Evaluation of Patients with alterations of consciousness and coma

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Coma: “Unarousable unresponsiveness in which the subjects lie with eyes closed”
- Plum and Posner- Diagnosis of Stupor and Coma

Other terms: obtundation, stupor
- fallen out of favour because of imprecision
- descriptive methods favoured
• Two components of conscious behavior
  ◦ sum of cognitive and affective function
  ◦ arousal- appearance of wakefulness

• Content depends on arousal but normal arousal does not guarantee normal content
Consciousness cannot be readily defined in terms of anything else

- A state of awareness of self and surrounding
• Ascending reticular activating system (ARAS)
  ◦ Activating systems of upper brainstem, hypothalamus, thalamus
  ◦ Determines the level of arousal
• Cerebral hemispheres and interaction between functional areas in cerebral hemispheres
  ◦ Determines the intellectual and emotional functioning
• Interaction between cerebral hemispheres and activating systems
Definitions of levels of arousal (consciousness) according to Plum-Posner

- **Alert (Conscious)** - Appearance of wakefulness, awareness of the self and environment
- **Lethargy** - mild reduction in alertness
- **Obtundation** - moderate reduction in alertness. Increased response time to stimuli.
- **Stupor** - Deep sleep, patient can be aroused only by vigorous and repetitive stimulation. Returns to deep sleep when not continually stimulated.
- **Coma (Unconscious)** - Sleep like appearance and behaviorally unresponsive to all external stimuli (Unarousable unresponsiveness, eyes closed)
• **Consciousness**: where is it localized?

  ◦ Ascending Reticular Activating System (ARAS) ‘core of the brainstem’
  ◦ receives input from numerous somatic afferents
  ◦ projects to midline thalamic nuclei (which are in a circuit with cortical structures) and limbic system

**ARAS:**

  ◦ alters effect of sensory stimuli ascending
  ◦ alters descending cortical stimulation
Coma implies dysfunction of:

- ARAS or
- Both hemi-cortices

Anatomically, this means

- central brainstem structures (bilaterally) from caudal medulla to rostral midbrain
- both hemispheres
• Plum and Posner 1982

  ◦ 500 consecutive cases of coma

    • 101 supratentorial (44/101 ICHemorrhages)
    • 65 subtentorial lesions (40/65 brainstem infarcts)
    • 326 diffuse or metabolic brain dysfunction
      ◦ 149 drug intoxication
• Primary CNS event versus secondary
• Implications:
  ◦ short and long-term outcome
  ◦ investigations
Abnormalities of respiration can help localize but almost always in the context of other signs

- Central-reflex Hyperpnea (midbrain-hypothalamus)
- Apneustic, cluster, Ataxic (lower pons)
- Loss of automatic breathing (medulla oblongata)
Systematic assessment of brainstem function via reflexes

Cranial Nerve Exam
- Pupillary light response (CN 2-3)
- Oculocephalic/calorics (CN 3,4,6,8)
- Corneal reflexes (CN 5,7)
- Gag reflexes (CN 9,10)
- Afferent Limb: Optic Nerve
- Efferent Limb: Parasympathetics via oculomotor
- Midbrain integrity/tectum
- Uncal Herniation (3\textsuperscript{rd} nerve dysfunction)
- Pupillary resistance to insult (Be aware of drug effects)
- **Pons**: pinpoint pupils
  - Symp. Dysfunction plus parasymp. irritation
- **Midbrain**: Large fixed pupils unresponsive to light, hippus
- Horner’s- symp. dysfunction
- Unilateral dilation: usually due to 3ʳᵈ nerve lesion

**Pupils: Localizing Value**
• **Afferent**: Trigeminal Nerve
• **Efferent**: Third Nerve (Bell’s Phenomenon and Facial Nerve (Eye closure))
• Tests dorsal midbrain (Bell’s) and pontine integrity (Eye closure)

**Corneal Reflex**
• Before maneuvers note resting position
  ◦ Midline
    • Deviation suggests frontal/pontine damage
  ◦ Conjugate
    • Dysconjugance suggests CN abnormalities
  ◦ Moving
    • Roving, dipping, bobbing
• Brisk rotation of head with eyes held open
• Watch for contraversive movements
• Next:
  ◦ **Flexion**: eyes deviate up and eyelids open (doll’s head phenomenon)
  ◦ **Extension**: eyes deviate downward

Oculcephalic Reflex
- Afferent: Glossopharyngeal
- Efferent: Vagus
- Taken in context of other findings

Gag Reflex
• Assess tone
• Response to painful stimuli
  ◦ none
  ◦ abnormal flexor
  ◦ abnormal extensor
  ◦ normal localization/withdrawal
• Avoid use of decerebrate/decorticate

Motor Examination
- Expanding lesions in lateral middle fossa
- Compression of hippocampal gyrus over free edge of tentorium

- Three stages described
  - Early *third nerve*
  - Late third nerve
  - Midbrain-Upper pons stage
- Akinetic mutism
- ‘Locked-in’ syndrome
- Catatonia (psychiatric, encephalitis, toxic)
- Conversion reactions

Coma Mimics
• Silent, immobile but alert appearing

• Usually due to lesion in bilateral mesial frontal lobes, bilateral thalamic lesions or lesions in peri-aqueductal grey (brainstem)

Akinetic Mutism
• Infarction of ventral pons (all descending motor fibers to body and face)

• May spare eye-movements

• Often spares eye-opening

• EEG is normal or shows alpha activity

“Locked-In’ Syndrome”
Coma

- State of reduced alertness and responsiveness from which you cannot be aroused

- Glasgow Coma Scale
  - Motor, verbal, eye opening
<table>
<thead>
<tr>
<th>GCS - Motor Response</th>
<th>Example</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Commands</td>
<td>Follows simple commands</td>
<td>6</td>
</tr>
<tr>
<td>Localizes Pain</td>
<td>Pulls examiner's hand away when pinched</td>
<td>5</td>
</tr>
<tr>
<td>Withdrawing from Pain</td>
<td>Pulls a part of body away when pinched</td>
<td>4</td>
</tr>
<tr>
<td>Abnormal Flexion</td>
<td>Flexes body inappropriately to pain</td>
<td>3</td>
</tr>
<tr>
<td>Abnormal Extension</td>
<td>Body becomes rigid in an extended position</td>
<td>2</td>
</tr>
<tr>
<td>No Response</td>
<td>Has no motor response to pinch</td>
<td>1</td>
</tr>
<tr>
<td>GCS - Eye-Opening</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-------------------</td>
<td>----------------</td>
<td>---</td>
</tr>
<tr>
<td>Spontaneous</td>
<td>Opens eyes on own</td>
<td>4</td>
</tr>
<tr>
<td>To Voice</td>
<td>Opens eyes when asked to in a loud voice</td>
<td>3</td>
</tr>
<tr>
<td>To Pain</td>
<td>Opens eyes when pinched</td>
<td>2</td>
</tr>
<tr>
<td>No Response</td>
<td>Does not open eyes</td>
<td>1</td>
</tr>
<tr>
<td>GCS - Verbal Response (Talking)</td>
<td></td>
<td></td>
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<td>-------------------------------</td>
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</tr>
<tr>
<td>Orientated</td>
<td>Carries on a conversation correctly and tells examiner where he is, who he is, and the month and year</td>
<td>5</td>
</tr>
<tr>
<td>Confused Conversation</td>
<td>Seems confused or disoriented</td>
<td>4</td>
</tr>
<tr>
<td>Inappropriate Words</td>
<td>Talks so examiner can understand him but makes no sense</td>
<td>3</td>
</tr>
<tr>
<td>Sounds</td>
<td>Makes sounds that examiner cannot understand</td>
<td>2</td>
</tr>
<tr>
<td>No Response</td>
<td>Makes no noise</td>
<td>1</td>
</tr>
</tbody>
</table>
- Global
  - Hypoglycemia, hypoxia

- CNS
  - Brainstem disease
  - Bilateral cortical disease
    - Unilateral should not present as coma
• Secondary to compression of the brainstem

• Primarily uncal vs. central
- Medial temporal lobe compresses brainstem
- Decreased responsiveness going into a coma
- Ipsilateral pupil dilated and nonreactive

**Uncal herniation**
Small, reactive

Diencephalon

Medium-sized, fixed

Midbrain

Ipsilateral dilated, fixed

Tectum

Dilated, Fixed

Midbrain

Pons

Myosis, pinpoint,
- Progressive loss of consciousness
- Decorticate posturing
- Irregular respirations

Central Herniation
Coma secondary to hemispheric hemorrhage may still have **localizing** features.

- Pupillary, muscle, and cranial nerve exam to determine central vs. focal.

- Pupillary response generally preserved in toxic metabolic coma.

**Clinical Features**
- Cervical spine immobilization if trauma suspected (multiple trauma)
- Pediatric coma commonly ingestion, infection, or abuse
- Seizures
Patients who survive coma do not remain in this state for \( >2–3 \) weeks, but develop a persistent unresponsive state in which sleep–wake cycles return.

After severe brain injury, the brainstem function returns with sleep–wake cycles, eye opening in response to verbal stimuli, and normal respiratory control.
Locked in syndrome

- Patient is awake and alert, but unable to move or speak

- Pontine lesions affect lateral eye movement and motor control

- Lesions often spare vertical eye movements and blinking
Vegetative

Locked-in

MIG-Roma
Confusional state

- Major defect: lack of attention
  - Disorientation to time, places, persons
  - Patient thinks less clearly and more slowly
  - Memory faulty (difficulty in repeating numbers (digit span))
- Misinterpretation of external stimuli
- Drowsiness may alternate with irritability
Markedly abnormal mental state
  ◦ Severe confusional state
  ◦ Visual hallucinations &/or delusions
    (complex systematized dream like state)
Loss of consciousness is common with SAH
- only about 1/2 of patients recover from the initial effects of the haemorrhage.

- **Causes of coma:**
  1. **Acute:** increase of ICP and
  2. **Late:** vasospasms, hyponatraemia
May cause a rapid decline in consciousness, from:

1. **Rupture into the ventricles**, subsequent herniation and brainstem compression.

2. **Cerebellar haemorrhage or infarction** with subsequent edema

3. **Direct brainstem compression**
The critical blood flow in humans required to maintain effective cerebral activity is about 20 ml/100 g/min and any fall below this leads rapidly to cerebral insufficiency.

The causes:
1. syncope in younger patients
2. cardiac disease in older patients.
Mass effects:
tumours, abscesses, haemorrhage, subdural, extradural haematoma, brainstem herniation → distortion of the RAS.
Raised intracranial pressure for brain tumors

- Herniation and loss of consciousness: mainly lesions located deeply, laterally, or in the temporal lobes.

- Rate of growth: slowly growing tumours may achieve a substantial size and distortion of cerebral structure without impairment of consciousness, in contrast to small rapidly expanding lesions with marked edema.
The leading cause of death below the age of 45, head injury accounts for 1/2 of all trauma deaths
- A major cause of patients presenting with coma.
- A history is usually available and, if not, signs of injury such as bruising of the scalp or skull fracture lead one to the diagnosis
Alcohol on the breath provides a direct clue to a cause of coma, evidence of head injury need not necessarily imply that this is the cause.

Epileptic seizure, may have resulted in a subsequent head injury.
Damage can be diffuse or focal.
Rotational forces of the brain cause surface cortical contusions and even lacerations, mostly fronto-temporal (irregular sphenoidal wing and orbital roof).
Subdural bleeding due to tearing of veins
Diffuse axonal injury is now seen as the major consequence of head injury and associated coma.

Mild degrees of axonal injury also occur with concussion and brief loss of consciousness.
Secondary damage can occur from parenchymal haemorrhage, brain edema, and vascular dilatation, all of which will lead to ↑ ICP and ↓ perfusion pressure, which can be accentuated by systemic hypoxia and blood loss.

Subdural and extradural haematomas causing impairment of consciousness followed by apparent recovery are important to diagnose, as they are readily treatable surgically.
Systemic infections may result in coma as an event secondary to metabolic and vascular disturbance or seizure activity.

Direct infections of the CNS (meningitis and encephalitis) can all be associated with coma.

**Meningitis:** the onset is usually subacute, with intense headache, fever, and neck stiffness. Meningococcal meningitis may be rapid in onset.
**Infections**

- **Diagnosis:** confirmed by identifying the changes in the CSF and by isolating the causative organism.

- **Prompt treatment** of acute meningitis is imperative and may precede diagnostic confirmation.

- **Encephalitis:** usually subacute, and often associated with fever and/or seizures, *herpes simplex* encephalitis may be explosive at onset, leading to coma within a matter of hours.
Septic patients

- Commonly develop an encephalopathy.
- In some patients this can be severe, with a prolonged coma.

- Lumbar puncture in such patients is usually normal or only associated with a mildly elevated protein level.

- EEG is valuable and is abnormal
Common cause of coma, with a period of unconsciousness following a single generalized seizure commonly lasting between 30 and 60 minutes.

Following status epilepticus, there may be a prolonged period of coma. *Search a trauma to the tongue or inside of the mouth.*

Seizures secondary to metabolic disturbances may have a longer coma.