# Here are possible causes of loss of consciousness (LOC) in Mrs. AB, a 65-year-old woman, 3 hours after hearing about her brother's death: ### 1. Neurogenic Causes \* Stroke (Ischemic or Hemorrhagic): Sudden emotional stress may elevate blood pressure and precipitate a cerebrovascular event. \* Transient Ischemic Attack (TIA): Brief, reversible reduction in cerebral perfusion. \* Seizure (with post-ictal confusion): Especially if she had prior epilepsy or a seizure threshold is lowered. ### 2. Cardiogenic Causes \* Vasovagal Syncope (Neurocardiogenic): Strong emotional stress (death of a loved one) can trigger fainting via vagus nerve stimulation. \* Arrhythmia (e.g., ventricular tachycardia): Can lead to decreased cerebral perfusion. \* Myocardial infarction: Especially stress-induced (Takotsubo cardiomyopathy) in elderly women. ### 3. Metabolic Causes \* Hypoglycemia: Especially in diabetics or those skipping meals under stress. \* Electrolyte Imbalance: Can lead to altered mental status or LOC. ### 4. Psychogenic Causes \* Psychogenic non-epileptic seizure (PNES): Stress-induced dissociation or conversion disorder. \* Acute stress reaction or catatonia: Seen in severe emotional trauma. ### 5. Other \* Head trauma: From a fall after initial syncope. \* Drug or alcohol intoxication: If there’s a history of use. Would you like a structured table summarizing these with key features for each? Loss of consciousness (LOC) is a state in which a person is unaware of themselves and their surroundings, and is unresponsive to external stimuli. ### Key Features: \* Sudden or gradual onset \* Temporary or prolonged duration \* Reversible or irreversible, depending on the cause ### Clinical Signs: \* Unresponsiveness \* No verbal or purposeful motor response \* May involve loss of postural tone (collapse) ### Causes are broadly grouped into: 1. Neurological (e.g., stroke, seizure) 2. Cardiac (e.g., arrhythmia, syncope) 3. Metabolic (e.g., hypoglycemia) 4. Psychiatric (e.g., conversion disorder) 5. Toxicologic (e.g., drug overdose) Would you like the differences between syncope and other causes of LOC? Physiologically, loss of consciousness (LOC) occurs when the brain — especially the cerebral cortex and/or reticular activating system (RAS) — does not receive adequate oxygen, glucose, or perfusion. ### Main Physiological Causes: 1. Reduced Cerebral Blood Flow \* Causes: Syncope (vasovagal, cardiac), shock \* Mechanism: Drop in blood pressure → ↓ brain perfusion 2. Decreased Oxygen Supply (Hypoxia) \* Causes: Respiratory failure, severe anemia \* Mechanism: Less oxygen in blood → brain tissue hypoxia 3. Decreased Glucose Supply (Hypoglycemia) \* Causes: Insulin overdose, fasting in diabetics \* Mechanism: Glucose is essential for brain function 4. Structural Brain Disorders \* Causes: Stroke, trauma, tumor \* Mechanism: Disrupts neurons in consciousness centers 5. Electrical Disturbance \* Causes: Seizure \* Mechanism: Abnormal neuronal firing disrupts normal consciousness 6. Toxins or Metabolic Disturbances \* Causes: Drugs, hepatic or uremic encephalopathy \* Mechanism: Toxic substances depress brain activity In short: Anything that reduces blood flow, oxygen, or glucose to the brain — or directly damages brain activity — can cause LOC. Want a visual diagram of this flow? The RAS (Reticular Activating System) is a network of nerve cells in the brainstem that plays a crucial role in maintaining consciousness, wakefulness, and alertness. RAS and Clinical Case Analysis

## 🔹 What is the RAS?

• Located in the brainstem, especially in the midbrain and upper pons.  
• Made up of interconnected neurons that project to the thalamus and then to the cerebral cortex.

## 🔹 How Does the RAS Work?

1. Receives Input  
 • From sensory systems (sight, sound, touch, etc.)  
 • From internal signals (like pain, movement)  
  
2. Activates the Thalamus  
 • The RAS sends signals to the thalamus, the brain’s relay station.  
  
3. Stimulates the Cerebral Cortex  
 • From the thalamus, signals go to the cerebral cortex, which controls awareness and thinking.  
  
4. Maintains Wakefulness  
 • As long as the RAS is active, a person remains awake and conscious.  
 • If it's damaged or suppressed (e.g., by trauma, drugs, or hypoxia), consciousness is lost.

## 🔹 Summary:

|  |  |
| --- | --- |
| Component | Role |
| Brainstem (RAS) | Receives and integrates sensory input |
| Thalamus | Relays signals from RAS to the cortex |
| Cerebral Cortex | Maintains conscious awareness |

## ✅ Pertinent Positives:

1. Known hypertension for 10 years (poorly controlled — stopped meds 1 year ago).  
2. Loss of consciousness for 3 hours.  
3. Emotional trigger (death of her younger brother).  
4. Incontinence (fecal and urinary) — suggests possible seizure or stroke.  
5. Chronic headache — for the past 2 months (possible warning sign of CNS pathology).

## ❌ Pertinent Negatives:

1. No body weakness  
2. No abnormal body movements (no observed seizure activity)  
3. No blurring of vision  
4. No similar illness in self or family  
5. No history of chronic illnesses like diabetes, cardiac, renal, or liver disease  
6. No trauma history  
7. No recent medication intake

## Differential Diagnosis Considerations

### 🔍 Stroke (Hemorrhagic or Ischemic)

✅ Supported by:  
 \* Hypertension (uncontrolled)  
 \* Sudden LOC  
 \* Headache (2-month history may suggest evolving pathology like aneurysm)  
 \* Incontinence  
❌ Not ruled out — remains a strong possibility

### 🔍 Seizure

✅ Supported by:  
 \* LOC with incontinence (common post-ictal feature)  
❌ Against:  
 \* No abnormal body movements reported  
 \* No history of seizures  
⚠️ Still possible (especially if focal seizure or unwitnessed convulsion)

### 🔍 Vasovagal Syncope

✅ Supported by:  
 \* Strong emotional trigger  
❌ Against:  
 \* LOC for 3 hours is too long (vasovagal typically <1 min)  
 \* Incontinence is unusual in simple syncope  
❌ R/O — unlikely

### 🔍 Cardiogenic Syncope/Arrhythmia

❌ Against:  
 \* No cardiac history  
 \* No palpitations or chest pain  
 \* No recent medication use  
⚠️ Still possible, but less likely without cardiac symptoms

### 🔍 Hypoglycemia

❌ Against:  
 \* No history of diabetes  
 \* No medication use  
❌ R/O

### 🔍 Psychogenic (e.g., Conversion Disorder)

✅ Supported by:  
 \* Emotional trigger  
❌ Against:  
 \* Incontinence not typical  
 \* Duration of LOC and age make this less likely  
❌ R/O — unlikely

### 🔍 Brain Tumor or Mass Lesion

✅ Supported by:  
 \* Headache history  
 \* New-onset LOC  
⚠️ Possible, though less acute presentation unless hemorrhage or seizure occurs

## ✅ Positive Physical Examination Findings

These are findings that support serious neurological pathology, most likely a stroke (probably hemorrhagic):

### 🔹 General & Vitals:

• Acutely sick-looking, comatose  
• BP = 190/110 mmHg → severe hypertension (risk factor for hemorrhagic stroke)  
• RR = 21/min, irregular & deep → possible central neurogenic pattern

### 🔹 CNS Findings (Highly Significant):

• GCS = 8/15 → Coma (severe impaired consciousness)  
• Fixed & dilated left pupil → suggests uncal herniation (compression of CN III)  
• Right facial deviation → suggests left-sided facial nerve weakness  
• Hypertonia on left → suggests right-sided brain insult  
• Reflexes: 3+ on left, Babinski upgoing → UMN lesion signs  
  
⮕ All point toward a right-sided cerebral lesion, possibly intracerebral hemorrhage with mass effect.

### 🔹 Other Systems:

• No signs of infection, trauma, or systemic illness  
• No murmurs or signs of cardiac source of emboli

### ✅ Summary of Key Positive Findings:

|  |  |  |
| --- | --- | --- |
| System | Positive Findings | Interpretation |
| Neuro | GCS 8, left fixed pupil, right facial deviation, left hypertonia, Babinski | Suggests right hemispheric lesion, likely hemorrhagic stroke with herniation |
| Vitals | BP 190/110, PR 108, RR 21 irregular | Hypertensive crisis, possible central brain dysfunction |
| General | Acutely ill, comatose | Emergency CNS pathology |

# Working Diagnosis and Investigation Plan

## Positive Findings and Pathophysiology

### 1. Coma (GCS = 8/15)

Cause: Disruption of the reticular activating system (RAS) or diffuse hemispheric dysfunction  
Pathophysiology: A large hemorrhage increases intracranial pressure (ICP) → decreases cerebral perfusion → compresses RAS → loss of consciousness

### 2. Fixed and Dilated Left Pupil

Cause: Uncal herniation  
Pathophysiology: Hemorrhage → brain tissue shifts (herniates) → compresses left oculomotor nerve (CN III) → pupil dilates and becomes non-reactive due to loss of parasympathetic fibers

### 3. Right Facial Deviation

Cause: Left facial muscle weakness  
Pathophysiology: Right cortical or internal capsule lesion affects corticobulbar tract → weakness of contralateral (left) lower face → unopposed muscles on right pull face → deviation to right

### 4. Hypertonia in Left Upper & Lower Limbs

Cause: Upper motor neuron lesion (UMN)  
Pathophysiology: Damage to motor tracts (corticospinal) in right hemisphere → loss of inhibition to spinal motor neurons → increased muscle tone (spasticity) on left

### 5. 3+ Reflexes and Positive Babinski (on Left)

Cause: Pyramidal tract dysfunction  
Pathophysiology: UMN damage → exaggerated reflexes and Babinski reflex (toes fan upward) due to loss of inhibitory control from cortex

### 6. BP = 190/110 mmHg

Cause: Chronic hypertension and/or Cushing reflex (ICP compensation)  
Pathophysiology: Chronic high BP causes small vessel rupture → intracerebral hemorrhage OR Increased ICP → sympathetic activation → systemic vasoconstriction → hypertension

### 7. Irregular, Deep Respirations

Cause: Central neurogenic breathing (e.g., Cheyne-Stokes or agonal)  
Pathophysiology: Raised ICP or brainstem dysfunction → disrupts respiratory centers in medulla/pons → abnormal breathing patterns

## ✅ Interpretation of Investigations

### 🔹 1. CBC

|  |  |  |
| --- | --- | --- |
| Test | Result | Interpretation |
| WBC | 11,000 | Normal – no infection |
| HCT | 36% | Low-normal – possible mild hemodilution |
| PLT | 212,000 | Normal – no thrombocytopenia |

### 🔹 2. Lipid Profile

|  |  |  |
| --- | --- | --- |
| Test | Result | Interpretation |
| Cholesterol | 180 mg/dL | Normal |
| HDL | 89 mg/dL | High – protective effect |
| LDL | 45 mg/dL | Low – could be genetic or due to malnutrition |

### 🔹 3. Renal Function Test (RFT)

|  |  |  |
| --- | --- | --- |
| Test | Result | Interpretation |
| Creatinine | 1.2 mg/dL | High-normal |
| BUN | 20 mg/dL | High-normal |

### 🔹 4. Liver Function Test (LFT)

|  |  |  |
| --- | --- | --- |
| Test | Result | Interpretation |
| SGOT | 30 U/L | Normal |
| SGPT | 29 U/L | Normal |
| ALP | 170 U/L | Elevated – may suggest bone turnover, age-related, or subclinical cholestasis (not acute issue here) |

### 🔹 5. RBS

105 mg/dL → Normal

### 🔹 6. CT Brain (Non-Contrast)

• Hyperdense area in putamen & thalamus (3 x 5 cm)  
→ Classic for hypertensive intracerebral hemorrhage (ICH)  
→ Putamen is the most common site  
→ Mass effect likely causing herniation signs (fixed pupil)

### 🔹 7. ECG & ECHO

• ECG: Normal  
• Echo: Left Ventricular Hypertrophy (LVH)  
→ Sign of chronic hypertension  
→ EF 60% = Normal systolic function

### 🔹 8. Coagulation Panel

• PT, PTT, INR: Normal → Coagulopathy unlikely

### 🔹 9. Serum Electrolytes & CXR

• Both normal → No metabolic or respiratory cause

✅ Summary of Significant Findings:  
  
| Finding | Interpretation |  
| -------------------------------- | -------------------------------------------------- |  
| CT Scan: Putaminal bleed | Confirms hypertensive ICH |  
| BP = 190/110 + LVH | Chronic hypertension as etiology |  
| Fixed pupil + coma + GCS 8 | Suggests mass effect with brain herniation |  
| Normal coagulation, glucose, ECG | Rules out metabolic, infectious, or cardiac causes |  
  
Would you like help forming the final diagnosis and management plan?  
Here’s a breakdown of the positive findings and their possible pathophysiology:  
  
1. WBC (11,000 cells/mm³)  
 \* Pathophysiology: Mild leukocytosis could be due to an infection, inflammation, or stress response. In the absence of infection or acute inflammation, it could also indicate a response to other conditions like recent trauma.  
  
2. HCT (36%)  
 \* Pathophysiology: On the lower end of normal; could suggest mild anemia, particularly if the patient has any symptoms like fatigue or weakness.  
  
3. PLT (212,000 cells/mm³)  
 \* Pathophysiology: Normal platelet count; no immediate concerns. Platelet count might reflect a balanced bone marrow production, not indicating bleeding or clotting risks.  
  
4. HDL (89 mg/dl)  
 \* Pathophysiology: Elevated HDL is generally considered protective against cardiovascular disease, reflecting healthy lipid metabolism and reduced risk of atherosclerosis.  
  
5. LDL (45 mg/dl)  
 \* Pathophysiology: Low LDL levels can be protective against heart disease, but extremely low levels might also signal malnutrition or other metabolic concerns.  
  
6. Cr (1.2 mg/dl)  
 \* Pathophysiology: On the upper end of normal. If the patient has kidney disease or symptoms of renal impairment, this could indicate reduced renal clearance.  
  
7. BUN (20 mg/dl)  
 \* Pathophysiology: Normal BUN but on the higher end; it could indicate mild dehydration or renal stress, especially in combination with elevated creatinine.  
  
8. ALP (170 u/L)  
 \* Pathophysiology: Elevated ALP could suggest liver or bone disease. The value is elevated beyond the normal range, potentially indicating cholestasis, bone disorders, or liver pathology.  
  
9. RBS (105 mg/dl)  
 \* Pathophysiology: Elevated blood sugar, although within the normal range, could indicate a risk for impaired glucose tolerance or prediabetes, especially if consistently elevated.  
  
10. CT Brain (3 x 5 cm hyperdense area in putamen and part of thalamus)  
 \* Pathophysiology: A hyperdense lesion on a CT scan could indicate hemorrhage, ischemia, or a calcified structure. The location in the putamen and thalamus suggests a possible vascular event like a stroke.  
  
11. LVH with EF of 60%  
 \* Pathophysiology: Left ventricular hypertrophy (LVH) can be due to chronic hypertension or other cardiac conditions. An EF of 60% is normal, indicating preserved systolic function, but LVH may increase the risk of arrhythmias or heart failure over time.  
  
Case summary  
This is a known hypertensive patient for the past 10 years who discontinued medication since a year back presented with loss of consciousness for the past 3 hours after she was told that her younger brother. He has fecal and urinary incontinence. Had history of headache since 2 weeks back. No other pertinent Hx.  
On examination ,acutely sick looking (coma)  
V/s; BP =190/110 mmHg, rtarm, supine position  
PR= 108 bpm, regular, full in volume  
RR= 21 breaths/min, irregular, deep  
To= 37.8 0c, axillary, in the morning  
SaO2 = 94 with atm air.  
CNS; - Comatose with GCS of 8/15 (E-2, V-3, M -3)  
Fixed and dilated left pupil, reactive and normal size right pupil  
Facial deviation to the right  
Hypertonic left upper and lower extremities, with comparable muscle bulk.  
Power is difficult to assess.  
Reflex is ¾ on her Lt Upper and lower extremities, with no clonus.  
Babiniski is upgoing.  
Non contrast enhanced CT scan of brain  
3 x 5 cm hyper dense are in the putamen and part of thalamus.  
She was diagnosed to have coma 2o to hemorrhagic stroke + Left sided facial palsy and is being managed with  
Coma care,  
NGT feeding  
Antihypertensive  
Close monitoring of BP  
Bedside physiotherapy  
  
MINI CASE  
What if she was a known cardiac patient and was found to have left sided body weakness while asleep. On physical exam, PR of 150 bpm, irregularly irregular