Coma

Description

Coma (Greek *koma* = deep sleep) – Unresponsive and unrousable state of unconsciousness. Usually equates to GCS<8. Secondary to bilateral cortical insult or RAS lesion. Other states:

- Stupor - Responsiveness is greatly diminished but may be roused by noxious stimuli.
- Obtundation - Reduced awareness to surroundings.
- Drowsiness - Mild obtundation, sleepiness.

Aetiology

- Trauma – Depressed skull #, ICH, SDH, SAH
- Toxic – EtOH, Drugs (opiates, BDZ, neuroleptics, GHB, MDMA), CO, solvents
- Metabolic – Hypo/hyperGlu, hypo/hyperNa, hypo/hyperCa, hypercapnia, hypoxia, hypothyroidism, acid-base disturbances, liver failure, renal failure, hypopituitarism
- Neurological - Epilepsy and status epilepticus, ↑ICP, obstructive hydrocephalus
- Ischaemic - Cerebral hypoperfusion, arrhythmias, CVA, hypertensive encephalopathy
- Infective - Meningitis, encephalitis, septicaemia, abscess, malaria, toxoplasmosis
- Auto-immune - Vasculitis
- Structural lesions – SOL
- Hysterical pseudo-coma.

Assessment

- AMPLE history if available
- Vital signs – looking for hypoxia, Cushing reflex, fever, BSL.
- GCS/AVPU
- Eyes:
    - Pupils - abnormal movements, size, response to light stimulus. Note:
        - Midrange pupils with response to light - diencephalic lesion.
        - Pupil fixed in mid-position with loss of light reflex=mid-brain lesion.
        - Small pupils with response to light – pontine lesions
        - Fixed dilatation - significant damage to the medulla/brainstem.
        - Blown=uncal herniation, temporal lobe over tentorium trapping IIIn
    - Corneal reflexes: Normally intact until very deep coma.
    - Fundoscopy: papilloedema
    - Eye movements:
        - Spontaneous eye movements
            - Conjugate deviation - ?focal hemispheric brainstem lesion
            - Depression of the eyes - lesion in the mid-brain (tectum)
            - Skew deviation of the eyes - pontomedullary junction lesion
            - Significant uncoordinated eye movements – IIIn/VIn
        - Reflex eye movements
            - Oculocephalic response (doll's eye) – brainstem functioning
            - Oculovestibular testing (ice water in ear canal):
                - Psychogenic - nystagmus - fast phase away from ear
                - Supratentorial lesion - conjugate movement to ear
                - Brainstem lesion - disconjugate/no response
- Posturing, focal/localizing signs

- Note breathing pattern (Kussmaul, Cheyne-Stokes – diencephalic, hyperventilation – midbrain or metabolic, apnoeas – Pons, agonal - medullary) & any odour of hepatic foetor, ketones, solvents, and alcohol
- Reflexes: tendon, plantar, corneal, gag, doll's eyes
- Neck stiffness
- HI: Battle's sign, panda eyes
- Skin - hyperpigmentation, rashes, signs of myxoedema, track marks, anaemia, jaundice & other liver disease stigmata, cherry-red discolouration (CO), clubbing

Investigations

- Bedside: BSL, Urine (culture & drug screen) & ECG
- Bloods: FBC, UEC, glucose, LFTs, coags, ABG (+CO-Hb), cultures, TFTs, CK/Trp, βHCG
- Radiology: Head CT, CXR, occ MRI
- Consider LP, paracetamol/EtOH level, malaria screen, EEG (NCSE) if indicated

Management

- Resuscitation with continuous monitoring – HR, RR, BP, SaO_2
    - Airway: intubate if GCS<8 or not ventilating adequately. Protect C-Spine if ?trauma
    - Breathing: High flow O_2
    - Circulation: IV access, fluids, support BP
    - Disability: Treat seizures **BDZ±phenytoin**, **glucose** if low, **thiamine** (Wernicke's)
- Consider trial of **naloxone** if opioid OD suspected
- **Mannitol** or **3% saline** if acute ↑ICP
- Neurosurgery for decompression, clot evacuation
- Antibiotics if suspect meningitis, aspiration, or sepsis
- Supportive care - Fluids, thermoreg, prevention of pressure sores and adequate nutrition
- Treat underlying condition if possible

Prognosis

Depends on the underlying cause, depth, duration and clinical signs.

- Head injury - prognosis is directly proportional to the GCS score
- Lack of brainstem and lateralising signs - cause most likely metabolic and ?reversible
- Drug overdose - good prognosis with proper treatment
- Coma >6hrs (not due to HI or OD) - only 10% chance of good recovery
- SAH & CVA - <5% good recovery
- Hypoxia or ischaemia (e.g. after cardiac arrest) - ~10% good recovery
- Coma > 24 hours - 10% recovery
- After 1 week - 3% good recovery
- After 7 days - high incidence of death/persistent vegetative state
- Absence of brainstem reflexes for 24hrs (without sedative drugs) - very little hope

Locked – in Syndrome

- Cognition preserved but infarction of ventral pons from basilar artery occlusion inhibits descending motor pathways to face & limbs. Spares eye innervation.

Persistent vegetative state (PVS)

May follow a coma. Apparent loss of cognition and external awareness, but non-cognitive brain function retained with normal or near-normal sleep-wake cycles. Gag, cough, sucking and swallowing reflexes may be preserved.