

Alcohol Intoxication

Alcohol shuts down the brain in top-to-bottom order: first judgment and coordination fail, then memory blacks out, then consciousness fades, and finally the brainstem stops telling the lungs to breathe.

THE STORY IN ONE SENTENCE

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“Alcohol works like a dimmer switch, turning off brain regions from the top down—judgment first, breathing last.”

Normal anatomy — the body's job

A healthy brain keeps a precise balance between glutamate (the brain's main go signal) and GABA (the brain's main stop signal). This balance keeps judgment, coordination, memory, consciousness, and breathing all in normal range.

BODY'S JOB

The frontal cortex handles judgment and impulse control—it pumps the brakes on risky actions. The cerebellum coordinates balance and smooth movement so you walk straight and speak clearly. The hippocampus forms new memories by turning what just happened into long-term storage. The reticular activating system in the brainstem keeps you awake and alert. The medulla controls automatic breathing by sending rhythmic signals to your diaphragm every few seconds.

1. The frontal cortex receives signals about risks and rewards, weighs them, and sends a go or stop command to your impulses—like a brake pedal for your actions.
2. The cerebellum takes movement commands from your motor cortex and smooths them out in real time, so you walk in a straight line and your speech comes out clear instead of jerky or halting.
3. The hippocampus takes what just happened—a conversation, a face, an address—and turns it into long-term storage, like hitting save on a computer file so you can recall it tomorrow.

4. The reticular activating system in the brainstem sends wake-up signals up to the entire cortex, keeping you conscious and able to respond to your surroundings—like a switch that keeps the lights on.
5. The medulla sends automatic rhythmic signals to your diaphragm and chest muscles every few seconds, making you breathe without thinking about it—even when you are asleep.

What breaks

Alcohol enhances GABA (the brain's main brake signal) and blocks glutamate (the brain's main go signal), so the whole brain slows down in a predictable top-to-bottom sequence. The higher thinking areas shut down first, and the life-support areas in the brainstem shut down last.

CLINICAL TERM

Ethanol potentiates GABA-A receptor activity and antagonizes NMDA glutamate receptors, producing dose-dependent central nervous system depression that progresses in a rostral-to-caudal pattern.

How the body compensates

Early on, the body tries to keep blood pressure and oxygen delivery up even as brain function declines. The most dangerous moment is when the person looks just drunk but is minutes away from breathing stopping completely.

1. {'mechanism': 'Sympathetic activation', 'physiology': 'The sympathetic nervous system (your fight-or-flight response) releases adrenaline to keep heart rate and blood pressure up, masking how slowed the brain really is—so the person may look alert but is actually severely intoxicated.'}
2. {'mechanism': 'Respiratory drive persistence', 'physiology': 'The respiratory drive center in the medulla keeps firing automatic breath commands—until the blood alcohol concentration crosses roughly 0.35 to 0.40 grams per deciliter, and then breathing slows or stops altogether.'}
3. {'mechanism': 'Critical warning signs', 'physiology': 'Big danger signs include: breathing rate below 8 breaths per minute, vomit present (risk of vomit entering the lungs), skin cold and clammy (body temperature dropping dangerously low), or the person cannot be roused to voice or painful touch (Glasgow Coma Scale 8 or below).'}- 4. {'mechanism': 'Coexisting hidden injuries', 'physiology': 'Hidden injuries often coexist: head trauma from falls, rhabdomyolysis (muscle breakdown releasing toxins into the blood) from lying still for hours, or low blood sugar (hypoglycemia) (low blood sugar) in a chronic drinker with depleted liver glycogen (stored sugar).'}-

5. {'mechanism': 'Rising blood alcohol trajectory', 'physiology': 'Know the trajectory: a person who is barely arousable now may stop breathing in the next 30 minutes if blood alcohol is still rising because the stomach is still absorbing alcohol. Peak blood alcohol typically occurs 30 to 90 minutes after the last drink.'}

Domino effect

1. Frontal cortex inhibition fails, so disinhibition and poor judgment appear. — Alcohol slows the frontal lobes first. These regions normally stop impulses and judge risk. When they go offline, the person loses self-control, speaks loudly, takes dangerous risks, and cannot judge consequences—like driving a car with no brakes.
2. Cerebellar depression causes loss of coordination (ataxia) (unsteady walk) and slurred speech (dysarthria) (slurred speech). — The cerebellum fine-tunes balance and smooth muscle coordination. Alcohol disrupts its output, so the person stumbles, sways, and slurs words because the brain cannot sequence muscle movements precisely—like a conductor losing the beat.
3. Hippocampal shutdown causes anterograde amnesia (blackout). — The hippocampus turns short-term experiences into long-term memory. When alcohol blocks NMDA receptors there, new memories cannot form—the person remains conscious and active but will remember nothing the next day, as if the save button on the computer stopped working.
4. Reticular activating system suppression causes altered mental status progressing to stupor and coma. — The reticular activating system in the brainstem keeps you awake. As alcohol concentration rises, this network shuts down, and the person becomes less and less arousable—first drowsy, then responding only to loud voice or pain, then unresponsive (Glasgow Coma Scale 8 or below).
5. Medullary respiratory center failure causes hypoventilation (slow breathing) or pauses in breathing (apnea) (no breathing). — The medulla's respiratory neurons fire rhythmically to drive each breath. At very high blood alcohol levels (often above 0.35 grams per deciliter), alcohol slows these neurons so much that breathing drops below 8 breaths per minute or stops entirely, causing low oxygen (hypoxia) (low blood oxygen) and death if not supported.
6. Aspiration of vomit causes acute respiratory distress and pneumonia. — Alcohol suppresses the gag reflex and cough reflex. If the person vomits while unconscious, stomach contents flow into the lungs instead of being expelled, causing chemical injury to the airways and bacterial infection (aspiration pneumonia) within hours.
7. Hypothermia worsens central nervous system depression and triggers cardiac arrhythmias. — Alcohol causes vasodilation (blood vessels widen), so heat radiates away from the core. If the person passes out in a cold environment, body temperature drops below 95 degrees Fahrenheit (35 degrees Celsius), which further slows brain activity and can trigger dangerous heart rhythms like ventricular fibrillation.

- 8.** Low blood sugar (Hypoglycemia) in chronic drinkers causes altered mental status and seizure risk. — Chronic alcohol use depletes liver glycogen (stored sugar), and alcohol itself blocks gluconeogenesis (the liver making new glucose). Blood sugar can drop dangerously low, mimicking or worsening the confusion and depressed consciousness—fingerstick glucose below 60 milligrams per deciliter requires immediate treatment with IV dextrose (sugar water).

Symptoms · what the patient feels, and why

- Disinhibition and impaired judgment: Alcohol slows down the prefrontal cortex (the front part of your brain that pumps the brakes on risky ideas). With that brake off, the person may say or do things they would never do sober—like driving drunk, starting fights, or making poor choices.
- Slurred speech (dysarthria): The cerebellum (the part of the brain at the back that fine-tunes your movements) helps your tongue and lips move smoothly together. Alcohol disrupts it, so words come out thick, garbled, or hard to understand—like trying to talk with a numb mouth.
- Loss of coordination (Ataxia) (unsteady walk, poor balance): Alcohol weakens the cerebellum's control over balance and movement. The person sways, staggers, or can't walk in a straight line—imagine trying to walk on a rocking boat when your balance system isn't working right.
- Involuntary eye jerking (Nystagmus) (jerky, back-and-forth eye movements): Your inner ear and cerebellum work together to keep your eyes steady when you move your head. Alcohol scrambles those signals, so the eyes drift to one side and then snap back quickly, over and over—like a broken tracking system.
- Anterograde amnesia (blackout—can't form new memories): Alcohol blocks NMDA receptors (special docking sites on memory-forming brain cells) in the hippocampus (the brain's memory recorder). New experiences during that time never get saved, so the person may talk and move around but later remember nothing—like a video camera that stopped recording even though it stayed on.
- Nausea and vomiting: Alcohol irritates the stomach lining and triggers the chemoreceptor trigger zone (a vomit sensor in the brainstem). The brain reads alcohol as a poison and tries to get rid of it by making you throw up.
- Flushed skin and feeling warm: Alcohol causes blood vessels in your skin to widen, so more warm blood flows near the surface. That makes you feel flushed and hot, even though you're actually losing body heat to the air—like opening the windows in a warm house in winter.

- Altered mental status progressing to stupor or coma: As blood alcohol climbs very high, it shuts down the reticular activating system (the brainstem's on-switch for wakefulness). The person becomes harder and harder to wake, then completely unresponsive—like turning down a dimmer switch until the lights go out.
- Respiratory depression (slow, shallow breathing): At very high levels, alcohol suppresses the medulla (the part of the brainstem that controls automatic breathing). Breaths become slow and shallow or stop altogether, so oxygen can't get into the blood—this is the main way people die from alcohol poisoning.
- Hypothermia (body temperature drops below 95 °F / 35 °C): Alcohol widens skin blood vessels, which lets body heat escape quickly. It also impairs the hypothalamus (your brain's thermostat), so you can't shiver or conserve heat properly. In a cold place, core temperature falls dangerously low—like leaving a heater running with all the doors open.
- Low blood sugar (Hypoglycemia) (blood sugar drops below 70 mg/dL): The liver normally releases stored sugar (glycogen breakdown) and makes new sugar (gluconeogenesis) to keep your blood glucose steady. Alcohol blocks both processes, especially if you haven't eaten, so blood sugar crashes—like a car running out of fuel because the tank is empty and the refinery is shut down.

Focused exam

- Blood alcohol concentration (BAC) - draw it first: The BAC tells you exactly how much alcohol is in the blood, measured in milligrams per deciliter (mg/dL) or as a percentage. 80 mg/dL (0.08%) is the legal driving limit in most states; 150–200 mg/dL usually causes confusion and big problems with balance; 300 mg/dL or higher carries serious risk of coma (being completely unresponsive); 400 mg/dL or higher can stop breathing and cause death. The number helps you know if what you see matches the dose—if someone has a BAC of 350 mg/dL but seems only mildly sleepy, they may drink so much regularly that their brain has adapted (tolerance).
- Breathing rate and depth - count breaths for a full minute: Alcohol slows down the medulla (the part of the brainstem at the base of the brain that automatically controls how fast and deep you breathe). If the rate drops below 12 breaths per minute or the breaths are shallow (not moving much air), the person is at high risk for breathing failure (not getting enough oxygen into the blood). This is the number-one way people die from alcohol poisoning.

- Level of consciousness – use a simple scale like AVPU (Alert, responds to Voice, responds to Pain, Unresponsive) or the Glasgow Coma Scale: Alcohol turns down brain activity in a predictable order as the dose goes up: first you feel relaxed, then confused, then very sleepy (lethargic), then hard to wake up (obtunded), then you slip into a stupor (respond only to painful stimuli like a hard pinch), and finally into a coma (no response at all). Knowing where the person is on that ladder tells you how deeply the brain is depressed and whether the airway is at risk—people who are barely responsive (obtunded) or worse often cannot protect their airway and may choke on vomit.
- Pupil size and light reflex: At typical alcohol doses, pupils stay normal size (3–5 mm) and still shrink quickly when you shine a light in them. If the pupils are pinpoint (very tiny, like the tip of a pen) or very dilated (wide open, bigger than 6 mm) and sluggish or do not react to light at all, think about other drugs mixed in—opioids (like heroin, fentanyl, or oxycodone) make pupils pinpoint; anticholinergic drugs (like diphenhydramine, scopolamine, or some plants) make them huge. Finding abnormal pupils completely changes your treatment plan because you may need to give an antidote like naloxone.
- Walking and balance – watch the person walk or try a tandem walk (heel-to-toe in a straight line) or Romberg test (stand with feet together, eyes closed): Alcohol messes up the cerebellum (the back part of the brain that keeps you balanced and coordinates smooth movement) very early, even at low doses. If the person stumbles, cannot walk heel-to-toe, or sways and nearly falls when standing still, you know the alcohol is affecting motor control. The worse the loss of coordination (ataxia) (unsteady, wobbly walking), the higher the dose or the more sensitive the person is.
- Skin color, temperature, and signs of vomiting (check breath, clothes, and mouth for vomit): Alcohol opens up (dilates) the small blood vessels in the skin, so the face and skin often look flushed (red and warm). But this also means heat escapes faster—if the person is in a cold place or lying outside, body temperature can drop dangerously low (hypothermia, core temperature below 95°F or 35°C). Blue or very pale skin may mean hypothermia or shock. Vomit on the person or a smell of vomit is a red flag: alcohol makes you throw up and also weakens the reflexes that protect your airway. If someone vomits while drowsy or unconscious, stomach contents can go into the lungs (aspiration), causing a dangerous lung infection or inflammation (aspiration pneumonitis or pneumonia).

Diagnostics · every test answers a question

- Blood alcohol concentration (BAC), serum → How much alcohol is in the blood right now, and does that number match how impaired the person looks?
- Fingertstick or serum glucose → Is the blood sugar low enough to cause or worsen confusion, and does it need immediate correction?

- Comprehensive metabolic panel (CMP): sodium, potassium, chloride, bicarbonate, blood urea nitrogen (BUN), creatinine, and glucose → Are there electrolyte imbalances (salts out of balance in the blood) or signs of kidney injury from dehydration, vomiting, or muscle breakdown?
- Serum or urine toxicology screen (comprehensive drug screen including opioids, benzodiazepines, amphetamines, cocaine, tricyclic antidepressants, acetaminophen, and salicylates) → Are there other drugs on board that could add to the sedation, make breathing worse, or cause different toxicity?
- Serum osmolality and calculated osmolal gap → Is there an unexplained difference between measured and calculated osmolality that suggests a toxic alcohol (methanol or ethylene glycol) is present?
- Creatine kinase (CK), serum → Is there muscle breakdown (rhabdomyolysis) from lying in one place too long, seizure, or trauma?
- Arterial or venous blood gas (with lactate) if the patient is very drowsy, breathing slowly, or has abnormal vital signs → Is the person holding onto carbon dioxide (CO₂) from slow breathing, and is there metabolic acidosis (acid building up in the blood) that suggests shock or toxic alcohol?
- Head CT scan (computed tomography, a detailed X-ray of the brain) if there is any concern for head trauma, focal neurologic signs (weakness or numbness on one side, unequal pupils, new slurred speech), or if the person does not wake up as expected → Is there bleeding inside the skull (intracranial bleeding (hemorrhage)) or swelling of the brain from a head injury?

Treatment logic

- Protect the airway with endotracheal intubation if the Glasgow Coma Scale (GCS) is 8 or lower, or if the person cannot protect the airway (no gag reflex, continuous vomiting, or witnessed aspiration) → Alcohol slows down the reticular activating system (the brain network that keeps you awake and alert) and the medulla (the brainstem center that controls breathing). When consciousness drops to stupor or coma, the muscles of the throat relax and the reflexes that keep food and vomit out of the lungs (gag and cough reflexes) stop working. Intubation means placing a plastic tube through the vocal cords into the windpipe (trachea) so you can deliver oxygen directly and use a ventilator to breathe for the person. This prevents aspiration (vomit going into the lungs), keeps oxygen levels safe, and buys time while the liver breaks down alcohol at a steady rate of about 15–20 mg/dL per hour (roughly one standard drink per hour).

- Give thiamine 100 mg intravenously (IV) or intramuscularly (IM) BEFORE giving any glucose or dextrose; follow with IV fluids (0.9% normal saline or lactated Ringer solution) at 100–200 mL per hour, plus folic acid 1 mg IV or by mouth daily, and a multivitamin → People who drink heavily for weeks, months, or years often have very low levels of thiamine (vitamin B₁) because alcohol damages the lining of the small intestine (where thiamine is absorbed), increases how much thiamine the kidneys throw away, and replaces food calories with alcohol calories (so they do not eat enough thiamine-rich foods like whole grains, pork, and legumes). Thiamine is essential for the brain to use glucose for energy. Without it, brain cells—especially in the thalamus and mammillary bodies (small structures deep in the middle of the brain that help with memory)—start to die, causing Wernicke encephalopathy: confusion, abnormal eye movements (nystagmus, which is bouncing or jerky eyes, or ophthalmoplegia, which is weak or paralyzed eye muscles), and loss of coordination (ataxia) (wobbly walking). If you give glucose (sugar) before thiamine, you can use up the last bit of thiamine the brain has and make Wernicke encephalopathy suddenly worse. If Wernicke encephalopathy is not treated quickly, it can turn into Korsakoff syndrome, a permanent condition with severe memory loss (anterograde amnesia—unable to form new memories) and confabulation (making up stories to fill memory gaps). Folic acid (vitamin B₉) deficiency also happens with heavy drinking and contributes to anemia (low red blood cells) and nerve damage (peripheral neuropathy, which causes numbness, tingling, or pain in the hands and feet). IV fluids correct dehydration from vomiting, sweating, and the diuretic effect of alcohol (alcohol suppresses antidiuretic hormone, or ADH, so you urinate more).
- Give dextrose 25–50 grams IV (one amp of D50W, which is 50% dextrose in water) if the fingerstick or serum glucose is below 70 mg/dL → Alcohol blocks the liver from making new glucose through a process called gluconeogenesis (making new sugar from other building blocks). Normally, when you have not eaten for several hours, the liver releases glucose into the blood to keep the brain fed. Alcohol shuts that down. If someone drinks heavily and skips meals, blood sugar can drop dangerously low (hypoglycemia). The brain depends entirely on glucose for energy—it cannot use fat or protein—so when glucose drops below 70 mg/dL, brain cells start to malfunction: confusion worsens, the person may have a seizure, and if it stays low, permanent brain damage or death can happen. Giving IV dextrose raises blood sugar within 1–2 minutes and often wakes the person up if low blood sugar (hypoglycemia) was the main problem.

- Give naloxone 0.4 to 2 mg IV, intramuscularly (IM), or by intranasal spray if opioid co-ingestion is suspected (clues: pinpoint pupils, breathing rate below 10 breaths per minute, deeply unresponsive, and not waking to loud voice or sternal rub) → Opioids (like heroin, fentanyl, oxycodone, morphine) bind to mu-opioid receptors in the medulla and spinal cord and powerfully depress breathing—often more than alcohol alone. When opioids and alcohol are taken together, the risk of stopped breathing (respiratory arrest) is much higher than with either drug alone because both act on the central nervous system in overlapping ways. Naloxone is a competitive antagonist—it kicks the opioid off the receptor and reverses the breathing depression, usually within 2–5 minutes. Pupils dilate, breathing rate picks up, and consciousness often improves. The effect lasts 30–90 minutes, so watch carefully—if the opioid is long-acting (like methadone or a fentanyl patch), the person can slip back into breathing depression when the naloxone wears off.
- Give fomepizole 15 mg per kilogram (mg/kg) IV as a loading dose over 30 minutes if toxic alcohol ingestion (methanol or ethylene glycol) is confirmed or strongly suspected based on osmolal gap above 10 mOsm/kg, anion-gap metabolic acidosis (acid building up in the blood), visual symptoms (methanol), or known ingestion history → Methanol (found in windshield washer fluid, racing fuel, and homemade or contaminated alcohol) and ethylene glycol (antifreeze, brake fluid) are themselves only mildly intoxicating, but the enzyme alcohol dehydrogenase in the liver converts them into highly toxic acids: methanol becomes formic acid (which damages the optic nerve and causes permanent blindness) and formaldehyde; ethylene glycol becomes glycolic acid and oxalic acid (which form sharp crystals in the kidneys and cause kidney failure). Fomepizole is a competitive inhibitor of alcohol dehydrogenase—it blocks the enzyme so the toxic alcohols cannot be converted into acids. The parent compounds (methanol and ethylene glycol) are then slowly eliminated by the kidneys, which is safe. Fomepizole is the preferred antidote because it is easier to use and safer than the older approach (IV ethanol infusion). In severe cases with too much acid in the blood (acidosis) already present (pH below 7.30) or very high levels, hemodialysis (an artificial kidney machine) is also needed to remove the toxic alcohols and acids quickly.

- Give a benzodiazepine—lorazepam 1–2 mg IV or by mouth, or diazepam 5–10 mg IV—if signs of alcohol withdrawal appear during observation: tremor (shaking hands), heart rate above 100 beats per minute, high blood pressure (systolic above 140 mmHg), heavy sweating, agitation, hallucinations (seeing bugs or shadows that are not there), or a seizure → When someone drinks alcohol daily for weeks or months, the brain adapts: it turns up the activity of glutamate (the main excitatory neurotransmitter, like the brain's gas pedal) and turns down the activity of GABA (gamma-aminobutyric acid, the main inhibitory neurotransmitter, like the brake). When alcohol is suddenly removed—whether the person stops drinking or is too sick to drink—the brake stays weak and the gas pedal stays floored. The result is hyperexcitability: tremor, sweating, fast heart rate, high blood pressure, anxiety, confusion, seizures, and in the worst cases delirium tremens (severe confusion, fever, hallucinations, and dangerous autonomic instability that can cause death). Benzodiazepines (like lorazepam and diazepam) bind to GABA-A receptors and boost the brake, restoring inhibition in the brain. This prevents seizures, treats agitation, lowers blood pressure and heart rate, and prevents progression to delirium tremens. Lorazepam is often preferred because it has a predictable duration and does not require the liver to activate it (important if the liver is damaged from chronic drinking). Diazepam is longer-acting and may be better for preventing repeated seizures.

- Before discharge, perform a brief intervention (5–15 minute structured conversation using the SBIRT framework: Screening, Brief Intervention, and Referral to Treatment) and refer to addiction medicine, psychiatry, or a primary care provider who can start medication-assisted treatment: naltrexone 50 mg by mouth once daily, or monthly injection (Vivitrol 380 mg IM); or acamprosate 333 mg tablets, two tablets three times daily (total 1998 mg per day); or disulfiram 250 mg by mouth once daily. Also connect the patient with social work or a care coordinator for housing assistance, mental health therapy (cognitive-behavioral therapy, CBT; or motivational enhancement therapy, MET), job or school support, and peer recovery support groups (such as Alcoholics Anonymous, AA; SMART Recovery; Refuge Recovery) → Alcohol use disorder is a chronic, relapsing brain disease—not a moral failing or lack of willpower. Repeated heavy drinking changes the reward circuitry in the brain (especially the mesolimbic dopamine pathway, which connects the ventral tegmental area in the midbrain to the nucleus accumbens in the front of the brain and drives cravings and reward, and the prefrontal cortex, which controls decision-making and impulse control) so that cravings become intense and decision-making is impaired. Without treatment, people cycle in and out of emergency departments, jails, and hospitals. Medication-assisted treatment works: naltrexone blocks mu-opioid receptors in the brain's reward system and reduces both craving and the pleasurable 'high' from drinking, making it easier to stay abstinent; acamprosate modulates glutamate and GABA transmission and reduces the physical and emotional discomfort (dysphoria, anxiety, insomnia) that lingers for weeks after stopping alcohol, which is a major driver of relapse; disulfiram inhibits aldehyde dehydrogenase (the enzyme that breaks down acetaldehyde, a toxic byproduct of alcohol), so if the person drinks while on disulfiram, they get a very unpleasant reaction (flushing, nausea, vomiting, headache, rapid heart rate) within 10–15 minutes, which creates a strong deterrent. Social determinants of health—stable housing, employment, supportive relationships, and treatment of co-occurring mental illness (depression, anxiety, PTSD, bipolar disorder)—are just as important as medication. Therapy helps people identify triggers, build coping skills, repair relationships, and find meaning and purpose beyond alcohol. Peer support groups offer community and accountability. Together, these interventions dramatically improve long-term recovery rates and reduce death, injury, and repeat emergency visits.

Medications

Thiamine (vitamin B1) · Water-soluble vitamin supplement

Thiamine is a helper the brain needs to turn sugar into energy and build the wrapping (myelin) around nerve wires. People who drink heavily often run low because alcohol stops the gut from soaking it up and the liver from storing it. When thiamine runs out, brain cells—especially in the thalamus (the relay station that keeps you alert and forms memories) and the cerebellum

(the balance center at the back of the brain)—start to die. We give thiamine before or with sugar water because flooding the brain with sugar when thiamine is already empty can push those cells over the edge and cause sudden, permanent brain damage (Wernicke encephalopathy). Thiamine refills the tank so the brain can safely use the sugar we give.

1. Thiamine enters the bloodstream from the IV line and travels to every cell.
2. Inside each cell, thiamine is turned into thiamine pyrophosphate (TPP), the active form that enzymes need to do their jobs.
3. TPP plugs into enzymes that run the Krebs cycle (the furnace that burns sugar for energy) and the pentose phosphate pathway (the assembly line that builds DNA and nerve insulation).
4. With TPP in place, brain cells can now safely use glucose (sugar from the IV) to make energy without making toxic waste (like lactate) that piles up and kills cells.
5. This prevents and reverses the confusion, frozen eye movements, and wobbly walk that signal Wernicke encephalopathy.

SAFETY WATCH

Very rarely, IV thiamine can trigger an allergic reaction (flushing, itching, tight throat, or anaphylaxis). Give it slowly over 30 minutes and watch closely for the first few minutes. · Always give thiamine BEFORE or WITH dextrose (sugar water), never after—giving sugar first when thiamine is low can cause sudden Wernicke encephalopathy. · Safe in pregnancy (Category A) and during breastfeeding.

Dextrose (intravenous glucose) · Simple sugar for energy replacement

Dextrose is pure glucose (the sugar the brain burns for fuel) dissolved in sterile water. Alcohol blocks the liver's assembly line (gluconeogenesis) that builds new glucose from stored parts, so blood sugar drops dangerously low (hypoglycemia). The brain depends completely on a steady glucose supply—it cannot store fuel or burn fat like muscles can. When glucose runs out, brain cells start misfiring, causing confusion, agitation, slurred speech, and seizures that look exactly like drunkenness. Giving IV dextrose instantly refills the fuel tank and wakes the brain back up. We give it through the vein (not by mouth) because intoxicated people cannot safely swallow and may choke or breathe the liquid into their lungs.

1. Dextrose enters the vein and dissolves into the bloodstream, raising blood glucose immediately.
2. Glucose crosses from blood into brain cells through GLUT-1 transporters (gate proteins that let glucose through the blood-brain barrier).

3. Inside the neuron, glucose enters glycolysis (the first stage of sugar burning), then the Krebs cycle (the main furnace), making ATP (adenosine triphosphate, the energy currency every cell uses).
4. ATP powers the sodium-potassium pumps (gates that move charged particles) that create the electrical charge neurons need to fire signals.
5. Confusion, combativeness, and slurred speech caused by low blood sugar (hypoglycemia) clear within 10–20 minutes as brain cells restart normal signaling.

SAFETY WATCH

Never give dextrose before thiamine in someone who drinks heavily or looks malnourished—flooding the brain with sugar when thiamine stores are empty can trigger sudden, permanent brain damage (Wernicke encephalopathy). · Giving too much dextrose too fast can cause a sugar overshoot (hyperglycemia) and make the body dump potassium into cells, lowering blood potassium (hypokalemia) and risking heart rhythm problems. Use D10 (10% dextrose) or D5 (5%) rather than D50 (50%) boluses when possible. · Check fingerstick glucose before and after to confirm low blood sugar (hypoglycemia) and guide dosing. · Extravasation (leaking out of the vein into tissue) can damage skin and tissue, especially with concentrated D50.

Folic acid (vitamin B9, folate) · Water-soluble vitamin supplement

Folic acid is a building block the body needs to copy DNA and make new red blood cells, white blood cells, and the insulation (myelin) that wraps nerve fibers. Alcohol blocks the gut from soaking up folate and the liver from storing and activating it. Over time, folate runs out, and the bone marrow cannot finish building red blood cells—they stay large, fragile, and too few (macrocytic anemia). The nervous system also suffers: low folate damages nerves in the arms and legs (causing numbness, tingling, and weakness) and raises homocysteine (a toxic amino acid linked to blood clots and brain fog). We give folic acid during acute intoxication to start refilling stores, prevent worsening anemia, and reduce the long-term nerve and heart damage seen in people with alcohol use disorder.

1. Folic acid enters the bloodstream and travels to the liver.
2. Liver enzymes (dihydrofolate reductase) convert folic acid into tetrahydrofolate (THF), the active helper molecule.
3. THF carries one-carbon units (tiny chemical pieces) needed to build the nucleotides (A, T, G, C) that form DNA.
4. In the bone marrow, THF lets early blood cells copy their DNA and divide normally, making healthy red blood cells instead of large, fragile ones.
5. Over weeks, anemia improves, energy returns, and nerve function steadies.

SAFETY WATCH

Always check vitamin B12 level before starting long-term folic acid, or give both together. Folate can correct the anemia of B12 deficiency but let the nerve damage (subacute combined degeneration of the spinal cord) keep getting worse unchecked. · High doses (above 1 mg per day) may rarely cause stomach upset, sleep trouble, or irritability. · Safe in pregnancy (in fact, essential to prevent spinal cord defects in the baby) and during breastfeeding. · Folic acid does NOT interact with alcohol, but continued heavy drinking will keep washing out any folate you give.

Naloxone (Narcan) · Opioid receptor blocker

Naloxone is used only when we suspect the person also took opioids (like heroin, fentanyl, oxycodone, or morphine) alongside alcohol. Opioids lock onto mu receptors (docking sites on brain cells that control pain and breathing) and turn down the breathing center in the brainstem, sometimes to the point where breathing stops. Naloxone is a key that kicks opioids off those receptors and blocks them from reattaching, instantly reversing the breathing suppression and sleepiness. We give it in suspected co-ingestion because alcohol and opioids together suppress breathing more than either drug alone (they team up), and naloxone can save a life. If no opioid is present, naloxone does nothing—it is completely safe and will not harm someone who is drunk on alcohol alone.

1. Naloxone is given IV, IM (into muscle), or intranasally (sprayed into the nose) and reaches the brain within 1–2 minutes (IV) or 3–5 minutes (nasal).
2. Naloxone molecules bind tightly to mu opioid receptors, kicking off any opioid molecules already attached.
3. With the receptors blocked, the opioid signal stops, and the brainstem breathing center resumes normal automatic breathing commands.
4. The person wakes up, pupils get less tiny (opioids cause pinpoint pupils; naloxone reverses this), and the breathing rate climbs back to 12–20 breaths per minute.
5. The effect lasts 30–90 minutes, shorter than most opioids, so repeat doses or a naloxone drip may be needed.

SAFETY WATCH

In opioid-dependent people, naloxone causes immediate withdrawal—be ready for agitation, combativeness, and vomiting. Use the lowest dose that brings breathing back (start 0.04–0.4 mg IV, go up slowly) rather than a full 2 mg bolus. · The effect wears off in 30–90 minutes, but many opioids (fentanyl, methadone, long-acting oxycodone) last much longer—watch for the person getting sleepy again and be ready to give more naloxone or start a drip. · Safe in pregnancy and does not harm a baby, though it may trigger opioid withdrawal in a mother and baby if both are opioid-dependent. · No serious side effects if given to someone without opioids on board—it simply does nothing.

Red flags

- Breathing rate below 8 breaths per minute or pauses between breaths
- Glasgow Coma Scale score of 8 or lower (won't open eyes even to pain, or no response at all)
- Known or suspected head injury, seizure, or fall—risk of brain bleed
- Vomiting while unconscious or very drowsy—can breathe vomit into the lungs and cause aspiration pneumonia
- Core body temperature below 95 °F (35 °C) or skin feels cold and clammy
- Fingertick blood glucose below 60 mg/dL

Nursing priorities

1. Position the patient on their left side in the recovery position (left lateral decubitus) if drowsy or vomiting, and keep suction at the bedside — Alcohol shuts down the gag reflex and airway protective reflexes; side-lying lets vomit drain out of the mouth instead of into the trachea (windpipe), preventing aspiration pneumonia and respiratory arrest
2. Check and record respiratory rate, oxygen saturation, and Glasgow Coma Scale score every 15 to 30 minutes until stable — Alcohol depresses the brainstem respiratory center in a dose-dependent way; respiratory rate below 8 or oxygen saturation below 90 percent signals severe CNS depression needing immediate airway support and possible intubation
3. Administer thiamine 100 mg intravenously or intramuscularly before giving any dextrose or glucose — Alcoholics are thiamine-deficient; giving glucose without thiamine first can trigger Wernicke encephalopathy (acute confusion, eye paralysis, loss of coordination (ataxia)) and permanent Korsakoff syndrome (irreversible memory loss)

4. Check fingerstick blood glucose immediately and treat low blood sugar (hypoglycemia) (glucose below 70 mg/dL) with dextrose 25 grams (50 mL of D50W) intravenously — Alcohol blocks the liver from making new glucose (gluconeogenesis) and chronic drinkers skip meals; low blood sugar (hypoglycemia) causes confusion and loss of consciousness that can mimic or worsen intoxication and cause permanent brain damage if untreated
5. Start an IV line and infuse 0.9 percent normal saline or lactated Ringer solution at 100 to 200 mL per hour — Alcohol is a diuretic (makes kidneys produce more urine) causing dehydration and electrolyte loss; IV fluids prevent low blood pressure (hypotension) (low blood pressure), support kidney blood flow (perfusion), and help the liver metabolize alcohol faster
6. Measure core body temperature with a rectal or esophageal thermometer if the patient was found outside or skin feels cold — Alcohol dilates blood vessels in the skin causing heat loss and impairs the hypothalamus (brain's thermostat); hypothermia (temperature below 95 °F) worsens CNS depression, causes arrhythmias, and can stop the heart
7. Reorient the patient frequently, keep the bed in low position with side rails up, and apply a bed alarm if the patient is confused or trying to get up — Alcohol causes loss of coordination (ataxia) (unsteady gait) and impairs judgment so patients do not realize they cannot walk; falls cause head injuries, hip fractures, and subdural hematomas that can be fatal
8. Prepare for endotracheal intubation if the Glasgow Coma Scale score is 8 or lower, or if the patient has no gag reflex and continues vomiting — A GCS of 8 or lower means the patient cannot protect the airway; without intubation the tongue falls back and blocks breathing or vomit enters the lungs causing respiratory arrest and death

Sources

- ACEP Clinical Policy on Acute Alcohol Intoxication
- NIAAA (National Institute on Alcohol Abuse and Alcoholism)
- ASAM Public Policy Statement on Alcohol Use Disorder
- AMA guidelines on screening and brief intervention for unhealthy alcohol use

Last reviewed: 2026-07-07