

Alcohol Use Disorder

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THE STORY IN ONE SENTENCE

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“Alcohol floods the brain's reward center with dopamine (the 'feel-good' messenger) and cranks up GABA (the 'calm-down' messenger) early, then the brain fights back by turning down its own calming signals and turning up its stress signals—so over time you need alcohol just to feel normal (dependence) and more of it to feel good (tolerance).”

Normal anatomy — the body's job

A healthy brain balances excitatory signals (glutamate, which activates neurons and sharpens focus) and inhibitory signals (GABA, which calms neurons and reduces anxiety), plus reward circuits (dopamine, which motivates behavior and marks experiences as 'do that again') and executive control (the prefrontal cortex, which applies the brakes to impulses and helps you plan ahead).

BODY'S JOB

Dopamine (the reward messenger) tells you what feels good and drives you to repeat behaviors that help you survive. GABA (the calming messenger) slows brain activity, lowers anxiety, and helps you relax. Glutamate (the activating messenger) speeds brain activity, sharpens focus, and keeps you alert. The prefrontal cortex (the very front of your brain, right behind your forehead) weighs short-term urges against long-term consequences, pumps the brakes on impulses, and helps you stick to plans even when something tempting is right in front of you.

1. The ventral tegmental area (VTA—a tiny cluster of dopamine-making neurons deep in the midbrain, just above the brainstem) sends dopamine to the nucleus accumbens (the brain's reward hub, sitting just above the brainstem) whenever you do something that feels good or helps you survive—eating, connecting with others, achieving a goal, or experiencing pleasure.
2. The nucleus accumbens tags that experience as 'good' and sends a signal up to the prefrontal cortex (the thinking and planning center at the very front of your brain, right behind your forehead) saying 'remember this—do it again.'
3. The prefrontal cortex weighs the signal against long-term consequences: 'Is this safe? Will it hurt me later? Do I have time for this?' It either gives the green light or applies the brakes, overriding the immediate urge in favor of a bigger goal.
4. Meanwhile, GABA neurons (scattered throughout the brain, especially in the cortex, amygdala, and cerebellum) act like a dimmer switch, calming overactive circuits and keeping anxiety, fear, and stress in check so you can think clearly and stay steady.
5. Glutamate neurons (found everywhere in the brain, especially in the cortex and hippocampus) act like the accelerator pedal, speeding up thinking, focus, and alertness so you can learn, react quickly, and stay sharp.
6. In a healthy brain, GABA and glutamate balance each other like a seesaw: enough glutamate to stay alert and focused, enough GABA to stay calm and relaxed, and the prefrontal cortex in charge of the final decision—deciding when to go and when to stop.

What breaks

Chronic alcohol exposure weakens (down-regulates) GABA receptors (the calming docking sites on neurons where GABA lands to slow things down) and strengthens (up-regulates) NMDA glutamate receptors (the activating docking sites where glutamate lands to speed things up). This means the brain becomes hyper-excitable—too easily activated and revved up—when alcohol is absent. At the same time, the prefrontal cortex (the brain region that stops impulses and manages stress) shrinks and loses gray matter (the neuron cell bodies that do the thinking) with repeated heavy drinking. This creates a vicious cycle: the person drinks to calm

stress and anxiety that the drinking itself has amplified, and the damaged prefrontal cortex can no longer pump the brakes to stop the cycle.

CLINICAL TERM

Neuroadaptation (the brain's compensatory remodeling in response to chronic alcohol exposure) shifts GABA-A receptor expression downward and NMDA glutamate receptor expression upward, producing hyperglutamatergic tone (excess excitatory signaling) during withdrawal. Chronic ethanol exposure also causes prefrontal cortex atrophy (shrinkage of the executive-control region) and hippocampal damage (injury to the memory-forming center), impairing insight, judgment, working memory, and the ability to inhibit drinking despite mounting medical, social, and legal consequences—the hallmark of Alcohol Use Disorder.

How the body compensates

Early on, the brain compensates by adjusting receptor numbers and neurotransmitter release so that the person feels nearly normal while drinking—tolerance (needing more alcohol to achieve the same calming or euphoric effect) masks the underlying damage. But tolerance is not protection—it's a warning sign. Sudden withdrawal or a medical crisis unmasks the compensatory changes, and withdrawal symptoms (tremor, seizures, delirium tremens) reveal just how dependent the brain has become.

1. The brain down-regulates (reduces the number of) its own GABA-A receptors and up-regulates (increases the number of) NMDA glutamate receptors to counterbalance alcohol's sedative effect. This means larger and larger doses of alcohol are needed to feel calm, relaxed, or 'normal'—this is tolerance. Tolerance is the brain trying to restore balance, but it also means the brain is now chemically remodeled and dependent.
2. When alcohol is suddenly stopped (or intake drops sharply), the unmasked hyperglutamatergic state (too much excitatory signaling with too little inhibitory tone) causes alcohol withdrawal syndrome: tremor (shaking hands), a fast heart rate (tachycardia) (heart rate over 100), high blood pressure (hypertension) (systolic blood pressure over 140), heavy sweating (diaphoresis) (drenching sweats), anxiety, insomnia, nausea, and overactive reflexes (hyperreflexia) (jumpy reflexes). In severe cases, generalized tonic-clonic seizures occur (usually 6–48 hours after the last drink), and delirium tremens (DTs) can develop 48–96 hours out—marked by confusion, visual or tactile hallucinations (seeing or feeling things that aren't there), and autonomic instability (wild swings in heart rate, blood pressure, and temperature) that can be fatal without treatment.

3. Chronic drinking depletes thiamine (vitamin B1) because alcohol damages the gut lining (reducing absorption) and increases the body's metabolic demand (alcohol breakdown uses up thiamine). Thiamine deficiency causes Wernicke encephalopathy: acute confusion, ophthalmoplegia (weakness or paralysis of the eye muscles, causing double vision or inability to move the eyes), and loss of coordination (ataxia) (unsteady, wide-based gait). If thiamine is not replaced immediately (IV thiamine before any glucose), the damage becomes permanent: Korsakoff syndrome—irreversible anterograde amnesia (inability to form new memories), retrograde amnesia (loss of old memories), and confabulation (unconsciously making up stories to fill memory gaps).
4. The liver tries to keep up by ramping up alcohol dehydrogenase (the main enzyme that breaks down alcohol in the liver) and activating the MEOS system (a backup alcohol-processing pathway involving the enzyme CYP2E1). But this produces toxic byproducts: acetaldehyde (a poison that damages DNA and proteins) and reactive oxygen species (unstable molecules that injure cells by stealing electrons). Over years, this leads to hepatic steatosis (fatty liver—fat droplets fill liver cells), alcoholic hepatitis (liver inflammation with fever, yellowing of the skin and eyes (jaundice), and abdominal pain), and eventually cirrhosis (permanent scarring that blocks blood flow through the liver and stops it from making proteins, clotting factors, and detoxifying ammonia).
5. Watch for red-flag emergencies: delirium tremens (DTs—confusion, hallucinations, autonomic instability, mortality up to 15% untreated), Wernicke encephalopathy (acute confusion, eye movement problems, loss of coordination (ataxia)—requires IV thiamine immediately), suicidal ideation (thinking about or planning suicide—present in up to 40% of people with AUD and a leading cause of death), and polysubstance overdose (combined use of alcohol with opioids or benzodiazepines, which can cause respiratory depression and death). Danger is highest during unsupervised withdrawal, after recent trauma or loss, and when co-occurring depression or PTSD is untreated.

Domino effect

1. Alcohol enhances GABA (the brain's main inhibitory neurotransmitter, which calms neurons) by binding to GABA-A receptors and making them more responsive, and it inhibits glutamate (the brain's main excitatory neurotransmitter, which activates neurons) by blocking NMDA receptors. This produces immediate anxiolysis (relief from anxiety) and euphoria (intense pleasure and relaxation). — The ventral tegmental area (VTA—a cluster of dopamine-making neurons deep in the midbrain) responds to alcohol by firing rapidly and sending a surge of dopamine to the nucleus accumbens (the brain's reward hub, sitting just above the brainstem). The nucleus accumbens tags alcohol as a powerful positive experience and signals the prefrontal cortex: 'Remember this—do it again.' This dopamine surge is the biological basis of reward and reinforcement, driving repeated drinking behavior.

2. Repeated alcohol exposure triggers neuroadaptation: GABA-A receptors down-regulate (the brain reduces their number and sensitivity) and NMDA glutamate receptors up-regulate (the brain increases their number and sensitivity), shifting baseline brain excitability upward. — The brain constantly seeks equilibrium (stable activity level). Because alcohol artificially boosts inhibition (via GABA) and suppresses excitation (via glutamate block), the brain compensates by reducing its own calming machinery and building more activating machinery. This is why tolerance develops—the same dose of alcohol now produces less calming effect, and the person needs more alcohol to feel relaxed or 'normal.' Tolerance is not safety; it's a sign the brain has chemically remodeled itself and is now dependent.
3. Without alcohol, the now-unopposed hyperglutamatergic state (excess excitatory tone with insufficient inhibitory tone) causes alcohol withdrawal syndrome: tremor (shaking, especially in the hands), a fast heart rate (tachycardia) (heart rate over 100), high blood pressure (hypertension) (systolic BP over 140), heavy sweating (diaphoresis) (heavy sweating), anxiety, insomnia, nausea, and overactive reflexes (hyperreflexia) (exaggerated reflexes). Severe withdrawal includes generalized tonic-clonic seizures (6–48 hours after last drink) and delirium tremens (DTs—48–96 hours out, with confusion, hallucinations, and autonomic instability). — The brain's compensatory up-regulation of excitatory circuits (more NMDA receptors, less GABA tone) is suddenly unmasked when alcohol—the sedative that was holding the system in check—is removed. Autonomic hyperactivity (overactive sympathetic nervous system—the fight-or-flight system) and CNS excitotoxicity (neuron injury from too much glutamate flooding the cells) produce the withdrawal syndrome. Untreated delirium tremens has a mortality rate of 5–15% due to cardiovascular collapse, hyperthermia (dangerously high body temperature), or seizure complications.
4. Chronic alcohol exposure damages the prefrontal cortex (the executive control center at the front of the brain, responsible for decision-making, impulse control, and planning) and the hippocampus (the memory center deep in the temporal lobe, responsible for forming and storing new memories). This impairs judgment, impulse control, working memory, and insight—making it nearly impossible to stop drinking despite mounting medical, financial, social, and legal consequences. — Gray matter atrophy (shrinkage of the neuron cell bodies) in the prefrontal cortex removes the brain's 'brakes' on impulsive behavior. The person knows intellectually that drinking is harmful—they can list the consequences—but they cannot override the craving, delay gratification, or plan effectively for long-term benefit over short-term relief. Hippocampal damage impairs memory consolidation, so the person may forget the severity of past consequences or fail to learn from repeated relapses. This is not a moral failure; it is structural brain damage that undermines the very capacities needed for behavior change.

5. Thiamine (vitamin B1) deficiency develops because alcohol impairs thiamine absorption in the small intestine (by damaging the gut lining and interfering with active transport) and increases thiamine consumption (the body uses more thiamine to metabolize alcohol). Thiamine is essential for glucose metabolism in neurons, so deficiency starves high-demand brain regions—the mammillary bodies (tiny memory relay stations at the base of the hypothalamus) and the thalamus (the sensory gateway in the center of the brain)—causing Wernicke encephalopathy and, if untreated, Korsakoff syndrome. — Neurons rely exclusively on glucose for energy, and thiamine is a required cofactor (helper molecule) for enzymes that break down glucose. Without thiamine, neurons cannot use glucose and begin to die. Wernicke encephalopathy presents acutely with the classic triad: confusion, ophthalmoplegia (weakness or paralysis of eye muscles—look for involuntary eye jerking (nystagmus), or jerky eye movements, and inability to move eyes outward), and loss of coordination (ataxia) (wide-based, unsteady gait). If IV thiamine is not given immediately—before any glucose infusion, which can precipitate acute crisis—the damage becomes permanent: Korsakoff syndrome, marked by irreversible anterograde amnesia (cannot form new memories), retrograde amnesia (loss of old memories), apathy, and confabulation (unconsciously fabricating stories to fill memory gaps without intent to deceive).
6. Co-occurring mental health disorders—major depressive disorder (MDD), post-traumatic stress disorder (PTSD), generalized anxiety disorder (GAD), and bipolar disorder—are present in 50–70% of people with AUD and independently predict relapse, worse functional outcomes, and higher suicide risk. — The brain circuits that regulate stress, reward, and emotion overlap heavily. Chronic stress and untreated depression or trauma keep cortisol (the body's main stress hormone, released by the adrenal glands) elevated, which sensitizes (makes more reactive) the amygdala (the brain's alarm center) and dysregulates the hypothalamic-pituitary-adrenal (HPA) axis (the body's stress-response system). This drives continued drinking as self-medication: alcohol temporarily calms the hyperactive stress system and numbs emotional pain, but it worsens depression and anxiety over time. Treating AUD without addressing co-occurring psychiatric illness leads to high relapse rates because the underlying driver—untreated mental suffering—remains.
7. Social determinants of health—housing instability, unemployment, food insecurity, trauma history (especially childhood trauma), incarceration, and lack of social support—independently predict AUD severity, treatment dropout, and relapse. — Chronic stress from unstable housing, violence, or financial crisis keeps the HPA axis activated, which sensitizes the brain's reward and stress circuits and makes alcohol cues more powerful and harder to resist. Without stable housing, employment, or a supportive social network, the person returns to alcohol as the only available coping mechanism. Recovery requires addressing both the biology (medication-assisted treatment, therapy) and the social environment (stable housing, job training, trauma-informed care, peer support) simultaneously—fixing one without the other usually fails.

- 8.** Long-term heavy drinking causes progressive liver damage: hepatic steatosis (fatty liver—fat droplets accumulate in hepatocytes), alcoholic hepatitis (acute inflammation with yellowing of the skin and eyes (jaundice), fever, and right upper quadrant pain), and cirrhosis (permanent scarring that blocks blood flow through the liver). Cirrhosis leads to portal high blood pressure (hypertension) (high pressure in the portal vein, which brings blood from the intestines to the liver), causing esophageal varices (swollen, fragile veins in the esophagus that can rupture and bleed massively), fluid buildup in the belly (ascites) (fluid accumulation in the abdomen), hepatic encephalopathy (confusion and altered mental status from ammonia buildup the liver can no longer clear), and hepatorenal syndrome (kidney failure triggered by severe liver failure). — The liver metabolizes alcohol via alcohol dehydrogenase into acetaldehyde (a highly toxic compound that damages DNA, proteins, and cell membranes) and then into acetate. Acetaldehyde triggers oxidative stress (floods cells with reactive oxygen species) and inflammation, directly injuring hepatocytes (liver cells). Over years, repeated injury causes fibrosis (scar tissue formation), which stiffens the liver and blocks blood flow. Once cirrhosis develops, the liver cannot synthesize clotting factors (leading to bleeding risk), albumin (leading to swelling (edema) and fluid buildup in the belly (ascites)), or detoxify ammonia (leading to encephalopathy). Portal high blood pressure (hypertension) shunts blood around the liver, causing varices that can rupture without warning and bleed fatally. Cirrhosis also dramatically increases the risk of hepatocellular carcinoma (liver cancer).

Symptoms · what the patient feels, and why

- **Tolerance:** The brain tries to balance out alcohol's calming effect by turning down its own 'slow-down' switches (GABA receptors—docking sites on nerve cells that respond to the brain's main calming messenger) and turning up its 'speed-up' switches (glutamate receptors—docking sites that respond to the brain's main exciting messenger). Over time, the same amount of alcohol does less, so a person needs to drink more and more to feel the same buzz.
- **Withdrawal symptoms:** When alcohol suddenly stops, all those 'speed-up' switches are still cranked high, but the braking effect of alcohol is gone. The brain races out of control, causing shaking hands (tremor), a pounding fast heart (tachycardia—heart beating over 100 times per minute), high blood pressure, heavy sweating, anxiety, trouble sleeping, and nausea. In severe cases, this overactive brain can trigger seizures (sudden electrical storms in the brain) or delirium tremens (DTs—dangerous confusion with hallucinations and unstable blood pressure and heart rate, usually starting 2 to 4 days after the last drink).

- Drinking more or longer than intended: Alcohol shrinks and weakens the front part of the brain (the prefrontal cortex—the area that controls impulses and plans ahead). At the same time, a deeper reward center (the nucleus accumbens—the brain's pleasure hub deep beneath the forehead) floods with dopamine (a brain messenger that says 'do that again!') every time the person drinks. Together, these changes make it almost impossible to stick to a plan like 'just one beer' once drinking starts.
- Persistent desire or unsuccessful efforts to cut down: The control circuits in the prefrontal cortex (the front of the brain that pumps the brakes on urges) are damaged, so willpower weakens. Meanwhile, the brain's stress and craving circuits (especially the amygdala, the brain's alarm center, and the anterior cingulate cortex, which flags conflicts between what you want and what you should do) become extra sensitive—they fire more easily, making every urge to drink feel overwhelming. Even when the person sees the harm and truly wants to quit, the brain's wiring works against them.
- Spending a lot of time obtaining, using, or recovering from alcohol: The brain's reward system (centered in the ventral tegmental area and nucleus accumbens—deep structures that drive motivation and pleasure) gets hijacked: it now treats alcohol as the top priority, above hobbies, friends, and work. Getting alcohol, drinking it, and then recovering from being drunk or withdrawing can eat up hours or days at a time.
- Cravings or strong urges to drink: Reminders of alcohol—seeing a bottle, smelling liquor, or feeling stressed—trigger a burst of dopamine (the brain's 'do that again' messenger) in the reward circuits, and that burst screams 'drink now!' The brain's glutamate system (the main excitatory messenger that controls excitement and learning) also goes into overdrive during stress or early withdrawal, making cravings feel impossible to ignore.
- Failure to fulfill major responsibilities at work, school, or home: Being drunk, going through withdrawal, or spending time hunting for the next drink all interfere with showing up and doing a good job. Chronic alcohol use also slows thinking, weakens memory, and clouds judgment because it damages the hippocampus (the brain's memory-maker) and the prefrontal cortex (the planning center), so even when the person is present, performance suffers.
- Continued use despite recurrent social or interpersonal problems: Damage to the prefrontal cortex (the front control region) reduces self-awareness (insight—the ability to see oneself clearly), so the person may not fully grasp how drinking is wrecking relationships. The brain also prioritizes short-term relief (the next drink calms anxiety or craving right now) over long-term consequences like broken friendships or family conflict.
- Giving up important activities because of alcohol: The reward system now associates pleasure almost exclusively with alcohol. Activities that used to feel fun—sports, hobbies, time with friends—no longer trigger enough dopamine (the feel-good messenger) to feel rewarding, so they are abandoned. Alcohol becomes the only thing that feels worth doing.

- Recurrent use in physically hazardous situations: The prefrontal cortex (the judgment and safety-checking area at the front of the brain) is impaired, and intense cravings override common sense. The person may drive drunk, operate dangerous machinery, swim while intoxicated, or take other life-threatening risks because the compulsion to drink—or the clouded thinking while drunk—blocks awareness of danger.

Focused exam

- AUDIT-C score 4 or higher in men or 3 or higher in women: This three-question tool catches unhealthy drinking before organs are damaged. The questions ask how often you drink, how many drinks you have, and how often you have six or more drinks at once. A positive score means the brain is being exposed to enough alcohol to change its chemistry and increase the risk of liver disease, heart problems, cancer, and accidents. The USPSTF (U.S. Preventive Services Task Force, the national expert panel that reviews evidence for screening tests) gives alcohol screening a Grade B recommendation, meaning every adult should be screened every year.
- Two or more 'yes' answers on the CAGE questionnaire (Cut down, Annoyed, Guilty, Eye-opener): This simple four-question screen reveals whether drinking has started to control you instead of the other way around. 'Have you ever felt you should cut down?' 'Have people annoyed you by criticizing your drinking?' 'Have you felt guilty about drinking?' 'Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover?' Two or more yes answers mean the reward circuit in your brain (the pathway that drives motivation and pleasure using dopamine, a chemical messenger) has been hijacked by alcohol. This is not a character flaw—it is a measurable change in brain wiring.
- Tremor, sweating, or fast heart rate when at rest or in the morning: These are early signs of alcohol withdrawal. Alcohol acts like a brake on the nervous system by boosting GABA (gamma-aminobutyric acid, the brain's main calming chemical). With repeated heavy drinking, the brain adapts by turning down its own GABA activity and turning up glutamate (the brain's main activating chemical) to balance things out. When you suddenly stop drinking, the brake is gone but the gas pedal is still pressed—your nervous system goes into overdrive. Heart rate climbs, hands shake, and sweating begins. If untreated, this can escalate within hours to days into seizures (sudden uncontrolled electrical storms in the brain) or delirium tremens (severe confusion, hallucinations, and dangerously high blood pressure and heart rate). Withdrawal seizures happen in 5 to 15 out of every 100 people who stop drinking suddenly after chronic heavy use, usually 12 to 48 hours after the last drink.

- Confusion, seeing or hearing things that are not real, or severe agitation: This is delirium tremens, or DTs—a life-threatening emergency. The brain's electrical activity spirals out of control because inhibitory (calming) signals are too weak and excitatory (activating) signals are too strong. People may see insects crawling on the walls, hear voices, or become so confused they do not know where they are. Blood pressure and heart rate spike. Without treatment in a hospital with benzodiazepines (medications that calm the overactive brain by mimicking GABA) and close monitoring, 5 to 15 out of every 100 people with DTs will die from heart arrhythmias (irregular heartbeats), respiratory failure (lungs stop working), or complications like aspiration pneumonia (food or vomit getting into the lungs).
- Memory blackouts during drinking or repeated failed attempts to cut back: Blackouts are periods where you cannot form new memories even though you are awake and moving around. Alcohol shuts down the hippocampus (the brain's memory-recording center), so the next day there are gaps—sometimes hours—you cannot remember. Blackouts mean alcohol levels in the brain were high enough to damage normal function. Repeated failed attempts to cut back or quit mean the prefrontal cortex (the brain region responsible for planning, self-control, and weighing consequences) has lost the ability to override the reward circuit. The mesolimbic dopamine pathway (the circuit from the ventral tegmental area to the nucleus accumbens that says 'I need this now' using dopamine) has become stronger than rational decision-making. This is one of the eleven DSM-5-TR (Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision, the official guidebook doctors use to diagnose mental health and substance use disorders) criteria for alcohol use disorder. Two or more of the eleven criteria in a twelve-month period equals alcohol use disorder.
- Job loss, damaged relationships, or legal problems directly caused by drinking: Alcohol use disorder does not just harm the body—it destroys the structure of daily life. Missing work, fighting with family, getting arrested for DUI (driving under the influence)—these are not separate problems; they are symptoms of the brain disease. The reward circuit has overridden the brain's ability to care about long-term goals, relationships, or safety. Social damage also creates a vicious cycle: losing your job or your family increases stress and isolation, which increases craving and relapse risk. Treatment must address both the brain changes and the wreckage in a person's life, or neither will improve.

Diagnostics · every test answers a question

- Liver enzymes: AST (aspartate aminotransferase), ALT (alanine aminotransferase), and GGT (gamma-glutamyl transferase) → Is chronic alcohol use damaging the liver—causing inflammation (hepatitis, where liver cells are injured and release enzymes into the blood) or scarring (cirrhosis, where scar tissue blocks blood flow and the liver cannot filter toxins)?
- Mean corpuscular volume (MCV), which measures the average size of your red blood cells → Is chronic alcohol use causing red blood cells to become larger than normal (macrocytosis)?

- Complete blood count (CBC) with platelets → Is alcohol suppressing the bone marrow or is advanced liver disease causing low platelet counts?
- Hepatitis B surface antigen (HBsAg) and hepatitis C antibody with reflex to HCV RNA if antibody is positive → Does the patient have a viral infection in the liver that is being accelerated by alcohol?
- Lipid panel (total cholesterol, LDL, HDL, and triglycerides) → Is alcohol causing the liver to overproduce triglycerides (a type of fat in the blood)?
- Thiamine (vitamin B1) level, though empiric IV or IM thiamine is given before the lab result returns → Is the person at risk for Wernicke encephalopathy (acute, reversible brain damage caused by thiamine deficiency)?
- Carbohydrate-deficient transferrin (CDT) if diagnosis is uncertain or monitoring abstinence is needed → Is there biochemical evidence of chronic heavy alcohol use over the past 2 to 3 weeks?
- Blood alcohol concentration (BAC) if intoxication or withdrawal severity assessment is needed → Is alcohol currently in the bloodstream, and at what level?

Treatment logic

- Screen all adults age 18 and older annually with AUDIT-C (men score 4 or higher, women 3 or higher equals positive) or single-question screen ('How many times in the past year have you had 5 or more drinks in a day for men, or 4 or more drinks in a day for women?'—one or more times equals positive). Then confirm diagnosis using DSM-5-TR criteria: 2 or more of 11 symptoms in a 12-month period equals alcohol use disorder. → Universal screening catches unhealthy drinking and alcohol use disorder before cirrhosis, pancreatitis, heart failure, or cancer develops. Detecting the problem early allows brief intervention—a 5 to 15 minute conversation by any clinician (doctor, nurse, pharmacist) using motivational interviewing techniques (asking open-ended questions, listening without judgment, and helping the person identify their own reasons to change). Brief intervention reduces drinking in 1 out of 4 people who screen positive but do not yet meet criteria for alcohol use disorder. For people with alcohol use disorder, screening opens the door to pharmacotherapy (medication treatment) and specialty referral. The USPSTF gives screening and brief intervention a Grade B recommendation, meaning there is high certainty of moderate benefit and it should be standard care for every adult every year, just like blood pressure screening.

- Naltrexone 50 mg by mouth once daily or 380 mg intramuscular injection once monthly (blocks mu-opioid receptors in the mesolimbic reward pathway, reducing the euphoria and reinforcement from drinking) OR acamprosate 666 mg by mouth three times daily (modulates GABA and glutamate activity in the brain, reducing craving and the discomfort of early abstinence) → Both medications are FDA-approved for alcohol use disorder and recommended as first-line by APA, ASAM, and the VA/DoD (Veterans Affairs and Department of Defense). Meta-analyses (studies that combine results from many trials) show naltrexone reduces heavy drinking days by about 4 days per month and cuts the chance of relapse to heavy drinking by 36 percent. Acamprosate is equally effective and is the better choice if liver disease is present because it is not metabolized by the liver. Naltrexone works by blocking the endogenous opioid (the body's own morphine-like chemicals) release that normally happens when you drink, so alcohol feels less rewarding. Acamprosate restores the balance between GABA (the brain's main calming chemical) and glutamate (the brain's main activating chemical) that is disrupted by chronic alcohol exposure. Both medications work best when combined with counseling, but medication alone is more effective than counseling alone. These are not 'willpower pills'—they change brain chemistry to give the prefrontal cortex (the front of the brain that handles decision-making and self-control) a fighting chance against the hijacked reward circuit.
- Benzodiazepine-based alcohol withdrawal protocol using CIWA-Ar (Clinical Institute Withdrawal Assessment for Alcohol, revised—a 10-item scale that scores tremor, sweating, agitation, headache, nausea, and hallucinations) with symptom-triggered dosing of lorazepam 1 to 2 mg or chlordiazepoxide 25 to 100 mg every 1 to 2 hours as needed until CIWA-Ar is below 8, plus thiamine 500 mg IV or IM three times daily for at least 3 days → Benzodiazepines are GABA-A receptor agonists, meaning they activate the same calming receptors that alcohol does. This prevents the uncontrolled excitatory rebound (the glutamate surge in the brain) that causes withdrawal seizures and delirium tremens. Symptom-triggered dosing—giving medication only when withdrawal symptoms appear rather than on a fixed schedule—uses 50 percent less total benzodiazepine and shortens hospital stay compared to fixed-schedule dosing, according to ASAM and APA guidelines. Withdrawal seizures occur in 5 to 15 percent of people who stop drinking abruptly after chronic heavy use; benzodiazepines reduce seizure risk to near zero. Thiamine prevents Wernicke encephalopathy (acute brain damage from thiamine deficiency). This is the only pharmacologic approach with strong evidence for managing acute alcohol withdrawal. The brain's GABA and glutamate systems recalibrate over 3 to 7 days, then benzodiazepines are tapered and stopped. Outpatient withdrawal management is safe for people with mild withdrawal (CIWA-Ar under 10), no history of seizures or DTs, and a supportive home environment. Inpatient management is needed for severe withdrawal, medical comorbidities (other diseases like heart failure or infection), or lack of social support.

- Screen for and treat co-occurring major depressive disorder, generalized anxiety disorder, PTSD (post-traumatic stress disorder), or bipolar disorder with evidence-based psychotherapy (cognitive-behavioral therapy, trauma-focused therapy such as prolonged exposure or EMDR—eye movement desensitization and reprocessing) and/or antidepressants (SSRIs like sertraline or escitalopram, SNRIs like venlafaxine) → About half of people with alcohol use disorder also have a co-occurring mental health disorder. Depression and anxiety are not just complications of drinking—they often came first and increase relapse risk by 2 to 3 times because the person uses alcohol to self-medicate (to numb emotional pain or quiet intrusive thoughts). Untreated trauma reactivates the amygdala (the brain's alarm center that flags danger and stores emotional memories) and the hypothalamic-pituitary-adrenal axis (the body's stress hormone system), which increases craving and impulsive drinking. Integrated treatment—addressing both the substance use disorder and the mental health disorder at the same time by the same clinical team—improves outcomes for both. SSRIs and SNRIs restore serotonin and norepinephrine signaling in the brain, reducing depression and anxiety. Cognitive-behavioral therapy teaches skills to challenge distorted thoughts ('I am worthless,' 'nothing will ever get better') and to break the link between emotions and drinking. Trauma-focused therapy processes traumatic memories so they lose their emotional charge. Treating the mental health disorder reduces the neurobiological drive to drink and gives the person a reason to stay sober.
- Address social determinants of health: connect to stable housing (transitional housing, sober living, Housing First programs), food assistance (SNAP—Supplemental Nutrition Assistance Program), income support (SSI/SSDI—Supplemental Security Income or Social Security Disability Insurance for people unable to work, TANF—Temporary Assistance for Needy Families), and job training or supported employment programs → Homelessness, food insecurity, and unemployment create chronic activation of the stress axis in the brain. The hypothalamus (a control center deep in the brain) releases CRH (corticotropin-releasing hormone), the pituitary gland (a small gland under the brain) releases ACTH (adrenocorticotrophic hormone), and the adrenal glands (glands on top of the kidneys) release cortisol (the body's main stress hormone). High cortisol strengthens the amygdala's emotional memory circuits and weakens the prefrontal cortex's executive control, which increases craving and impulsive relapse. Studies show that providing stable housing through Housing First programs (giving people an apartment with no requirement to be sober first) reduces heavy drinking by 40 to 50 percent and increases engagement in treatment by nearly 60 percent. A safe place to sleep, regular meals, and a daily routine allow the prefrontal cortex to recover from alcohol-induced atrophy (shrinkage). MRI studies show the prefrontal cortex begins to regrow within 6 to 12 months of abstinence—but only if the environment is stable. This is not just social work; it is neuroscience-informed care. Addressing social determinants is harm reduction and is as evidence-based as any medication.

- Mutual-support groups (Alcoholics Anonymous, SMART Recovery—Self-Management and Recovery Training, Refuge Recovery, LifeRing) or individual cognitive-behavioral therapy (CBT), motivational enhancement therapy (MET), or contingency management (rewarding abstinence with vouchers or prizes) → Psychosocial interventions build new neural pathways that compete with alcohol's reward signal. Cognitive-behavioral therapy identifies triggers (people, places, emotions, times of day) and teaches coping skills (delay, distraction, calling a friend, urge surfing—riding out a craving like a wave without acting on it). MET uses reflective listening and open-ended questions to resolve ambivalence ('part of me wants to quit, part of me does not') and strengthen internal motivation. Contingency management provides immediate, tangible rewards (gift cards, vouchers) for verified abstinence (negative urine or breathalyzer tests), which activates dopamine release in the nucleus accumbens (the brain's reward center) and reinforces sobriety. Mutual-support groups provide social connection, which triggers oxytocin and dopamine—natural feel-good chemicals that compete with alcohol's effects. Hearing stories from people further along in recovery activates mirror neurons (brain cells that fire both when you act and when you watch someone else act) and builds hope. These interventions work through neuroplasticity—the brain's ability to rewire itself. The more you practice a new behavior (calling a friend instead of drinking, going to a meeting instead of a bar), the stronger that neural pathway becomes and the weaker the old drinking pathway becomes.
- Disulfiram 250 mg by mouth daily (blocks aldehyde dehydrogenase, the enzyme that breaks down acetaldehyde—a toxic byproduct of alcohol—so drinking causes immediate flushing, nausea, vomiting, headache, and chest pain) → Disulfiram creates a strong deterrent by making drinking physically unpleasant and even dangerous (severe reactions can cause low blood pressure (hypotension)—low blood pressure, arrhythmias—irregular heartbeats, and respiratory depression—slow, shallow breathing). It works best in people with high motivation, external accountability (a family member observes daily dosing), and low impulsivity. It does not reduce craving—it works by creating a consequence. Some studies show it is as effective as naltrexone when adherence is supervised. It is not first-line because adherence is poor when unsupervised (people simply stop taking it before drinking), and it requires abstinence before starting (if you drink while on disulfiram, you can become very sick). It is contraindicated (should not be used) in people with heart disease, liver disease, psychosis, or pregnancy.

Medications

Naltrexone · Opioid receptor blocker

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.

1. You take naltrexone as a 50 mg pill once daily, or get a monthly 380 mg shot (Vivitrol) into your buttock muscle that slowly releases over 4 weeks.
2. The drug travels through your bloodstream to your brain within 1 hour.
3. Naltrexone molecules land on and cover mu-opioid receptors, blocking them like a key stuck in a lock.
4. When alcohol enters your system, it can't activate those receptors to release dopamine (the pleasure messenger).
5. Without the dopamine reward, your brain doesn't learn to crave alcohol as strongly, and drinking loses its appeal over time.
6. The medicine stays in your system blocking receptors for 24 hours (pill form) or 30 days (injection form).

SAFETY WATCH

NEVER start naltrexone if you're taking any opioid pain medicine (like oxycodone, hydrocodone, morphine, tramadol, or buprenorphine) or if you've used opioids in the past 7 to 10 days—it will cause sudden, severe withdrawal with vomiting, diarrhea, body aches, and panic. · Tell your doctor if you have hepatitis or liver failure right now; naltrexone can stress the liver further. Your doctor will check liver blood tests before starting and every few months. · If you need surgery or emergency pain relief, naltrexone blocks opioid painkillers, so doctors must use higher doses or different pain medicines. Wear a medical alert bracelet. · The injection site (buttock) may be sore, hard, or have a lump for weeks; rarely it can cause serious tissue damage or infection. · Common mild side effects: nausea, headache, dizziness, tiredness, trouble sleeping. These usually fade after the first week.

Acamprosate (Campral) · Glutamate modulator and GABA restorer

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.

1. You take two 333 mg tablets three times a day (total 1998 mg daily), with or without food.

2. Acamprosate is absorbed in your intestine and travels to your brain within 3 to 8 hours.
3. The drug weakly blocks overactive NMDA glutamate receptors, dialing down the 'speed-up' signals.
4. At the same time, it helps GABA-A receptors work better, boosting the 'slow-down' signals.
5. This rebalancing reduces the brain hyperactivity that causes cravings, anxiety, and sleeplessness after you quit drinking.
6. The drug does NOT get you high, does NOT interact with alcohol, and takes 5 to 7 days to build up and start working fully.

SAFETY WATCH

Tell your doctor if you have kidney disease. Acamprosate leaves your body through urine, so if your kidneys don't work well (creatinine clearance below 30 mL/min), the drug builds up and can be dangerous. Your doctor may lower the dose or choose a different medicine. · Most common side effect is diarrhea (watery stools), which usually gets better after the first week. Taking it with food may help. · Other mild side effects: stomach cramps, gas, itching, dizziness, mild anxiety. Rarely causes depression or suicidal thoughts—tell your doctor right away if your mood crashes. · Safe in pregnancy (Category C—some animal studies show risk, but human data limited). Discuss with your OB if pregnant or planning pregnancy. · No abuse potential; you can stop it suddenly without withdrawal symptoms if needed.

Disulfiram (Antabuse) · Aldehyde dehydrogenase inhibitor (alcohol-sensitizing agent)

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.

1. You take one 250 mg tablet daily (sometimes 500 mg, based on doctor's decision), usually in the morning.
2. Disulfiram is absorbed in your intestine and carried to your liver within a few hours.
3. The drug permanently locks onto aldehyde dehydrogenase, shutting it down. Your liver must make brand-new enzyme molecules over the next 1 to 2 weeks to recover.

4. If you drink alcohol anytime in the next 1 to 2 weeks, acetaldehyde shoots up 5 to 10 times higher than normal in your blood.
5. This floods your body with toxins, causing flushing (red, burning face and chest), pounding headache, nausea, vomiting, chest tightness, rapid heartbeat, low blood pressure, and confusion. The reaction lasts 30 minutes to several hours depending on how much you drank.
6. Because the enzyme is permanently blocked, even stopping disulfiram today means a reaction can still happen if you drink within 2 weeks.

SAFETY WATCH

NEVER give disulfiram to someone who is drunk, who didn't agree to take it, or who can't understand the risks. Informed consent is required by law and ethics. · Dangerous or forbidden in patients with heart disease (coronary artery disease, heart failure), stroke history, severe lung disease, kidney failure, liver failure, psychosis, or seizure disorders—the alcohol reaction can trigger heart attack, dangerous heart rhythms, or stroke. · Hidden alcohol is everywhere: cough syrup, mouthwash, vanilla extract, vinegar sauces, communion wine, some desserts, aftershave, hand sanitizer. Even small amounts can cause a reaction. Read all labels. · Liver damage risk: Check liver blood tests before starting, then monthly for 6 months, then every 3 to 6 months. Stop immediately if liver enzymes rise or you develop yellow skin or eyes (jaundice), dark urine, or severe belly pain. · Never take disulfiram with metronidazole (antibiotic) or certain other drugs—causes psychosis-like confusion. · Pregnancy category C—can harm the developing baby. Use effective birth control or avoid if pregnant or breastfeeding. · Common mild side effects: metallic or garlic-like taste in mouth, drowsiness, skin rash, headache. Rarely causes nerve damage (tingling, numbness in feet) or vision changes if used for many months.

Benzodiazepines (lorazepam, diazepam, chlordiazepoxide) · GABA-A receptor positive allosteric modulators (brain-calming agents)

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.

1. The doctor chooses a benzodiazepine based on how sick you are: lorazepam (Ativan) 1-2 mg IV or by mouth every 6-8 hours if liver is damaged; diazepam (Valium) 10-20 mg IV or by mouth every 6-8 hours if liver is healthy (longer-acting, smoother); or chlordiazepoxide (Librium) 25-100 mg by mouth every 6 hours.
2. The drug is absorbed quickly (within 30 minutes by mouth, instantly by IV) and travels to your brain.
3. Benzodiazepine molecules land on GABA-A receptors and change their shape, making them open chloride channels more often when GABA binds.
4. Chloride (a negatively charged particle) floods into nerve cells, making them less excitable—like lowering the voltage in a circuit.
5. This calms the electrical storm in your brain, stops seizures, lowers blood pressure and heart rate, reduces shaking and sweating, and prevents DTs.
6. The doctor adjusts the dose based on your withdrawal symptoms (using a scale called CIWA-Ar) and tapers it gradually over 3 to 7 days as your brain re-adapts.
7. After acute withdrawal, the benzodiazepine is stopped completely to avoid dependence on a new drug.

SAFETY WATCH

DANGEROUS combination: Benzodiazepines plus opioids (pain pills, heroin, fentanyl) or alcohol can stop your breathing and kill you. If you're taking opioids, the doctor will monitor you in the hospital or lower doses carefully. · **Breathing problems:** If you have sleep pauses in breathing (apnea) (stop breathing in your sleep) or COPD (chronic lung disease), benzodiazepines can make it worse. The doctor may use a lower dose and watch you closely. · **Pregnancy risk:** Benzodiazepines in the first trimester (first 3 months) slightly raise the risk of cleft lip/palate (facial birth defect). Later in pregnancy, they can cause floppy baby syndrome and withdrawal in the newborn. If you're pregnant, the doctor weighs the risk of untreated withdrawal (which can kill you and the baby) against the drug risk. · **Older adults:** Much higher risk of falls, confusion, memory problems, and prolonged sedation. Use the lowest possible dose. · **Taper slowly:** After withdrawal ends, the doctor reduces the dose gradually over days to avoid rebound seizures or anxiety. NEVER stop suddenly. · **Do not drive or operate machinery while taking benzodiazepines—they slow your reaction time and impair judgment.** · **Liver disease:** Lorazepam is safest because it's processed by the kidneys, not the liver. Diazepam and chlordiazepoxide can build up dangerously if your liver is failing.

Thiamine (Vitamin B1) · Essential vitamin (coenzyme for glucose metabolism in the brain)

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.

1. In the emergency room or hospital, you get 100 mg of thiamine by IV (into a vein) or IM (shot into a muscle) BEFORE any IV fluids with glucose (sugar). Giving glucose first without thiamine can trigger sudden, severe Wernicke encephalopathy.
2. Thiamine is absorbed immediately (IV) or within 30 minutes (IM) and rushes to your brain.
3. The vitamin enters brain cells and becomes thiamine pyrophosphate (TPP), the active form.
4. TPP acts as a helper molecule (coenzyme) for enzymes that break down glucose into energy. Without TPP, glucose piles up unused and brain cells suffocate.
5. Restoring TPP allows brain cells to make energy again, stopping cell death within hours to days.
6. You continue 100 mg thiamine daily by mouth for weeks to months to rebuild stores and prevent relapse, plus eat thiamine-rich foods (whole grains, pork, legumes, fortified cereals).

SAFETY WATCH

Extremely safe. Thiamine is a water-soluble vitamin, so excess is peed out—almost no risk of overdose. · VERY RARE allergic reaction: Anaphylaxis (severe allergic shock with throat swelling, trouble breathing, and blood pressure drop) has happened with IV thiamine, especially if given too fast. The doctor gives it slowly and watches you for 15 minutes after the first dose. · ALWAYS give thiamine BEFORE glucose (sugar) in malnourished or alcohol-dependent patients. Glucose without thiamine can exhaust the tiny bit of thiamine left and trigger sudden Wernicke encephalopathy. · No drug interactions, safe in pregnancy and breastfeeding, safe in children and elderly. · Oral thiamine is very cheap (pennies per pill) and available over the counter, but absorption is poor in active drinkers, so IV/IM dosing is critical during acute illness.

Red flags

- 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

Nursing priorities

1. 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. — Alcohol withdrawal progresses in stages: mild symptoms (6 to 12 hours—tremor, anxiety, nausea), seizures (12 to 48 hours—sudden brain overactivity when alcohol's calming effect is gone), then delirium tremens (48 to 96 hours—autonomic storm with fever, a fast heart rate (tachycardia) over 120, high blood pressure (hypertension), confusion). CIWA-Ar scores guide symptom-triggered benzodiazepine dosing to prevent progression and save lives.

2. 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. — Alcohol enhances GABA (the brain's calming neurotransmitter) and blocks glutamate (the brain's excitatory signal). When alcohol is removed, glutamate floods the brain causing hyperactivity—tremors, seizures, a fast heart rate (tachycardia), high blood pressure (hypertension). Benzodiazepines mimic alcohol's GABA effect, calming overactive neurons and preventing seizures and delirium tremens, which kill 5 to 15 percent of untreated patients.
3. 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. — Chronic alcohol blocks thiamine (vitamin B1) absorption in the gut and storage in the liver. Thiamine is essential for brain glucose metabolism. Deficiency causes Wernicke encephalopathy (confusion, abnormal eye movements called involuntary eye jerking (nystagmus), unsteady walking called loss of coordination (ataxia)) and Korsakoff syndrome (permanent short-term memory loss). Giving IV dextrose without thiamine first burns up the last thiamine stores and can trigger or worsen Wernicke encephalopathy, causing irreversible brain damage.
4. 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. — Severe withdrawal and delirium tremens trigger autonomic hyperactivity—the sympathetic nervous system (fight-or-flight) runs wild without alcohol's brake. This causes a fast heart rate (tachycardia), high blood pressure (hypertension), hyperthermia (high body temperature), and fast breathing (tachypnea) (fast breathing). Uncontrolled autonomic storm leads to arrhythmias (irregular heartbeat), stroke, heart attack, and multi-organ failure. Frequent vitals catch deterioration early so you can escalate benzodiazepine doses or transfer to ICU.
5. 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. — Alcohol withdrawal lowers the seizure threshold by removing alcohol's inhibitory effect on the brain. Generalized tonic-clonic seizures (full-body convulsions, also called grand mal) peak 12 to 48 hours after the last drink. Seizures cause low oxygen (hypoxia) (low oxygen to the brain), aspiration (vomit going into lungs), injury from falling, and—if prolonged—status epilepticus (a seizure that will not stop) (continuous seizing that damages the brain). Benzodiazepines stop seizures by boosting GABA and calming overexcited neurons.

6. 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. — Delirium tremens causes visual hallucinations (patients see bugs, shadows, or people that are not there) and tactile hallucinations (feeling things crawling on skin). The confused, terrified patient may try to flee, pull out IV lines, or become combative. A calm environment reduces sensory overload that worsens delirium. Reorientation helps the patient anchor to reality. Close observation prevents self-harm and allows rapid response if condition deteriorates.
7. 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. — Chronic alcohol use and acute withdrawal cause dehydration from vomiting, sweating, and poor oral intake. Alcohol also acts as a diuretic (makes you urinate more), flushing out electrolytes. Low magnesium worsens tremor and lowers seizure threshold. Low potassium causes arrhythmias (dangerous heart rhythms). Low phosphate causes muscle weakness and confusion. Correcting dehydration and electrolytes stabilizes vital signs and supports safe withdrawal.
8. 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). — Detox alone has high relapse rates—60 to 90 percent of patients return to drinking within a year without ongoing treatment. Naltrexone blocks opioid receptors in the brain's reward pathway, reducing alcohol cravings and the pleasurable buzz from drinking. Acamprosate restores balance between GABA and glutamate, easing post-withdrawal anxiety and insomnia that trigger relapse. Disulfiram causes severe nausea, vomiting, and flushing if the patient drinks (blocks alcohol breakdown), creating a deterrent. Counseling and peer support address psychological, social, and behavioral drivers of addiction, giving the patient skills and community to sustain recovery long-term.

Sources

- 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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