APPROACH TO COMA AND ALTERED CONSCIOUSNESS

COMMON PRESENTATION TO E.D. → SPECTRUM RANGING FROM SLEEPINESS TO FRANK COMA.

PATHOPHYSIOLOGY:

- CONSCIOUSNESS contains properties of:
  - AROUSAL → awareness of one's self or surroundings
  - COGNITION → combination of orientation, judgment and memory
- The ASCENDING RETICULAR ACTIVATING SYSTEM is the neuroanatomic sturcutre primarily responsible for arousal → controls input of somatic and sensory stimuli to the cerebral cortex
- The cognition centres are mostly in the cortex
- Insults to the cerebral cortex and brainstem can EACH independently cause depressed consciousness or coma
  - Vulnerable to metabolic derangements, toxins or mechanical injury
- Causes can be broken down into a FEW GENERAL CATEGORIES:
  - Metabolic/systemic:
    - Hypoxia, hypoperfusion (shock), infection (esp if CNS involved), toxin/drug effect, electrolytes, glucose
  - Structural:
    - Most commonly arising from head trauma (subdural, extradural, contusion), stroke (thromboembolic, septic/fat emboli, cerebral sinus thrombosis), haemorrhage (SAH, pontine, cerebellar, ICH), tumour, infection (abscess, subdural empyema)
    - Ischaemic strokes will only depress consciousness if insult is MASSIVE
- SPECIAL POPULATIONS:
  - ELDERLY → susceptible to minor infections, drug changes
  - IMMUNOCOMPROMISED → vulnerable to multitude of opportunistic pathogens

HISTORY:

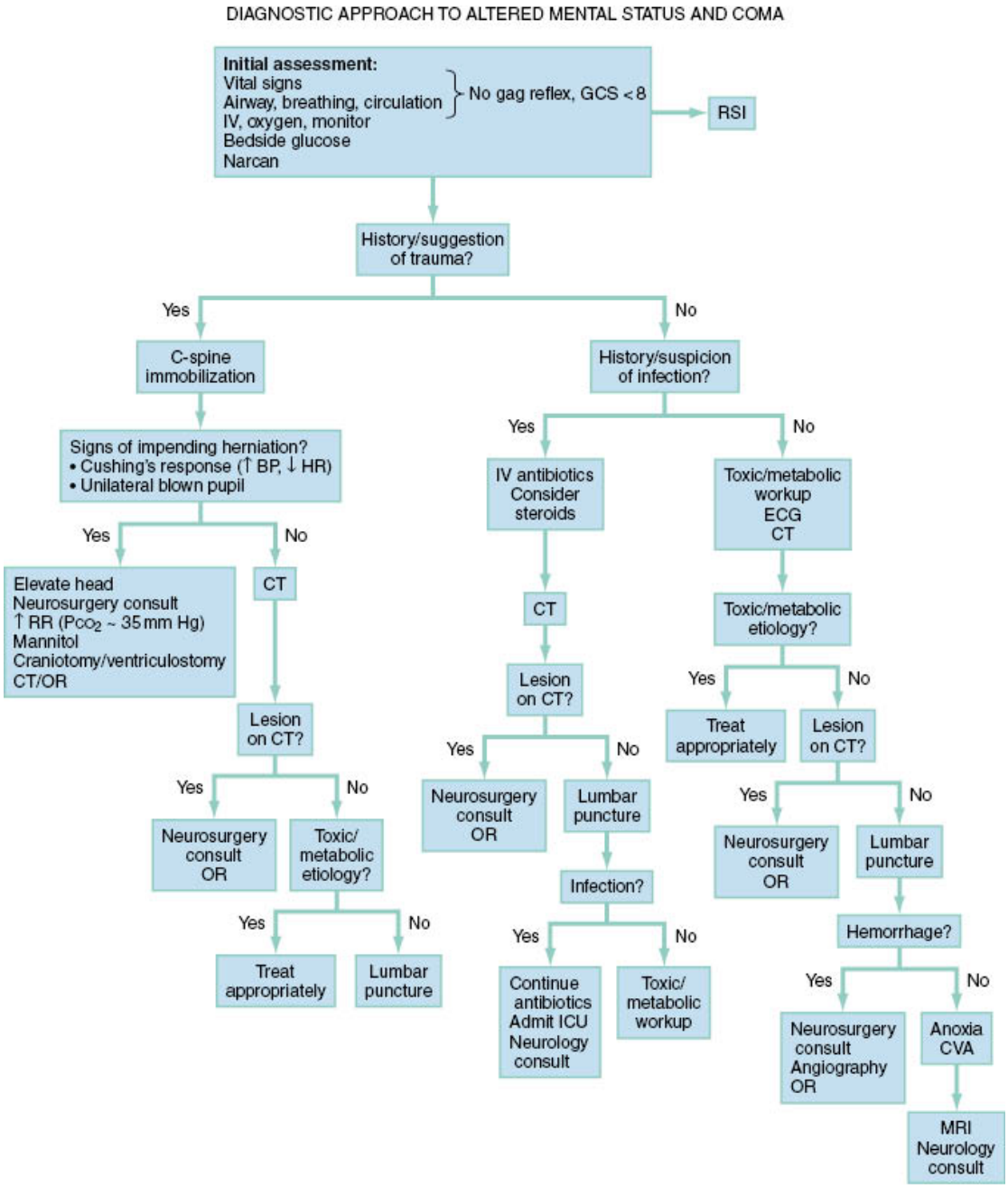
- From family/carers/friends → less interactive, more sleepy, difficult to rouse
- Comorbidities, trauma, immunocompromise
- Symptoms prior → headache, focal weakness, incoordination, visual disturbances
- Causes vary with age:
  - Infant → infection, trauma/NAI, metabolic
  - Child → toxic ingestion
  - Adolescent → toxic ingestion, recreational drug use, trauma
  - Elderly → medication changes, OTC meds, infection, stroke

PHYSICAL EXAMINATION:

- Severity of presenting symptoms dictates the speed needed for stabilization and diagnosis
- VITAL SIGNS:
  - Significant hypotension → shock

    - Cushing reflex (↑BP, ↓HR) → severe ICP elevation
    - Both extremes of temperature can cause alteration of consciousness
    - Anomalies in respiration can point to CNS/toxic-metabolic cause → hyperventilation, Kussmauls breathing, Cheyn-Stoke
- A rapid, directed neurologic screening examination can determine whether the patient has a significant focal motor deficit
- Head-to-toe → pupillary reflexes, evidence of head trauma (haemotympanum, scalp haematoma). Nuchal rigidity, chest exam, evidence of ascites, peripheral stigmata of liver disease, skin rashes/lesions
- GCS → central to any assessment (WE ALL KNOW THIS!):
    - Eyes, voice, best motor
    - Eyes (spontaneous 4, to voice 3, to pain 2, none 1)
    - Voice (oriented 5, confused 4, inappropriate words 3, incomprensible 2, none 1)
    - Best motor (obeys 6, localizes 5, withdraws 4, abnormal flexion 3, abnormal extension 2, none 1)
- Pupils:
    - Unilateral dilatation of a pupil and loss of reactivity in a comatose patient are ominous signs of UNCAL HERNIATION → immediate neurosurgery consultation
    - In setting of trauma, a unilateral third cranial nerve palsy suggests IPSILATERAL COMPRESSIVE LESION
    - Cranial nerve VI palsies are NON-LOCALISING as it has a long intracranial course
- Oculocephalic (DOLL'S EYES) reflex:
    - Only once C-spine cleared
    - In those who maintain forward gaze despite head turning → unlikely to have brainstem lesion
- Oculovestibular reflex (cold-water caloric testing)
    - Cannot be voluntarily resisted
    - Iced water into external auditory canal results in slow conjugate deviation of gaze toward the side of the stimulus followed by corrective nystagmus toward the midline → if there is no response, brainstem dysfunction is possible

DIAGNOSTIC ALGORITHM



- History and exam should be used to direct the approach to diagnostic testing
- Early neuroimaging (non-contrast CT brain) to exclude structural cause
- Bedside glucose
- Serum electrolytes
- Leukocytosis not helpful, but leucopenia suggests immunocompromise and should direct investigation toward and infectious cause
- TFT → myxoedema coma
- Ammonia levels have not been shown to be a reliable marker in the setting of depressed consciousness
- ECG → toxidromes (TCA), electrolyte anomalies (↑K), hypothermia, ischaemia

- EEG if non-convulsive status is suspected

EMPIRICAL MANAGEMENT:

- Establish ABCs
- Early intubation unless coma is reversible (i.e. hypoglycaemia, opioids)
    - Consider lignocaine pre-treatment of cords if ICP elevation is suspected
- DEXTROSE, NALOXONE, THIAMINE → treats three important causes of altered consciousness
- Consider mannitol empirically if transtentorial herniation is suspected
- Broad spectrum antibiotics suggested and should not be delayed if meningoencephalitis is suspected