Assessment of Coma

Dr Martin Hughes
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Example 1

- 68 year old woman, found collapsed by son
- Was well about 1 hour previously
- No medical history available
- Asked to take for scan, GCS 3
- Airway patent, breathing 35 / minute, 130/80, 90/min
Example 2

- 58 yo alcoholic
- Recovering from pneumococcal pneumonia
- Sedation stopped, not waking up
Example 3

- 46 year old man with abdominal pain
- Smoker and drinker, FH cerebrovascular disease
- ?gastritis, in medical wards
- Sudden LOC (spoken to by nurses 5 minutes earlier)
Example 4

- 48 y.o man
- OOH cardiac arrest
- Admitted for cooling
- Post cooling still GCS 3
- Facial twitching ? myoclonus
Plan

• Basic approach to comatose patient
• System to allow accurate assessment of coma and diagnosis of the cause of coma
• Allow appropriately directed further investigation
• No CT interpretation
• Not about outcome
• Not about management of specific diseases
Basic Approach

• Airway with cervical spine: patency and protection. Act if necessary
• Breathing: SpO2, respiratory rate, respiratory examination. Act if necessary
• Circulation: BP, HR, capillary refill. Act if necessary
• Get history
• Glucose, ECG, CxR, Bloods, ABGs, tox screen
General examination

- Skin: rash, anaemia, cyanosis, jaundice, spiders, track marks
- Temp: infection, hypothermia
- CVS: infection, Addison's, arrhythmia
- RS: hypoxia or hypercarbia. Remember neurogenic pulmonary oedema
- Abdomen: ascites, organomegaly
- Thyroid
Basic Approach - Trauma

• GCS 15, age < 65, no clinical evidence skull #, no retrograde amnesia, no seizure, no significant mechanism of injury or serious assault

• GCS 13 or 14 but 15 within 2 hours of injury and above

• Otherwise CT
Consciousness

- Wakefulness (arousal, vigilance, alertness)
- Awareness (of self or environment)
- No awareness without wakefulness
- Wakefulness can occur without awareness (vegetative state)
Wakefulness

• Linked to ARAS
• In pons and midbrain
• Projects to diencephalon (thalamus and hypothalamus) and cortex
Awareness

- Needs functioning cortex and subcortical connections
- Needs wakefulness
What is Coma?

- No awareness or arousal
- Lasts > 1h (cf syncope and concussion)
- No spontaneous speech or movement, eyes shut
- No eye opening to verbal command (E2)
- Noxious stimuli: vocalisation limited or absent (V2)
- Noxious stimuli: motor activity absent/abnormal/reflexive (not purposeful or defensive) (M4)
Coma

• No sleep wake cycles
• Transitional
• Injury or functional disruption of bilateral cortical structures or ARAS
How can we classify it?

- Direct brainstem
- Indirect brainstem
- Generalised neuronal dysfunction
What are the causes?

• Commonly stroke, cranial trauma and drugs
• ‘Medical’: CVA 50%, hypoxic / ischaemic 20%, infective / metabolic encephalopathies the rest
• Depends on location: when looked for subclinical status epilepticus up to 8% in ICU coma
• HOW DO WE MAKE THE DISTINCTION, AND WHY?: direct brainstem, indirect brainstem, generalised neuronal dysfunction
Generalised Neuronal Dysfunction

- Drugs
- Anoxia
- Epilepsy: may be subclinical
- CO
- SIRS
- Hypoxia
- Hypercapnia
- Glucose
- Sodium
- Liver
- Kidneys
- Hypothermia
- Hypertension
- Hypotension
- Hypercalcaemia
- Wernicke’s
- Thyroid, adrenal, pituitry
Examination
# Glasgow Coma Scale

<table>
<thead>
<tr>
<th>Eye opening</th>
<th></th>
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</thead>
<tbody>
<tr>
<td>Spontaneous</td>
<td>=4</td>
</tr>
<tr>
<td>To speech</td>
<td>=3</td>
</tr>
<tr>
<td>To pain</td>
<td>=2</td>
</tr>
<tr>
<td>None</td>
<td>=1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Verbal response</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Oriented</td>
<td>=5</td>
</tr>
<tr>
<td>Confused</td>
<td>=4</td>
</tr>
<tr>
<td>Inappropriate</td>
<td>=3</td>
</tr>
<tr>
<td>Incomprehensible sounds</td>
<td>=2</td>
</tr>
<tr>
<td>None</td>
<td>=1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Motor response</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Obeys</td>
<td>=6</td>
</tr>
<tr>
<td>Localizing</td>
<td>=5</td>
</tr>
<tr>
<td>Normal Flexion</td>
<td>=4</td>
</tr>
<tr>
<td>Abnormal Flexion (decorticate)</td>
<td>=3</td>
</tr>
<tr>
<td>Extension (decerbrate)</td>
<td>=2</td>
</tr>
<tr>
<td>None</td>
<td>=1</td>
</tr>
</tbody>
</table>

**Total** = 15
EYES

• ARAS near centres for pupillary function and eye movements
• Pupillary responses
• Oculocephalic reflexes
• Corneal reflexes
• Eye deviation and movements
• Fundus
Pupillary responses

- Preserved in lesions above thalamus and below pons
- If they are normal it is unlikely to be direct or indirect brainstem injury (as the cause of coma)
- Beware drugs (tricyclics, anticholinergics, amphetamines, carbamazepine)
Pupillary responses

- Large fixed bilateral: tectal (dorsal mid brain)
- Mid size fixed bilateral: mid brain
- Pin point bilateral: pons
- Small reactive: diencephalon
- Large fixed unilateral: III nerve
Pupillary responses

• Need bright light and sometimes a magnifying glass
• Generally intact in metabolic/toxic/generalised neuronal dysfunction
• Local injury and drugs can interfere
Oculocephalic reflexes

- Centres for eye movement adjacent to centres for arousal: useful guide to brainstem disease
- ‘Doll’s eye’ reflex
- Only in a coma
- Remain fixed on the current point of focus initially
- Then follow the head
Oculocephalic reflexes

• If normal pontomedullary junction to level of oculomotor nucleus in the brainstem intact
• Abnormality means brainstem dysfunction, normal means brainstem ok
• Can substitute caloric reflexes (neck disorders, more sensitive. More effort. Identifies INO)
Corneal reflexes

- Intact pons: closure
- Intact pons and midbrain: Bell’s phenomenon
Lateral Conjugate Gaze Deviation

- Lesion from frontal (eye) fields to PPRF in brainstem
- Destructive or excitatory
- Away from hemiparesis if hemispheric, towards hemiparesis if brainstem
- Basically it is a frontal or brainstem stroke, or the patient is fitting
- (Failure of lateral gaze may indicate central herniation causing bilateral VI nerve palsies)
Disconjugate gaze deviation

- III nerve
- VI nerve
- Brainstem
Downward deviation

- Not especially helpful
- Bilateral thalamic and subthalamic (dorsal mid brain) lesions
- E.g. acute obstructive hydrocephalus
- Some metabolic encephalopathies, e.g. hepatic encephalopathy
- Ocular bobbing is pontine
Upward Deviation

- Not helpful
- Sleep
- Brainstem
- Seizures
- Bilateral hemispheric damage
Roving eye movements

- Slow, conjugate, lateral, to and fro
- Generalised neuronal dysfunction, usually due to DRUGS
- (Occasionally bilaterally hemispheric)
Ping pong movements

• Bilateral hemispheric
INO

- Caloric stimulation
- Near eye moves to cold water, far eye stationary
- Damage to MLF i.e. pons
Fundus
Fundus
Fundus
Other brainstem signs

- Grimacing
- Gag
- Breathing
Meningism

- SAH
- Meningitis
- Meningoencephalitis
General Neurological Examination

- Tone, power, reflexes
- Identifies lateralising signs: hemispheric lesions
- In general plantars do not help
- Signifies hemispheric lesions but may be old
- Decerebrate, decorticate
Respiratory pattern

• Cheyne –Stokes: bilateral diencephalic or hemispheric injury (LVF, COPD, drugs depressing response to CO2, ventilatory over support)
• Hyperventilation: pons or midbrain injury (ARF, shock, fever, acidosis, psychiatric disease)
• Apneustic breathing: pons
• Ataxic: medulla, usually preterminal
Autonomic respiratory centers
  Pneumotaxic center
  Apneustic center

Pons

Medulla oblongata

Olive

Fourth ventricle

Reticular formation

(a) Longitudinal section (cut-away)
In reality you need: good torch, cotton wool or swab, opthalmoscope, tendon hammer

- Pupils, size and reaction
- Eye deviation and eye movements
- Oculocephalic reflex
- Corneal reflex
- Cough/gag, grimacing, breathing
- Fundi
- Meningism
- Neurology: GCS, tone, power, reflexes
- About 4 minutes
Examination
Why examine?

• If brainstem signs, it is direct or indirect injury (almost always)
• If there are brainstem signs and lateralising signs or III nerve palsy, it is a hemispheric lesion: urgent CT scan, measures for ICP
• If there are brainstem signs alone it is direct injury: CT may help
Why examine?

- If there are no brainstem signs it is a generalised cause of coma: look for it, identify it
Generalised Neuronal Dysfunction

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- Glucose

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Why examine?

• You may identify seizures, including subclinical status
• You may identify meningism
• More commonly you can direct your investigation appropriately
• Even more commonly you can do nothing except watchful waiting while treating the underlying disease
Example 1

- 68 year old woman, found collapsed by son
- Was well about 1 hour previously
- No medical history available
- Asked to take for scan, GCS 3
- Airway patent, breathing 35 / minute, 130/80, 90/min
Example 1

- No peripheral focal neurology identifiable
- Pupils pinpoint
- No corneal or oculocephalic reflexes
- DIAGNOSIS
Example 2

- 58 yo alcoholic
- Recovering from pneumococcal pneumonia
- Sedation stopped, not waking up
Example 3

• 46 year old man with abdominal pain
• Smoker and drinker, FH cerebrovascular disease
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• Sudden LOC (spoken to by nurses 5 minutes earlier)
• GCS 3, no focal neurology, brainstem signs or meningism, CVS stable
• DIAGNOSIS
Example 4

- 48 y.o man
- OOH cardiac arrest
- Admitted for cooling
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- Facial twitching ? myoclonus
- Lateral conjugate gaze deviation
- DIAGNOSIS
Summary

• Clinical assessment of coma is useful
• It can lead to unexpected diagnoses
• It leads to the appropriate investigation, including no investigation
• It should be as routine as respiratory examination in respiratory failure