Acute ischemic stroke | Occur in shock, whether septic, hypovolemic, or cardiogenic |

**2. Measure Level of Consciousness**

Assess the depth of coma using the Glasgow Coma Scale (GCS):

| Eye opening (E) | Verbal response (V) | Motor response (M) |
|---|---|---|

Record each component separately (e.g., E2 M4 V2 = GCS 8).

**Also note the type of motor response to pain:**

- Abnormal flexion (decorticate) or extension (decerebrate) → severe brain injury.
- No response → deep coma or brain death.

**3. Pupillary Examination**

**Examine size, symmetry, and light reaction of pupils.**

- Equal and reactive pupils → (metabolic or toxic causes)
- Unequal pupils (anisocoria) → Structural lesion, Uncal herniation.
- Pinpoint pupils → pontine hemorrhage or opioid toxicity.

| NOTE |
|---|
| Use a penlight to check direct and consensual response.<br>If no reaction, recheck in a dark room, sometimes sluggish reactivity may be overlooked. |

## 4. Eye Movements and Brainstem Reflexes

**Eye signs are the window to the brainstem — essential for localizing coma.**

### A. Eye gaze (conjugate eye movements)

- Examine for abnormal eye gaze

### B. Oculocephalic Reflex (Doll's Eye Test)

Turn the head briskly from side to side.

- **Normal**: eyes move in the opposite direction of head movement → brainstem intact.
- **Absent movement**: indicates brainstem dysfunction (structural cause).

### C. Corneal Reflex:

Touch the cornea gently with sterile gauze.

- **Normal**: patient blinks.
- **Absent**: brainstem involvement.

## 5. Motor System Examination

Evaluate tone, reflexes, and motor response to pain.

### A. Tone

- **Increased tone (hypertonia):** points to a structural lesion.
- **Decreased tone (flaccidity):** often due to metabolic cause.

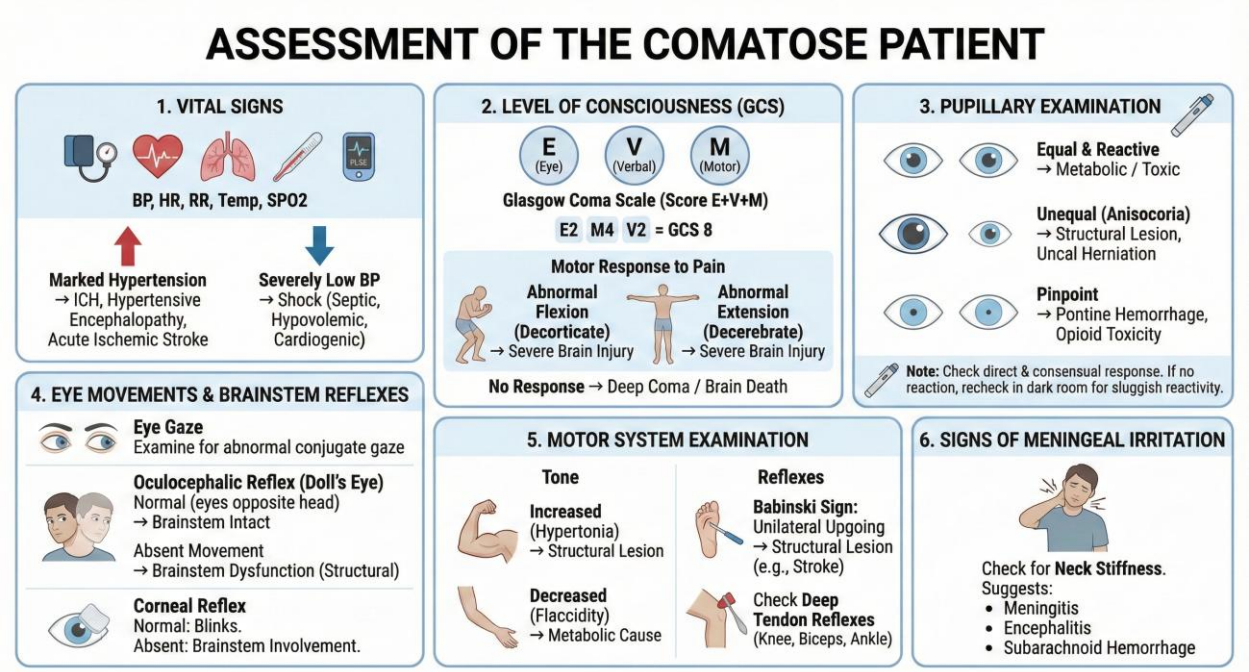
### B. Reflexes

- **Test plantar reflex (Babinski sign):** unilateral upgoing response → structural lesion e.g stroke
- **Check deep tendon reflexes** (knee, biceps, ankle).

## 6. Signs of Meningeal Irritation

**Check for neck stiffness.**

- Presence of these signs suggests meningitis/encephalitis or subarachnoid hemorrhage.




## CLINICAL PEARLS

- ❖ Always reassess GCS, pupils, and vital signs — they can change within minutes.
- ❖ Examine from head to toe, but prioritize neurological and vital signs findings
- ❖ Combine clinical examination with history — together they guide diagnosis before investigations.

## Step 3 – Investigations?

Investigations in a comatose patient must be systematic, prioritized, and time-sensitive.

**Important:**

- Investigations should never delay resuscitation.
- Always stabilize the Airway, Breathing, and Circulation (ABC) before starting diagnostic workup.

## Basic Laboratory Investigations:

**1. Blood Glucose** → Always the first test for any unconscious patient.

**2. CBC** → Detect infection (leukocytosis), anemia, or bleeding tendency (platelet count).

**3. Serum Electrolytes ($Na^+$, $K^+$, $Ca^{2+}$, $Mg^{2+}$, $Cl^-$)** → any disturbances can directly impair brain function.

- Hyponatremia → cerebral edema → coma.
- Hypernatremia → dehydration → confusion.
- Hypocalcemia → seizures.

**4. Renal Function Tests (RFTs):**

- Urea, Creatinine, BUN → evaluate for Uremic encephalopathy.

**5. Liver Function Tests (LFTs):**

- AST, ALT, Bilirubin, albumin → to detect Hepatic encephalopathy and underlying pre-existing liver disease.

**6. Arterial Blood Gas (ABG):**

Checks for acidosis, hypoxia, or $CO_2$ retention.

- Metabolic acidosis → e g DKA, sepsis and renal failure
- Respiratory acidosis → respiratory failure ($Co_2$ narcosis)

## Imaging and Specialized Investigations

After stabilization and initial lab results, proceed with imaging to identify structural lesions or confirm the metabolic hypothesis.

**1. CT Brain:**

- First-line imaging in all suspected coma due to structural causes
- Detects: Intracranial hemorrhage, Infarction, Brain edema, Tumor or mass effect, Midline shift or herniation.

**Note:** Always do CT before lumbar puncture if there's any sign of raised ICP.

**2. MRI Brain:**

- **More sensitive for**: brainstem or small ischemic lesions, diffuse axonal injury, PRES, Encephalitis or early infarction.

**3. MRV** → if suspect cerebral venous thrombosis

**3. Lumbar Puncture (LP):**

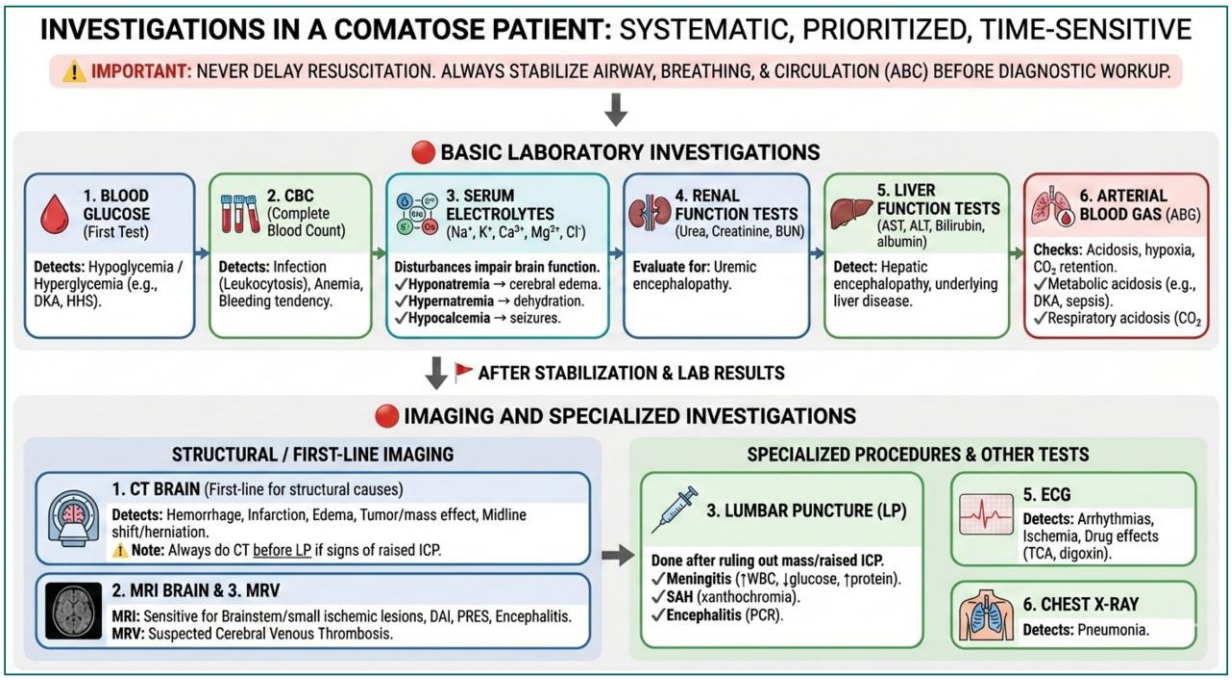
- Done after ruling out mass lesion or raised ICP.

**- Diagnostic for:**

- ❖ Meningitis (↑WBCs, low glucose, high protein).
- ❖ Subarachnoid hemorrhage (xanthochromia).
- ❖ Encephalitis (PCR for viruses).

**5. ECG** → detects arrhythmias, ischemia, drug effects (TCA, digoxin).

**6. Chest X-Ray** → detects pneumonia




## Step 4 – How to manage?

- ❖ In all cases, Management of ABC is vital, urgent intubation is needed if GCS is ≤ 8 to protect the airway.
- ❖ Admit all cases at the ICU and treat the cause.