Delirium

CHAPTER 11

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Definition and Aetiology

Delirium is an acute neuropsychiatric syndrome characterised by the onset of fluctuating impairment in attention, awareness and cognition. It is usually triggered by one or more pathophysiological stressors, although ~20% of cases have no determinable precipitant.[1] Delirium is a medical emergency as it can indicate serious underlying pathology and is associated with adverse outcomes.

Delirium develops from the interplay of predisposing vulnerability factors and one or more acute stressors (precipitating factors) (Table 1). Overall risk of developing delirium depends on the number, type and severity[2] of predisposing factors, particularly age and cognitive impairment.[3] In lower risk individuals, a more severe acute insult is usually necessary to precipitate delirium, whereas those with a higher burden of predisposing factors may develop delirium after minor insults.[2]

Predisposing risk factors	Precipitating factors
Demographics • Older age • Lower educational attainment	*Systemic illness or organ dysfunction* • Unplanned hospital admission • Severe acute presenting illness • Infection • Neurological injury e.g. stroke • Uncontrolled pain • Dehydration and acute kidney injury • Hypoxia • Metabolic abnormalities e.g. blood glucose, electrolyte derangements, metabolic acidosis
Co-morbidities • Cognitive impairment and dementia • Cumulative comorbidities • Specific co-morbidities including cardiovascular disease, pulmonary disease (obstructive sleep apnoea or chronic obstructive pulmonary disease), central nervous system disorders (including previous delirium), diabetes, chronic kidney disease, anaemia, malignancy, chronic pain, terminal illness	*Surgical factors* • Surgery type – emergency procedure, thoracic or aortic surgery, major abdominal surgery, major joint surgery • Intraoperative blood loss and haemodynamics • Duration of surgery • Anaesthesia type and depth
Functional impairment • Frailty • Sensory impairment (vision and hearing)	*Medication or substance related* • Opioids • Anticholinergics • Benzodiazepines • Psychoactive drugs • Alcohol or recreational drug intoxication or withdrawal • Nicotine withdrawal
Psychiatric comorbidity • Depression, other psychiatric disorders or traits (broadly defined, anxiety, neuroticism, and low conscientiousness)	*Nutritional status* • Malnutrition • Vitamin deficiency e.g. B1
Medications and drugs • Polypharmacy • Specific medications including opioids, anticholinergics, steroids, benzodiazepines and other psychoactive drugs (e.g. gabapentin, leviteracetam, antidepressants) • Alcohol or illicit drug use	*Iatrogenic and environmental* • ICU admission • Mechanical ventilation • Invasive devices and instrumentation e.g. intravenous lines, indwelling catheters • Poor sleep and day-night orientation • Restraint use • Immobility
Nutritional status • Malnutrition or undernutrition (chronic)	*Other* • Constipation • Urinary retention

Table 1: Predisposing and precipitating factors for delirium. List not exhaustive, adapted from Ormseth et al, 2023.[4]

Epidemiology

Delirium is common amongst hospital inpatients across all services. A 2025 systematic review and meta-analysis of medically hospitalised older adults aged ≥ 60 years yielded a pooled prevalence of 23.6% and incidence of 13.5%.[5] Delirium rates are highest in intensive care units (35.7%[6] and likely higher in ventilated patients), older post-operative patients (incidence depends on surgery type, range 13-32%[7]), and palliative care inpatients in the weeks prior to death (up to 88%).[8]

Delirium rates are lower in community settings compared to hospitals, although recognition may be reduced due to social and other factors e.g. whether there is a co-resident caregiver. A systematic review reported delirium prevalence of 0.2-8.3% in community-dwelling adults aged ≥65 years, with higher prevalence in those who were frail and receiving home care or primary care.[9] Prevalence of delirium in nursing home residents varies from 1.4-70.3% depending on diagnostic criteria, screening tools utilised and baseline population characteristics.[10]

The global economic burden associated with delirium is significant. Estimates vary, but cost of inpatient delirium in the United States alone may be as high as US$82.4 billion.[11] For hospitalised patients the presence of delirium increases the cost associated with admission and is highest for individuals with delirium superimposed on dementia.[12]

Pathophysiology

The pathophysiology of delirium is complex and current understanding limited. This is because multiple aetiological factors usually underpin the clinical syndrome (both predisposing and precipitating), so unravelling contributory neurobiological mechanisms is challenging.[3] To date, no common pathway has been identified, although several pathophysiological mechanisms are implicated as listed below and illustrated in Figure 1.

1. Neuroinflammation
2. Cerebral vascular dysfunction
3. Altered brain metabolism
4. Neurotransmitter imbalance
5. Impaired neuronal network connectivity

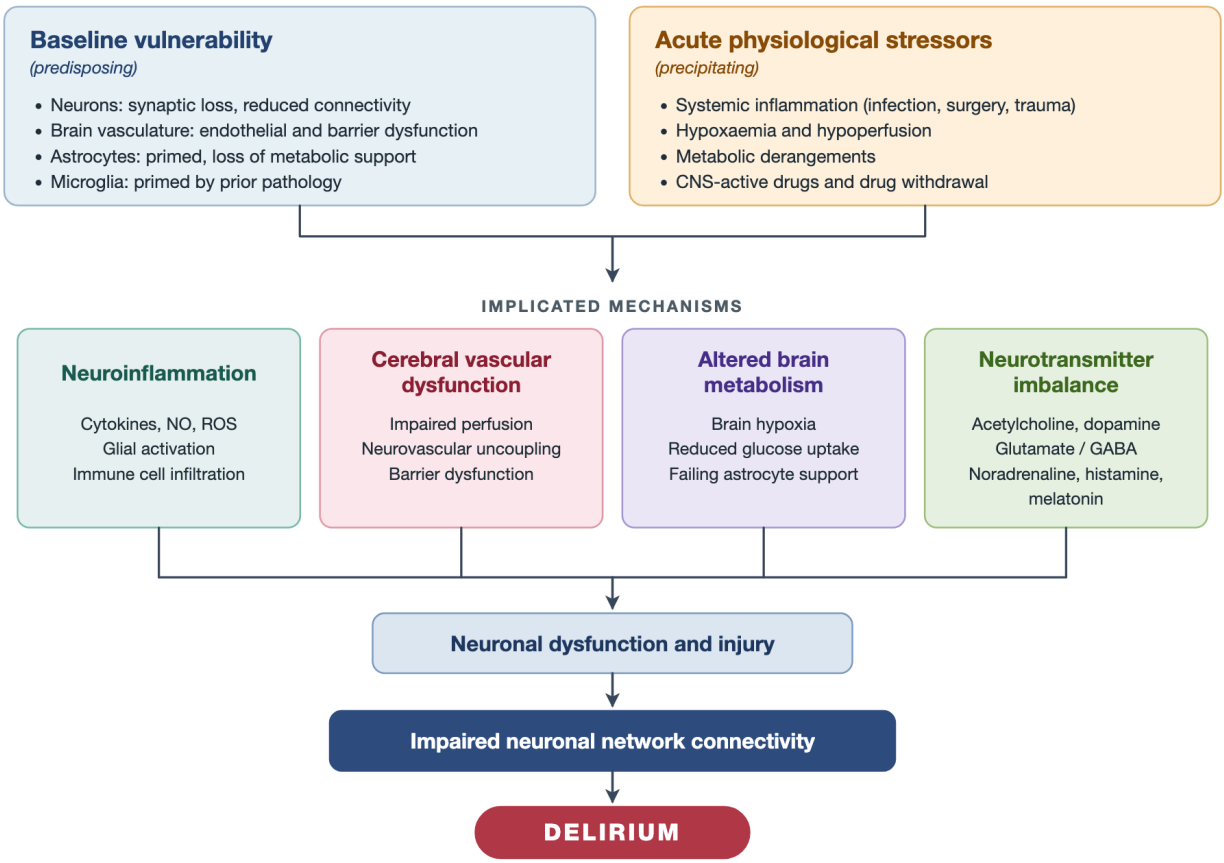


Figure 1: Pathophysiological mechanisms implicated in delirium, adapted from Cunningham et al 2023.[13] NO, nitric oxide; ROS, reactive oxygen species.

Presentation and Course of Illness

Delirium can present with a mix of motor, psychiatric and cognitive symptoms. The hallmarks are:

1. Acute onset (hours to days) and
2. Fluctuating level of consciousness and cognition (particularly attention).

The heterogeneity and fluctuating nature of symptoms can contribute to misdiagnosis or the diagnosis being missed or delayed.[14]

There are three motor subtypes: hyperactive, hypoactive,[15] and mixed (features of both hyper-and hypoactive).

- Hyperactive: increased motor activity (wandering, agitation, picking) and 'excess' or abnormal sensory experiences, such as hallucinations, delusions, aggression, irritability and being overly talkative/noisy.[16] This subtype may be misdiagnosed as a psychotic illness, mania or anxiety disorder. Given these symptoms may interfere with delivery of health care, patients with this presentation are more quickly identified as delirious, although they only account for 25% of cases.[17]

- Hypoactive: is the more common presentation (~75%),[17] characterised by reduced movement/activity and withdrawal/loss of awareness. The patient may appear sleepy or listless, psychomotor retarded, and have reduced speech.[16] A high index of suspicion is needed to diagnose hypoactive delirium, as the patient's behaviour is not usually disruptive to care. They may be misdiagnosed with depression or dementia.

Course of illness

The clinical features of delirium usually develop over hours to days. While delirium is considered a transient state, duration is variable and can be prolonged. Approximately one in five older hospital patients with delirium will have some residual symptoms at six months.[18] It is important that patients and their family/carers are informed so that realistic expectations are set regarding trajectory.

Delirium is associated with numerous short and long-term adverse outcomes. During the acute episode, risk of hospital acquired complications e.g. aspiration, pressure areas, falls, dehydration, reduced mobility and function are increased, resulting in longer length of hospital stay. Post delirium, the risk of cognitive decline and dementia, hospital readmission, institutionalisation, impaired quality of life and mortality are increased,[19] with one-year mortality approximately double that of patients without delirium.[20] Older age, frailty, dementia comorbidity, hypoactive subtype, prolonged delirium and delirium severity are predictors of poorer outcomes.[21] Social determinants of health may impact diagnosis, treatment and outcomes of delirium with worse outcomes in minority or disadvantaged patient cohorts.[22]

Assessment and Diagnosis

The gold standard for delirium diagnosis is clinical assessment. The DSM-5 criteria[23] outline the key features (Table 2). Criterion E may not always be met.

DSM-5 Criteria for Delirium
Criterion A – **Disturbance in attention** (i.e. reduced ability to direct, focus, sustain, and shift attention) **and awareness** (reduced *orientation to the environment*).
Criterion B – The disturbance **develops over a short period of time** (usually hours to a few days), represents an **acute change from baseline** *attention and awareness*, and tends to **fluctuate** in severity during the course of a day.
Criterion C - An **additional disturbance in cognition** (e.g. memory deficit, disorientation, language, visuospatial ability, or perception)
Criterion D- The disturbances in Criteria A and C are **not better explained by** a pre-existing, established or evolving **neurocognitive disorder** and **do not occur in the context of a severely reduced level of arousal** such as coma.
Criterion E- There is **evidence** from the history, physical examination or laboratory findings that the disturbance is a direct physiological consequence **of another medical condition, substance intoxication or withdrawal** (i.e. due to a drug of abuse or to a medication), **or exposure to a toxin**, or is due to multiple aetiologies.

Table 2: DSM-V Criteria for Delirium[23]

To diagnose delirium:

- Take a history from the patient
- Assess their level of attention/arousal and other cognitive deficits
- Evaluate mental state abnormalities (e.g. psychotic, emotional and behavioural signs)
- Conduct a physical examination to identify underlying causes.
- Take a collateral history (e.g. from family/carers, general practitioner, medical records) to establish baseline cognition and function, symptom onset and identify fluctuating level of consciousness and cognition.

Screening tools may help in settings with a high prevalence of delirium to identify cases early and trigger comprehensive clinical assessment. Which tool is best (most sensitive and specific) depends on the setting of care, administration time, ease of use and whether staff require training to use the tool.[24] A validated tool used internationally is the 4AT (www.the4AT.com).[25, 26] This 2-minute 4-item tool requires no training to administer. It has psychometric evidence for use with people with dementia and those of culturally and linguistically diverse (CALD) backgrounds.[27]

Monitoring tools can be used for serial screening or once delirium has been diagnosed to track symptom resolution. Options include the Single Question in Delirium[28] (SQiD), modified Richmond Agitation Screening Scale (mRASS),[29] Delirium Observation Screening Scale (DOSS),[30] Nurses' delirium screening checklist (Nu-DESC)[31] and the Recognising Acute Delirium as Part of Your Routine (RADAR).[32] A positive result should trigger diagnostic assessment.[3]

The differential diagnosis of delirium is broad and includes cognitive disorders and mental illness (Table 3).

	Delirium	**Dementia**	**Depression**	**Schizophrenia (psychotic episode)**
Age of first episode	Usually middle to older age	Older adults	Any age	Most common in young adults; second smaller peak of onset in late life
Onset	Sudden, acute (hours-days)	Slow, insidious (months-years)	Variable: Gradual/subacute (weeks-months)	Variable: Subacute (weeks), often with previous episodes
Course	Fluctuates	Non-fluctuant*, slowly progressive	Stable	Variable, but generally not fluctuating
Duration	Days- weeks	Years	Months- years	Months- years

Reversibility	Usually reversible	Irreversible	Yes	Usually reversible
Attention	Impaired, distracted	Normal (until late in illness course)	Normal (unless comorbid catatonia or psychosis)	Varies depending on nature of psychotic symptoms
Level of consciousness	Altered	Normal until late*	Normal	Normal
Psychotic symptoms (hallucinations and delusions)	Common. Often changeable, brief, poorly formed and unelaborated	May occur but often later in illness	Uncommon (unless psychotic depression and with this presentation usually mood-congruent)	Common. Usually complex, structured and similar over the course of an episode
Involuntary movements	Tremors, picking	Absent until late	Absent	May have extrapyramidal side effects from antipsychotic treatment

*Exception is dementia with Lewy bodies

Table 3. Differential diagnosis of delirium

Investigations

Suspected delirium should trigger broad investigations (Table 4) for common causes as well as tests targeting the patient's presenting symptoms, medical history and physical examination.

- Bloods: electrolytes (includes glucose and renal function), liver function tests, calcium, magnesium, phosphate, full blood count, thyroid function studies, CRP
- Drug levels (e.g. lithium, digoxin) where relevant
- Urinalysis and culture
- Bladder scan and postvoid residual (for suspected urinary retention)
- Chest X-ray
- Electrocardiogram

Table 4. Initial investigations for causes of delirium

Additional tests may be indicated such as blood cultures (if fever or sepsis), neuroimaging (e.g. after a fall, focal neurological signs, taking an anticoagulant),[33] electroencephalogram (suspicion for epileptic activity), and per rectum examination (or, if not possible, abdominal x-ray for constipation). A lumbar puncture should not routinely be performed as it is invasive, may increase distress and confers risk of adverse events.[33]

Underlying acute causes of delirium may include the Five Ps (pee, poo, pain, pus and poison) and **PINCHES ME:**[34]

P: Pain- it may be difficult for people with delirium, dementia or impaired communication to report pain, assess for changes in facial expression, behaviour and function

I: Infection– while urinary tract infections are often considered, dental or atypical presentations of cerebral/respiratory infections may be missed

N: Nutrition

C: Constipation- this is common in older people and inconsistently monitored

H: Hydration

E: Endocrine and electrolyte

S: Stroke

M: Medication- check for drugs with anticholinergic properties (see medicheck.com). Consider also supratherapeutic drug levels (e.g. lithium), substance use and withdrawal.

E: Environment- overstimulating noisy environments and sensory impairment (poor vision or hearing, language barriers for CALD people) can lead to misinterpreting the environment.

Neuropsychiatric aspects of treatment

1. Prevention

Prevention of delirium, especially in high-risk populations, has the strongest evidence. Multicomponent interventions have been shown to reduce the incidence of delirium in hospitalised older patients by ~30%.[35] Although specific interventions differ, common elements include;

- maintaining adequate hydration and nutrition,
- supporting regular bladder and bowel function,
- early mobilisation and physiotherapy,
- non-pharmacological measures to improve sleep,
- reorientation (including delivered by educated family/carers),[36]
- cognitive stimulation,
- addressing hypoxia,
- pain management
- correcting sensory impairment (e.g. through visual and hearing aids).[3]

Iatrogenic factors which may trigger delirium should be avoided where possible, such as physical restraint (including bed rails), certain medications (e.g. benzodiazepines), intravenous lines and indwelling catheters.[37] Proactive geriatric medicine consultation may reduce the incidence of delirium in high-risk surgical populations by identifying and addressing risk factors for delirium, including medication review and reducing exposure to deliriogenic medications.[38, 39]

There is limited evidence for medication preventing delirium. Dexmedetomidine may reduce the incidence of delirium compared to placebo,[40, 41] and is recommended in preference to other sedating agents for patients in critical care settings or undergoing major surgery.[39] Findings are mixed regarding the effectiveness of melatonin for delirium prevention,[33] with some evidence in surgical settings.[42] Recent studies suggest potential for orexin antagonists (lemborexant and suvorexant) in delirium prevention,[43, 44] but more randomised clinical trials are needed. There is also emerging evidence for intranasal insulin to prevent post operative delirium but larger multicentre trials are needed in a wider range of surgical populations.[45, 46] Evidence does not support the use of antipsychotics,[40, 47] or ketamine[48] to prevent delirium.

1. Management

- Treat the precipitating factor(s)

Delirium management involves identifying and treating the underlying causes of delirium (see PINCHES-ME). Evidence for delirium management is less strong than for delirium prevention, but many of the non-pharmacological strategies for prevention are also applicable.[49] Multicomponent interventions reduce the duration and severity of delirium and incidence of pressure injuries, but there is no clear evidence for improved length of hospital stay or mortality, return to independent living or falls.[49]

Although many non-pharmacological strategies reflect good quality clinical care, there are challenges with implementation[3] and inconsistent delivery in busy ward environments. Training and education in delirium for healthcare workers may be inadequate, leading to limited awareness and skill, and unhelpful attitudes with resulting lack of ownership of delirium care.[3] However, educational interventions may be effective, with a systematic review reporting benefits in at least one measure in 90% of studies.[50] Initiatives linking staff education to action, such as the Think, Investigate, Manage, Engage (TIME) bundle, can lead to improved assessment of delirium triggers and exacerbating factors.[51]

- Symptomatic treatment

Delirium can be distressing for both the patient and their family/carers. Communication is important, including providing information about delirium, the management of current symptoms and reducing risk of complications.[3, 39] Family and carers may also assist in implementing non-pharmacological strategies, e.g. reorientation,[36] hydration.

There is no consistent evidence that any medication reduces delirium duration, severity or complication rates.[3] Antipsychotics, benzodiazepines, cholinesterase inhibitors, melatonin and melatonin receptor agonists, clonidine and dexmedetomidine have been studied. Dexmedetomidine may confer benefit in mechanically ventilated patients in intensive care (reduced delirium duration, earlier extubation),[52] although a meta-analysis noted limited study quality.[41]

The routine use of antipsychotics for treating delirium is not supported by evidence from several meta-analyses and systematic reviews,[47, 53-55] with lack of association between use and change in severity or duration of delirium.[54] Antipsychotics are also associated with adverse effects including falls, sedation and cardiac effects (e.g. QT prolongation).[54, 55] However, short-term use may be considered when there is risk of the patient harming themselves or others, or severe distress, and other strategies have been ineffective.[33, 39] In such circumstances, the potential harm versus

benefit should be considered and informed consent obtained from the patient/family/carer.[56] Use of antipsychotics for delirium should be reviewed daily with cessation as soon as clinically appropriate.[33] Starting doses should be low, i.e. aripiprazole 5mg, haloperidol 0.5-1mg, olanzapine 2.5mg, quetiapine 12.5-25mg, risperidone 0.25-0.5mg and ziprasidone 20mg.[57] Benzodiazepines should be avoided[33, 39] as adverse events are common and long acting benzodiazepines may promote delirium.[58-60]

- Follow-up

Recovery from delirium is variable and can be prolonged. The patient may need additional supervision or support with daily functioning after discharge. Clinicians should educate the patient and their family/carers about risk of recurrence, strategies to mitigate risk factors and encourage prompt assessment of any acute change in mental state or cognition. Follow-up is important,[56] as delirium can accelerate the course of cognitive decline[33] and patients may develop depression.[61]

Chapter Summary

Definition & Classification: Delirium is a syndrome characterised by acute onset and fluctuating impairment in attention, awareness and cognition which may be caused by medical illness, substances or surgery.

Epidemiology: Delirium rates are highest in older adults and in particular settings, such as intensive care units, postoperative and palliative care wards, and nursing homes.

Clinical Presentation: Delirium may present with a mix of motor, cognitive and psychiatric symptoms. Additional features include sudden onset and fluctuating course and severity.

Pathophysiology: Various neurobiological mechanisms have been proposed for delirium, but no common pathway identified.

Course of illness and prognosis: While delirium is largely reversible with treatment of underlying factors, it may persist in 20% of older inpatients. Worse outcomes are associated with hypoactive delirium, comorbid dementia, older age, frailty and prolonged or severe delirium. Importantly, delirium may accelerate cognitive decline and increase risk of dementia.

Assessment and Diagnosis: Clinical assessment (history, physical examination and cognitive testing) alongside collateral history (to establish onset, fluctuation and baseline cognition) may establish the diagnosis of delirium. The DSM-5 diagnostic criteria outlines key features. Screening tools may be helpful to detect delirium in high-risk populations.

Investigations: Investigations for underlying causes of delirium are guided by the patient's presenting symptoms, medical history and physical findings.

Neuropsychological Profile: Impaired attention is a component of the diagnostic criteria for delirium, but other cognitive domains, such as orientation, may be affected. Disturbed level of consciousness is a key feature.

Management: Treatment of the underlying cause(s) of delirium is the basis of management. There is good evidence that multicomponent non-pharmacological interventions may prevent delirium. There is no good evidence for any medications to treat delirium and none are approved for this indication. However, antipsychotics may be used cautiously short-term for severe distress or when there is risk of the patient harming themselves or others. Follow-up after delirium is indicated given risk of cognitive impairment.

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