

Alcohol Use Disorder

A plain-English guide for you and your family

WHAT IS HAPPENING

A healthy brain keeps three main chemical messengers balanced: dopamine (the pleasure and motivation signal), GABA (the calming signal that slows things down), and glutamate (the activating signal that speeds things up). This balance keeps your mood steady, your thinking clear, and your self-control strong. Alcohol floods the GABA system, which is why it feels calming at first—it temporarily quiets stress and anxiety. But the brain adapts: over months to years, it turns down its own calming signals and turns up the stress signals to try to stay in balance. Eventually, the brain becomes dependent on alcohol just to feel normal. Without it, withdrawal symptoms kick in—shaking, sweating, fast heartbeat, anxiety, and in severe cases, life-threatening seizures or delirium tremens (confusion, hallucinations, and dangerously unstable vital signs). The prefrontal cortex (the front part of the brain that controls impulses and weighs consequences) gets damaged by chronic alcohol exposure, so the very brain region you need to stop drinking is the one alcohol harms most. This creates a trap: drinking briefly fixes the craving and discomfort that drinking itself created. Social factors—trauma, unstable housing, job loss, isolation—make the circuit damage worse and recovery harder. Effective treatment tackles both the biology (medication to ease cravings and withdrawal, therapy for co-occurring depression or PTSD) and the social environment (stable housing, job support, trauma care) at the same time, because fixing one without the other usually fails.

What you might feel

- Tolerance
- Withdrawal symptoms
- Drinking more or longer than intended
- Persistent desire or unsuccessful efforts to cut down
- Spending a lot of time obtaining, using, or recovering from alcohol
- Cravings or strong urges to drink
- Failure to fulfill major responsibilities at work, school, or home
- Continued use despite recurrent social or interpersonal problems

- Giving up important activities because of alcohol
- Recurrent use in physically hazardous situations

Call the doctor right away if...

- Delirium tremens (DTs): severe confusion, seeing or feeling things that are not there (hallucinations), unstable vital signs (fever, fast heart over 120, high or swinging blood pressure), usually 2 to 4 days after the last drink—life-threatening and requires intensive care with IV benzodiazepines to calm the brain
- Seizures during withdrawal: full-body convulsions (generalized tonic-clonic or grand mal seizures), typically 6 to 48 hours after stopping alcohol—require emergency benzodiazepines and hospital admission to prevent status epilepticus (a seizure that will not stop) (seizures that do not stop)
- Wernicke encephalopathy: sudden confusion, paralyzed or wobbly eye movements (ophthalmoplegia), and unsteady walking (ataxia) from severe thiamine (vitamin B1) deficiency—give IV thiamine immediately before any glucose to prevent permanent memory damage (Korsakoff syndrome)
- Suicidal thoughts or suicide attempt: alcohol use disorder increases suicide risk tenfold—screen every visit, ask directly about plans to harm oneself, and arrange immediate safety (remove means, involve family, consider hospitalization)
- Mixing alcohol with opioids or benzodiazepines: both types of drugs slow breathing and brain activity; together they can stop breathing entirely (respiratory arrest) and cause death—always ask about all drugs and pills

What the care team will do

- Assess for withdrawal using the CIWA-Ar (Clinical Institute Withdrawal Assessment for Alcohol, revised) score every 1 to 4 hours depending on severity—check tremor, sweating, agitation, headache, nausea, hallucinations, anxiety, and orientation. A score of 8 or higher signals moderate withdrawal needing medication; 15 or higher means severe withdrawal.
- Administer benzodiazepines (most often lorazepam 1 to 4 mg IV or PO, diazepam 10 to 20 mg IV or PO, or chlordiazepoxide 50 to 100 mg PO) according to CIWA-Ar score or fixed-schedule protocol. Give lorazepam or oxazepam if liver disease is present because they bypass liver metabolism.
- Give thiamine 100 mg IV or IM before any IV dextrose (sugar water), then continue thiamine 100 mg daily by mouth for at least 3 to 5 days. Add folate 1 mg daily and a multivitamin.

- Monitor vital signs every 15 to 30 minutes in severe withdrawal or delirium tremens—watch for fever over 100.4°F (38°C), heart rate over 100 (tachycardia), systolic blood pressure over 140 or swinging by more than 20 points, respiratory rate over 20, and oxygen saturation below 94 percent. Place on continuous telemetry (heart monitor) if available.
- Maintain seizure precautions—pad side rails, keep suction and oxygen at bedside, turn the patient on their side if a seizure starts, note seizure time and duration, and do not insert anything into the mouth. Give lorazepam 2 to 4 mg IV push (over 2 minutes) for active seizure; repeat once if seizure continues after 5 minutes.
- Reorient the patient frequently if confused, keep the room calm and well-lit, and assign one-to-one observation if hallucinations or severe agitation are present. Explain all procedures simply and reassure the patient they are safe.
- Encourage oral fluids if the patient can swallow safely, or start IV normal saline at 100 to 150 mL/hour if dehydrated, vomiting, or unable to drink. Monitor intake and output (I&O), and check electrolytes daily—especially potassium, magnesium, and phosphate—and replace as ordered.
- Offer naltrexone 50 mg daily by mouth or monthly IM injection (380 mg Vivitrol), acamprosate 666 mg three times daily by mouth, or—if the patient requests it and understands the risks—disulfiram 250 mg daily by mouth after 12 hours alcohol-free. Coordinate with addiction counselor or social worker for outpatient follow-up and mutual-support groups (AA, SMART Recovery).

Medicines you may be given — and why

Naltrexone

Naltrexone sits on special spots in your brain called mu-opioid receptors (tiny docking stations on nerve cells that normally create pleasure feelings). By covering these spots, naltrexone blocks alcohol from triggering the reward rush that makes you want to drink more. Think of it like putting tape over a light switch so you can't flip it on. The drug doesn't make you sick or fix cravings directly—it just takes away the 'buzz' feeling that keeps the cycle going. This makes it easier to say no because drinking doesn't feel as rewarding anymore.

Acamprosate (Campral)

Long-term heavy drinking throws your brain's 'on-off switches' out of balance. Glutamate is the brain's 'speed-up' messenger, and GABA (gamma-aminobutyric acid) is the 'slow-down' messenger. After months or years of alcohol flooding your brain, your brain compensates by turning the glutamate system way up and the GABA system way down. When you stop drinking, your brain stays stuck in overdrive—racing thoughts, jumpy nerves, intense cravings. Acamprosate gently nudges both systems back toward normal, calming the glutamate storm

and helping GABA work again. Think of it like a thermostat resetting your brain's temperature after alcohol kept cranking the heat.

Disulfiram (Antabuse)

Disulfiram sabotages one key step in how your liver breaks down alcohol. Normally, alcohol is broken into acetaldehyde (a toxic chemical) and then quickly broken again into harmless vinegar-like stuff by an enzyme called aldehyde dehydrogenase. Disulfiram blocks that enzyme permanently, so acetaldehyde piles up in your blood like a backed-up drain. Within 10 minutes of drinking even a sip of alcohol, you feel flushed, hot, nauseous, dizzy, your heart races, your head throbs, and you may vomit. It's so unpleasant that the fear of this reaction helps motivated people stay away from alcohol. Think of it like a strict alarm that punishes any attempt to drink.

Benzodiazepines (lorazepam, diazepam, chlordiazepoxide)

When you suddenly stop drinking after heavy, long-term use, your brain goes haywire. Alcohol normally slams the brakes on brain activity by boosting GABA (gamma-aminobutyric acid, the calming messenger) and blocking glutamate (the revving-up messenger). Your brain adapts by cranking glutamate way up and GABA way down to stay balanced while alcohol is present. When alcohol leaves, your brain is suddenly all gas pedal and no brakes—nerve cells fire wildly, causing shaking, sweating, hallucinations, seizures, and sometimes delirium tremens (DTs), a life-threatening state of confusion, fever, and dangerously fast heartbeat. Benzodiazepines are emergency brakes. They boost GABA's calming power, slowing nerve firing back to safe levels and preventing seizures and DTs during the first 3 to 7 days of withdrawal.

Thiamine (Vitamin B1)

Thiamine is a vitamin your brain absolutely needs to burn sugar (glucose) for energy. Long-term heavy drinking causes thiamine deficiency in two ways: alcohol damages your stomach and intestine so you can't absorb thiamine from food, and malnutrition (skipping meals, eating mostly empty calories) means you're not getting enough thiamine in the first place. Without thiamine, brain cells—especially in the memory and coordination centers—starve and start dying within days. This causes Wernicke encephalopathy, a medical emergency with confusion, strange eye movements, and stumbling that can progress to coma or death. If not treated fast, it turns into Korsakoff syndrome, permanent severe memory loss where you can't form new memories and make up stories to fill the gaps (confabulation). Thiamine given early reverses Wernicke encephalopathy and prevents Korsakoff syndrome.

Where this information comes from

- APA DSM-5-TR 2022
- ASAM National Practice Guideline for Alcohol Use Disorder 2020

- SAMHSA TIP 45 Detoxification and Substance Abuse Treatment 2015
- VA/DoD Clinical Practice Guideline for Substance Use Disorders 2021

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