

Alcohol Withdrawal & Delirium Tremens

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THE STORY IN ONE SENTENCE

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“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.”

Normal anatomy — the body's job

GABA (the brain's main brake signal) and glutamate (the brain's main gas signal) together keep nerve cells firing at the right speed. Chronic heavy alcohol use suppresses glutamate and boosts GABA, so the brain fights back by removing some GABA receptors and adding more glutamate receptors. Keep that picture in your head, because when alcohol disappears suddenly, this adapted brain is left with too much gas and too little brake — that is the upstream break we are about to trace.

BODY'S JOB

The brain's chemical brake-and-gas system keeps nerve cells firing at the right speed — not too fast, not too slow. GABA is the brake, quieting nerve signals so you can sleep, stay calm, and not shake. Glutamate is the gas, waking nerve cells up so you can think, move, and respond. When these two balance perfectly, your heart rate stays steady, your blood pressure stays normal, your temperature stays right, and your thoughts stay clear. Alcohol is a powerful brake booster: it makes GABA brakes stronger and weakens the glutamate gas. Over weeks and months of heavy drinking, the brain fights back by removing some GABA brake receivers from nerve cell surfaces and adding more glutamate gas receivers, trying to keep everything running normally despite the alcohol. This adaptation is why a chronic drinker can seem almost normal even with high alcohol levels — their brain has reset its balance point. The danger comes when alcohol suddenly vanishes: the brakes are weak and the gas pedal is huge, so the brain races out of control.

1. GABA-A receptors on nerve cells catch GABA molecules, which open tiny gates that let chloride (negatively charged particles) flow into the cell.
2. NMDA receptors (the glutamate receivers most involved in learning and excitement) catch glutamate molecules, which open gates that let sodium and calcium (positively charged particles) flow into the cell.
3. Alcohol boosts GABA's calming effect and blocks glutamate's waking effect.
4. Over weeks of chronic heavy alcohol use, the brain downregulates GABA-A receptors (removes some from nerve cell surfaces) and upregulates NMDA glutamate receptors (adds more to nerve cell surfaces).
5. When alcohol is suddenly removed, the adapted brain has weak GABA braking and strong glutamate acceleration.

What breaks

The brain's chemical balance breaks when alcohol — the molecule that was holding the adapted system together — suddenly disappears. Imagine your car's engine has been tuned to run only with the parking brake halfway on. The mechanic removed some brake pads and boosted the engine to keep the car moving normally. Now someone suddenly releases the parking brake: the car lurches forward out of control. That is what happens in the brain during alcohol withdrawal. The GABA brake is weak, the glutamate gas is strong, and the sympathetic nervous system (the body's fight-or-flight alarm system, powered by the locus coeruleus in the brainstem, a tiny alarm center deep in the back of the brain that releases norepinephrine, and the medulla at the base of the brain that controls heart rate and blood pressure) explodes into action. Every nerve circuit fires too fast: thinking circuits in the prefrontal cortex (the front of the brain that guides clear thought) produce confusion, sensory circuits in the visual cortex

(the back of the brain that processes what you see) and somatosensory cortex (the strip along the top that processes touch and body sensation) produce hallucinations, motor circuits in the cerebellum (the back lower part that smooths movement) and basal ganglia (deep brain structures that coordinate movement) produce tremor, temperature circuits in the hypothalamus (the brain's thermostat, deep in the center) produce fever, and heart and blood vessel circuits in the medulla produce racing pulse and high blood pressure. This is not one organ failing — it is the central control system losing its ability to regulate the entire body.

CLINICAL TERM

Delirium tremens occurs 48 to 96 hours after abrupt cessation of alcohol in patients with chronic heavy use (typically above 5 drinks per day for men or 4 for women, sustained for weeks to months), especially if early withdrawal (tremor, anxiety, nausea, mild high blood pressure (hypertension), a fast heart rate (tachycardia)) was untreated or undertreated. The adapted brain suddenly faces unopposed glutamate excitation at NMDA receptors throughout the cerebral cortex, limbic system (emotional and memory centers including the hippocampus, the memory-maker deep in the temporal lobe, and amygdala, the alarm center that flags danger), and brainstem, plus a surge of sympathetic activity driven by norepinephrine (the body's main fight-or-flight chemical) released from the locus coeruleus (the brainstem's alarm center that releases norepinephrine into the brain). That break does not stay local. The downstream signals you will actually see at the bedside are confusion, agitation, visual and tactile hallucinations (seeing bugs on the skin, feeling crawling sensations), coarse tremor, profuse heavy sweating (diaphoresis) (heavy sweating), high blood pressure (systolic often above 160 mmHg), a fast heart rate (heart rate often above 120 bpm), and hyperthermia (temperature above 100.4°F, sometimes climbing above 104°F). Read all the findings backwards from there — they all point to this one broken thing upstream: loss of inhibitory tone and autonomic hyperactivity.

How the body compensates

The body tries to keep blood pressure, heart rate, and oxygen delivery normal even as the brain spirals out of control. Early on, vital signs may look stable or even high-normal because the sympathetic system is compensating hard. This is the dangerous middle: the patient looks okay on the monitor but is actually on the edge of collapse. The job is to recognize the

trajectory toward decompensation before the crash — because once compensation fails, the window to reverse it narrows to minutes.

1. {'compensation': 'The sympathetic nervous system (driven by the locus coeruleus in the brainstem and the medulla at the base of the brain) raises heart rate and blood pressure by releasing norepinephrine (the fight-or-flight chemical that speeds the heart and tightens blood vessels) to maintain blood flow (perfusion) (blood flow) to vital organs.', 'tradeoff': 'This keeps the brain, heart, and kidneys fed with oxygen for now, but the heart is working much harder than normal — oxygen demand rises, and if the heart cannot keep up, arrhythmias (abnormal heart rhythms like atrial fibrillation (an irregular, quivering heartbeat) or ventricular a fast heart rate (tachycardia)) or myocardial starved blood flow (ischemia) (not enough blood reaching heart muscle) can start.'}
2. {'compensation': 'Respiratory rate increases (often above 20 breaths per minute) to blow off carbon dioxide and compensate for metabolic acidosis (acid building up in the blood) (acid buildup from agitation, muscle breakdown, and poor tissue blood flow (perfusion)).', 'tradeoff': 'Faster breathing helps keep blood pH closer to normal (7.35 to 7.45), but if the patient tires from exhaustion or aspirates (breathes in vomit or saliva), respiratory failure and low oxygen (hypoxia) (low oxygen, SpO₂ below 90%) can happen suddenly.'}
3. {'compensation': 'The liver tries to clear rising lactate (from anaerobic metabolism when cells burn sugar without enough oxygen) and myoglobin (muscle protein released during rhabdomyolysis).', 'tradeoff': 'Chronic heavy alcohol use has already damaged the liver (fatty liver, alcoholic hepatitis, or cirrhosis); it cannot handle the extra load, so lactate and toxins build up, worsening too much acid in the blood (acidosis) and confusion.'}
4. {'compensation': 'The kidneys (specifically the glomeruli, the tiny filters in each nephron, and the tubules, the tubes that reabsorb water and salts) try to filter myoglobin and maintain electrolyte balance.', 'tradeoff': 'Myoglobin clogs the kidney's tiny tubules, causing acute tubular tissue death (necrosis) (ATN, death of the tubule cells) and acute kidney injury (AKI, creatinine rising above 1.5 mg/dL or doubling from baseline); dehydration from sweating and fever makes this worse, and once the kidneys fail, potassium rises (hyperkalemia, above 5.5 mEq/L) and other electrolytes spiral out of control, increasing arrhythmia risk.'}
5. {'compensation': 'Agitation and tremor are the brain's attempt to discharge excess electrical activity through the motor cortex (the strip along the top front that controls voluntary movement) and basal ganglia (deep structures that coordinate movement).', 'tradeoff': 'Sustained muscle contraction breaks down muscle cells (rhabdomyolysis), releasing potassium, phosphate, creatine kinase (CK, a muscle enzyme often above 5,000 U/L, sometimes above 50,000), and myoglobin into the blood — this can stop the heart or destroy the kidneys.'}

6. {'compensation': "Fever and sweating are the hypothalamus's response to the metabolic storm and sympathetic surge.", 'tradeoff': "Hyperthermia (body temperature above 104°F or 40°C) denatures (breaks) proteins and enzymes, causing permanent brain damage; profuse sweating causes severe dehydration (often 4 to 10 liters fluid deficit) and electrolyte loss, worsening low blood pressure (hypotension) (low blood pressure, systolic below 90 mmHg) and arrhythmias.'}
7. {'compensation': "Hallucinations and confusion reflect the brain's attempt to make sense of chaotic sensory input from the visual cortex (the back of the brain) and somatosensory cortex (the strip along the top that processes touch).", 'tradeoff': "Disorientation and agitation increase the risk of self-injury, violent behavior, pulling out IV lines or breathing tubes, aspiration, and inability to cooperate with life-saving treatment.'}
8. {'compensation': "Watch for the crash point before it happens: a patient with normal blood pressure but worsening agitation, rising heart rate (climbing above 120 bpm), or new tremor is not stable — they are compensating on the edge of collapse.", 'tradeoff': "Trend vital signs and mental status over time, not just one snapshot. Decompensation can be sudden: ventricular arrhythmias (ventricular a fast heart rate (tachycardia) or ventricular fibrillation, where the bottom chambers quiver instead of pump), aspiration pneumonia (vomit or saliva entering the lungs, causing low oxygen (hypoxia) and sepsis), cardiovascular collapse (shock, cardiac arrest), and status epilepticus (a seizure that will not stop) (seizures lasting longer than 5 minutes or repeated seizures without recovery between, causing brain swelling and lactic acidosis (acid from oxygen-starved tissues)). Untreated delirium tremens carries a mortality (death rate) of approximately 15%; even with aggressive treatment, 1 to 5% die. The window to reverse the domino effect closes fast once compensation fails.'}

Domino effect

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. — 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. — 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. — 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. — 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. — 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. — 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. — 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%. — 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%.

Symptoms · what the patient feels, 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.
- **High blood pressure (top number often over 160 mmHg):** The same surge of norepinephrine and epinephrine squeezes blood vessels tighter, raising pressure inside them. This is the body's attempt to keep blood flowing to vital organs during what it perceives as a crisis.
- **High fever (100.4–104°F or 38–40°C):** Constant muscle shaking and agitation generate heat like an engine running too fast. The hypothalamus (the brain's temperature control center deep in the brain) is also thrown off by the glutamate surge, and dehydration makes it harder for the body to cool itself by sweating.
- **Generalized tonic-clonic seizures (whole-body convulsions):** Unopposed glutamate lowers the seizure threshold, meaning the brain is much more likely to fire in abnormal synchronized waves. GABA (the brain's main calming signal) is also very low because alcohol is gone. These waves cause the muscles to contract violently all at once. Without treatment, seizures can become continuous—a life-threatening emergency called status epilepticus (a seizure that will not stop).
- **Insomnia and severe sleep disturbance:** The brain is in a state of hyperarousal—glutamate is too high and GABA is too low. The thalamus (the brain's relay station that filters signals) and the reticular activating system (the wakefulness switch in the brainstem) stay turned on. The person cannot fall asleep or stay asleep, even though they are exhausted.

Focused exam

- Tremor — fast, fine shaking in the hands, arms, or whole body: When someone who drinks heavily every day suddenly stops, their brain loses the calming brake that alcohol provided. Without that brake, glutamate (the brain's main 'go' chemical that makes nerve cells fire) runs wild, causing muscles to twitch too fast. This shows up as shaking. The tremor is one of the first warning signs that the brain is in withdrawal overdrive.
- Confusion and disorientation — not knowing where you are, what day it is, or who people are: Alcohol used to keep the brain's excitement in check. When it disappears, the brain floods with too much glutamate — like pressing the gas pedal with no brakes. That flood scrambles thinking and memory. This confusion is the hallmark of delirium tremens (DTs), the most dangerous form of alcohol withdrawal. It is a medical emergency.
- Hallucinations — seeing, feeling, or hearing things that are not really there (such as bugs crawling on the walls or voices): The brain's wiring is firing so fast that it creates false signals. You might see bugs crawling on the walls or feel things touching your skin when nothing is there. These hallucinations are classic for delirium tremens and mean the brain is in a hyperactive, dangerous state.
- Fast heart rate (tachycardia, over 100 beats per minute) and high blood pressure (over 140/90 mmHg): The sympathetic nervous system (the part of the brain and nerves that controls your heart and blood vessels in 'fight or flight' mode) goes into overdrive. It dumps norepinephrine and epinephrine (adrenaline chemicals) into the blood like a car revving too high. This drives up heart rate and blood pressure. Doctors call this an 'autonomic storm' — the body's automatic controls are spinning out of control.
- Heavy sweating (diaphoresis) and fever (body temperature over 100.4 °F or 38 °C): The hypothalamus (the brain's thermostat, a region deep in the brain that controls body temperature) loses its fine tuning, and the sympathetic surge makes you sweat buckets and run hot. These are more signs that the automatic systems in your body are out of balance. Together with the fast heart and high blood pressure, they paint the picture of a hyperadrenergic storm — too much adrenaline flooding the system.
- Severe restlessness, agitation, and overwhelming anxiety — unable to sit still, pacing, or thrashing: Without enough GABA (gamma-aminobutyric acid, the brain's main calming chemical), glutamate takes over and the brain cannot put the brakes on movement or emotion. The patient may thrash, pace, or seem unable to sit still. This extreme agitation raises the risk of injury, choking (aspiration), and putting dangerous stress on the heart.

Diagnostics · every test answers a question

- Comprehensive metabolic panel (CMP) plus magnesium and phosphate → Are the blood salts (electrolytes) out of balance in a way that makes seizures or dangerous heart rhythms more likely?
- Blood alcohol level and urine drug screen → Is the patient drunk right now, or is this withdrawal? Are other drugs involved?
- Thiamine level (vitamin B1) — but always give thiamine immediately without waiting for the lab result → Is the patient low on thiamine, which the brain needs to make energy?
- Liver function tests (AST, ALT, bilirubin, INR) and complete blood count (CBC) → Has chronic drinking damaged the liver or bone marrow? Is the blood able to clot normally?
- Chest X-ray, urinalysis, and blood cultures if infection is suspected → Is there a hidden infection (pneumonia, bladder infection, or bacteria in the blood) that brought the patient to the hospital and triggered withdrawal?
- Head CT scan (without contrast dye) if the patient has new weakness on one side, a history of falling or hitting the head, or focal neurologic signs → Is there bleeding in the brain, a stroke, or a mass that could look like or coexist with delirium tremens?
- Electrocardiogram (ECG) to check for arrhythmias and QTc prolongation → Is the heart's electrical rhythm abnormal or at risk for a life-threatening arrhythmia?
- Arterial or venous blood gas if the patient has rapid breathing, low oxygen saturation (below 94%), or suspected acid-base disturbance → Is the patient's blood too acidic or too basic, and is oxygen delivery adequate?

Treatment logic

- 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.
- 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.

- 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.
- 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.
- 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.
- Haloperidol 2–5 mg IV or IM every 30–60 minutes, only after the patient has received adequate benzodiazepines, and only to control severe hallucinations or agitation that persists despite GABA-enhancing agents → Haloperidol blocks dopamine D2 receptors in the mesolimbic pathway (the brain's emotion and perception circuit), which can quiet hallucinations and extreme agitation when used alongside benzodiazepines.
- Identify and treat the medical condition that triggered withdrawal: antibiotics for pneumonia or sepsis, IV fluids for dehydration, blood clot prevention (DVT prevention (prophylaxis) with heparin 5,000 units subcutaneous twice daily or enoxaparin 40 mg subcutaneous once daily), aspiration precautions (keep head of bed up at 30–45 degrees, suction airway as needed), and management of any trauma, bleeding, or surgical complications → Delirium tremens usually happens when a heavy drinker is hospitalized for infection, injury, or surgery and suddenly cannot drink. Treating the pneumonia, repairing the fracture, or draining the abscess reduces the body's overall stress and improves survival. Supportive care (IV fluids, nutrition, preventing blood clots and bedsores) keeps other organ systems stable while the brain recovers.

Medications

Benzodiazepines (lorazepam or diazepam) · GABA-A receptor positive allosteric modulator (sedative-hypnotic)

When someone drinks alcohol every day for weeks or years, the brain changes to adapt. The brake system—GABA-A receptors, which calm brain cells—turns down. The gas pedal—glutamate NMDA receptors, which speed up brain cells—turns up. When alcohol suddenly stops, the brain is left with weak brakes and a floored gas pedal. Brain cells fire wildly. This wild firing causes seizures, confusion, seeing things that are not there, a racing heart, high blood pressure, fever, and sweating. Benzodiazepines latch onto GABA-A receptors and make the remaining GABA work much stronger. This restores the brake and slows the dangerous overactivity. Lorazepam (Ativan) or diazepam (Valium) is given through an IV—a tube into a vein. Start with 2 to 4 milligrams every 5 to 15 minutes. Keep going until the heart rate, blood pressure, sweating, and agitation come down. The goal is to calm the autonomic storm—the overactive nervous system controlling heart and blood vessels—and prevent seizures.

1. Alcohol withdrawal leaves GABA-A receptors downregulated—fewer receptors, less sensitive—and glutamate NMDA receptors upregulated—more receptors, more active.
2. The benzodiazepine molecule binds to a specific pocket on the GABA-A receptor. This is not the same spot where GABA itself lands.
3. This binding changes the shape of the receptor. When GABA then attaches, the chloride channel—a pore in the brain cell membrane—opens wider and stays open longer.
4. More chloride ions—negatively charged particles—flow into the brain cell. This makes the inside more negative. The cell becomes less likely to fire.
5. Brain cells slow down across the brain. This reduces seizure risk, autonomic overactivity, and agitation.
6. Lorazepam has a half-life of 10 to 20 hours and no active breakdown products. It works predictably even when the liver is damaged. Diazepam works faster—it is fat-loving and crosses into the brain in 1 to 3 minutes. But it has active breakdown products that last days, which can cause prolonged sleepiness.

SAFETY WATCH

Respiratory depression (slowed breathing): high doses can suppress the brainstem respiratory center—the part of the brainstem that controls breathing. This is especially true when combined with opioids or in patients with lung disease. Watch oxygen level and breathing rate. Be ready to support breathing or reverse with flumazenil. But flumazenil can trigger seizures in withdrawal, so use it only if life-threatening breathing failure occurs. · Low blood pressure (Hypotension) (low blood pressure): benzodiazepines relax blood vessels. In dehydrated or septic patients, pressure can drop dangerously. Give IV fluids alongside the drug. · Paradoxical agitation: rarely, benzodiazepines can worsen confusion or aggression. This is more common in elderly patients or those with brain injury. · Aspiration risk: heavy sedation blunts protective airway reflexes. If the patient vomits, stomach contents can enter the lungs and cause aspiration pneumonia—lung infection from inhaled vomit. Position the patient on their side if heavily sedated. · Accumulation with repeated dosing: diazepam's long-acting breakdown products build up over days. This risks prolonged sleepiness. Lorazepam is safer in liver disease because it is cleared by the kidneys through simple conjugation—a straightforward chemical attachment that the kidneys can handle.

Phenobarbital · Barbiturate (GABA-A receptor agonist and glutamate antagonist)

Phenobarbital is a backup when benzodiazepines alone cannot control severe withdrawal. It works at two sites. First, it directly opens the chloride channel on GABA-A receptors—even without GABA present. Second, it blocks glutamate NMDA receptors, the gas pedal. This double action calms the brain more deeply. Phenobarbital also lasts much longer in the body—half-life 80 to 120 hours—so one dose keeps working for days. It is given as a loading dose, 10 to 20 milligrams per kilogram of body weight. This is divided into smaller IV doses over an hour. Then it is maintained with daily dosing if needed. Because it works even when GABA is scarce, it can rescue patients who are refractory—not responding—to benzodiazepines.

1. Phenobarbital binds to a barbiturate site on the GABA-A receptor. This is distinct from both the GABA site and the benzodiazepine site.
2. It forces the chloride channel to open even if GABA is absent. Chloride ions flood into brain cells and silence them.
3. It also blocks NMDA receptors—glutamate-gated channels that let calcium and sodium into brain cells. Blocking these stops the excitatory signal.
4. The combination of enhanced inhibition—GABA boost—and reduced excitation—glutamate block—stops the runaway cell firing that drives delirium tremens.
5. Phenobarbital also induces liver enzymes over days, which can speed the breakdown of other drugs. But in acute withdrawal, this effect is not yet relevant.

SAFETY WATCH

Severe respiratory depression: phenobarbital suppresses the brainstem respiratory center more than benzodiazepines. Patients often need ICU monitoring. Some require intubation—a breathing tube—and mechanical ventilation. · Low blood pressure (Hypotension): it relaxes blood vessels and can drop blood pressure, especially when loaded rapidly. Give fluids and monitor closely. · Prolonged sedation: the long half-life means the patient may stay sleepy for days. This is acceptable in ICU settings but requires careful monitoring for aspiration and pressure ulcers. · Paradoxical excitement in children: rarely, phenobarbital causes hyperactivity instead of sedation in young patients. · Drug interactions: phenobarbital induces cytochrome P450 enzymes. This can lower levels of many medications—warfarin, phenytoin, oral contraceptives—over days to weeks. In the acute setting, this is usually not a concern.

Propofol · General anesthetic (GABA-A receptor agonist)

Propofol is used in the ICU for the sickest patients—those whose withdrawal is so severe that benzodiazepines and phenobarbital together are not enough. It binds to GABA-A receptors and greatly amplifies chloride flow. The result is deep sedation or even general anesthesia. Propofol is given as a continuous IV drip, starting at 5 to 10 micrograms per kilogram per minute. Then it is adjusted up or down, so it can be changed quickly. It works within seconds and wears off within minutes when stopped. This gives very tight control. The patient usually requires a breathing tube—endotracheal intubation—because propofol can completely suppress spontaneous breathing.

1. Propofol binds to the beta subunit of GABA-A receptors. This increases the time the chloride channel stays open.
2. Chloride ions rush into brain cells, hyperpolarizing them—making the inside very negative—so they cannot fire.
3. The combined effect suppresses nearly all brain activity. This controls even the most dangerous autonomic surge—heart rate over 140, systolic blood pressure over 180, severe fever.
4. Propofol is broken down rapidly by the liver and also in the blood and other tissues. Its effect ends minutes after the infusion stops.

SAFETY WATCH

Respiratory arrest: propofol will stop breathing. The patient must be intubated and on a ventilator before or immediately after starting the infusion. · Low blood pressure (Hypotension): propofol dilates blood vessels and suppresses the heart's pumping strength. Blood pressure can drop dangerously. Patients often need vasopressors—norepinephrine, vasopressin—to keep pressure up. · Propofol infusion syndrome: rare but deadly. It is seen after high doses—above 80 micrograms per kilogram per minute—for more than 48 hours. It causes metabolic acidosis (acid building up in the blood)—blood becomes too acidic—rhabdomyolysis—muscle breakdown—high potassium (hyperkalemia)—high potassium—heart failure, and kidney failure. Monitor creatine kinase, lactate, and triglycerides daily. Stop propofol if triglycerides rise sharply or if too much acid in the blood (acidosis) develops. · Hypertriglyceridemia: propofol is suspended in a lipid—fat—emulsion. This delivers a large calorie and fat load. Triglycerides can rise over 500 milligrams per deciliter, risking pancreatitis—inflamed pancreas. Check levels every 24 to 48 hours. · Infection risk: the lipid emulsion can support bacterial growth if sterile technique is broken. Change IV tubing every 6 to 12 hours and discard unused propofol after opening. · Green urine: harmless, due to phenol breakdown products. Tell the team so they do not misinterpret it as a sign of infection or other problem.

Thiamine (vitamin B1) · Vitamin supplement (cofactor for cellular energy metabolism)

Chronic alcohol use drains thiamine because alcohol blocks its absorption in the gut. The liver cannot store much. Thiamine is required for three key enzymes that turn glucose into energy inside the mitochondria—the power plants of cells. Without thiamine, brain cells—especially in the thalamus (the brain's sensory relay station), mammillary bodies (small round structures at the base of the brain involved in memory), and brainstem—run out of energy and die. This causes Wernicke encephalopathy: confusion, eye movement problems, and unsteady walking. If untreated, this leads to Korsakoff syndrome—permanent memory loss. Thiamine 500 milligrams IV three times daily for at least 3 days replaces the shortage before glucose is given. Giving glucose without thiamine forces the brain to try to burn it without the needed helper. This can trigger or worsen Wernicke encephalopathy. After 3 days, switch to 100 milligrams IV or by mouth daily until the patient resumes a normal diet.

1. Thiamine is absorbed into cells and converted to thiamine pyrophosphate, or TPP, the active form.
2. TPP binds to three key enzymes. Pyruvate dehydrogenase turns pyruvate into acetyl-CoA to enter the energy cycle. Alpha-ketoglutarate dehydrogenase works in the Krebs cycle to make NADH. Transketolase makes ribose for DNA and RNA in the pentose phosphate pathway.

3. With these enzymes activated, brain cells can fully metabolize glucose into ATP—the energy molecule cells use for everything.
4. Brain cells in the thalamus and mammillary bodies have very high energy demands. They are the first to fail without thiamine, leading to petechial hemorrhages—tiny bleeds—and cell death.
5. Replacing thiamine prevents new damage and may partially reverse early Wernicke encephalopathy if given within hours to a few days. Permanent damage—Korsakoff syndrome—cannot be reversed.

SAFETY WATCH

Anaphylaxis: rare—1 in 10,000—but life-threatening allergic reaction with IV thiamine. Have epinephrine ready. Some clinicians give a small test dose or administer it slowly over 30 minutes. · Always give thiamine BEFORE or WITH glucose: giving glucose first can exhaust the tiny remaining thiamine stores and trigger Wernicke encephalopathy. · Local irritation: IV thiamine can burn at the injection site. Dilute in 50 to 100 milliliters saline and infuse over 30 minutes if possible.

Magnesium sulfate · Electrolyte replacement (divalent cation, NMDA receptor antagonist, and cofactor)

Chronic alcohol use causes the kidneys to waste magnesium. Poor diet means little magnesium comes in. Low magnesium—low magnesium (hypomagnesemia), serum level below 1.7 milligrams per deciliter—makes withdrawal worse. Magnesium normally blocks NMDA glutamate receptors, acting like a natural brake on excitation. It is required for hundreds of enzymes, including those that make ATP and regulate potassium and calcium channels. Without magnesium, brain cells are more excitable. The heart rhythm becomes unstable—risk of torsades de pointes, a deadly arrhythmia. Muscles cramp and twitch. Potassium cannot stay inside cells, so potassium levels drop no matter how much you replace. Magnesium sulfate 2 grams IV over 15 minutes, then 1 to 2 grams every 6 hours until serum level is above 2 milligrams per deciliter, replaces the shortage. Oral magnesium oxide, 400 milligrams once or twice daily, continues replacement after the acute phase.

1. Magnesium ions— Mg^{2+} —sit in the pore of NMDA receptors at rest. They physically block calcium from entering brain cells even when glutamate binds.
2. Magnesium stabilizes the resting membrane potential by helping the sodium-potassium pump work. This keeps potassium inside cells and prevents spontaneous firing.
3. In the heart, magnesium regulates calcium channels and repolarization—the recovery phase after each heartbeat. This prevents long QT interval and torsades de pointes.
4. Magnesium is a helper for enzymes that make and use ATP. Muscle and nerve function improve once levels are normal.

5. Magnesium also calms vascular smooth muscle—the muscle in blood vessel walls. This reduces blood pressure slightly and prevents spasm.

SAFETY WATCH

Hypermagnesemia (too much magnesium, level above 4 milligrams per deciliter): causes muscle weakness, loss of deep tendon reflexes, slowed heart rate, low blood pressure. At very high levels—above 12 milligrams per deciliter—respiratory paralysis and cardiac arrest can occur. This is rare with normal kidney function because the kidneys excrete excess magnesium quickly. In kidney failure, reduce the dose or avoid IV magnesium. · Rapid infusion: giving magnesium faster than 1 gram per 10 minutes can cause flushing, low blood pressure, and nausea. Infuse over at least 15 minutes. · Calcium antagonism: very high magnesium can block calcium channels too much, causing profound weakness. If this happens, give calcium gluconate 1 to 2 grams IV to reverse the effect. · Check levels: recheck serum magnesium every 6 to 12 hours during replacement to avoid overshooting.

Potassium chloride · Electrolyte replacement

Alcohol causes the kidneys to lose potassium. Vomiting or diarrhea makes it worse. Low potassium—low potassium (hypokalemia), level below 3.5 milliequivalents per liter—destabilizes the heart's electrical system. This causes arrhythmias, especially dangerous when the autonomic storm of withdrawal is already stressing the heart. Potassium is the main positive ion inside cells. The gradient—difference in concentration—between inside and outside the cell determines how easily brain cells and heart muscle cells can fire and recover. Low potassium also weakens skeletal muscles and slows gut movement. Replace with potassium chloride 10 to 20 milliequivalents per hour IV—never faster, or it can stop the heart. The target is a serum level above 4 milliequivalents per liter. In less urgent cases, give oral potassium chloride 20 to 40 milliequivalents two to four times daily.

1. Potassium enters the bloodstream from the IV and is taken up by cells through sodium-potassium pumps. These pumps trade 3 sodium ions out for 2 potassium ions in, powered by ATP.
2. The high inside-cell potassium concentration restores the normal resting membrane potential—around -70 to -90 millivolts. This makes cells stable and ready to respond appropriately to signals.
3. In the heart, potassium channels open during repolarization—after each beat. Potassium flows out and resets the cell. If potassium is low, repolarization is delayed. This shows as a long QT interval on the ECG and increases the risk of torsades de pointes.
4. Replacing potassium shortens the QT interval and stabilizes heart rhythm.

SAFETY WATCH

High potassium (Hyperkalemia) (level above 5.5 milliequivalents per liter): causes life-threatening arrhythmias—peaked T waves, wide QRS, sine-wave pattern, then ventricular fibrillation and cardiac arrest. Give potassium slowly, no faster than 10 to 20 milliequivalents per hour through a central line—or 10 milliequivalents per hour through a peripheral IV. Recheck levels every 4 to 6 hours. · Peripheral IV infiltration: potassium is very irritating to tissues. If it leaks out of the vein—extravasates—it causes severe pain, blistering, and tissue death. Use a central line for high-dose replacement if possible. · Renal failure: if the kidneys are not working—creatinine above 3 milligrams per deciliter, urine output below 0.5 milliliters per kilogram per hour—they cannot excrete potassium. Levels can rise dangerously. Give smaller doses and check levels every 2 to 4 hours, or avoid IV potassium altogether and rely on oral if the gut works. · Never give potassium as a bolus—rapid push. It will stop the heart immediately.

Sodium chloride and dextrose (IV fluids) · Crystalloid fluid and glucose replacement

Patients in alcohol withdrawal are often dehydrated from poor intake, vomiting, sweating, and fever. Dehydration drops blood volume. This stresses the heart and kidneys and makes low blood pressure worse when sedatives are given. Normal saline—0.9% sodium chloride—or lactated Ringer's solution replaces lost fluid and sodium. After thiamine is given, add dextrose—5% dextrose in half-normal saline—to provide glucose, the brain's only fuel. Chronic drinkers often have low blood sugar—low blood sugar (hypoglycemia)—from poor nutrition and depleted liver glycogen stores. Typical starting rate: 100 to 150 milliliters per hour IV. Adjust based on urine output, blood pressure, and heart rate. The goal is urine output above 0.5 milliliters per kilogram per hour and stable blood pressure without needing vasopressors.

1. Crystalloid fluid enters the bloodstream and increases circulating volume within minutes.
2. Increased volume stretches baroreceptors—pressure sensors—in the carotid arteries and aorta. These signal the brain to reduce sympathetic outflow. This lowers heart rate and blood pressure toward normal.
3. Sodium and water distribute across the blood vessel and tissue spaces. About 1/3 stays in blood vessels. 2/3 moves into tissues, correcting dehydration at the cellular level.
4. Glucose is taken up by brain cells—which do not require insulin for glucose transport via GLUT1 and GLUT3 transporters—and other cells. It enters glycolysis and the Krebs cycle to produce ATP.
5. Correcting low blood sugar prevents further brain injury and supports the energy-intensive work of recovering from withdrawal.

SAFETY WATCH

Fluid overload: giving too much fluid, especially in heart failure or kidney failure, causes pulmonary edema (fluid flooding the lungs)—fluid in the lungs—making breathing difficult. Monitor lung sounds, oxygen level, and jugular venous pressure. Reduce rate if crackles are heard or oxygen drops. · Low sodium (Hyponatremia) (low sodium): if you give too much free water—like dextrose 5% in water—without adequate sodium, serum sodium can drop below 135 milliequivalents per liter. This causes confusion, seizures, and brain swelling. Use normal saline or half-normal saline with dextrose. · High sodium (Hypernatremia) (high sodium): over-correcting with normal saline can raise sodium above 145 milliequivalents per liter, causing confusion and seizures. Check sodium every 6 to 12 hours during aggressive fluid replacement. · High blood sugar (Hyperglycemia): patients with diabetes or stress high blood sugar can see blood sugar rise above 200 milligrams per deciliter with dextrose infusions. Check glucose every 4 to 6 hours and use insulin if needed.

Haloperidol · First-generation (typical) antipsychotic (dopamine D2 receptor antagonist)

Haloperidol blocks dopamine D2 receptors in the brain. This can calm severe agitation and hallucinations. In alcohol withdrawal, the main problem is too much glutamate and too little GABA. But some patients also have very high dopamine activity. Dopamine is a brain messenger involved in reward, movement, and mood. Very high dopamine worsens paranoia, visual hallucinations, and violent agitation. Haloperidol is used ONLY as an add-on to benzodiazepines, never alone. It does NOT fix the GABA-glutamate imbalance. It can actually LOWER the seizure threshold—make seizures more likely. Typical dose: 0.5 to 5 milligrams IV or IM—into the muscle—every 30 to 60 minutes as needed for agitation that persists despite adequate sedation with benzodiazepines. Use the lowest effective dose for the shortest time.

1. Haloperidol binds tightly to dopamine D2 receptors, preventing dopamine from activating them.
2. Blocking D2 receptors in the mesolimbic pathway reduces positive psychotic symptoms—hallucinations, delusions, paranoia.
3. Blocking D2 receptors in the striatum suppresses overactive movement circuits. This can help with restlessness, but also risks extrapyramidal side effects—involuntary muscle stiffness, tremor.
4. Haloperidol has minimal effect on GABA or glutamate systems. It does NOT address the core problem of alcohol withdrawal.

SAFETY WATCH

QT prolongation and torsades de pointes: haloperidol, especially IV, can prolong the QT interval on the ECG—the time the heart takes to reset electrically. If the QT interval exceeds 500 milliseconds, the risk of torsades de pointes—a fatal arrhythmia—rises sharply. Check a baseline ECG and recheck daily. Avoid haloperidol if the QTc is over 500 milliseconds or if the patient is also on other QT-prolonging drugs—methadone, ondansetron, azithromycin. Correct potassium and magnesium before giving haloperidol. · Neuroleptic malignant syndrome, or NMS: rare but deadly. NMS causes fever—often above 104°F—muscle rigidity, confusion, and autonomic instability—wildly swinging blood pressure and heart rate. Creatine kinase rises above 1,000 units per liter from muscle breakdown. Stop haloperidol immediately. Give cooling, IV fluids, and dantrolene—a muscle relaxant—or bromocriptine—a dopamine booster. · Extrapyramidal symptoms: acute dystonia—sustained muscle spasm, often of the neck, eyes, or jaw—can occur within hours to days. Treat with benztropine 1 to 2 milligrams IV or diphenhydramine 25 to 50 milligrams IV. Akathisia—inner restlessness, inability to sit still—and parkinsonism—tremor, stiffness, slow movement—can develop over days to weeks. · Lowers seizure threshold: haloperidol and other antipsychotics increase seizure risk, which is already high in alcohol withdrawal. Use only when absolutely necessary and ensure adequate benzodiazepine coverage. · Low blood pressure (Hypotension): haloperidol can block alpha-1 adrenergic receptors, causing blood vessels to dilate and blood pressure to drop, especially with IV dosing. Monitor closely and give fluids if needed. · Aspiration risk: heavy sedation blunts protective airway reflexes. This increases the risk of inhaling vomit into the lungs. Position the patient on their side if heavily sedated.

Folic acid (vitamin B9) · Vitamin supplement (cofactor for DNA synthesis and methylation)

Chronic alcohol use drains folic acid because alcohol blocks its absorption in the small intestine. Heavy drinkers often eat poorly. Folic acid is needed to make new DNA and RNA when cells divide. It is required to convert homocysteine—a potentially toxic amino acid—into methionine—an amino acid and methyl donor. Without folic acid, red blood cells grow too large and do not divide properly. This causes macrocytic anemia—low red blood cell count with large, fragile cells. Brain cells also need folic acid to maintain myelin—the insulation around nerves—and to produce neurotransmitters. Replacing folic acid with 1 milligram by mouth or IV daily prevents worsening anemia and supports brain recovery. Folic acid is often given alongside thiamine because both are commonly depleted together.

1. Folic acid is absorbed in the jejunum—middle part of the small intestine—and converted in the liver to tetrahydrofolate, or THF, the active form.

2. THF donates one-carbon units needed to build purines and pyrimidines—the building blocks of DNA and RNA.
3. Without adequate folate, DNA synthesis slows. Cells in the bone marrow cannot complete division. They become large and fragile. Many die before leaving the marrow, causing anemia.
4. THF also converts homocysteine to methionine, preventing homocysteine buildup. High homocysteine damages blood vessels and brain cells.

SAFETY WATCH

Masking vitamin B12 deficiency: giving folic acid can improve the anemia of B12 deficiency without treating the brain and nerve damage, which continues to worsen silently. Always check vitamin B12 level and replace if low—level below 200 picograms per milliliter—before starting folic acid. · Generally very safe: folic acid has no significant toxicity even at high doses, because excess is excreted in the urine.

Phosphate (sodium phosphate or potassium phosphate) · Electrolyte replacement

Chronic alcohol use and poor nutrition often cause low phosphate—hypophosphatemia, level below 2.5 milligrams per deciliter. Phosphate is essential for making ATP—the energy currency of cells. It is part of the backbone of DNA and RNA. Severe low phosphate—below 1.0 milligram per deciliter—causes muscle weakness, including the diaphragm. This risks breathing failure. It causes rhabdomyolysis—muscle breakdown releasing myoglobin that clogs the kidneys. It causes red blood cell destruction (hemolysis)—red blood cells rupture—and heart failure. In alcohol withdrawal, the stress and high energy demand make low phosphate especially dangerous. Replace with sodium phosphate or potassium phosphate 0.16 to 0.32 millimoles per kilogram IV over 6 hours. Target a serum level above 2.5 milligrams per deciliter. Oral replacement—Neutra-Phos or K-Phos 1 to 2 packets or tablets three times daily—is safer for mild shortage.

1. Phosphate enters the bloodstream and is taken up by cells via sodium-phosphate cotransporters.
2. Inside cells, phosphate is added to ADP—adenosine diphosphate—to make ATP—adenosine triphosphate—the molecule that powers almost every cellular process.
3. Phosphate is also required for 2,3-diphosphoglycerate, or 2,3-DPG, in red blood cells. 2,3-DPG helps hemoglobin release oxygen to tissues. Low phosphate lowers 2,3-DPG. Tissues become starved for oxygen even if the blood oxygen level looks normal.
4. Phosphate stabilizes cell membranes and is part of the DNA and RNA backbone. Replacing it supports cell division and repair.

SAFETY WATCH

Hyperphosphatemia (level above 4.5 milligrams per deciliter): causes calcium to fall out of the blood and deposit in tissues—soft-tissue calcification. This leads to severe low calcium, causing tetany, seizures, and arrhythmias. Give phosphate slowly and recheck levels every 6 to 12 hours. · Low calcium (Hypocalcemia): phosphate binds calcium in the blood. Replacing phosphate too fast can drop calcium below 8.5 milligrams per deciliter. This causes tingling around the mouth, muscle spasms—Chvostek sign, Trousseau sign—and seizures. Monitor calcium and correct if it drops. · Renal failure: if the kidneys are not working, they cannot excrete phosphate. Levels can rise dangerously. Use lower doses or oral replacement only. · IV infiltration: phosphate solutions are irritating. Leaking out of the vein can cause tissue death. Use a central line for large doses if possible.

Red flags

- 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.

Nursing priorities

1. Start continuous pulse oximetry, cardiac telemetry (heart rhythm monitoring), and automated blood pressure checks every 15 minutes until the patient is stable. — 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. — 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. — 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.
4. Place the patient in a quiet, well-lit room with constant observation; remove any objects that could be used for harm; use calm verbal redirection and avoid physical restraints unless the patient is an immediate danger to self or staff. — Delirium causes confusion and fear; hallucinations and agitation can lead to combative behavior, self-injury, or pulling out IV lines; a calm environment reduces sensory overload and brain irritation, while restraints can worsen agitation and increase the risk of rhabdomyolysis (muscle breakdown) and aspiration.
5. Check core temperature every hour and use cooling measures—remove blankets, apply ice packs to the groin and armpits, give IV fluids—if temperature rises above 101°F (38.3°C). — The autonomic storm disrupts the brain's thermostat (hypothalamus); high fever above 102.2°F signals severe instability, raises the risk of seizures and organ failure, and can cause permanent brain damage if not controlled quickly.
6. Draw blood for glucose, electrolytes (sodium, potassium, magnesium, phosphate), liver function, and complete blood count; correct magnesium below 2.0 mg/dL and potassium below 3.5 mEq/L immediately. — Low magnesium and low potassium lower the seizure threshold and make the heart prone to dangerous rhythms; chronic alcohol use depletes these minerals through vomiting, diarrhea, and poor absorption; correcting them makes benzodiazepines work better and reduces seizure risk.
7. Keep IV access secure with at least one large-bore (18-gauge or larger) peripheral IV or consider a central line if the patient is combative or veins are poor; flush the line every 4 hours. — Delirium tremens requires large, repeated IV benzodiazepine doses and continuous IV fluids; losing IV access during agitation delays life-saving treatment and forces dangerous delays or risky intramuscular injections that absorb unpredictably.

- 8.** Assess for aspiration risk before giving anything by mouth; keep the patient NPO (nothing by mouth) or give only small sips if alert enough to protect the airway; keep suction equipment at the bedside. — Confusion and decreased level of consciousness raise the risk of aspiration (breathing in stomach contents or saliva), which causes aspiration pneumonia and respiratory failure; vomiting is common in alcohol withdrawal and can lead to choking if the patient cannot protect the airway.

Sources

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