

Alcohol Intoxication

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

THE ONE-LINE TRUTH

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 chain — how it unfolds

1. Frontal cortex inhibition fails, so disinhibition and poor judgment appear. — why: 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). — why: 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). — why: 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. — why: 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). — why: 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. — why: 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. — why: 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. — why: 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).

Spot it — what you see, 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.

Red flags — act now

- 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

Treat it — in this order

1. 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).

2. 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).
3. 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.

4. 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.
5. 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.

Your top priorities at the bedside

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 — why: 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 — why: 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 — why: 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)