

Alcohol Intoxication

A plain-English guide for you and your family

WHAT IS HAPPENING

A healthy brain balances two kinds of chemical signals: glutamate (which speeds things up) and GABA (which slows things down). This balance keeps your judgment, coordination, memory, consciousness, and breathing all working smoothly. Alcohol is a depressant—a drug that slows brain activity. It shuts down brain regions in order from highest to lowest. The frontal cortex (the front part of your brain that controls judgment and self-control) goes offline first, so you lose your ability to stop risky impulses and make careful decisions. Next, the cerebellum (the back of your brain that controls balance and smooth movement) stops working well, causing stumbling, swaying, and slurred speech. Then the hippocampus (the memory-forming center deep in your brain) fails, leading to blackouts—you stay awake and moving but cannot form new memories, so you remember nothing the next day. As the blood alcohol level keeps rising, the reticular activating system (the brainstem network that keeps you awake) turns off and you become unresponsive. Finally, the medulla (the breathing control center at the base of your brainstem) shuts down, and breathing slows or stops completely. Every symptom you see—loss of self-control, unsteady walk, confusion, unconsciousness, slow breathing—is a predictable step in this top-down shutdown. Social factors matter enormously: a person who drinks alone, has untreated depression, or lacks a safe ride home faces far higher risk of aspiration (vomit entering the lungs), hypothermia (body temperature dropping dangerously low), or traumatic brain injury from falls. Treat the body and the social context together, or progress stalls.

What you might feel

- Disinhibition and impaired judgment
- Slurred speech (dysarthria)
- Loss of coordination (Ataxia) (unsteady walk, poor balance)
- Involuntary eye jerking (Nystagmus) (jerky, back-and-forth eye movements)
- Anterograde amnesia (blackout—can't form new memories)
- Nausea and vomiting
- Flushed skin and feeling warm

- Altered mental status progressing to stupor or coma
- Respiratory depression (slow, shallow breathing)
- Hypothermia (body temperature drops below 95 °F / 35 °C)
- Low blood sugar (Hypoglycemia) (blood sugar drops below 70 mg/dL)

Call the doctor right away if...

- 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
- Fingerstick blood glucose below 60 mg/dL

What the care team will do

- Position the patient on their left side in the recovery position (left lateral decubitus) if drowsy or vomiting, and keep suction at the bedside
- Check and record respiratory rate, oxygen saturation, and Glasgow Coma Scale score every 15 to 30 minutes until stable
- Administer thiamine 100 mg intravenously or intramuscularly before giving any dextrose or glucose
- 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
- Start an IV line and infuse 0.9 percent normal saline or lactated Ringer solution at 100 to 200 mL per hour
- Measure core body temperature with a rectal or esophageal thermometer if the patient was found outside or skin feels cold
- 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
- 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

Medicines you may be given — and why

Thiamine (vitamin B1)

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.

Dextrose (intravenous glucose)

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.

Folic acid (vitamin B9, folate)

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.

Naloxone (Narcan)

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.

Where this information comes from

- 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

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