ALTERED MENTAL STATUS AND COMA

DISORDERS OF CONSCIOUSNESS DIVIDED INTO PROCESSAL THAT AFFECT AROUSAL OR CONTENT OF CONSCIOUSNESS OR BOTH

AROUSAL BEHAVIOURS INCLUDE WAKEFULNESS AND BASIC ALERTNESS → RETICULAR ACTIVATING SYSTEM RESPONSIBLE (MIDBRAIN, PONS, MEDULLA)

CONTENT OF CONSCIOUSNESS RESIDES IN CORTEX → INCLUDES SELF-AWARENESS, LANGUAGE, REASONING AND SPATIAL AWARENESS

PSYCHIATRIC DISORDERS AND ALTERED MENTAL STATES MAY SHARE FEATURES SUCH AS HALLUCINATIONS OR DELUSIONS → DISCRIMINATING FEATURES ARE SHOWN BELOW

Table 162-1 Features of Delirium, Dementia, and Psychiatric Disorder

Characteristic	Delirium	Dementia	Psychiatric Disorder
Onset	Over days	Insidious	Sudden
Course over 24 h	Fluctuating	Stable	Stable
Consciousness	Reduced or hyperalert	Alert	Alert
Attention	Disordered	Normal	May be disordered
Cognition	Disordered	Impaired	May be impaired
Orientation	Impaired	Often impaired	May be impaired
Hallucinations	Visual and/or auditory	Often absent	Usually auditory
Delusions	Transient, poorly organized	Usually absent	Sustained
Movements	Asterixis, tremor may be present	Often absent	Absent

MENTAL STATUS EVALUATION DIVIDED INTO SIX CATEGORIES (BELOW):

Table 162-2 Six Elements of Mental Status Evaluation

Appearance, behavior, and attitude
Is dress appropriate?
Is motor behavior at rest appropriate?
Is the speech pattern normal?
Disorders of thought
Are the thoughts logical and realistic?
Are false beliefs or delusions present?
Are suicidal or homicidal thoughts present?
Disorders of perception
Are hallucinations present?
Mood and affect
What is the prevailing mood?
Is the emotional content appropriate for the setting?
Insight and judgment
Does the patient understand the circumstances surrounding the visit?
Sensorium and intelligence
Is the level of consciousness normal?
Is cognition or intellectual functioning impaired?

DELIRIUM:

- Aka → acute confusional state, acute cognitive impairment, altered mental state → all refer to a TRANSIENT DISORDER WITH IMPAIRMENT OF ATTENTION AND COGNITION
- Alerting functions are working, but the patient has difficulty focussing, shifting or sustaing attention → confusion may fluctuate

PATHOPHYSIOLOGY:

- FOUR GENERAL CAUSES:
  - Primary intracranial disease
  - Systemic diseases that secondarily affect the CNS
  - Exogenous toxins
  - Drug withdrawal

CLINICAL FEATURES OF DELIRIUM:

- Generally develops over days → attention, perception, thinking and memory are all altered
- Activity levels MAY BE EITHER INCREASED OR DECREASED and the patient may fluctuate rapidly between the HYPOACTIVE AND HYPERACTIVE STATE
- Symptoms may be intermittent
- SLEEP-WAKE CYCLES ARE DISRUPTECD → agitation at night "sundowning" is common
- Hallucinations tend to be visual

DIAGNOSIS:

- OBTAIN HISTORY FROM CAREGIVERS
- Acute onset of attention deficits and cognitive abnormalities fluctuating in severity throughout the day and worsening at night is VIRTUALLY DIAGNOSTIC OF DELIRIUM
- EVALUATION IS DIRECTION AT DISCOVERING AN UNDERLYING PROCESS → PNEUMONIA, UTI, METABOLIC, INTRACEREBRAL
- MMSE is a tool for detecting delirium, but does not detect mild impairment
- DEPRESSION MAY MIMIC HYPOACTIVE DELIRIUM → withdrawal, slowed speech and poor results on cognitive testing → rapid fluctuation in symptoms delineates delirium from depression

TREATMENT:

- Aimed at underlying cause, with common medical causes being listed below
- Environmental manipulations (adequate lighting, psychosocial support) → aid adjustment to hospital environment
- Sedation may be needed in cases of severe agitation → benzodiazepines, butyrophenones. SEDATION OR RESTRAINT ARE NO SUBSTITUTE FOR DIAGNOSTIC ACTIVITIES OR ILLNESS-TARGETED THERAPY

- Unless a rapidly reversible cause is found → patient should be admitted for further treatment and investigation

Table 162-5 Important Medical Causes of Delirium in Elderly Patients	
Infectious	Pneumonia
	Urinary tract infection
	Meningitis or encephalitis
	Sepsis
Metabolic/toxic	Hypoglycemia
	Alcohol ingestion
	Electrolyte abnormalities
	Hepatic encephalopathy
	Thyroid disorders
	Alcohol or drug withdrawal
Neurologic	Stroke or transient ischemic attack
	Seizure or postictal state
	Subarachnoid hemorrhage
	Intracranial hemorrhage
	Central nervous system mass lesion
	Subdural hematoma
Cardiopulmonary	Congestive heart failure
	Myocardial infarction
	Pulmonary embolism
	Hypoxia or Co_2 narcosis
Drug-related	Anticholinergic drugs
	Alcohol or drug withdrawal
	Sedatives-hypnotics
	Narcotic analgesics
	Polypharmacy

DEMENTIA:

- Implies a loss of mental capacity
- Largest categories are IDIOPATHIC (ALZHEIMER'S DISEASE) OR VASCULAR DEMENTIA → diagnoses of exclusion
- Typical course is slow with insidious symptom onset → presentation to ED is usually precipitated by a sentinel event

PATHOPHYSIOLOGY:

- Pathophysiology of Alzheimer's disease is complex → reduction of neurons in cerebral cortex, increased amyloid deposition and production of neurofibrillary tangles and plaques
- Pathology of vascular dementia → cerebrovascular disease with multiple infarctions
- Different causes of dementia are listed below:

Table 162-6 Classification of Dementia by Cause
Degenerative
Alzheimer's disease
Huntington's disease
Parkinson's disease, others
Vascular
Multiple infarcts
Hypoperfusion (cardiac arrest, profound hypotension, others)
Subdural hematoma
Subarachnoid hemorrhage
Infectious
Meningitis (sequelae of bacterial, fungal, or tubercular)
Neurosyphilis
Viral encephalitis (herpes, human immunodeficiency virus), Creutzfeldt-Jakob disease
Inflammatory
Systemic lupus erythematosus
Demyelinating disease, others
Neoplastic
Primary tumors and metastatic disease
Carcinomatous meningitis
Paraneoplastic syndromes
Traumatic
Traumatic brain injury
Subdural hematoma
Toxic
Alcohol
Medications (anticholinergics, polypharmacy)
Metabolic
Vitamin B_{12} or folate deficiency
Thyroid disease
Uremia, others
Psychiatric
Depression (pseudodementia)
Hydrocephalic
Normal-pressure hydrocephalus (communicating hydrocephalus)
Noncommunicating hydrocephalus

CLINICAL FEATURES:

- Impairment of memory (especially recent memory) is gradual and progressive and remote memories are often preserved
- Progression leads to loss of reading, decreased performance in social situations and loss of direction
- Late stages → extreme disorientation, inability to perform self-care and personality changes
- THE POSSIBILITY OF A CONCURRENT MEDICAL CONDITION SUDDENLY CAUSING COGNITIVE FUNCITONING TO DETERIORATE

SHOULD BE STRONGLY CONSIDERED AND OFTEN IS THE THRUST OF INVESTIGATION IN THE ED
    - UTI, CCF, HYPOTHYROIDISM ARE THREE COMMON EXAMPLES OF CONDITIONS THAT WILL CAUSE A MILDLY DEMENTED BUT FUNCTIONAL INDIVIDUAL TO RAPIDLY DECLINE
- Depression may coexist or MIMIC dementia → PSEUDODEMENTIA → treatable cause of dementia

TREATMENT:

- Antipsychotics for management of psychosis/nonpsychotic behaviours
    - Should be reserved for those with persistent psychotic symptoms or those with extreme disruptive behaviour
- Treatment of vascular dementia limited to management of risk factors
- Normal pressure hydrocephalus should be considered if urinary incontinence and gait disturbance develop early in the disease process

COMA:

- Coma is a state of reduced alertness and responsiveness from which the patient cannot be aroused
- GCS is a widely used scoring system:

Table 162-7 Glasgow Coma Scale

Component	Score	Adult	Child <5 y	Child >5 y
Motor	6	Follows commands	Normal spontaneous movements	Follows commands
	5	Localizes pain	Localizes to supraocular pain (>9 mo)	
	4	Withdraws to pain	Withdraws from nail bed pressure	
	3	Flexion	Flexion to supraocular pain	
	2	Extension	Extension to supraocular pain	
	1	None	None	
Verbal	5	Oriented	Age-appropriate speech/vocalizations	Oriented
	4	Confused speech	Less than usual ability; irritable cry	Confused
	3	Inappropriate words	Cries to pain	Inappropriate words
	2	Incomprehensible	Moans to pain	Incomprehensible
	1	None	No response to pain	
Eye opening	4	Spontaneous	Spontaneous	
	3	To command	To voice	
	2	To pain	To pain	
	1	None	None	

PATHOPHYSIOLOGY:

- Multiple potential causes of coma (listed below)
- Pathophysiology is complex → deficiency of substrate needed for neuronal function (hypoxia, hypoglycaemia)
    - Systemic causes → brain is globally affected

- Primary CNS problems → brainstem disorders. Can be indicated with hemiparesis or cranial nerve abnormalities

Table 162-8 Differential Diagnosis of Coma
Coma from causes affecting the brain diffusely
Encephalopathies
Hypoxic encephalopathy
Metabolic encephalopathy
Hypoglycemia
Hyperosmolar state (e.g., hyperglycemia)
Electrolyte abnormalities (e.g., hypernatremia or hyponatremia, hypercalcemia)
Organ system failure
Hepatic encephalopathy
Uremia/renal failure
Endocrine (e.g., Addison disease, hypothyroidism, etc.)
Hypoxia
CO_2 narcosis
Hypertensive encephalopathy
Toxins
Drug reactions (e.g., neuroleptic malignant syndrome)
Environmental causes—hypothermia, hyperthermia
Deficiency state—Wernicke encephalopathy
Sepsis
Coma from primary CNS disease or trauma
Direct CNS trauma
Diffuse axonal injury
Subdural hematoma
Epidural hematoma
Vascular disease
Intraparenchymal hemorrhage (hemispheric, basal ganglia, brainstem, cerebellar)
Subarachnoid hemorrhage
Infarction
Hemispheric, brainstem
CNS infections
Neoplasms
Seizures
Nonconvulsive status epilepticus
Postictal state

- **HERNIATION SYNDROMES:**
  - **Models for alterations in consciousness, but their mechanisms are unknown → vascular compression due to local cerebral oedema or local increased ICP may be an underlying mechanism**
  - UNCAL HERNATION:
    - Medial temporal lobe shifts to compress upper brainstem → progressive drowsiness followed by unresponsiveness.
    - Ipsilateral pupil is sluggish, eventually becoming dilated
    - Hemiparesis may develop ipsilateral to the mass
  - CENTRAL HERNIATION:
    - Progressive LOC, loss of brainstem reflexes, decorticate posturing and irregular respiration

- In extreme uncontrolled ICP elevation, CPP is diminished as the ICP approaches MAP

CLINICAL FEATURES:

- Clinical features vary BOTH WITH DEPTH OF COMA AND THE CAUSE
- TOXIC-METABOLIC COMA:
  - Plethora of causes
  - Movements are symmetric without evidence of hemiparesis
  - Pupillary responses are preserved
  - DTR are normal
    - A notable exception is coma due to barbiturate poisoning → MIMICS BRAIN DEATH → large pupils, no extraocular movements, flaccid paralysis, apnoea
- COMA FROM SUPRATENTORIAL LESIONS:
  - I.E. lesions of the hemispheres → progressive hemiparesis or asymmetric muscle tone and reflexes → can manifest by asymmetric responses to stimuli or asymmetric extensor or flexor postures
  - Coma without lateralising signs may result form decreased cerebral perfusion secondary to increased ICP
  - HT and bradycardia → CUSHING REFLEX → raised ICP
- COMA FROM INFRATENTORIAL LESIONS:
  - Posterior fossa lesions
  - An expanding lesions may cause abrupt coma, abnormal extensor posturing, loss of pupillary reflexes and loss of extraocular movements
  - The anatomy of the posterior fossa leaves little room to accommodating expanding mass
  - Pontine haemorrhage is differentiated by PINPOINT PUPILS
- PSEUDOCOMA:
  - Psychogenic
  - Pupillary responses, extraocular movments, muscle tone and reflexes are shown to be intact
  - Tests of particular value:
    - Response to MANUAL EYE OPENING → should be little response in truly comatose patient
    - Avoidance of gaze on assessment of extraocular movements
    - Nystagmus with caloric testing

DIAGNOSIS:

- Stabilisation, diagnosis and treatment actions are performed simultaneously
- Reversible causes (↓BSL, opiates) should always be considered
- Tempo of onset of coma is of great diagnostic value
  - Abrupt → CNS failure (Seizures, ICH, stroke)
  - Progressive → neoplasm, ↑BSL
- The goal of the clinician is to rapidly determine if the CNS dysfunction is from diffuse impairment of the brain or if signs point to a focal region of CNS dysfunction

    - Asymmetry of pupils, corneal reflexes, posturing, reflexes → focal lesion suggested
- CT scanning mandated in all patients → suspect basilary artery thrombosis in those with NORMAL CT → HYPERDENSE BASILAR ARTERY
- In children, toxic ingestions, infections and child abuse have HIGHER PREVALENCE
- NONCONVULSIVE STATUS OR SUBTLE STATUS EPILEPTICUS MAY BE AN UNDER-RECOGNISED CAUSE OF COMA

TREATMENT:

- Identification and treatment of underlying cause
    - Evaluation for readily reversible causes, such as hypoglycaemia and opioid toxicity are paramount
- Consider THIAMINE in malnourished/alcoholics prior to glucose administration
- If raised ICP considered a culprit → elevate bed to aid venous drainage, avoid noxious stimuli, mannitol (0.5-1g/kg), hyperventilation to normocarbia if intubated (most cases), dexamethasone if tumour considered culprit lesion