

Disorders of Consciousness and Mental Status

1. Know the anatomic structures necessary to maintain consciousness
Brainstem reticular activating system (above midpons), ascending projections to thalamus, and cerebral hemispheres
2. Outline the initial steps in the evaluation of a comatose patient
Emergency management - (a) ensure patency of airway and adequacy of ventilation and circulation (ABCs), may need to intubate or place tracheostomy, ventilation > 8/min, maintain pulse/BP with fluids, pressors or anti-arrhythmic drugs. (b) insert IV and get blood for labs, check blood levels of glucose, electrolytes, hepatic/renal function, PT and PTT, CBC. (c) IV infusion of dextrose (25 g), thiamine (100 mg), and naloxone (.4-1.2 mg) (d) Blood gases and pH, help find metabolic causes (e) treat seizures if present H&P - (a) History - onset: sudden often vascular origin (stroke or SAB), rapid progression from hemispheric signs to coma in minutes to hrs suggest intracerebral hemorrhage, protracted course suggest tumor, abscess, chronic subdural, preceded by confusion/delirium suggest metabolic. (b) General Physical- signs of trauma, BP, temperature, signs of meningeal irritation, fundoscopic exam (papilledema/retinal hemorrhage) (c) Neuro exam - Pupils (normal, thalamic (small but reactive), fixed and dilated, fixed midsized, pinpoint, asymmetric); EOM (oculocephalic/doll-head maneuver or oculovestibular cold water caloric); Motor response to Pain (supraorbital, sternum, nail bed)
3. Know how to perform the neurologic exam on a comatose patient
4. Be able to differentiate structural from metabolic causes of coma
Sudden onset suggest vascular origin (stroke or SAB), rapid progression (min-hrs) of hemispheric signs (i.e. hemiparesis, hemisensory deficit, aphasia) suggest intracerebral hemorrhage, protracted course (days-weeks) suggest tumor, abscess or chronic subdural, Coma preceded by confusional state/agitated delirium w/o lateralizing signs probably metabolic.
Metabolic coma - no focal signs (hemiparesis, hemisensory loss, aphasia) and usually no LOC (except SAR), history reveals progressive somnolence or toxic delirium followed by gradual descent into coma, symmetric neurological exam supports metabolic coma, Asterixis/myoclonus/tremor preceding coma suggest metabolic, finding reactive pupils in presence of otherwise impaired brainstem function is the hallmark of metabolic encephalopathy
5. Be able to recognize transtentorial and foramen magnum herniation syndromes
Supratentorial structural lesions - usually have hemispheric disorder, aphasia w/ dominant hemispheric lesion, agnosia w/ non dominant hemisphere, as mass expands, somnolence supervenes due to compression of contralateral hemisphere and downward pressure on diencephalon, progressive compression of thalamus, midbrain, pons, and medulla, reveal dysfunction at successively lower levels, once pontine level reached fatal outcome inevitable, when medial part of temporal lobe herniates across cerebellar tentorium it exerts direct pressure on rostral brainstem cause signs of oculomotor and midbrain compression (ipsilateral pupillary dilation and adduction of eyes) Subtentorial structural lesions - sudden onset coma w/ focal brainstem signs, w/ focal midbrain lesion pupillary function lost (pupils midsized and nonreactive), pinpoint pupils seen w/ pontine hemorrhage, conjugate gaze deviation away from side of lesion toward hemiparesis suggest subtentorial lesion, ventilatory patterns may be abnormal and variable

6. Be aware of conversion reactions and appreciate how historical and physical findings may be helpful in suggesting the diagnosis

Conversion reactions - obvious loss of neurologic function in absence of organic neurologic disease, often have antecedent stressor and unconscious secondary gain, patient does not consciously realize the nonorganic basis of illness. (a) pseudo seizures bizarre episodes w/o incontinence or self injury, commonly also suffer from true epileptic seizures. (b) pseudocoma -light coma, responsive to noxious stimulus and subtle avoidance of physical threat. (c) pseudoparalysis - weakness is variable, little-resistance to passive movement, intact resistance to gravity. (d) pseudosensory loss - may include blindness, deafness, anesthesia (e) pseudoataxia - have swooping dives and thrusts, sustained monopodal postures, and clutching to surfaces, rarely falls.

7. Know the difference between delirium and dementia

Delirium - impaired level of consciousness, acute/subacute course, fluctuating course, hyperactivity, usually reversible. AKA acute confusional state, refers to acute, global disorder of thinking and perception characterized by impaired consciousness and inattention., may be restless, agitated, and combative. Cause of delirium = Vascular, Infectious, Traumatic, Autoimmune, Metabolic/toxic, Iatrogenic, Neoplastic, Seizures (VITAMINS)

Dementia - no impairment of consciousness, chronic course, steadily progressive course, no autonomic hyperactivity, usually irreversible.

8. Know the differential diagnosis of dementia, especially the reversible causes of dementia

DDX: non-reversible = Alzheimer's, Lewy body, Multiinfarct (vascular)

Reversible = normal pressure hydrocephalus, intracranial mass lesion, Vit b12 deficiency, hypothyroidism, neurosyphilis,

- 9. Recognize the clinical presentation of Alzheimer's dementia

Initially impairment of recent memory, then progresses to disorientation to time then place. Aphasia/anomia/acalculia may develop. Initially depression then gives way to agitated and restless state. Later develop apraxias and visuospatial disorientation, pt becomes easily lost. Late stages lose social graces, may have psychosis w/ paranoia, hallucinations, or delusions. May also see seizures, extrapyramidal rigidity and bradykinesia. .

10. Know the difference between aphasia, apraxia, and agnosia

aphasia = loss or impairment of language function as result of damage to specific language centers in dominant hemisphere.

apraxia = inability to perform learned motor tasks despite sufficient memory and sensorimotor function to understand command, usually due to lesion in dominant inferior parietal lobe. .

Agnosia = inability to recognize a specific sensory stimulus despite preserved sensory function, i.e. inability to recognize visual stimulus even though visual acuity normal, lesion usually in occipitotemporallobe.

11. Know the difference between dysarthria and aphasia

Dysarthria = language function intact, but patient unable to articulate speech, result of supranuclear, nuclear, or peripheral lesion of lower CN or lesions of bulbar musculature or neuromuscular junction.

12. Know the clinical findings and anatomy of Broca's, Wernicke's and conduction aphasia
Broca's = expressive aphasia, comprehension preserved, repetition and fluency lost,
inferior frontal lobe
Wernicke's = receptive aphasia, fluency preserved, comprehension and repetition lost, superior temporal gyrus/lobe
Arcuate fasciculus = conductive aphasia, comprehension and fluency preserved, repetition lost, runs from temporal lobe, via parietal lobe, to frontal lobe.