

Alcohol Withdrawal & Delirium Tremens

Delirium tremens is the brain's electrical storm when alcohol vanishes after weeks or months of heavy use — without treatment, 1 in 7 patients die.

THE ONE-LINE TRUTH

Delirium tremens is a brain emergency that strikes 2 to 4 days after the last drink, marked by severe confusion, seeing or hearing things that are not there, and an autonomic storm — the body's normal control over heart rate, blood pressure, and temperature collapses.

The chain — how it unfolds

1. Chronic heavy alcohol use (typically weeks to months, often above 5 drinks per day for men or 4 for women) downregulates GABA-A receptors and upregulates NMDA glutamate receptors across the cerebral cortex, limbic system (including the hippocampus, the memory-maker deep in the temporal lobe, and amygdala, the alarm center that flags danger), and brainstem. — why: Alcohol chronically enhances GABA (the brain's main inhibitory signal that opens chloride channels and calms nerve firing) and blocks glutamate at NMDA receptors (the brain's main excitatory signal that opens sodium and calcium channels); the brain adapts by reducing GABA-A receptor density on nerve cell surfaces and increasing NMDA receptor density to maintain normal nerve firing in the presence of alcohol.
2. Abrupt alcohol cessation (or sharp reduction) removes inhibition, leaving the brain in a hyperexcitable state 48 to 96 hours later. — why: When alcohol is removed suddenly, GABA activity collapses (fewer GABA-A receptors, no alcohol boost) and glutamate activity explodes (more NMDA receptors, no alcohol block), because the brain's receptors are still adapted to alcohol's presence — this creates an electrical and chemical storm throughout the cortex, limbic system, and brainstem.

3. Confusion, agitation, hallucinations (visual, tactile, or auditory), coarse tremor, profuse heavy sweating (diaphoresis) (drenching sweats), a fast heart rate (tachycardia) (heart rate often above 120 bpm), high blood pressure (hypertension) (systolic often above 160 mmHg), and fever (often above 100.4°F, sometimes above 104°F) appear. — why: The hyperexcitable brain misfires everywhere: confusion and agitation from chaos in the prefrontal cortex (the front that guides clear thought) and limbic system (emotional centers), hallucinations from sensory cortex overstimulation (visual cortex at the back producing visions of bugs or snakes, somatosensory cortex producing crawling sensations), tremor from motor cortex and cerebellar overdrive, and sweating, fast heart rate, high blood pressure, and fever from sympathetic surge (norepinephrine release from the locus coeruleus in the brainstem and medulla at the base of the brain) in response to perceived threat.
4. Electrolyte depletion (hypokalemia, low magnesium (hypomagnesemia), hypophosphatemia) and thiamine deficiency worsen brain and heart instability. — why: Chronic heavy alcohol use and poor nutrition deplete potassium (target above 4 mEq/L), magnesium (target above 2 mg/dL), and phosphate (target above 2.5 mg/dL); low potassium and magnesium destabilize cardiac cell membranes (increasing risk of torsades de pointes and ventricular fibrillation) and lower the seizure threshold, and thiamine deficiency prevents neurons (especially in the mammillary bodies and thalamus) from using glucose for energy, risking Wernicke encephalopathy (confusion, eye movement problems, unsteady walk) and permanent Korsakoff syndrome (memory loss) if untreated.
5. Rhabdomyolysis (muscle breakdown with CK often above 5,000 U/L, sometimes above 50,000), acute kidney injury (creatinine rising, urine output dropping below 0.5 mL/kg/hr), arrhythmias (atrial fibrillation, ventricular a fast heart rate (tachycardia), torsades), and seizures (generalized tonic-clonic) follow. — why: Agitation and tremor cause sustained muscle contraction, releasing myoglobin (muscle protein that clogs kidney tubules, causing acute tubular tissue death (necrosis) and AKI); electrical chaos in the motor cortex and limbic system triggers seizures; norepinephrine surge and electrolyte shifts (especially low potassium and magnesium) provoke dangerous heart rhythms.
6. Aspiration (breathing in vomit or saliva), hyperthermia (temperature above 104°F causing protein denaturation and brain damage), and cardiovascular collapse (shock, cardiac arrest) threaten life. — why: Confusion and vomiting increase the risk of aspiration, causing aspiration pneumonia, low oxygen (hypoxia) (SpO₂ below 90%), and respiratory failure; fever above 104°F denatures enzymes and can cause permanent brain damage; the heart's compensatory a fast heart rate (tachycardia) eventually exhausts, and arrhythmias or myocardial starved blood flow (ischemia) lead to shock or cardiac arrest.

7. Without treatment, status epilepticus (a seizure that will not stop) (seizures lasting more than 5 minutes or repeated without recovery) or cardiovascular collapse causes death in approximately 15% of untreated patients. — why: Uncontrolled seizures cause brain swelling (cerebral swelling (edema)), lactic acidosis (acid from oxygen-starved tissues) (pH dropping below 7.2 as cells burn sugar without oxygen), hyperthermia, and respiratory failure; arrhythmias (ventricular fibrillation, asystole), aspiration pneumonia, or shock lead to cardiac arrest — this is why untreated delirium tremens kills 1 in 7 patients.
8. Benzodiazepines (lorazepam or diazepam), thiamine, electrolyte repletion (IV potassium, magnesium, phosphate), IV fluids, and continuous monitoring reverse the domino chain and drop mortality below 5%. — why: Benzodiazepines (lorazepam 2 to 4 mg IV every 15 to 30 minutes or diazepam 5 to 10 mg IV every 5 to 15 minutes) are GABA-A receptor agonists (they boost GABA's calming effect) that restore inhibitory tone and calm the excitatory storm — titrate to light sedation (RASS 0 to -2, calm or drowsy but arousable) using symptom-triggered protocols (CIWA-Ar score guiding dose); thiamine 500 mg IV three times daily for 3 to 5 days (given before glucose to prevent Wernicke encephalopathy) restores cellular glucose metabolism in the mammillary bodies and thalamus; correct potassium above 4 mEq/L (often requiring 40 to 80 mEq over 24 hours), magnesium above 2 mg/dL (2 to 4 grams IV), and phosphate to normal (often requiring oral or IV repletion); IV fluids (normal saline or lactated Ringer's, often 4 to 10 liters in the first 24 hours) address dehydration and support kidney blood flow (perfusion); cooling blankets, ice packs, or evaporative cooling treat hyperthermia (target below 102°F); and continuous telemetry (heart rhythm monitoring), pulse oximetry (oxygen level monitoring), and frequent reassessment catch arrhythmias, respiratory failure, and worsening agitation early. This aggressive, protocol-driven care in an ICU or monitored setting drops mortality to 1 to 5%.

Spot it — what you see, and why

- Confusion and disorientation: When alcohol is stopped suddenly, the brain chemical glutamate (which excites nerve cells) surges unchecked. This flood of signals scrambles the thinking and memory centers in the prefrontal cortex (the front of the brain that handles thinking and planning) and the hippocampus (the memory hub deep in the brain), so the person cannot keep track of time, place, or what is happening around them.
- Agitation and combativeness: The amygdala (the brain's alarm center that flags danger) and the limbic system (emotion centers deep in the brain) become overactive, and the prefrontal cortex (the front of the brain that pumps the brakes on impulses) is overwhelmed. The result is extreme restlessness, irritability, and even violent outbursts.

- Visual, auditory, or tactile hallucinations: The visual cortex (the part of the brain that processes what you see), the auditory cortex (the part that processes sound), and the sensory cortex (the part that handles touch) fire too fast and create false signals. The person may see bugs crawling on the walls, hear voices that are not there, or feel things crawling on their skin—even though nothing is really there.
- Coarse tremor (shaking): The motor cortex (the part of the brain that controls movement) and the cerebellum (the balance and coordination center at the back of the brain) are overstimulated by glutamate. This causes rhythmic, jerky muscle contractions, most visible as shaking hands or arms.
- Profuse sweating: The sympathetic nervous system (the body's fight-or-flight system) goes into overdrive and floods the sweat glands with signals driven by norepinephrine (a stress messenger chemical). The person drenches their clothes as if they just ran a race, even lying still in bed.
- Fast heart rate (often over 100–120 beats per minute): A surge of norepinephrine and epinephrine (adrenaline) forces the heart to beat faster to pump blood. Dehydration from sweating and the body's overheated state also contribute. The heart races as if the person is in danger, even though they are lying down.

Red flags — act now

- Temperature above 102.2°F (39°C) or continuing to rise despite cooling measures—signals severe autonomic instability and high risk of organ failure.
- New-onset seizures or any seizure lasting longer than 5 minutes—warns of possible progression to status epilepticus (a seizure that will not stop), a brain emergency requiring immediate intervention.
- Systolic blood pressure over 180 mmHg or heart rate over 140 beats per minute despite benzodiazepines—means sedation is inadequate and the patient is at high risk for stroke, dangerous heart rhythms, or cardiovascular collapse.
- Worsening confusion or decreased responsiveness after starting treatment—may indicate a second problem such as infection, head injury, hepatic encephalopathy (liver failure causing brain toxicity), or Wernicke encephalopathy from thiamine deficiency.
- Dark (tea-colored) urine, rising creatinine in blood tests, or tender, swollen muscles—suggests rhabdomyolysis, where muscle is breaking down and releasing toxins that can shut down the kidneys.

Treat it — in this order

1. IV benzodiazepines — lorazepam 2–4 mg IV every 5–15 minutes or diazepam 5–10 mg IV every 5–15 minutes, using a symptom-triggered protocol (guided by CIWA-Ar score) until the patient is calm, tremor stops, heart rate and blood pressure normalize, and CIWA-Ar score is below 10 → Benzodiazepines plug into the GABA-A receptors (the brain's main calming switches) and turn up the 'brake' signal. That calms the wild glutamate storm, stops seizures, slows the heart, drops blood pressure, and prevents progression to the worst stages of delirium tremens. This is the single most important treatment and saves lives.
2. IV thiamine 500 mg three times daily for at least 3–5 days, started before or at the same time as any IV sugar (dextrose) → Thiamine is a helper molecule (cofactor) that the brain needs to turn glucose into energy. Without thiamine, giving sugar can push the brain over the edge into Wernicke encephalopathy (sudden confusion, trouble walking, and abnormal eye movements). If not treated immediately, this can become permanent memory loss (Korsakoff syndrome). Thiamine prevents this brain injury.
3. IV magnesium sulfate 2 g over 15–30 minutes, then 1–2 g every 6 hours to keep blood magnesium above 2 mg/dL; also replace potassium (goal above 4 mEq/L), phosphate (goal above 2.5 mg/dL), and calcium (goal ionized calcium 4.5–5.3 mg/dL) according to lab results → Magnesium stabilizes the outer shell of nerve cells and helps the pumps that move sodium and potassium in and out. Potassium sets the resting voltage of cells. Phosphate is needed to make ATP (the energy currency). Calcium keeps the heart's electrical system steady. Replacing these electrolytes lowers seizure risk and prevents dangerous heart rhythms.
4. Phenobarbital loading dose 10–15 mg/kg IV (up to 1 gram total) or propofol infusion 5–50 mcg/kg/min if benzodiazepines alone do not control the agitation, hallucinations, or seizures → Phenobarbital opens GABA-A channels even longer than benzodiazepines and has strong anti-seizure effects. Propofol boosts GABA and also blocks NMDA glutamate receptors. Both provide deeper brain suppression when the GABA receptors are so worn down that benzodiazepines are not enough. This is reserved for refractory (resistant) cases.
5. Dexmedetomidine infusion 0.2–1.5 mcg/kg/h as an adjunct to benzodiazepines in the ICU → Dexmedetomidine is an alpha-2 agonist that turns down the sympathetic 'fight or flight' system in the locus coeruleus (a cluster of cells in the brainstem that pumps out norepinephrine). It reduces heart rate, blood pressure, agitation, and delirium without suppressing breathing as much as propofol or benzodiazepines. It is useful when you need to calm the autonomic storm but want the patient to wake up for neuro checks.

Your top priorities at the bedside

1. Start continuous pulse oximetry, cardiac telemetry (heart rhythm monitoring), and automated blood pressure checks every 15 minutes until the patient is stable. — why: Delirium tremens causes an autonomic storm—the nervous system loses control over heart rate, blood pressure, and breathing; dangerous heart rhythms, blood pressure spikes above 180 mmHg, and low oxygen from aspiration or fast breathing can happen suddenly and need immediate detection.
2. Administer IV thiamine 500 mg slowly over 30 minutes before or at the same time as any IV dextrose (sugar) solution. — why: Alcoholics are thiamine-deficient; giving sugar without thiamine first pushes the brain to burn the last thiamine stores and can trigger Wernicke encephalopathy, causing permanent brain damage to memory and coordination centers.
3. Give IV benzodiazepines—lorazepam 2 to 4 mg or diazepam 5 to 10 mg—every 5 to 15 minutes using CIWA-Ar (Clinical Institute Withdrawal Assessment for Alcohol, revised) scores to guide dosing until tremor stops, heart rate drops below 100, and the patient is calm but arousable. — why: Alcohol withdrawal removes GABA (the brain's brake chemical) suddenly; benzodiazepines replace that brake and stop the runaway nerve firing that causes seizures, hallucinations, and autonomic instability; symptom-triggered dosing prevents under-treatment and life-threatening complications.