

Delirium

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Delirium = Acute Brain Failure

Presentation

Functional
Impairment

Impaired
swallow

Drowsiness

Agitation

Memory

Aggression

Impairment

Confusion

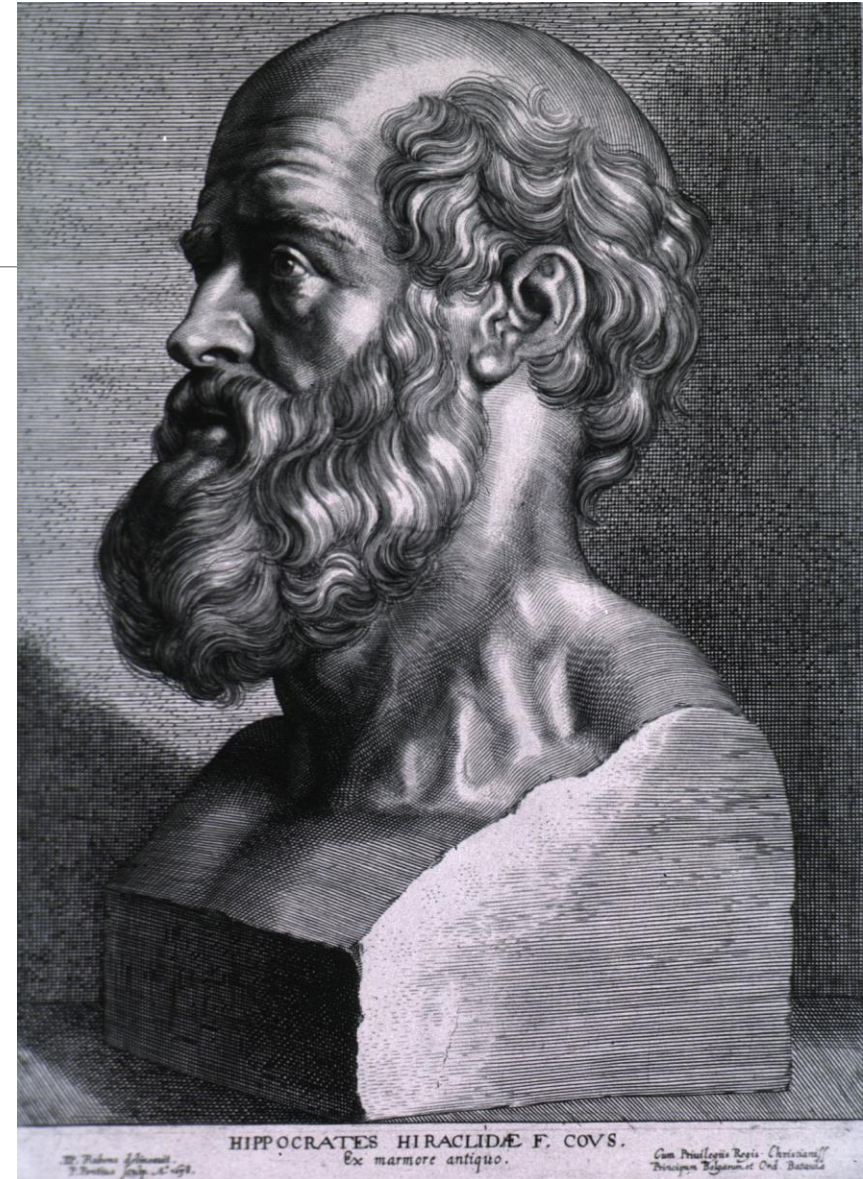
Delusions

Hallucinations

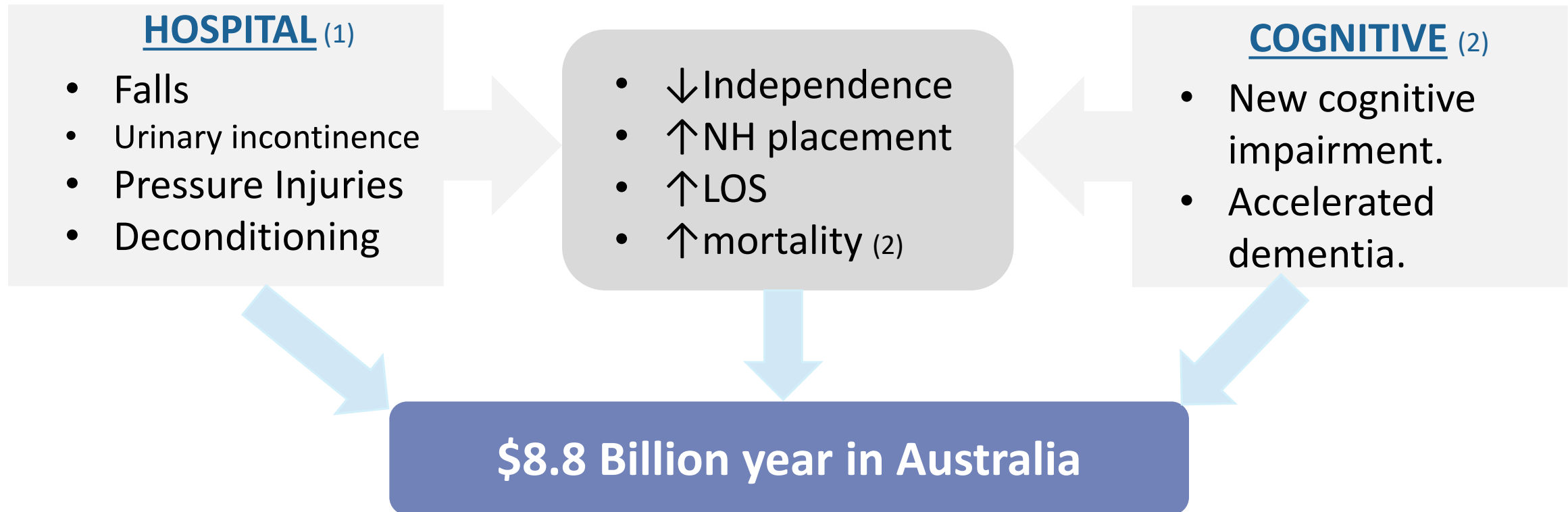
Falls

Background

- First described by Hippocrates 500BC
 - “Phrenitis” and “Lethargus”
- Clinical diagnosis(1):
 1. Disturbance in attention and awareness
 2. Acute and fluctuating
 3. Additional disturbance in cognition
 - Memory, orientation, language, visuospatial, perception
 4. Not due to another pre-existing, established or evolving neurocognitive disorder.
 5. Direct physiological consequence of another medical condition.
- Phenotypes: hyperactive, hypoactive, mixed.

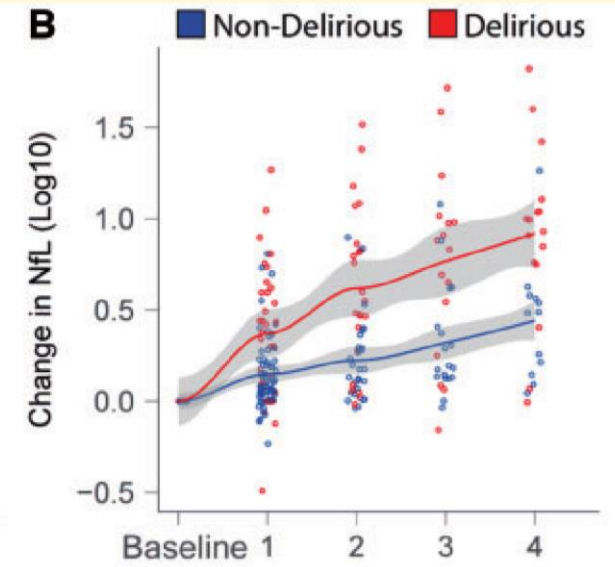
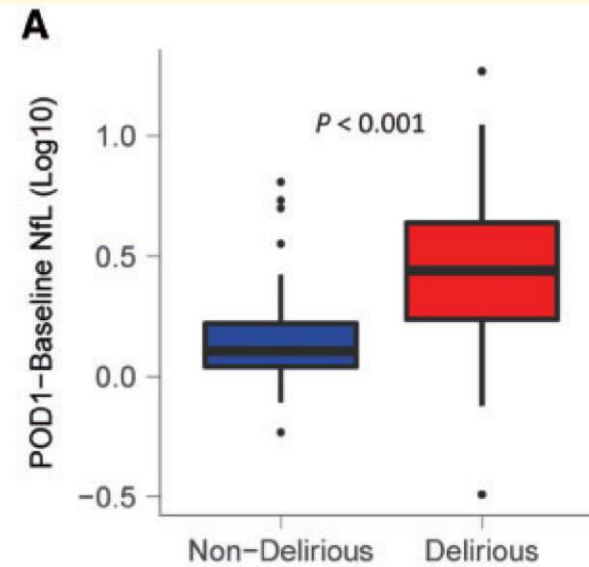
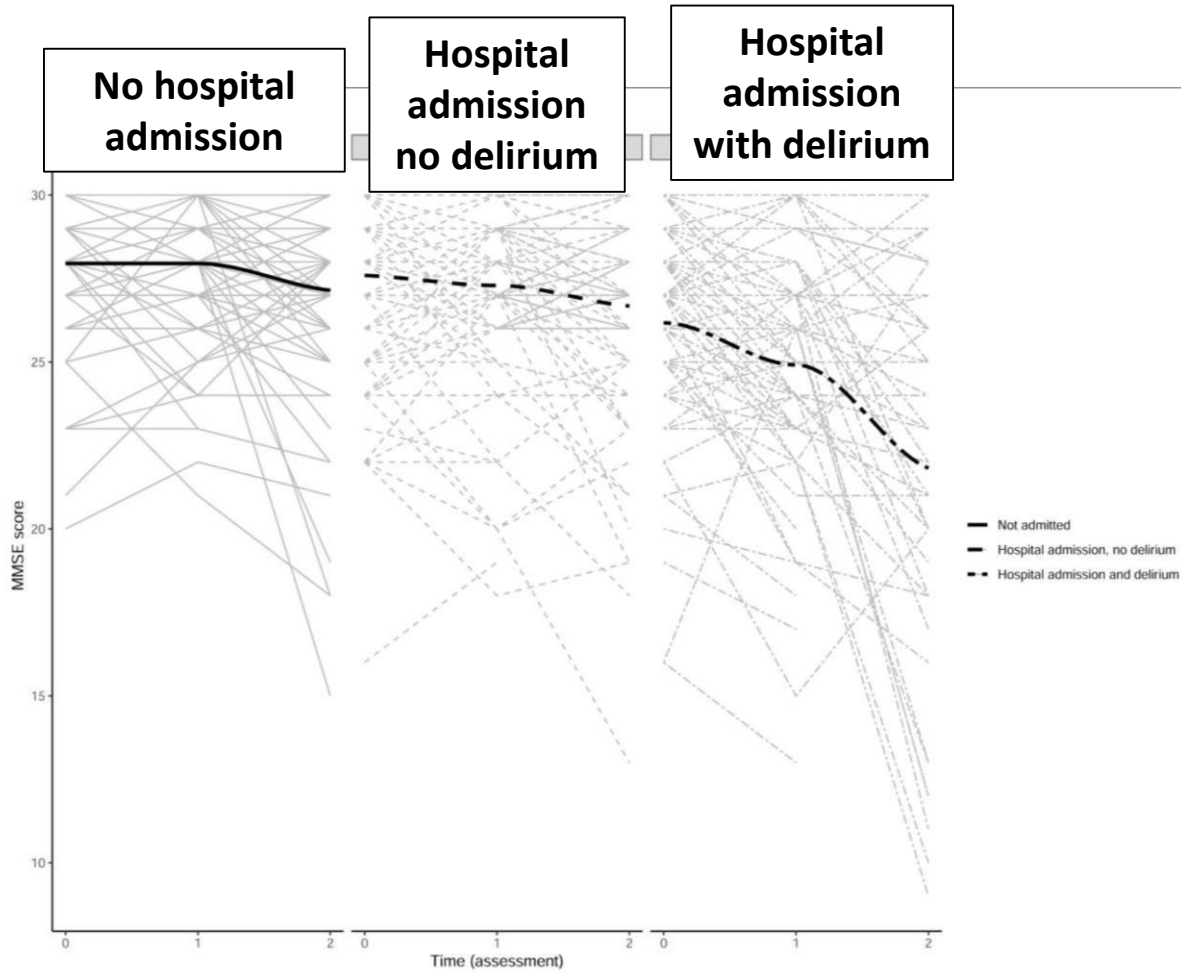


Complications of Delirium



Increased duration of delirium are associated with worse outcomes.

Is delirium a modifiable risk factor for dementia?



Incidence of delirium

Delirium is common:

Population	Prevalence (%)	Incidence (%)
Geriatric Medicine	25	20-29
General Medicine	18-35	11-14
ICU	7-50	19-82
Dementia	18	56
Orthopaedic Surgery	17	12-51
Cardiac Surgery	-	11-46
Non-cardiac Surgery	-	11-46
Palliative care, cancer	-	47

CASE 1

- Mrs A, 87F from HLCNH, presents with delirium “calling out”
 - Mx: Dementia, IHD, HTN, visual impairment secondary to glaucoma.
 - Mobilises 2 assist 4WW, requires assistance all pADLs.
 - O/E: afebrile, HD stable, abdomen tender LLQ
 - Ix: Bloods – NAD, bladder scan 40mL, U/A clear
 - AXR – faecal loading +++

IMPRESSION: Delirium secondary to constipation.

- Discharged with post-enema with laxatives.

CASE 2

- Mrs B, 83F from home, presents with delirium – “drowsy, slurred speech, disorientated”.
 - Mx: HTN
 - High functioning, cognitively intact, independent all ADLs.
 - O/E afebrile, HD stable, hypoactive, nil focal neurology/infectious source.
 - Bloods – NAD, bladder scan 40mL, U/A 10-100WCC, - nitrites.
 - CXR clear, CTB nil acute.

IMPRESSION: Delirium secondary to UTI -> admit.

- Day 1: remains drowsy, 2 assist mobility, ?dysarthria
- Day 2: MRI -> internal capsule stroke
- 4 weeks later, post-rehabilitation discharged to RACF.

Why do people become delirious?

Predisposing factors:

- Dementia
- Cognitive impairment
- History of delirium
- Functional impairment
- Visual impairment
- Hearing impairment
- Medical comorbidities
- Alcohol misuse
- Age >75 years

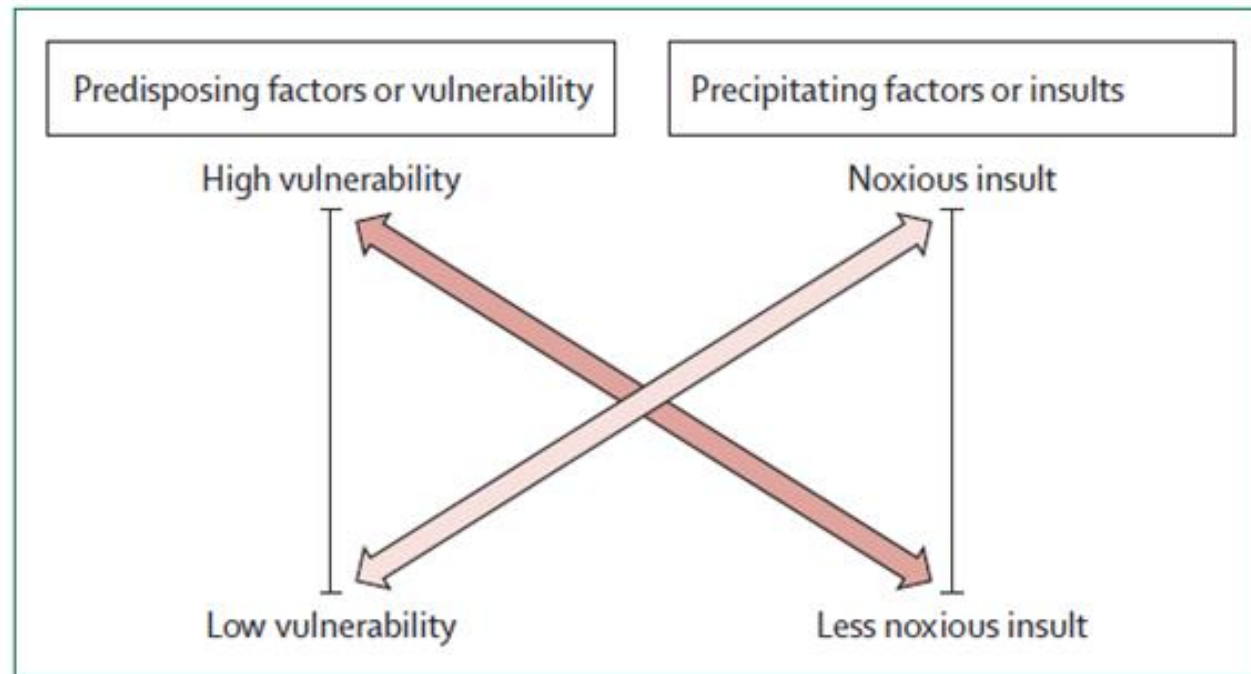


Figure: Multifactorial model of delirium in older people

Precipitating factors:

- Drugs
- Infection (bugs)
- Metabolic
- Brain disorders
- Systemic organ failure
- Urinary retention
- Constipation
- Environmental
- Surgery



**Clinical Care
Standards**

Delirium Clinical Care Standard

Screen patients for delirium



- 1** A patient presenting to hospital with one or more key risk factors for delirium receives cognitive screening using a validated test. In addition, the patient and their carer are asked about any recent changes (within hours or days) in the patient's behaviour or thinking.
-

Delirium Detection

4AT (screening)

CAM / DSMV (screening and diagnosis)

Figure 1. 4AT assessment sticker

4AT Delirium assessment tool (65 years and over)

Has your patient been more **confused, sleepy or drowsy**? Place this sticker in the notes and complete to assess for delirium.

	Circle score for each section
1 Alertness	
Normal (fully alert, but not agitated)	0
Mild sleepiness for <10 seconds after waking, then normal	0
Clearly abnormal	4
2 AMT4 Ask your patient the following: age, date of birth, name of hospital/building, current year	
No mistakes	0
1 mistake	1
2 or more mistakes or untestable	2
3 Attention Ask your patient to list the months of the year backwards	
7 months or more correctly	0
Starts, but scores <7 months/refuses to start	1
Untestable (cannot start because unwell, drowsy)	2
4 Acute change or fluctuating course <i>Evidence of significant change or fluctuation in alertness, cognition, other mental function arising over the last 2 weeks and still evident in last 24 hours</i>	
No	0
Yes	4

4 or above - possible delirium - use the Delirium pathway

1-3 - possible cognitive impairment

0 - delirium or severe cognitive impairment unlikely (but delirium still possible if 4 information incomplete)

Total score

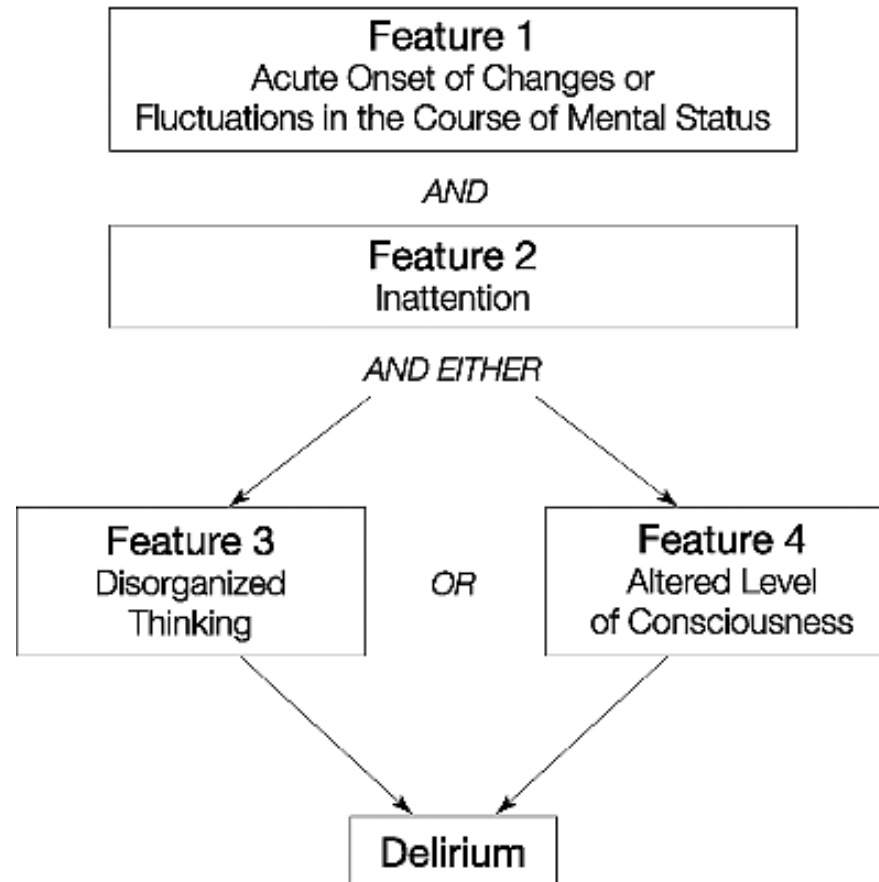
Adapted from MacLulich A (2014). See full delirium guideline on intranet.

Diagnose delirium



- 2** A patient with cognitive impairment on presentation to hospital, or who has an acute change in behaviour or cognitive function during a hospital stay, is promptly assessed for delirium by a clinician trained and competent in delirium diagnosis and in the use of a validated diagnostic tool. The patient and their carer are asked about any recent changes in the patient's behaviour or thinking. The patient's diagnosis is discussed with them and is documented.

CAM (Confusion Assessment Method)



DSM V

- A) Disturbed level of attention and awareness
- B) Develops over a short period, represents an acute changes, fluctuates
- C) Change in cognition
- D) No better explained by pre-existing, established neurocognitive disorder
- E) Evidence of an underlying cause

Features	Delirium	Dementia
Onset	Acute	Gradual
Course	Fluctuating	Progressive
Duration	Days – weeks	Months - years
Consciousness	Altered	Clear
Attention	Impaired	Normal (unless severe)
Psychomotor changes	Increased or decreased	Often normal
Hallucinations	Common	Usually only advanced disease
Reversibility	Usually	Rarely

Prevent delirium



- 3** A patient at risk of delirium is offered a set of interventions to prevent delirium and regular monitoring for changes in behaviour, cognition and physical condition.

Non Pharmacological Prevention/ Management

- Personal profile (sun flower tool)
- Behaviour chart (to identify triggers)
- Family involvement
- Hydration / Nutrition (chart)
- Mobility and falls assessment
- Bladder and bowel function
- Sleep hygiene
- Sensory input
- Pain management
- Promoting cognition: reorientate, reassure

30-40% of delirium is preventable



Investigate and treat the precipitants



- 4** A patient with delirium is offered a set of interventions to treat the causes of delirium, based on a comprehensive assessment.

Patient Assessment

History and Examination

- Medication review (especially anticholinergics / sedatives / opioids)
- Alcohol, smoking and benzodiazepine use (consider withdrawal)
- Ask about pain/discomfort (e.g. urinary retention, constipation)
- Collateral history from family / nursing staff / carers
- OE: Vitals, hydration status, infectious sources, focal neurology, urinary retention

Delirium Investigations

Consider based on the history and clinical assessment

- FBC, EUC, CMP, LFTs, glucose
- TFTs, B12 level (if not recently checked)
- Drug levels if relevant (e.g. digoxin, lithium, anticonvulsants)
- Septic screen (U/A +/- MSU, blood cultures if febrile)
- CXR
- Bladder scan
- ECG / troponin (if indicated) – atypical presentation of AMI

Delirium Investigations – 2nd Line

- ABG
- AXR
- CT Brain (if **focal neurology** or suspected **head trauma**)
 - Hufschmidt 2008 – retrospective study of 294 patients:
 - No focal abnormalities **and** fever or dehydration → 96% scans normal
 - No focal abnormalities **and** baseline dementia → 98% scans normal
- EEG
 - Exclude occult seizures / non-convulsive status epilepticus
 - Almost always abnormal in delirium (diffuse slowing) – therefore can help differentiate from psychiatric conditions
- Lumbar Puncture
 - Suspected infective / inflammatory / paraneoplastic processes
- MRI

Reduce the risk of complications



- 5** A patient with delirium receives care based on their risk of falls and pressure injuries.

Pharmacotherapy in Delirium

Antipsychotics = no evidence



- 6** Treatment with an antipsychotic medicine is only considered if a patient with delirium is distressed and the cause of their distress cannot be addressed and non-drug strategies have failed to ease their symptoms.

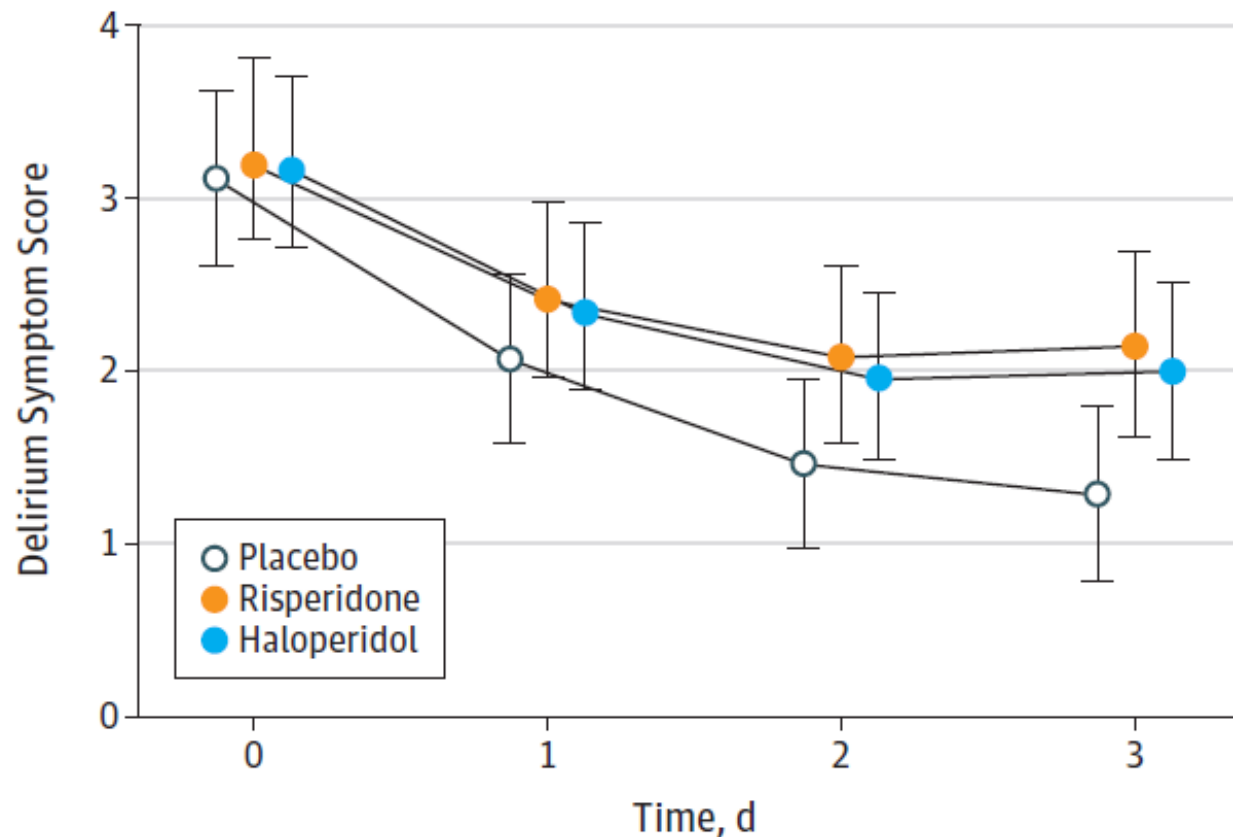
Efficacy of Oral Risperidone, Haloperidol, or Placebo for Symptoms of Delirium Among Patients in Palliative Care

A Randomized Clinical Trial

JAMA Intern Med. doi:10.1001/jamainternmed.2016.7491

Published online December 5, 2016.

Meera R. Agar, PhD; Peter G. Lawlor, MB; Stephen Quinn, PhD; Brian Draper, MD; Gideon A. Caplan, MBBS; Debra Rowett, BPharm; Christine Sanderson, MPH; Janet Hardy, MD; Brian Le, MBBS; Simon Eckermann, PhD; Nicola McCaffrey, PhD; Linda Devilee, MBus; Belinda Fazekas, BN; Mark Hill, PhD; David C Currow, PhD



Antipsychotics

- Reserve for patients with **severe agitation** where required for:
- Investigation & treatment of underlying conditions
- Reducing risk of harm to self or others
- Severe, **distressing** psychotic symptoms
- No role in hypoactive delirium
- Avoid in Parkinsons disease / LBD (risk of ESPE)
- Start low, go slow
- Oral route where possible

Antipsychotics: Side Effects

- Extrapyraxidal side effects
- Both typical & atypical attach to D2 receptor
- Atypical occupy D2 receptor transiently and then rapidly dissociate → less likely to cause EPS
- QT prolongation
- Prolong duration of delirium
- Increased risk of falls & fractures
- Orthostatic hypotension, seizures, disturbed glucose & lipid metabolism
- Increased risk of stroke & death

The extremely agitated patient (chemical restraint)

1. Get help – security
2. If sedation is required – try oral first:
 - Antipsychotics:
 - Risperidone 0.25 – 0.5mg PO (tablet or liquid)
 - Haloperidol 0.25 - 0.5mg PO / SC / IM
 - Quetiapine 12.5 – 25mg PO
 - Benzodiazepines – lorazepam 0.5 – 1 mg PO
3. Parenteral sedation:
 - Haloperidol 0.5mg IM
 - Midazolam 1 - 2mg IM – with close monitoring of airway

Discharging a patient with delirium



- 7** Before a patient with current or resolved delirium leaves hospital, the patient and their carer are involved in the development of an individualised care plan and are provided with information about delirium. The plan is developed collaboratively with the patient's general practitioner and describes the ongoing care that the patient will require after they leave hospital. It includes a summary of any changes in medicines, strategies to help reduce the risk of delirium and prevent complications from it, and any other ongoing treatments. This plan is provided to the patient and their carer before discharge, and to their general practitioner and other ongoing clinical providers within 48 hours of discharge.

Initial check for acute, life-threatening causes

Identify & treat causes / optimise conditions for brain recovery

Detect & treat distress / agitation

Consider dementia / Follow-up

Delirium 8

Prevent complications

Rehabilitation during delirium

Monitor for recovery

Communicate with patient & carers

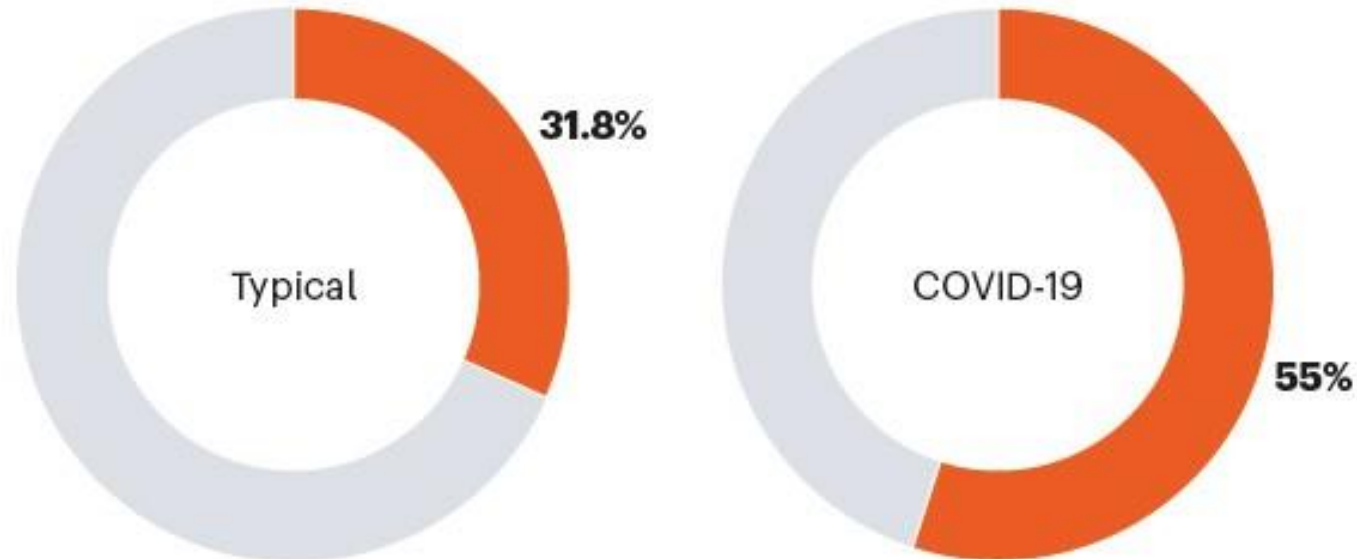
Summary:

1. Delirium = acute brain failure, causes dementia and is a medical emergency
2. If in doubt, assume delirium, not dementia
3. Screening, prevention and management of delirium are everyone's responsibility
4. Diagnosing delirium is just the beginning

COVID and delirium

HOW COMMON IS DELIRIUM?

Typically, almost one-third of people who are critically ill will have an episode of delirium; for COVID-19, the proportion rises to more than half.



Mechanisms of cognitive decline in delirium

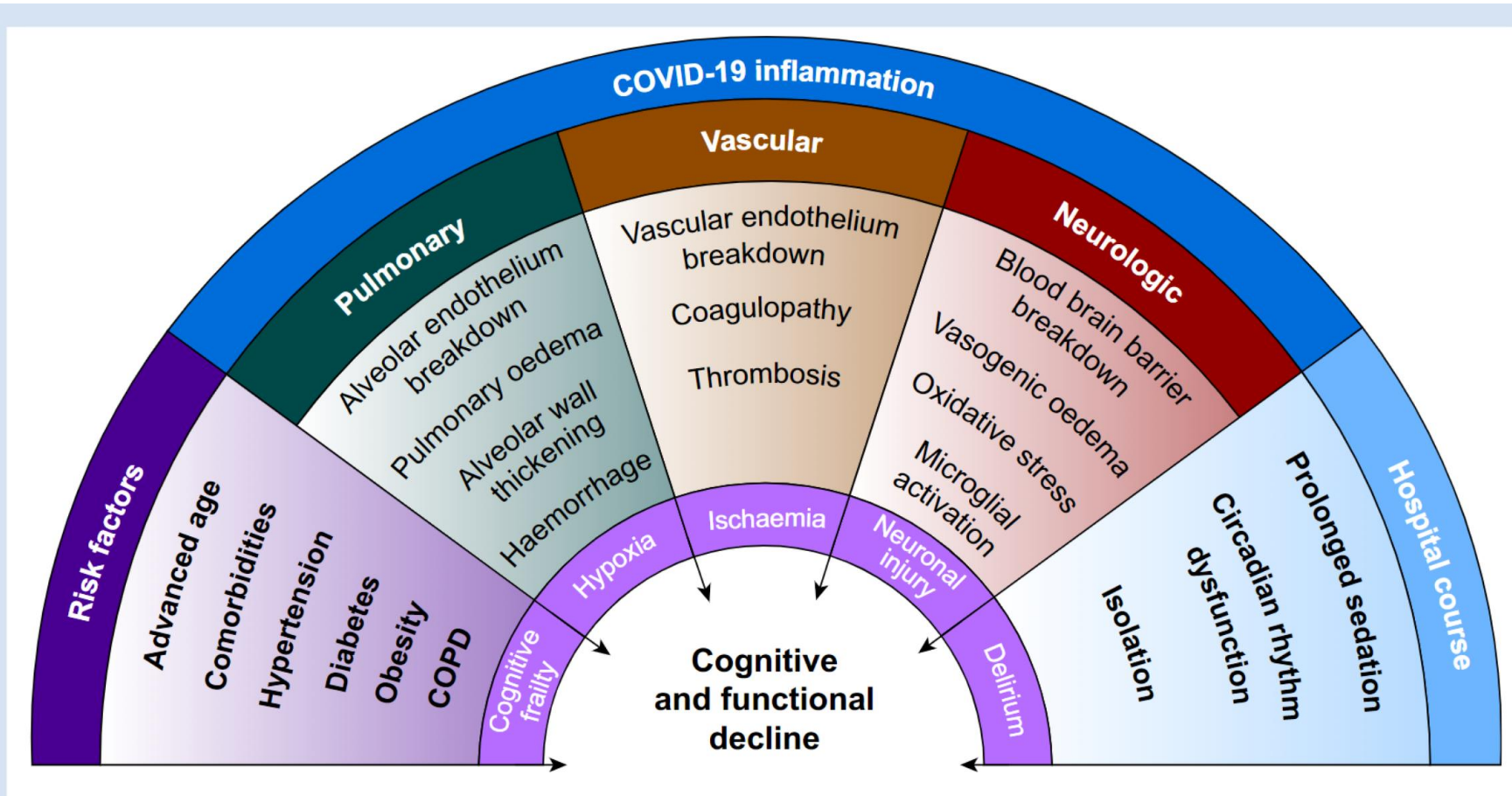


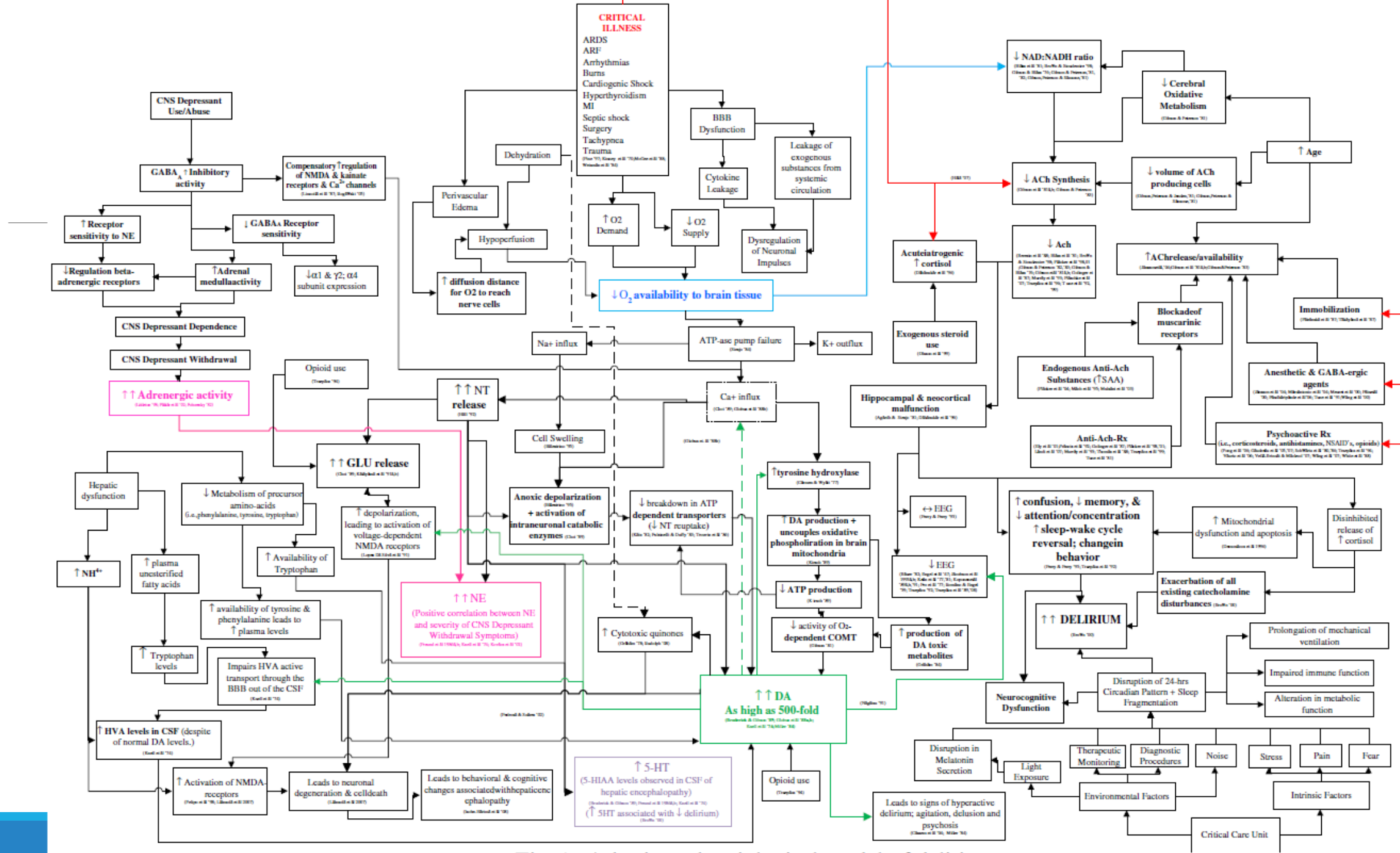
Fig 1. Wheel of factors contributing to long-term cognitive and functional decline in COVID-19 survivors. COPD, chronic obstructive pulmonary disease; COVID-19, coronavirus disease 2019.

COVID-19 and Delirium

- COVID-19 and delirium commonly co-exist and can be challenging to manage
 - Barriers to non-pharmacological management / basic communication
 - Balancing the risks of chemical restraint and respiratory depression
- KEY POINTS:
 - 1) Consider a **RAPID swab** in patients with acute behavioural disturbance who are unable to maintain COVID-19 isolation precautions.
 - 2) Patients with cog impairment who are unable to maintain isolation precautions must be reviewed urgently
 - 3) Prevention should be prioritised and chemical restraint is a last resort
 - 4) Safe prescription of chemical restraint.

Untangling the pathophysiology of delirium

- Delirium is one of the most complicated condition to unravel.
- Multifactorial
- There are ≥ 1 underlying medical conditions
- Multiple predisposing factors
- Inaccessibility of the CNS



Pathophysiology

Neurotransmitter dysregulation

- Cholinergic deficiency / dopamine excess / GABA agonists

Functional dysconnectivity

- Default mode network abnormalities

Inflammatory hypothesis – cytokines IL-1 β , IL-6, TNF α , IFN

- Altered BBB permeability to cytokines with ageing, diabetes, dementia

Aberrant stress response / excess cortisol

Abnormal melatonin secretion

Cerebral hypometabolism

- Reduced cerebral blood flow seen on SPECT scans
- Reduced cerebral glucose metabolism seen on FDG-PET scans

