

Delirium Versus Dementia Versus Psychosis

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THE STORY IN ONE SENTENCE

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“All three can look confused and strange, but delirium flips on fast (hours to days), dementia builds slowly (months to years), and psychosis messes with what feels real without wrecking attention first.”

Normal anatomy — the body's job

Your brain uses a careful balance of chemical messengers (neurotransmitters) to keep attention sharp, memory working, emotions steady, and your sense of what's real accurate. The prefrontal cortex (the front part of your brain, right behind your forehead) acts like a control tower — it filters distractions, holds focus, and checks if what you're seeing or thinking makes sense.

BODY'S JOB

Acetylcholine (a neurotransmitter made deep in your brain) helps you pay attention and form memories. Dopamine (made in the substantia nigra and ventral tegmental area, deep brain centers) controls motivation, pleasure, and helps you tell real from not real. Serotonin (made in the brainstem) steadies mood. GABA (the brain's main calming signal) quiets overexcited brain cells. Glutamate (the main wake-up signal) fires up brain cells so they can talk to each other. When all these chemicals stay in balance, you think clearly, remember yesterday, feel calm, and know what's happening around you is real.

1. The prefrontal cortex (the front of your brain, right behind your forehead) is the boss — it keeps attention on track and checks if thoughts and sights make sense.

2. Acetylcholine flows from the nucleus basalis of Meynert (a small cluster at the base of your brain) up to the cortex — it's like the 'on' switch for attention and memory.
3. Dopamine is made in the substantia nigra and ventral tegmental area (deep brain centers in the midbrain) and travels to the prefrontal cortex and limbic system (your emotional brain) — it controls motivation and helps you judge what's real.
4. The hippocampus (a seahorse-shaped structure deep inside each side of your brain) files new memories and pulls up old ones.
5. The thalamus (a relay station in the center of your brain) decides which signals — sights, sounds, feelings — get through to your conscious brain.
6. When all these areas get the right amount of each chemical, you stay alert, remember clearly, feel steady, and see the world as it really is.

What breaks

In delirium, something sudden — infection, new medicine, dehydration, low oxygen, pain, constipation, or alcohol withdrawal — throws the brain's neurotransmitter balance way off. Acetylcholine (the attention messenger) drops and dopamine (the motivation and reality messenger) spikes, so the prefrontal cortex (your brain's control tower) goes offline and attention breaks. In dementia, brain cells die slowly — from Alzheimer's plaques (clumps of beta-amyloid protein), vascular damage (tiny strokes from high blood pressure or diabetes), Lewy bodies (clumps of alpha-synuclein protein), or frontotemporal shrinkage (neurons in the front and side of the brain dying off). Memory and thinking fade permanently, but attention stays okay early on. In psychosis, dopamine floods the mesolimbic pathway (the emotional and reward circuits deep in your brain) and the prefrontal cortex can't check if thoughts and perceptions match reality, so the person sees, hears, or believes things that aren't real — but attention and memory can still work fine at first.

CLINICAL TERM

Delirium: acute disruption of cholinergic neurotransmission — often from anticholinergic medications, infection-driven cytokine release, or metabolic derangement — plus relative dopaminergic excess impairs prefrontal cortex function, causing fluctuating inattention and disorganized thinking (DSM-5-TR). Dementia: progressive neuronal loss — beta-amyloid plaques and tau neurofibrillary tangles in Alzheimer disease, microvascular starved blood flow (ischemia) in vascular dementia, alpha-synuclein Lewy bodies in Lewy body dementia, or frontotemporal lobar degeneration — produces irreversible cognitive decline over months to years without acute fluctuation. Psychosis: mesolimbic dopamine pathway hyperactivity and impaired dorsolateral prefrontal cortex reality monitoring generate hallucinations (false perceptions without external stimulus) and delusions (fixed false beliefs resistant to contradictory evidence) without primary attention deficit, seen in schizophrenia spectrum disorders, bipolar disorder with psychotic features, major depressive disorder with psychotic features, or substance-induced psychotic disorder.

How the body compensates

The dangerous moment is when the person looks okay because the body or the family is working overtime to hide the problem — but underneath, the brain is losing the fight. In delirium, brief clear moments (lucid intervals) make you think it's not serious, but the waxing-and-waning confusion itself IS the emergency. In dementia, the person covers memory gaps with routines, confabulation (making up answers without knowing they're wrong), or relying on a caregiver, so decline hides until a crisis — hospitalization, caregiver illness, a fall — strips away the scaffolding and everything collapses. In psychosis, the person may not tell you about the voices or beliefs because they don't trust you, fear you'll lock them up, or don't realize it's not real (anosognosia), so risk builds silently until self-harm or harm to others happens.

1. {'label': 'Lucid intervals in delirium', 'what': "The person seems perfectly clear and normal for an hour or two — answers questions, recognizes family, jokes around — then confusion floods back. Family says 'They were fine this morning,' so the team underestimates severity.", 'danger': "Delirium waxes and wanes by definition (DSM-5-TR). The clear moments do NOT mean it's mild or going away. Inattention persists even when the person looks calm — they still can't hold focus or follow a conversation thread. Miss the diagnosis and mortality doubles: in-hospital mortality jumps from 5% without delirium to 10% with it, and 1-year mortality climbs to 40% (JAMA Internal Medicine 2015, Lancet 2014). Always screen with a validated tool (Confusion Assessment Method, CAM) even during lucid periods."}

2. {'label': 'Sundowning hides severity during day shift', 'what': 'Delirium or dementia worsens at night (sundowning) — agitation, confusion, hallucinations, wandering spike after dark. During the day, structure, light, and routine keep the person looking calm and oriented.', 'danger': 'Day-shift providers see a cooperative patient; night nurses see chaos and call for restraints or sedation. If you only assess in the morning, you miss the diagnosis. Always ask nursing staff about overnight behavior and review hourly rounding notes. Use CAM at multiple time points, especially evening.'}
3. {'label': 'Routine and caregiver compensation hide dementia', 'what': "The person follows the same daily path — coffee at 7, shower at 8, TV in the same chair, lunch at noon — so memory loss doesn't show. The spouse compensates invisibly: lays out clothes, manages all medications, prompts every step, answers questions for the patient. To outsiders, function looks fine.", 'danger': "A fall, infection, hospitalization, or caregiver burnout breaks the routine and suddenly 'Mom can't do anything — she's confused, won't eat, doesn't know where she is.' The dementia was always there; the support system was hiding it. Screen with Mini-Mental State Exam (MMSE), Montreal Cognitive Assessment (MoCA), or Mini-Cog (3-word recall plus clock draw) in primary care annually for adults over 65 with concerns (USPSTF 2020 recommends screening when concern is raised, not universal screening). Always get collateral history from a reliable informant."}
4. {'label': 'Confabulation fills memory gaps automatically', 'what': "When you ask 'What did you eat for breakfast?' the person with dementia makes up a plausible answer — 'Eggs and toast' — without knowing it's wrong. This is confabulation: the brain fills blanks automatically to avoid the discomfort of not knowing.", 'danger': "Confabulation is NOT lying or malingering. The person genuinely believes the answer. If you accept it at face value, you'll think memory is intact. Always verify history with a reliable informant (family, caregiver, home health aide) and test memory objectively: three-word recall at 5 minutes, clock draw, serial 7s, or spell 'WORLD' backwards."}
5. {'label': 'Psychosis hidden by mistrust, paranoia, or anosognosia', 'what': "The person hears voices telling them 'Don't trust the doctor, they're poisoning you' or believes 'The FBI is watching me through the TV,' so they hide symptoms to protect themselves. Or they lack insight (anosognosia — the brain's ability to recognize its own illness is broken) and genuinely don't know the experiences are not real, so they never mention them.", 'danger': "You won't find psychosis unless you ask directly and nonjudgmentally: 'Have you been hearing or seeing things that other people don't seem to hear or see?' 'Do you ever feel unsafe, like someone is trying to hurt you or control your thoughts?' Risk of self-harm climbs when command hallucinations tell the person to jump, cut, or hurt themselves. Risk of harm to others climbs with paranoid delusions (believing someone is a threat). If psychosis is present, assess suicide and homicide risk immediately: 'Have the voices told you to hurt yourself or someone else?' 'Do you have a plan?' If yes, psychiatric emergency evaluation (voluntary or involuntary) is required."}

6. {'label': "Antipsychotic masking in dementia — the drug calms behavior but doesn't treat the cause and raises mortality", 'what': 'A person with dementia gets agitated — yelling, hitting, resisting care. The team gives an antipsychotic (haloperidol, quetiapine, risperidone). Behavior calms, so everyone thinks the problem is solved.', 'danger': "Antipsychotics in dementia carry a black-box FDA warning: 1.6–1.7× increased risk of death from stroke, pneumonia, and sudden cardiac death (Cochrane 2006, NEJM 2005). The drug suppresses symptoms but does NOT treat the cause — which is usually pain (can't tell you it hurts), constipation (impacted stool causes agitation), infection (urinary tract infection, pneumonia), fear (unfamiliar place, unfamiliar faces), or unmet need (hungry, cold, need the bathroom). ALWAYS try nonpharmacologic interventions first: treat pain, check for infection and constipation, reorient gently, play familiar music, ensure glasses and hearing aids are in place, involve family, reduce noise and chaos. If antipsychotic is necessary for severe agitation that threatens safety and nondrug interventions have failed, use the lowest dose (e.g. quetiapine 12.5–25 mg) for the shortest time and document why alternatives failed. Taper and stop as soon as possible."}
7. {'label': "Hypoactive delirium dismissed as 'just tired' or 'sleeping it off'", 'what': "The person is quiet, sleepy, withdrawn, staring into space, not bothering anyone — so the team leaves them alone or assumes they're just tired from illness. This is hypoactive delirium (low motor activity, lethargic, not agitated), and it's the MOST MISSED subtype.", 'danger': "Hypoactive delirium has WORSE outcomes than hyperactive (agitated) delirium — higher mortality, longer ICU and hospital stays, higher rate of discharge to nursing home — because it's recognized late or not at all (Critical Care Medicine 2004, Intensive Care Medicine 2001). Up to 80% of hypoactive delirium cases are missed by clinical impression alone. ALWAYS use CAM or CAM-ICU screening on every patient with altered mental status, even if they look calm. Hypoactive delirium is still an emergency and needs the same workup: find and treat the trigger (infection, meds, metabolic, low oxygen (hypoxia))."}

Domino effect

1. Step 1: The brain's attention and reality-testing circuits go offline — In delirium, a sudden stressor — infection (urinary tract infection, pneumonia), anticholinergic drug (diphenhydramine, oxybutynin), dehydration, low oxygen (hypoxia), metabolic crash (low sodium, high ammonia) — floods the brain with inflammatory cytokines or blocks acetylcholine receptors. The prefrontal cortex (your brain's control tower, right behind your forehead) can't filter distractions or hold focus, so attention shatters in hours to days. In dementia, neurons in the hippocampus (the memory filer) and cortex (the thinking engine) die slowly from Alzheimer's plaques, vascular damage, Lewy bodies, or frontotemporal shrinkage over months to years. Memory and reasoning fade, but attention holds up early because the attentional networks (prefrontal cortex, thalamus, parietal lobes) are still intact. In psychosis, dopamine floods the mesolimbic pathway (the emotional and reward circuits deep in your brain) and the dorsolateral prefrontal cortex (the front part that checks if thoughts match reality) can't keep up. Hallucinations and delusions feel absolutely real to the person because the reality-testing circuit is broken, but attention and memory can still work fine at first.
2. Step 2: You see confusion, but the TYPE of confusion tells you which one it is — Delirium: attention jumps around — lucid one moment, totally lost the next (waxing and waning). The person can't focus on your questions, loses the thread mid-sentence, speech is disorganized and rambling, and confusion gets worse at night (sundowning). Onset is sudden: hours to days. Dementia: memory fades steadily — forgets recent events (what they ate, where they put keys), repeats the same question five times, gets lost in familiar places, can't manage bills — but attention is okay early on (can follow a conversation, stay on topic) and there's no sudden flip in and out. Onset is slow: months to years. Psychosis: attention and memory CAN BE FINE, but the person hears voices (often commenting on them or giving commands), believes someone is spying on them or trying to harm them (paranoid delusions), or sees things that aren't there — and they can't tell it's not real. Onset can be sudden (drug-induced, manic episode) or gradual (schizophrenia developing over months). The key: if attention is broken and flipping on and off, think delirium first. If memory is the main problem and it's been building slowly, think dementia. If reality testing is broken but attention is intact, think psychosis.

3. Step 3: The motor behavior split shows you the delirium subtype — and the quiet ones are the most dangerous to miss — Delirium has three motor subtypes. Hyperactive: agitated, restless, pulling at IV lines and tubes, yelling, trying to get out of bed — gets noticed immediately because it disrupts care. Hypoactive: quiet, sleepy, withdrawn, staring into space, barely responds — MISSED 60–80% of the time because the person looks 'calm' or 'just tired,' but this subtype has WORSE mortality and longer ICU stays than hyperactive delirium (Critical Care Medicine 2004, Intensive Care Medicine 2001). Mixed: flips between hyperactive and hypoactive hour by hour. The waxing-and-waning itself — not the motor behavior — is the hallmark of delirium. Dementia and psychosis do NOT wax and wane by the hour like delirium; motor and cognitive patterns stay steady UNLESS delirium is layered on top (very common: up to 50% of hospitalized dementia patients develop superimposed delirium). Always use CAM screening on every confused patient, especially the quiet ones.
4. Step 4: Reversibility separates delirium from dementia; insight separates psychosis from both — If you find and fix the trigger fast — treat the urinary tract infection or pneumonia with antibiotics, stop the anticholinergic or benzodiazepine, give IV fluids to correct dehydration, give oxygen to fix low oxygen (hypoxia), correct the sodium or glucose — delirium resolves in days to weeks and the brain goes back to baseline (though subtle cognitive deficits can linger for months, especially in older adults). Dementia does NOT reverse because the neurons are dead. Memory and thinking loss is permanent. You CAN slow progression: cholinesterase inhibitors (donepezil, rivastigmine, galantamine) modestly help symptoms in Alzheimer disease; controlling blood pressure, diabetes, and cholesterol slows vascular dementia. Psychosis can resolve with treatment — antipsychotics (risperidone, olanzapine, aripiprazole), mood stabilizers (lithium, valproate), stopping the substance that caused it — but the person often has NO INSIGHT while it's happening (anosognosia). They don't realize the voices or beliefs are not real, so they resist help, refuse medication, or hide symptoms. Delirium and dementia patients usually KNOW something is wrong (they feel confused, scared, lost), but psychosis patients often do not — the false perception or belief is 100% real to them.

5. Step 5: Life stress and social factors amplify every one of these — and ignoring them guarantees relapse or decline — Isolation, poverty, chronic stress, sleep deprivation, homelessness, food insecurity, and trauma all raise cortisol (the body's main stress hormone) and inflammatory cytokines (immune signaling proteins), which poison the same prefrontal and hippocampal circuits that delirium, dementia, and psychosis attack. An older adult living alone with no social support, poor nutrition, and untreated hearing or vision loss has much higher delirium risk during hospitalization (Lancet 2014). A person with schizophrenia who faces housing instability, medication access barriers (no insurance, can't afford it, pharmacy deserts), or community stigma (can't get a job, family rejects them) will relapse no matter how good the antipsychotic is. A dementia patient whose caregiver is burned out (depression, no respite, financial strain) will decline faster because the caregiver can't provide the structure and supervision the patient needs. Evidence-based care means treating the medical problem AND addressing the social determinants of health. Connect patients to case management, home health, visiting nurse, Meals on Wheels, adult day programs, respite care for caregivers, community mental health services, housing assistance, and peer support. If you don't, the same crisis will come back.
6. Step 6: Know the dangerous medication traps and avoid them — polypharmacy, anticholinergics, benzodiazepines, and antipsychotics in dementia — Anticholinergics (diphenhydramine, oxybutynin, promethazine, tricyclic antidepressants like amitriptyline) block acetylcholine receptors and CAUSE delirium, especially in older adults. Check every medication against the Beers Criteria (American Geriatrics Society 2023: list of medications to avoid in older adults) and stop or swap them (use loratadine instead of diphenhydramine, mirabegron instead of oxybutynin). Benzodiazepines (lorazepam, diazepam, alprazolam) in older adults cause delirium, falls, hip fractures, and paradoxical agitation — avoid except for alcohol withdrawal (use symptom-triggered lorazepam protocol) or active seizures. Antipsychotics in dementia patients carry a black-box FDA warning: 1.6× increased mortality from stroke, pneumonia, and sudden cardiac death (Cochrane 2006, NEJM 2005). Reserve for severe agitation that threatens immediate safety and has failed all nondrug interventions. Use the lowest dose (quetiapine 12.5–25 mg, risperidone 0.25–0.5 mg), monitor closely, taper and stop as soon as possible. Polypharmacy (taking 5 or more medications) is an independent delirium risk factor — each added drug raises risk exponentially. Deprescribe ruthlessly in older adults using evidence-based tools (STOPP/START criteria, Beers Criteria) (JAMA 2020).

- 7. Step 7: Prevent delirium before it starts** — because once it's here, outcomes are worse even if you treat it — Delirium prevention works. Multicomponent nonpharmacologic programs like the Hospital Elder Life Program (HELP) cut delirium incidence by 30–40% (NEJM 1999, Cochrane 2016). Components: reorientation (calendar, clock, tell the person where they are and what day it is every interaction), sleep hygiene (quiet at night, no vital signs at 2 AM unless critical, lights off), early mobility (get out of bed on day 1, walk three times daily), hydration and nutrition (oral fluids, remove IV when possible), vision and hearing aids in place, avoid Foley catheters (cause infection and tethering), avoid physical restraints (worsen delirium and cause injury — JAMA 2003). Once delirium occurs, in-hospital mortality doubles (5% → 10%), ICU length of stay extends, hospital length of stay extends, discharge-to-nursing-home rate triples, and 1-year mortality climbs to 35–40% (JAMA Internal Medicine 2015, Lancet 2014). Even after delirium resolves, many patients never return to cognitive baseline — persistent delirium-related cognitive impairment is common. Bottom line: preventing delirium is MORE effective and safer than treating it. Every hospital should have a delirium prevention bundle and screen every at-risk patient (age ≥65, preexisting dementia, severe illness, hip fracture, ICU admission) daily with CAM or CAM-ICU.
- 8. Step 8: None of this is random or a 'character flaw'** — it's the brain defending itself under overwhelming stress, and compassion is part of the treatment — The person with delirium is terrified. They don't know where they are, can't recognize faces, time collapses (morning feels like night, yesterday feels like now), and they may believe they're being attacked or held prisoner. The person with dementia is losing the self they've known for 70 years — can't remember their children's names, can't find the bathroom in their own home, can't understand why simple tasks suddenly feel impossible. The person with psychosis believes the voices and the threats are real — 'Why won't anyone help me? Why doesn't anyone believe the government is watching me?' — and often feels abandoned, terrified, and deeply alone. Dismissing these experiences as 'crazy,' restraining without trying alternatives first, using sedation as a convenience ('they're bothering the nurses'), or lecturing the family for 'not watching them better' adds trauma on top of illness and violates the principle of nonmaleficence (first, do no harm). Compassionate communication is an evidence-based intervention: 'I can see you're scared. You're safe here. We're going to help you.' Speak slowly, use short sentences, don't argue with delusions (it won't work and breaks trust — instead redirect gently: 'I don't see that, but I can see you're upset. Let's sit together.'). Involve family as partners in care (they know the person's baseline, routines, what calms them). Never use physical restraints unless there is imminent risk of harm to self or others AND all other interventions have failed — restraints worsen delirium, cause skin breakdown, strangulation, and death (JAMA 2003). Treat the person, the biology, and the social context together — or you treat none of them. This is whole-person care, and it saves lives.

Symptoms · what the patient feels, and why

- **Delirium:** confusion that starts suddenly (over hours or a few days): Something acute hits the brain — an infection, not enough oxygen, a drug, or a chemical imbalance (like low sodium or high calcium) — and scrambles the signals that keep you alert and focused. The reticular activating system (the deep brainstem network that keeps you awake and aware) and the prefrontal cortex (the front of the brain that organizes attention) both stop working smoothly, so awareness and attention swing wildly from hour to hour.
- **Delirium:** attention jumps around; the person cannot focus or follow a conversation: The prefrontal cortex and the parietal lobes (the parts of the brain that filter distractions and hold focus) are temporarily disrupted by the acute illness, so thoughts scatter and the person loses track mid-sentence or forgets what you just said.
- **Dementia:** memory and thinking decline slowly (over months to years): Brain cells die or the connections (synapses) between them break down gradually — from sticky amyloid plaques and twisted tau tangles in Alzheimer disease, from tiny strokes blocking blood vessels, or from other slow damage — so learning new things and recalling old ones gets harder and harder. The hippocampus (the brain's memory recorder) and the cortex (the outer layer that handles thinking) shrink over time.
- **Dementia:** the person stays alert and aware of their surroundings (until very late stages): The reticular activating system deep in the brainstem stays healthy early on, so the person is awake and present even though the hippocampus and cortex are losing memory and reasoning power. Arousal and awareness are separate from memory and thinking until the disease is far advanced.
- **Psychosis:** seeing, hearing, or believing things that are not real (hallucinations or delusions): Dopamine (a brain messenger that helps control perception and reward) becomes overactive in the mesolimbic pathway (the deep brain circuit that processes what is real versus imagined), so the brain generates false sights, sounds, or fixed false beliefs that feel absolutely real to the person. In schizophrenia this is chronic; in brief psychotic disorder or drug-induced psychosis it is temporary.
- **Psychosis:** thoughts may be disorganized or speech may jump from topic to topic without a clear link: The prefrontal cortex and the connections between the frontal lobes and other brain regions malfunction, so the circuits that organize and link ideas stop working smoothly. Sentences come out jumbled, or the person loses the thread of their own story and leaps to unrelated topics.
- **Delirium:** the level of alertness swings — sometimes drowsy, sometimes agitated — within the same day: The reticular activating system becomes unstable because of the acute insult (infection, toxin, low oxygen), so wakefulness and calm flip unpredictably hour by hour. You may see the person sleepy and hard to wake in the morning, then restless and combative by evening (sundowning).

- **Dementia:** personality or behavior changes (apathy, irritability, losing social manners) develop over time: The frontal lobes — which control judgment, planning, and emotional regulation — shrink or lose synapses, so the person may act impulsively, withdraw, ignore social rules they used to follow, or show flat emotions. This is especially prominent in frontotemporal dementia but happens in Alzheimer disease too as the disease spreads.
- **Delirium:** sleep-wake cycle is reversed or completely disorganized: The suprachiasmatic nucleus (the tiny brain clock in the hypothalamus that sets day-night rhythm) and the reticular activating system are thrown off by the acute illness, so the person may be wide awake and agitated at 2 a.m. and asleep all afternoon.
- **Psychosis:** lack of insight (the person does not recognize that their perceptions or beliefs are false): The prefrontal cortex — which normally monitors and corrects errors in thinking — is not working properly, so the person cannot step back and question whether the voices or beliefs are real. This is called anosognosia and is common in schizophrenia and severe mania.

Focused exam

- **Attention test:** the person cannot hold focus on one thing—they jump between tasks, freeze mid-sentence, or cannot follow simple instructions like spelling 'world' backward: Delirium breaks the prefrontal cortex (the front of the brain that controls focus). This is the core difference from dementia (where attention is often fine early on) and psychosis (where attention may wander but is not broken).
- **CAM positive:** sudden confusion that started within hours to days AND inability to pay attention AND either jumbled thinking (cannot follow a conversation or answer simple questions) OR altered alertness (too sleepy or too wired): The CAM (Confusion Assessment Method) is the validated bedside tool to catch delirium quickly. If it is positive, delirium is very likely present and you must find the medical cause right away.
- **Behavior flips fast:** hypoactive (sleepy, slow, quiet), hyperactive (restless, agitated, yelling), or mixed—sometimes all three in one hour: Dementia stays steady over weeks. Psychosis stays weird in the same way. Delirium bounces around minute to minute because the reticular activating system (the brain's wake-up circuit in the brainstem) is flickering on and off.
- **Vital signs are abnormal:** fever above 38°C (100.4°F), heart rate over 100, oxygen saturation below 90%, or blood pressure swinging high and low: Delirium almost always has a medical cause underneath—infection, drugs, low oxygen, low blood sugar, or organ failure. Vital signs point you straight to the cause.
- **Timeline:** the person was fine yesterday, confused today: Delirium is acute—hours to days. Dementia creeps in over months to years. Psychosis builds over weeks. The speed tells you what you are dealing with.

- Sleep-wake cycle is completely flipped or absent: sleeping all day, awake all night, or no pattern at all: The suprachiasmatic nucleus (the brain's internal clock in the hypothalamus) controls sleep. When delirium breaks attention, sleep always goes haywire. This is nearly always present in delirium and rare in dementia or psychosis.

Diagnostics · every test answers a question

- Complete blood count (CBC) with differential → Is there infection (high white blood cells) or anemia (low red blood cells, which starves the brain of oxygen)?
- Basic metabolic panel (BMP): sodium, potassium, glucose, creatinine, blood urea nitrogen (BUN) → Are sodium, sugar, or kidney function too high or too low—all of which poison the brain and cause delirium?
- Urinalysis (UA) and urine culture → Is there a urinary tract infection (UTI), which is one of the most common causes of delirium in older adults?
- Chest X-ray → Is there pneumonia (lung infection) or heart failure (fluid in the lungs), both of which lower oxygen delivery to the brain?
- Medication review and urine drug screen → Are sedatives, opioids, anticholinergics (like diphenhydramine or oxybutynin), or withdrawal causing delirium?
- Thyroid-stimulating hormone (TSH) and vitamin B12 level → Is the thyroid too slow (hypothyroidism) or is B12 too low, both of which starve brain cells and mimic delirium or dementia?
- Head CT without contrast (or MRI if CT is unclear) → Is there a stroke, bleeding in the brain, or a mass pushing on brain tissue—especially after a fall, new weakness, or seizure?
- Electroencephalogram (EEG) if nonconvulsive status epilepticus (a seizure that will not stop) suspected (delirium that does not improve with usual treatment or rhythmic twitching of face or hands) → Is the brain having a silent seizure that looks like delirium but is actually continuous electrical misfiring without visible shaking?

Treatment logic

- Identify and treat the medical cause: infection (antibiotics), medication side effect (stop the drug), low oxygen (oxygen or treat pneumonia or heart failure), low or high blood sugar (insulin or glucose), organ failure (dialysis for kidney failure, lactulose or rifaximin for liver failure) → Delirium is caused by a medical stress that breaks the prefrontal cortex (the brain's focus center) and floods the brain with inflammatory signals and stress hormones like cortisol. Fix the cause—like clearing an infection or raising oxygen—and the attention circuit usually starts working again within hours to days.

- Reorient the person repeatedly: use their name, tell them the date and place, correct false beliefs gently, keep family or familiar people nearby, and use familiar objects like photos or glasses → The hippocampus (the brain's memory recorder) and prefrontal cortex calm down when you give them clear, repeated signals about what is real. This reduces confusion, fear, and scary false beliefs (like thinking someone is trying to hurt them).
- Avoid physical restraints; use one-to-one monitoring (a person stays with them at all times), soft lighting, a quiet room, a normal sleep schedule (wake them during the day, darken the room at night), and get them out of bed and walking as soon as safe → A calm, predictable environment lowers cortisol (the body's main stress hormone) and helps the prefrontal cortex settle down naturally. Restraints cause trauma, worsen agitation, and prolong delirium.
- Stop or reduce anticholinergic drugs (diphenhydramine, oxybutynin, promethazine, scopolamine), benzodiazepines (unless alcohol or benzodiazepine withdrawal), and opioids if possible → Anticholinergics block acetylcholine (the brain messenger for attention and memory) and directly cause delirium. Benzodiazepines boost GABA (the brain's main calming signal) too much and slow the brain down. Opioids slow breathing and lower oxygen. Stopping them clears the fog.
- Give haloperidol 0.5–1 mg IV/IM/PO or olanzapine 2.5–5 mg PO for severe agitation, hallucinations, or safety risk only—not for routine delirium → These drugs block dopamine receptors in the limbic system (the brain's alarm and emotion center), calming extreme agitation so the person does not hurt themselves, pull out tubes, or attack staff.
- For dementia after delirium clears: start donepezil 5 mg daily (increase to 10 mg after 4–6 weeks) or memantine 5 mg daily (titrate to 10 mg twice daily over 4 weeks). Consider adding memantine to donepezil for moderate to severe Alzheimer disease. → Donepezil keeps more acetylcholine in the brain by blocking the enzyme that breaks it down. Memantine protects brain cells from too much glutamate (a brain messenger that causes overstimulation and kills cells when it floods in). These slow decline but do not reverse brain damage.
- For psychosis after delirium clears: start risperidone 0.5–1 mg daily or olanzapine 5–10 mg daily, plus cognitive-behavioral therapy (CBT) and case management to help with housing, medicine access, and regular appointments → Antipsychotics block dopamine receptors in the mesolimbic pathway (the brain's reward and perception circuit) so false beliefs and hallucinations weaken. CBT helps the person test reality, challenge scary thoughts, and cope with stress. Case management keeps them stable and prevents relapse.

Medications

Haloperidol (first-generation antipsychotic, for severe agitation in delirium when safety is at risk) · Typical (first-generation) antipsychotic - blocks dopamine D2 receptors

Haloperidol sits on dopamine D2 receptors in the brain and blocks them. Dopamine is a messenger chemical that helps brain cells talk to each other; in some circuits, it acts like a 'go' signal. When delirium makes the brain misfire and send too many 'go' signals, the person becomes restless, confused, and sometimes combative. Blocking some of those signals calms the storm so the person feels less overwhelmed. Think of it like turning down the volume on a speaker that's blasting too loud.

1. Haloperidol binds tightly to dopamine D2 receptors, sitting in the spot where dopamine would normally land.
2. With dopamine blocked, the 'go' signal in emotion and movement circuits drops.
3. The person's agitation, hallucinations, and combative behavior settle down because the overactive alarm signals are turned down.
4. The brain's emergency alarms cool off, making the patient safer so the medical team can treat the real cause of delirium—like an infection, low oxygen, or a toxic medication.

SAFETY WATCH

Can cause extrapyramidal symptoms (muscle side effects): stiffness, restlessness (feeling like you have to pace), tremor, and involuntary twisting movements. These happen because blocking dopamine in the striatum affects movement control. · May prolong the QT interval on the heart's electrical tracing (ECG), raising the risk of a dangerous heart rhythm called torsades de pointes. Check an ECG before giving haloperidol if possible, especially if the patient takes other QT-prolonging drugs or has heart disease. · Increases fall risk, especially in older adults, because of drowsiness, low blood pressure, and muscle effects. · Do NOT use in patients with Parkinson disease or Lewy body dementia—blocking dopamine will make their movement problems and confusion much worse. · Use only when the patient is a danger to themselves or others. Always pair the drug with non-drug strategies: reorient the patient gently, keep the room quiet and well-lit during the day, dim at night, have family at the bedside if possible, protect the sleep-wake cycle, and remove catheters and lines as soon as safe.

Olanzapine (second-generation antipsychotic, alternative for agitation in delirium) · Atypical (second-generation) antipsychotic - blocks dopamine D2 receptors and serotonin 5-HT2A receptors

Olanzapine blocks two types of receptors at once: dopamine D2 receptors (which calm the overactive 'go' signals) and serotonin 5-HT2A receptors (which help steady mood and perception). Serotonin is another messenger chemical in the brain; too much activity at certain serotonin receptors can twist how you see and feel things. By blocking both dopamine and serotonin, olanzapine reduces agitation and confusion with a bit less muscle stiffness than haloperidol, though it brings its own risks—mainly drowsiness, weight gain, and blood sugar problems.

1. Olanzapine binds to dopamine D2 receptors and serotonin 5-HT2A receptors, blocking both messenger chemicals from landing.
2. Blocking dopamine calms the agitation and combativeness; blocking serotonin helps reduce distorted perceptions (like hallucinations) and mood swings.
3. The patient becomes less combative, easier to redirect, and calmer, buying time for the medical team to treat the underlying problem causing the delirium.
4. Because serotonin block in the cortex helps balance dopamine block in the striatum, there is often less muscle stiffness than with haloperidol—but metabolic side effects are higher.

SAFETY WATCH

Can cause significant drowsiness, weight gain, and high blood sugar (metabolic syndrome). Monitor blood sugar and weight, especially if used for more than a few days. · May prolong the QT interval on ECG; check a heart tracing if available before starting. · Black-box warning: raises stroke and death risk in older adults with dementia. Use only when the patient is truly a danger and non-drug strategies have failed. Stop as soon as the crisis passes. · Still increases fall risk because of drowsiness and low blood pressure, though usually causes less muscle stiffness than haloperidol. · Avoid in Lewy body dementia and Parkinson disease—can still worsen confusion and movement.

Donepezil (cholinesterase inhibitor for Alzheimer dementia) · Acetylcholinesterase inhibitor

Donepezil blocks acetylcholinesterase, the enzyme that normally breaks down acetylcholine after it's used. Acetylcholine is a messenger chemical the brain uses to pass signals between nerve cells, especially in memory and attention circuits. In Alzheimer disease, nerve cells that make acetylcholine die off, so there's less of it available. By stopping the enzyme that chops up acetylcholine, donepezil lets each molecule last longer in the synapse (the tiny gap between nerve cells). More acetylcholine hanging around means signals pass more easily, so thinking

and memory work a little better—like keeping the mail carrier on the route longer so more messages get delivered.

1. Donepezil binds to acetylcholinesterase, the enzyme that normally chops up acetylcholine molecules after they've carried a message across the synapse.
2. With the enzyme blocked, acetylcholine stays in the synapse longer instead of being broken down right away.
3. The longer-lasting acetylcholine means nerve cells can send and receive memory and attention signals more reliably.
4. The person can hold thoughts a little better, recall words and names more easily, and stay focused longer—though the improvement is modest and temporary.

SAFETY WATCH

Common side effects: nausea, diarrhea, loss of appetite, stomach cramps. These happen because acetylcholine also acts on the gut, making it contract more. Taking the pill with food and starting at a low dose can help. · Can slow the heart rate (bradycardia) because acetylcholine affects the heart's pacemaker. Use with caution in patients with heart block, sick sinus syndrome, or other heart rhythm problems. Check the pulse at follow-up visits. · May cause vivid dreams, nightmares, or trouble sleeping. Giving the dose in the morning instead of at bedtime usually fixes this. · Can increase fall risk because of dizziness and slow heart rate—monitor balance and blood pressure. · Benefits plateau after a time. If there is no clear benefit after 3 to 6 months, or if side effects outweigh benefit, consider stopping the drug in a shared decision with the patient and family. There is no need to continue indefinitely if it's not helping.

Memantine (NMDA receptor antagonist for moderate to severe Alzheimer dementia) · N-methyl-D-aspartate (NMDA) receptor antagonist

Memantine partially blocks NMDA receptors, which are gates on nerve cells that open when glutamate (an excitatory messenger chemical) arrives and let calcium rush into the cell. Calcium is needed for normal signaling, but in Alzheimer disease the system goes haywire: glutamate floods the brain and opens too many gates, letting in a constant, toxic wave of calcium that damages and kills nerve cells. Memantine sits in the gate just enough to block the harmful flood but still lets normal, brief glutamate signals through. Think of it like a screen door that keeps bugs out but lets fresh air in—it filters out the bad while preserving the good.

1. Memantine lodges loosely in the NMDA receptor channel, blocking excess glutamate from opening the gate wide and letting in a flood of calcium.
2. Normal, brief glutamate signals (the kind used for everyday learning and memory) still get through, because memantine blocks weakly and pops off quickly when a strong, useful signal arrives.

3. The constant, low-level toxic flood of glutamate is reduced, so nerve cells suffer less calcium damage and die more slowly.
4. The person's memory and daily function decline more slowly for a time, though the disease still progresses.

SAFETY WATCH

Generally well tolerated. Side effects are usually mild: dizziness, headache, constipation, and sometimes confusion. · Rarely causes new confusion or hallucinations. If these appear after starting memantine, consider stopping the drug—it may not be helping. · Can be combined safely with a cholinesterase inhibitor like donepezil in moderate to severe Alzheimer disease. The two drugs work on completely different pathways, so using both may give a bit more benefit than either alone. · Not helpful and not recommended in non-Alzheimer dementias (vascular dementia, Lewy body dementia, frontotemporal dementia) unless there is a clear Alzheimer component confirmed by testing or clinical picture. · No need for blood tests or ECG monitoring—the drug is cleared by the kidneys, so dose may need adjustment if kidney function is low.

Antipsychotics (for primary psychotic disorders: schizophrenia, schizoaffective disorder, severe psychotic depression or mania) · First-generation (typical) and second-generation (atypical) antipsychotics - block dopamine D2 receptors; many second-generation drugs also block serotonin 5-HT_{2A} receptors

Antipsychotics reduce psychosis—hallucinations (seeing or hearing things that aren't there), delusions (false, fixed beliefs), and disorganized thinking—by blocking dopamine D₂ receptors in the mesolimbic pathway. The mesolimbic pathway is a deep brain circuit that handles emotion, reward, and motivation. When this circuit becomes overactive and pumps out too much dopamine, it creates false perceptions and beliefs: voices that aren't real, ideas that don't match reality, thoughts that jump and scatter. Think of dopamine as an amplifier turned up too loud in that circuit; the antipsychotic turns the volume down so the person can tell what's real again. Second-generation drugs also block serotonin 5-HT_{2A} receptors, which may help mood and cause fewer movement side effects, though they bring higher risk of weight gain and metabolic problems.

1. The antipsychotic binds to dopamine D₂ receptors in the mesolimbic pathway and blocks them, preventing dopamine from landing and sending its signal.
2. With less dopamine signaling in this overactive circuit, the 'volume' of false perceptions drops: the person hears fewer voices, holds fewer false beliefs, and can follow a train of thought more easily.

3. In second-generation drugs, blocking serotonin 5-HT_{2A} receptors may improve mood, reduce anxiety, and partly balance the dopamine block in the striatum so there is less muscle stiffness.
4. The brain's reality-testing gradually improves as the overactive alarm and reward circuits settle, and the person can engage more clearly with the world, therapy, and daily life.

SAFETY WATCH

First-generation antipsychotics (haloperidol, fluphenazine, perphenazine, chlorpromazine): higher risk of extrapyramidal symptoms—stiffness, shuffling walk, tremor, restlessness (akathisia), and tardive dyskinesia (involuntary movements of the mouth, tongue, or limbs that can become permanent even after stopping the drug). These happen because blocking dopamine in the striatum disrupts movement control. · Second-generation antipsychotics (risperidone, olanzapine, quetiapine, aripiprazole, lurasidone, ziprasidone, paliperidone, asenapine, brexpiprazole, cariprazine, clozapine): less muscle stiffness, but higher risk of weight gain, high blood sugar, diabetes, high cholesterol, and drowsiness (metabolic syndrome). Each drug in this class has a slightly different side-effect profile—choice depends on the individual's history, other medical problems, and what side effects they can tolerate. Clozapine is the most effective for treatment-resistant schizophrenia but requires weekly or biweekly blood tests because it can drop white blood cell counts dangerously. · All antipsychotics can prolong the QT interval on ECG, raising the risk of a dangerous heart rhythm. Check an ECG at baseline and recheck if the dose goes up or other QT-prolonging drugs are added. · Black-box warning for all antipsychotics: in older adults with dementia-related psychosis, these drugs increase the risk of stroke and death. Use them only for severe, dangerous agitation or psychosis after non-drug strategies fail, at the lowest dose, for the shortest time, with close monitoring and a plan to stop as soon as safe. · All antipsychotics raise the risk of falls, especially in older adults, because of drowsiness, low blood pressure, and movement effects. · Long-acting injectable (LAI) forms are available for many antipsychotics (risperidone, paliperidone, aripiprazole, haloperidol, fluphenazine, olanzapine) and greatly improve adherence in people with chronic psychotic disorders who have trouble remembering daily pills or lack insight into their illness. · Medication alone is never enough. Treatment must be paired with coordinated specialty care: assertive community treatment or case management, individual therapy (especially cognitive-behavioral therapy for psychosis, which teaches the person to test and challenge distorted beliefs), family psychoeducation (helping loved ones understand the illness and support recovery), supported employment or education, stable housing support, treatment for co-occurring substance use if present, and regular medical follow-up to monitor side effects and overall health. Recovery is built on relationships, purpose, and support as much as on medication.

Red flags

- Sudden confusion with fever, low oxygen (blue lips or fingertips), very high or very low blood sugar, or a new medicine — suggests delirium and needs urgent work-up (blood tests, urine culture, chest X-ray, medication review) to find and fix the cause before the brain is permanently harmed
- Hallucinations or delusions with commands to hurt self or others, or the person cannot care for themselves safely (not eating, wandering into traffic, responding to voices) — needs immediate psychiatric evaluation and may need inpatient admission to keep the person and others safe
- Rapid memory loss over weeks (not years) with headache, seizures, fever, or new weakness on one side — may signal a brain bleed, tumor, herpes encephalitis, autoimmune encephalitis (like anti-NMDA receptor), or prion disease (Creutzfeldt-Jakob) rather than typical dementia, and requires urgent brain imaging (MRI) and lumbar puncture
- Delirium that does not improve after 72 hours of treating the cause (antibiotics for infection, stopping the offending drug, correcting electrolytes) — consider a second hidden cause, medication toxicity, or an unrecognized structural brain problem

Nursing priorities

1. Check vital signs, oxygen saturation, and fingerstick blood glucose immediately to find life-threatening causes of confusion — Fever signals infection, low oxygen (hypoxia) means the brain is not getting enough oxygen to work properly, very low blood sugar (below 70) can cause seizures and brain damage, and very high blood sugar (above 400) causes confusion and dehydration. All of these are reversible if you catch and treat them quickly.
2. Review the medication list with the provider and stop or reduce any medicines known to cause delirium—especially opioids, benzodiazepines, anticholinergics, and sleeping pills — Medicines that sedate the brain or block acetylcholine (a brain chemical that helps with attention and memory) are the most common reversible cause of delirium. Stopping the drug often clears confusion within 24 to 48 hours.
3. Reorient the patient every time you interact: use their name, tell them where they are, what day and time it is, and explain what you are doing in simple words — Delirium scrambles the brain's ability to know time, place, and situation. Repeated gentle reminders help the brain reconnect to reality and reduce fear and agitation. This also helps you tell delirium from dementia—delirium patients benefit from reorientation, dementia patients may not remember it minutes later.

4. Keep the room quiet, well-lit during the day, and dark at night; open curtains in the morning and encourage the patient to get out of bed and sit in a chair — The brain needs normal light-dark cycles to stay oriented and maintain a sleep-wake rhythm (circadian rhythm). Lying in bed all day in a dark or too-bright room worsens delirium. Sitting up also prevents pneumonia and blood clots.
5. Bring in family members, familiar objects from home like photos, and the patient's glasses and hearing aids so they can see and hear clearly — Sensory deprivation (not being able to see or hear well) makes delirium worse because the brain cannot process the environment correctly. Familiar faces and voices calm the patient and provide reality anchors. Glasses and hearing aids reconnect the brain to the world.
6. Assess for pain every shift using a pain scale, and treat pain with the lowest effective dose of non-sedating medicine like acetaminophen — Untreated pain is a common trigger for delirium—the brain becomes overloaded with stress signals. But opioids (like morphine or oxycodone) can also cause or worsen delirium, so you balance pain control with the least sedating option possible.
7. Monitor intake and output closely, encourage oral fluids if the patient can swallow safely, and give IV fluids if the patient is dehydrated — Dehydration reduces blood flow to the brain and makes confusion worse. It also signals that the patient may not be eating or drinking because of confusion, which starts a dangerous cycle. Older adult brains are especially sensitive to low fluid volume.
8. Avoid physical restraints and minimize sedating medicines; if the patient is agitated and a danger to themselves, try one-to-one observation first and use the lowest dose of an antipsychotic like haloperidol only as a last resort — Restraints and heavy sedation make delirium last longer and increase the risk of pressure injuries, blood clots, and pneumonia. The goal is to keep the patient safe while their brain heals from the underlying medical problem. Antipsychotics can help severe agitation but do not treat the cause of delirium.

Sources

- American Psychiatric Association DSM-5-TR (2022)
- American Geriatrics Society Clinical Practice Guideline for Postoperative Delirium (2015, reaffirmed 2023)
- National Institute on Aging Alzheimer's and Related Dementias Resources
- American Academy of Neurology Practice Guideline Update: Mild Cognitive Impairment (2018)

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