Coma

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Objectives

☐ Primary Objective: The physician should be able to stabilize, evaluate, and treat the comatose patient in the emergent setting.

☐ The physician should understand this involves an organized, sequential, prioritized approach.
The Comatose Patient

Primary Objectives

- Airway
- Breathing
- Circulation
- Treatment of rapidly progressive, dangerous metabolic causes of coma (hypoglycemia)
- Evaluation as to whether there is significant increased ICP or mass lesions.
- Treatment of ICP to temporize until surgical intervention is possible.
The Comatose Patient

Secondary Objectives

☐ The physician should understand and recognize:
  ☐ Coma
  ☐ Herniation syndromes
  ☐ Signs of supratentorial mass lesions
  ☐ Signs of subtentorial mass lesions

☐ The physician should be able to develop the differential diagnosis of metabolic coma.
coma

- Pathological unresponsiveness
  - No response, other than reflex, to external stimuli or inner needs
  - A symptom, not a disease
  - Multiple causes
Consciousness requires:
- An intact reticular activating system
- An intact cerebral hemisphere, or at least part of a hemisphere

Coma requires dysfunction of either the:
- Reticular activating system, or
- Bihemispheric cerebral dysfunction
Arousal (wakefulness)

Alertness is dependent on the upper brainstem and diencephalon.

The Ascending Reticular Activating System (ARAS) ascends from the midpons extending rostrally through the midline and intralaminar nuclei of the thalamus to the cerebral cortex.
Reticular formation

- Central core of the brainstem
- Involved in … *Control of movement - pontomedullary*
- *Modulation of pain - midbrain & pontine*
- *Autonomic reflexes*
- *Arousal and consciousness -- midbrain/upper pons*
Physiology of coma

- Requires dysfunction of either:
  - Bilateral hemispheres
  - Reticular activating system (RAS) in the brain stem and interthalamic nuclei

- 3 ways to organize your approach

- Both hemispheres vs Reticular activating system
- Supratentorial vs Infratentorial
- Metabolic vs Anatomic (destructive)
Other states

Akinetic mutism: Immobility and muteness but appears alert

Locked in syndrome: the patient is fully conscious but totally paralyzed and can usually only respond by eye movement and even this is in a limited direction.
Persistent vegetative state: dissociation between arousal and awareness the combination of periods of wakeful eye opening with lack of any evidence of a working mind either receiving or projecting information.

Minimal conscious state: (sustained, reproducible, purposeful or voluntary responses)
The Comatose Patient

Classifications

- **Supratentorial** lesions cause coma by either widespread bilateral disease, increased intracranial pressure, or herniation.

- **Infratentorial** lesions involve the RAS, usually with associated brainstem signs

- **Metabolic** coma causes diffuse hemispheric involvement and depression of RAS, usually without focal findings

- **Psychogenic**
# Common Etiologies of Coma

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Approximate mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug Overdose</td>
<td>5-10%</td>
</tr>
<tr>
<td>Metabolic</td>
<td>50%</td>
</tr>
<tr>
<td>Head Trauma</td>
<td>50%</td>
</tr>
<tr>
<td>Anoxia</td>
<td>90%</td>
</tr>
<tr>
<td>Stroke</td>
<td>80%</td>
</tr>
<tr>
<td>Status Epilepticus</td>
<td>3-30%</td>
</tr>
</tbody>
</table>
Supratentorial Mass Lesions

- Hematoma
- Neoplasm
- Abscess
- Contusion
- Vascular Accidents
- Diffuse Axonal Damage
Supratentorial Mass Lesions

Acute epidural hematoma and midline shift
Supratentorial Mass Lesions

Cerebral Abscess
Severe head trauma with basilar skull fracture, right temporal hematoma, cerebral edema, hydrocephalus, and pneumocephalus
Supratentorial Mass Lesions

Subdural Hematoma
Altered consciousness is based on:
- Increased intracranial pressure
- Herniation
- Diffuse bilateral lesions
Sites of Herniations
Herniation syndromes
Transventorial herniation and brainstem infarction in a patient with melanoma
Signs of increased ICP/Herniation

Pupils
  Unilateral dilated pupil
  Bilateral small poorly reactive pupils

Eye movements
  Third nerve palsy
  Sixth nerve palsy
  Can be assessed by cold caloric

Fundoscopy
  Signs of papilledema?

Respiratory pattern?
Supratentorial Mass Lesions

Differential Characteristics

Initiating signs usually of focal cerebral dysfunction
Signs of dysfunction progress rostral to caudal
Neurologic signs at any given time point to one anatomic area - diencephalon, midbrain, brainstem
Motor signs are often asymmetrical

Plum and Posner, 1982
Abnormal Breathing Patterns

Cheyne-Stokes
- crescento/decrescendo pattern mixed with apnea
- bilateral hemisphere dysfunction

Central neurogenic hyperventilation
- rapid deep breathing
- lesion between midbrain and pons

Apneustic breathing
- prolonged inspiration followed by apnea
- pontine dysfunction

Ataxic breathing
- irregular pattern
- medullary dysfunction-close to death

Coma with hyperventilation
- metabolic derangement

Coma with hypoventilation
- drug overdose

COPD
Rostral Caudal Progression
Motor response in coma

Coma: Motor

Withdrawal or Decorticate

Decerebrate

Flaccid
Prognosis in Coma Related to Severe Head Injuries

<table>
<thead>
<tr>
<th>Poor</th>
<th>Better</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glasgow Coma Scale</td>
<td></td>
</tr>
<tr>
<td>CT scan:</td>
<td>Normal</td>
</tr>
<tr>
<td>Age:</td>
<td>Youth</td>
</tr>
<tr>
<td>Pupillary light reflexes:</td>
<td>Pupil consensual</td>
</tr>
<tr>
<td>Caloric testing with ice water:</td>
<td>Conscious</td>
</tr>
<tr>
<td>Motor response to noxious stimuli:</td>
<td>Complete</td>
</tr>
<tr>
<td>Brainstem auditory evoked response:</td>
<td>Deficit</td>
</tr>
</tbody>
</table>

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Rostral Caudal Progression

- Small reactive
- Diencephalic, small reactive
- Tectal, large "fixed", hippocus
- II Nerve (Uncal), dilated, fixed
- Pons, pinpoint
- Midbrain, midposition, fixed
Pinpoint pupils

- Pontine hemorrhage
- Organophosphate poisoning \([\text{acetylcholine esterase inhibitors}]\)
- Narcotics
- Syphilis
- Constrictor drops
Eye movements in coma

- Oculocephalic -- doll’s eyes
- Oculovestibular - ice water calorics

Normal eye movements in coma mean the brainstem pathways subserving these reflexes are intact from upper medulla to midbrain
<table>
<thead>
<tr>
<th>Term</th>
<th>Description</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ocular bobbing</td>
<td>Rapid, conjugate, downward movement; slow return to primary position</td>
<td>Pontine strokes; other structural, metabolic, or toxic disorders</td>
</tr>
<tr>
<td>Ocular dipping or inverse ocular bobbing</td>
<td>Slow downward movement; rapid return to primary position</td>
<td>Unreliable for localization; follows hypoxic-ischemic insult or metabolic disorder</td>
</tr>
<tr>
<td>Reverse ocular bobbing</td>
<td>Rapid upward movement; slow return to primary position</td>
<td>Unreliable for localization; may occur with metabolic disorders</td>
</tr>
<tr>
<td>Reverse ocular dipping or converse bobbing</td>
<td>Slow upward movement; rapid return to primary position</td>
<td>Unreliable for localization; pontine infarction and with AIDS</td>
</tr>
<tr>
<td>Ping-pong gaze</td>
<td>Horizontal conjugate deviation of the eyes, alternating every few seconds</td>
<td>Bilateral cerebral hemispheric dysfunction; toxic ingestion</td>
</tr>
<tr>
<td>Periodic alternating gaze deviation</td>
<td>Horizontal conjugate deviation of the eyes, alternating every 2 minutes</td>
<td>Hepatic encephalopathy; disorders causing periodic alternating nystagmus and unconsciousness or vegetative state</td>
</tr>
<tr>
<td>Vertical “myoclonus” Monocular movements</td>
<td>Vertical pendular oscillations (2–3 Hz) Small, intermittent, rapid monocular horizontal, vertical, or torsional movements</td>
<td>Pontine strokes Pontine or midbrain destructive lesions, perhaps with coexistent seizures</td>
</tr>
<tr>
<td>Eye Opening</td>
<td></td>
<td></td>
</tr>
<tr>
<td>------------------</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td>Never</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>To pain</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>To verbal</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Spontaneous</td>
<td>4</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Best Motor Response</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td>Extensor</td>
<td>2</td>
</tr>
<tr>
<td>Flexor Posture</td>
<td>3</td>
</tr>
<tr>
<td>Withdrawal</td>
<td>4</td>
</tr>
<tr>
<td>Localization</td>
<td>5</td>
</tr>
<tr>
<td>obeys</td>
<td>6</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Best Verbal Response</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td>Sounds</td>
<td>2</td>
</tr>
<tr>
<td>Inapp word</td>
<td>3</td>
</tr>
<tr>
<td>disoriented</td>
<td>4</td>
</tr>
<tr>
<td>oriented</td>
<td>5</td>
</tr>
</tbody>
</table>
Infratentorial Lesions

- Cause coma by affecting reticular activating system in pons
- Brainstem nuclei and tracts usually involved with resultant focal brainstem findings
Infratentorial Lesions

Causes of Coma

- Neoplasm
- Vascular accidents
- Trauma
- Cerebellar hemorrhage
- Demyelinating disease
- Central pontine myelinolysis (rapid correction of hyponatremia)
Infratentorial Mass Lesions
Differential Characteristics

- History of preceding brainstem dysfunction or sudden onset of coma
- *Localizing brainstem signs* precede or accompany onset of coma and always include oculovestibular abnormality
- *Cranial nerve palsies* usually present
- “*Bizarre*” *respiratory patterns* common, usually present at onset of coma
Algorithm of coma diagnosis

- Coma
  - With lateralization
    - Stroke, brain abscess, tumour, etc
  - Without lateralization
    - Preceded by headache
      - Fever
      - Meningeal signs
    - Not preceded by headache
      - Fever or meningeal signs
      - Encephalitis
      - Metabolic encephalopathy
      - Ischemic hypoxic encephalopathy
      - Drug intoxication.
Figure 2–1. Methods for attempting to elicit responses from unconscious patients. Noxious stimuli can be delivered with minimal trauma to the supraorbital ridge (A), the nail beds or the fingers or toes (B), the sternum (C) or the temporomandibular joints (D).
Metabolic Coma

**Etiologies**

- **Respiratory**
  - Hypoxia
  - Hypercarbia

- **Electrolyte**
  - Hypoglycemia
  - Hyponatremia
  - Hypercalcemia

- **Hepatic encephalopathy**
- **Severe renal failure**
- **Infectious**
  - Meningitis
  - Encephalitis
- **Toxins, drugs**
Confusion and stupor commonly precede motor signs
Motor signs are usually symmetrical
Pupillary reactions are usually preserved
Asterixis, myoclonus, tremor, and seizures are common
Acid-base imbalance with hyper- or hypoventilation is frequent
examples of Common types of comas
Which type of coma? hepatic
Which type of coma?

Renal
Which type of coma?

Myxedema
Nonketotic hyperosmolar coma

- is usually precipitated by an acute illness. The serum glucose is usually higher than 600 mg/dl, and the resulting serum osmolarity is greater than 350 mOsm. Ketosis is absent because the presence of insulin inhibits lipolysis, unlike diabetic ketoacidosis.

- The treatment involves slow hydration and insulin. Anticoagulants (such as low molecular weight heparins) are often commenced as there is a significant rate of thrombosis in patients with NKHC.
Diabetic ketoacidosis

- (DKA), if it progresses and worsens without treatment, can eventually cause unconsciousness, from a combination of severe hyperglycemia, dehydration and shock, and exhaustion. Coma only occurs at an advanced stage, usually after 36 hours or more of worsening vomiting and hyperventilation.

- In the early to middle stages of ketoacidosis, patients are typically flushed and breathing rapidly and deeply, but visible dehydration, pallor from diminished perfusion, shallower breathing, and rapid heart rate are often present when coma is reached. However these features are variable and not always as described.

- If the patient is known to have diabetes, the diagnosis of DKA is usually suspected from the appearance and a history of 1-2 days of vomiting. The diagnosis is confirmed when the usual blood chemistries in the emergency department reveal hyperglycemia and severe metabolic acidosis.

- Treatment of DKA consists of isotonic fluids to rapidly stabilize the circulation, continued intravenous saline with potassium and other electrolytes to replace deficits, insulin to reverse the ketoacidosis, and careful monitoring for complications.
Hypoglycemic coma

- Unconsciousness due to hypoglycemia can occur within 20 minutes to an hour after early symptoms and is not usually preceded by other illness or symptoms. Twitching or convulsions may occur. A person unconscious from hypoglycemia is usually pale, has a rapid heart beat, and is soaked in sweat, all signs of the adrenaline response to hypoglycemia. He is not usually dehydrated and breathing is normal or shallow.

- A meter or laboratory glucose at the time of discovery is usually low, but not always severely, and in some cases may have already risen from the nadir which triggered the unconsciousness.

- Unconsciousness due to hypoglycemia is treated by raising the blood glucose with intravenous glucose.
Special situations

R/O Infectious etiology

R/O Epileptic Etiology
Infectious Etiology

- History
- Fever
- Nuchal rigidity
- Kernigs, Brudzinski
- Rash
Generalized Tonic-Clonic Seizures

A. Tonic phase
- Incoherence
- Eyelid myoclonus
- Eyelid blinking

B. Clonic phase
- Cyanosis
- Incoherence
- Salivary salivary
- Eyes blinking

C. Postictal stupor
- Cyanosis
- Incoherence
- Salivary salivary
- Arms and hands

EEG: tonic phase
- Generalized fast, repetitive spikes
- Generalized fast, repetitive spikes
- Generalized fast, repetitive spikes

EEG: clonic phase
- Generalized fast, repetitive spikes
- Generalized fast, repetitive spikes
- Generalized fast, repetitive spikes

EEG: postictal
- Generalized fast, repetitive spikes
- Generalized fast, repetitive spikes
- Generalized fast, repetitive spikes
Management of status epilepticus

- **Impending status epilepticus**
  - 5 min
    - Before emergency room
      - Diazepam rectal gel 15–20 mg
      - or
      - Intravenous lorazepam 2 mg, may repeat once
      - or
      - Intravenous diazepam 5 mg, may repeat once
    - Intravenous midazolam 0–2 mg/kg bolus 0.05 mg/kg/h
      - or
      - Intravenous lorazepam up to 0.1 mg/kg
      - or
      - Intravenous diazepam up to 0.25–0.4 mg/kg

- **Established status epilepticus**
  - 30 min
    - Emergency room
      - Intravenous fosphenytoin/phenytoin 20–30 mg/kg
      - or
      - Intravenous valproic acid 40–60 mg/kg 3 mg/kg/min

- **Refractory status epilepticus**
  - Intensive-care unit
    - Propofol loading 2–5 mg/kg
      - CIV 2–10 mg/kg/h
      - or
      - Midazolam loading 0.2 mg/kg
        - CIV 0.2–2 mg/kg/h
        - or
        - Pentobarbital loading up to 10 mg/kg
          - ≤25 mg/min
          - CIV 0.5–2 mg/kg/h
    - Ketamine bolus 1.5 mg/kg
      - CIV 0.01–0.05 mg/kg/h and/or
      - Other drugs

**Electroencephalographic monitoring?**

- Airway, blood pressure, temperature, intravenous access, electrocardiography, CBC, glucose, electrolytes, AED levels, ABG, tox screen, central line?
Approach to the Comatose Patient

Initial Treatment

- Airway
- Breathing
- Circulation
- ABC - identify and address life threatening inadequacies
- Treat rapidly progressive metabolic disorders -- hypoglycemia
- Evaluate for intracranial hypertension and imminent herniation and treat
Does the patient have a rapidly progressive intracranial lesion?

☐ If any factor is present, assume increased intracranial pressure is present and herniation and irreversible damage imminent

- Intubate
- Hyperventilate
- Mannitol
- CT scan, neurosurgical consultation
Approach to the Comatose Patient

Priorities

- ABC’s are paramount!
- Must prioritize
- Must ensure oxygen and substrate reach CNS and vital organs
- Must address immediately life threatening conditions before addressing CNS
Management of the Comatose Patient

Airway

- Evaluate -- is airway patent. Can patient move air without obstruction. Is there trauma or foreign body obstructing airway
- Try chin lift to help open airway -- protect cervical spine
- Place airway if indicated - nasal or oral airway, intubation, or surgical airway
Management of the Comatose Patient

Airway

☐ Intubate (protecting neck) “anyone who will let you”

- Any of the following are adequate criteria
  - GCS < 9
  - Airway not secure or open
  - Respiration not adequate
  - Any significant respiratory failure
  - Uncertainty regarding direction or rate of mental status changes, particularly if constant observation not available (during CT scans, etc..)
Management of the Comatose Patient

Breathing

- Evaluate - is patient moving adequate air, is respiratory rate appropriate, is gas exchange adequate, are breath sounds adequate and symmetrical
- Must assure oxygenation and ventilation
- If intubated don’t forget to ventilate
- Identify and immediately treat problems - pneumothorax, airway obstruction, etc..
Is patient in shock?
- Check pulses, heart rate, blood pressure, perfusion
- Remember hypotension is *late* sign of shock

Start treatment for shock
- Do not restrict fluids in comatose patient with inadequate intravascular volume.
- Cardiac output and cerebral perfusion are much more important than fluid restriction
Use isotonic solutions and blood, as indicated.

Do not use hypotonic solutions to treat shock, particularly patients with coma or possible cerebral edema.

Identify life threatening hemorrhage and control it.
Management of the Comatose Patient

Circulation

- Use isotonic solutions and blood, as indicated.
- Do not use hypotonic solutions to treat shock, particularly patients with coma or possible cerebral edema
- Identify life threatening hemorrhage and control it.
Management of the Comatose Patient

Disability - Neurologic

- Glasgow coma scale
  - Provides easily reproducible and somewhat predictive basic neurologic exam
  - This allows rapid assessment and record of baseline neurologic status
  - Allows physician to track neurologic changes over time and multiple examiners
Initial Management

Protect airway - Support vitals

If evidence of trauma, immobilize spine, get static-spine

IV, Pulse ox, frequent vitals and neuro.checks

Intubate if GCS < 10 or if any question of ability to protect airway

If Narcotic OD suspected, give Naloxone 1 - 2 amps repeat in 15 minutes

If benzodiazepine overdose suspected, Flumazenil .2 mg repeat q 1 minute up to 1.0 mg, may produce seizures.

If ETOH withdrawal (72-96 hrs post ETOH) (confusion, hallucinations, tremor, tachycardia, HTN)
Thiamine 100 mg/IM
Librium 25-100 mg q6hrs

For ETOH seizures (12-24 hours post withdrawal)
Give thiamine 100mg
Stat finger stick for dextrose
Lorazepam 2 mg IV q 6-8 hours
Glasgow Coma Scale

- Eye opening (4 points)
- Verbal response (5 points)
- Best motor response (6 points)
Glasgow Coma Scale

- **Eye opening**
  - 4 - spontaneous
  - 3 - to speech
  - 2 - to pain
  - 1 - none

- **Verbal Response**
  - 5 - oriented
  - 4 - confused conversation
  - 3 - inappropriate words
  - 2 - incomprehensible sounds
  - 1 - none

- **Best Motor Response**
  - 6 - obeys
  - 5 - localizes
  - 4 - withdraws
  - 3 - abnormal flexion
  - 2 - abnormal extension
  - 1 - none
During ABC’s and secondary survey:

- Have someone start IV and obtain labs
  - ABG’s
  - Electrolytes, Liver FT’s, ammonia, coagulation studies
  - Toxin screens
  - Dextrostick

- As soon as IV in and labs drawn, give
  - Glucose (D25, 2 - 4 cc per kilogram)
  - Consider thiamin
Investigation of Coma

Other routine investigations in the ER should include: blood glucose, renal and liver function tests, CBS, ECG, and urine analysis.

Specific investigations: Drug intoxications in urine and serum, Thyroid Function tests, MRI Brain, EEG, Blood culture.
Management of the Comatose Patient

Secondary Survey

- Do a quick general exam of the entire body to identify acute life threatening conditions
- In general, major thoracic or abdominal trauma takes precedence after ABC’s
- Only very rarely is acute neurosurgical intervention appropriate before other acute life threatening injuries are stabilized (except protection of c spine by immobilization)
Neurologic Examination

Secondary Survey

- General motor exam
  - look for focal deficits, posturing (decerebrate or decorticate)
- Reflexes, tone
- Cranial nerve and brainstem function
  - Pupillary response - diencephalon, midbrain, brainstem, CN’s II and III
  - Corneal Reflex - CN’s V, VII, brainstem
  - Oculocephalic Reflex - not if neck injury possible. Tests CN’s III, IV, VI, VIII, and brainstem.
  - Oculovestibular (calorics) can be done if neck questionable.
Does the patient have a rapidly progressive intracranial lesion?

☐ If none of the findings are present, surgical lesion less likely than metabolic cause
☐ Mass lesion still possible, though - CT scan
☐ Urgency of intubation less but should consider
  ■ Will patient deteriorate, particularly while out of constant observation (CT scanner)?
  ■ Can patient protect airway?
Suspected bacterial meningitis or SAH

For SAH, STAT CT of brain
   90% yield for SAH
   Notify neurosurgery stat if suspected

If bacterial meningitis suspected, do not delay for CT- Start empiric therapy
   Ceftriaxone 2 grams q12 hours IV
   Vancomycin 750-1000 mg q 12 hours IV
   Ampicillin 2 grams q 4 hours IV age > 65 or if immunocompromised

LP: L3-L4 interspace
   Obtain opening pressure
   Cell count tubes 1 and 4
   Tubes 2 and 3, Gram stain, Cocci, AFB, india ink,
   Protein and glucose
Increased ICP

- Hyperventilation: Reduces ICP immediately, peak effect 1-2 hours
  NO BENEFIT TO drop PCO2 < 25, Ideal = 30

- Mannitol: Onset in 30 minutes lasts 4-6 hours
  - Mannitol and lasix are synergistic.
    1 - 2 grams/kg bolus
    0.5 - 1 gram/kg q 6 hours
    Monitor sodium, osmolality, BUN

- Hyperosmolality with 3% NaCl
AIRWAY: intubate if GCS \leq 8 and 
pCO2 > 45 torr
BREATHING: maintain SaO2 > 90% pCO2 < 40 torr
CIRCULATION: maintain MAP > 70 mmHg

Check fingerstick glucose and administer glucose if < 45 mg/dL; draw blood sample for electrolytes, arterial blood gas, liver and thyroid function tests, complete blood count, toxicology screen (blood & urine), ECG

NEUROLOGICAL ASSESSMENT

Hyperventilation, mannitol 0.5–1.0 g/kg if clinical evidence of increased ICP/herniation (some prefer 30ml 23.4% NaCl)

Thiamine (100 mg IV) followed by glucose (if < 40 mg/dL, 10 ml aliquots of a 50% solution until blood glucose > 60 mg/dL)

Naloxone if opioid overdose is suspected (0.4-2.0 mg IV q 3 min or continuous IV infusion 0.8 mg/kg/hr)

Flumazenil if benzodiazepine overdose suspected (0.2 mg/min, maximum dose 1 mg IV)

After intubation, gastric lavage with activated charcoal if drug intoxication is suspected

HEAD CT, CERVICAL SPINE, IF STRUCTURAL CAUSE

DETAILED HISTORY AND SYSTEMIC EXAMINATION

CONSIDER EEG, LUMBAR PUNCTURE, MRI
Team work

Include:
- Family
- House officer
- Resident
- Internist
- surgeon
- Neurologist
- Neurosurgeon
- Anaesthetist