

Opioid Use Disorder

Medication-assisted treatment (buprenorphine, methadone, or naltrexone) cuts the risk of death in half—50 percent fewer deaths.

THE STORY IN ONE SENTENCE

Medication-assisted treatment (buprenorphine, methadone, or naltrexone) cuts the risk of death in half—50 percent fewer deaths.

“Opioid Use Disorder means the brain's reward circuits get hijacked and rewired so they fire only for the drug, not for normal life.”

Normal anatomy — the body's job

Your brain makes natural opioids (endorphins and enkephalins) that lock onto opioid receptors on brain cells in the reward center, helping you feel pleasure, cope with pain, and stay motivated.

BODY'S JOB

The ventral tegmental area and nucleus accumbens (the brain's reward hub) release dopamine (a feel-good messenger) when you do something good—eat, laugh, accomplish a goal. The prefrontal cortex (the front of your brain) weighs options and pumps the brakes on impulses. The locus coeruleus (a tiny alarm center in the brainstem) stays quiet when you're calm and fires norepinephrine (a wake-up chemical) only when you need to be alert. Natural opioids fine-tune this system, keeping pleasure, pain, and motivation in balance.

1. Normal reward: you eat, laugh, or finish a task → the ventral tegmental area releases dopamine → you feel satisfied.
2. Natural opioids (endorphins, enkephalins) lock onto opioid receptors → they gently boost pleasure and dampen pain.
3. The prefrontal cortex checks: Is this safe? Is this worth it? → it says yes to healthy rewards and no to dangerous impulses.
4. The locus coeruleus stays quiet → you feel calm, your heart rate is steady, and stress hormones stay low.

5. Result: you feel good from normal life, you can delay gratification, and you cope with everyday stress without needing a drug.

What breaks

Opioid drugs flood the brain's opioid receptors with a signal 10 to 100 times stronger than anything natural, so the brain turns down its own opioid production and shrinks the number of receptors to protect itself. Now normal rewards feel empty, the prefrontal cortex loses control over impulses, and the locus coeruleus fires wildly during withdrawal, causing pain, sweating, and terror.

CLINICAL TERM

Chronic opioid exposure down-regulates μ -opioid receptors and suppresses endogenous opioid synthesis in the mesolimbic reward pathway (ventral tegmental area, nucleus accumbens). The prefrontal cortex exhibits decreased activity and impaired executive control. During withdrawal, the locus coeruleus becomes hyperactive, releasing excess norepinephrine and driving autonomic dysregulation (tachycardia, heavy sweating (diaphoresis), dilated pupils (mydriasis), gastrointestinal distress). Tolerance develops as receptor density falls and intracellular signaling adapts. Social determinants—trauma, poverty, housing instability, criminal-legal involvement—amplify craving, relapse risk, and mortality.

How the body compensates

The body tries to restore balance by turning down opioid receptors and ramping up the alarm system, but these workarounds trap the person in a cycle: they need the drug to feel normal, and stopping triggers brutal withdrawal that feels impossible to endure without help.

1. The brain down-regulates (shrinks) opioid receptors to protect itself from overstimulation—this buys short-term stability but makes the person dependent on the drug to feel okay.
2. The locus coeruleus turns up its activity to counterbalance the calming effect of opioids—this keeps the person awake and breathing while on the drug, but it backfires during withdrawal, causing pain, sweating, and panic.
3. The person uses more drug, more often, to chase the original high and to avoid withdrawal—this looks like control at first, but tolerance keeps climbing and the window between 'enough' and 'overdose' shrinks, especially with fentanyl.
4. The prefrontal cortex tries to plan and resist cravings, but it's outgunned by the hyperactive reward circuit—the person knows using is dangerous, but the craving feels like survival.

5. Biggest danger: fatal overdose from respiratory depression (breathing stops), especially after a period of abstinence (jail, detox, or hospital) when tolerance drops and the person uses the old dose. Fentanyl (50–100 times stronger than morphine) in street drugs makes overdose unpredictable and fast.
6. Other serious harms: hepatitis C and HIV from shared needles; bacterial endocarditis (heart-valve infection); skin and soft-tissue infections (abscesses, cellulitis); housing loss; criminal-legal involvement; and social isolation as relationships break under the weight of the disorder.

Domino effect

1. Opioid drugs flood the brain's opioid receptors with a signal far stronger than natural endorphins. — Pills, heroin, or fentanyl lock onto μ -opioid receptors in the ventral tegmental area and nucleus accumbens (the reward center) and trigger a dopamine surge 10 to 100 times bigger than anything from food, love, or accomplishment—the brain learns that the drug is the most important thing in the world.
2. The brain down-regulates opioid receptors and stops making its own endorphins. — To protect itself from overstimulation, the brain shrinks the number of receptors and turns off natural opioid production—now normal rewards (a good meal, a hug, a victory) feel empty and joyless without the drug.
3. Tolerance develops: each dose feels weaker, so the person needs more to feel normal or high. — With fewer receptors and altered cell signaling, the same dose of opioid produces a smaller effect—the person chases the original feeling by using more, more often, and the cycle tightens.
4. The prefrontal cortex loses control over impulses and planning. — Chronic opioid exposure weakens activity in the prefrontal cortex (the front of the brain that weighs consequences and says no)—cravings override logic, and the person uses even when they know it will destroy their job, health, or family.
5. Withdrawal punishes any attempt to stop: pain, sweating, vomiting, shaking, and terror. — When the drug wears off, the locus coeruleus (the brainstem alarm center) rebounds and fires wildly, dumping norepinephrine into the body—heart races, pupils dilate, muscles ache, gut cramps, and the person feels like they're dying, so they use again to make it stop.
6. Loss of control: the person keeps using despite knowing the harm. — The hijacked reward circuit makes the drug feel more urgent than food, safety, or relationships—the person isn't weak or bad; their brain chemistry is broken, and willpower alone can't override a malfunctioning mesolimbic system.

7. Life crumbles: money vanishes, relationships break, work disappears, housing is lost, infections spread, and overdose looms. — Every dollar goes to drugs; family and friends pull away from the chaos; employers fire someone who can't show up; needles spread hepatitis C, HIV, and endocarditis; and fentanyl-laced street drugs make every use a gamble with death—social determinants (poverty, trauma, homelessness, criminal-legal involvement) accelerate the spiral.
8. Medication-assisted treatment, therapy, and social support repair the circuit and cut death risk in half. — Buprenorphine or methadone (partial or full opioid agonists) steady the receptors without causing a high, blocking withdrawal and cravings so the prefrontal cortex can re-engage; naltrexone (an opioid blocker) prevents relapse after detox; therapy rebuilds coping skills; housing, jobs, and peer support give reasons to stay well—and overdose deaths drop 50 percent because the brain gets the chemistry it needs while the person rebuilds a life worth living.

Symptoms · what the patient feels, and why

- Strong cravings for opioids: Opioids flood the brain's mu-opioid receptors (special locks on nerve cells that control reward and pain), releasing huge bursts of dopamine (the brain's main feel-good messenger). Over time, the brain learns to need the drug to feel normal, so when it's not there, the reward system screams for it—that scream is the craving.
- Using more opioids than planned or for longer than planned: Opioids hijack the prefrontal cortex (the front part of the brain that plans and stops impulses). When that control center weakens, the person can no longer reliably put the brakes on use, even when they meant to stop or cut back.
- Tolerance (needing more of the drug to feel the same effect): The brain's mu-opioid receptors get less sensitive after being constantly activated, and some receptors even disappear inside the cell. To get the same buzz or pain relief, the person has to take a bigger dose.
- Withdrawal symptoms when stopping (sweating, shaking, nausea, muscle aches, runny nose, fast heart): Opioids normally quiet the locus coeruleus (a tiny alarm cluster deep in the brainstem that releases norepinephrine, the body's main stress chemical). When the drug is gone, that alarm center fires wildly, flooding the body with norepinephrine and causing all the miserable flu-like symptoms.
- Pinpoint pupils (tiny, constricted pupils) when recently using: Opioids turn on mu-opioid receptors in the brainstem's Edinger-Westphal nucleus (the nerve cluster that controls the muscle that shrinks the pupil). This keeps the pupil clamped down tight, even in dim light.
- Track marks, skin infections, or scars (especially on arms, hands, or legs): Repeated injections damage veins and break the skin barrier. Dirty needles or bacteria on the skin can seed infections (abscesses, cellulitis, even heart-valve infection) at injection sites.

- Giving up important activities (work, school, hobbies, time with family) to use or recover from opioids: The midbrain dopamine reward circuit (ventral tegmental area and nucleus accumbens) now values the drug above natural rewards like friends or goals. The person's priorities flip because the brain marks the drug as the most important thing for survival.
- Continuing to use despite harm (legal trouble, broken relationships, health problems): Opioids weaken the prefrontal cortex's ability to weigh consequences and override impulses. Even when the person knows use is wrecking their life, the craving signal and weakened self-control make stopping incredibly hard.
- Slowed, shallow breathing: Opioids turn down the brainstem's respiratory center (the cluster of neurons that tells the lungs to breathe). High doses can slow breathing so much that oxygen drops dangerously low.
- Confusion, extreme sleepiness, or can't be woken up: Too much opioid in the bloodstream over-activates mu-opioid receptors all over the brain, dampening consciousness and arousal. This is overdose—the brain is so sedated that the person may stop breathing.

Focused exam

- Pinpoint pupils (miosis): Opioids lock onto mu receptors in the brainstem (the base of the brain that runs automatic body functions) and squeeze the pupils tight. Tiny pupils mean opioid is in the body right now.
- Track marks, scars, or skin-popping sites on arms, legs, groin, or neck: Scars show where the drug was injected. Old scars mean months or years of use; fresh red or infected marks mean recent injection and high risk of overdose or infection today.
- Slow breathing (respiratory rate < 12 breaths/min) or shallow breaths: Opioids shut down the brainstem's breathing center (the part that tells the lungs when to breathe). Slow or shallow breathing means the person may stop breathing entirely—this is how most people die from opioid overdose.
- Dilated pupils, goosebumps, sweating, yawning, or muscle cramps: These are withdrawal signs. When opioid receptors (the brain's locks that opioids fit into) suddenly go empty, the sympathetic nervous system (the fight-or-flight system) fires too hard. The body sweats, shakes, and aches.
- Heart murmur or fever (temp > 38.0°C / 100.4°F): People who inject drugs can push bacteria from the skin straight into the bloodstream, where it lands on the heart valves and grows (infective endocarditis). A new murmur plus fever means the heart may be infected and needs urgent blood cultures and antibiotics.
- Skin abscesses, cellulitis, or necrotizing fasciitis: Dirty needles or skin-popping (injecting under the skin instead of into a vein) can introduce bacteria into tissue. Red, swollen, or blackening skin means infection spreading fast and needs antibiotics or surgery.

Diagnostics · every test answers a question

- Urine drug screen (immunoassay) → Is there opioid in the body right now?
- Hepatitis C antibody (anti-HCV) and HCV RNA if antibody positive → Does the person have hepatitis C from sharing needles?
- HIV antibody/antigen combination test (4th generation) → Is there HIV infection from needle sharing or sex?
- Blood cultures (two sets from different sites) → Is bacteria in the bloodstream (bacteremia or endocarditis)?
- Echocardiogram (transthoracic or transesophageal) → Is there a vegetation (clump of bacteria and clot) on a heart valve?
- Liver function tests (AST, ALT, bilirubin, INR) → Is the liver damaged from hepatitis, alcohol, or acetaminophen (if mixed opioids were used)?
- Clinical Opiate Withdrawal Scale (COWS) score → How severe is the withdrawal right now?
- Pregnancy test (urine hCG) in anyone with a uterus of childbearing age → Is the person pregnant (which changes opioid treatment and safety counseling)?

Treatment logic

- Buprenorphine/naloxone sublingual tablet or film (starting 2–4 mg, titrated to 12–24 mg daily) or long-acting buprenorphine injection (monthly) → Buprenorphine is a partial agonist—it sits on the brain's mu opioid receptors (the locks that opioids fit into) and activates them just enough to stop withdrawal and craving, but not enough to cause a high or dangerous breathing slowdown. The naloxone in the combo pill blocks misuse by injection. The brain's reward circuit (ventral tegmental area and nucleus accumbens) quiets down, so the person can think clearly and make choices instead of chasing the next dose.
- Methadone (starting 10–30 mg daily, titrated slowly to 60–120 mg daily at a certified opioid treatment program) → Methadone is a full mu opioid agonist that stays in the body a long time (half-life ~24–36 hours). It fills the brain's opioid receptors steadily all day, stopping withdrawal and craving without peaks or valleys. It is more tightly controlled than buprenorphine and requires daily witnessed dosing at first.
- Naloxone nasal spray (4 mg) or auto-injector (2 mg IM) given to the patient, household members, and friends with training on how to use it → Naloxone is an opioid antagonist—it kicks opioids off the mu receptor and reverses respiratory depression (slow or stopped breathing) within 2–5 minutes. It buys 30–90 minutes for emergency help to arrive. It has saved thousands of lives.

- Naltrexone (oral 50 mg daily or extended-release injection 380 mg monthly) after full detox and 7–10 days opioid-free → Naltrexone blocks all opioid receptors completely, so if the person uses opioids they feel nothing. This removes the reward. It works best for highly motivated people with strong support and no access to opioids.
- Psychosocial support: cognitive-behavioral therapy (CBT), contingency management (rewards for clean urine), motivational interviewing, peer recovery coaching, or mutual-help groups (12-step, SMART Recovery) → Therapy helps rewire the prefrontal cortex (the front of the brain that plans, decides, and pumps the brakes on impulses) and the limbic system (the emotion and reward centers). It teaches new coping skills, rebuilds motivation, and addresses trauma or co-occurring depression and anxiety.
- Harm reduction supplies: fentanyl test strips, clean syringes, alcohol wipes, safe disposal containers, and education on safer injection technique → Fentanyl test strips detect fentanyl in street drugs before use, preventing surprise overdoses. Clean needles stop HIV, hepatitis C, and skin/blood infections. These measures keep people alive and healthier while they work toward recovery.
- Treat co-occurring conditions: depression (SSRI like sertraline 50–200 mg daily), anxiety (CBT, buspirone, or short-term hydroxyzine), insomnia (sleep hygiene, trazodone 50–100 mg at bedtime), and chronic pain (physical therapy, non-opioid analgesics, nerve blocks) → Many people with opioid use disorder started using to numb emotional or physical pain. Treating the underlying depression (low serotonin and norepinephrine in the brain), anxiety (overactive amygdala, the brain's alarm center), or pain removes the fuel that keeps the addiction circuit running.

Medications

Buprenorphine (Suboxone, Sublocade) · Partial opioid agonist

Buprenorphine latches onto the mu opioid receptors (the same brain locks that heroin, fentanyl, and prescription painkillers open) but only turns the key partway. This gives just enough signal to quiet cravings and stop withdrawal pain without causing a high or slowing breathing dangerously. Because it sticks so tightly, it also blocks full opioids from working if someone tries to use them, like a key stuck in a lock that won't let another key fit.

1. Buprenorphine binds very tightly to mu opioid receptors throughout the brain and spinal cord.
2. It only partly activates these receptors — enough to stop withdrawal and cravings but not enough to cause euphoria or dangerous breathing slowdown.
3. Because it sticks so firmly, it kicks off any full opioid (like heroin or fentanyl) already sitting on the receptor and blocks new doses from working.

4. The steady, low-level receptor signal calms the craving and stress circuits without feeding addiction.
5. Over time, this stable signal lets the brain start to heal and rebalance its natural opioid and dopamine systems.

SAFETY WATCH

Starting buprenorphine too soon after using a full opioid can trigger precipitated withdrawal (sudden, severe withdrawal) because it kicks the full opioid off the receptor. Wait until moderate withdrawal has started (usually 12–24 hours after short-acting opioids, 24–72 hours after long-acting ones like methadone) before the first dose. · Mixing buprenorphine with benzodiazepines (like Xanax or Valium) or alcohol raises the risk of slowed breathing and overdose, especially early in treatment. · The film or tablet under the tongue must dissolve completely — swallowing it whole wastes most of the medicine because the liver breaks it down before it reaches the brain. · Long-acting injection (Sublocade) can only be started after the person is stable on the film or tablet for at least a week, and it keeps working for a month or more, so dose changes are slow. · Buprenorphine can cause constipation, headache, and sweating, though usually milder than full opioids. · Liver function should be checked before starting and periodically during treatment, especially if the person has hepatitis or heavy alcohol use. · Pregnancy is not a reason to stop buprenorphine — keeping the mother stable protects both her and the baby. The newborn may need monitoring for neonatal opioid withdrawal syndrome, which is milder and safer than uncontrolled opioid use during pregnancy.

Methadone · Full opioid agonist

Methadone fully activates the mu opioid receptors (the brain's main opioid locks), giving a complete signal that stops withdrawal and cravings without causing a high when taken once daily at a steady dose. Because it works slowly and lasts 24–36 hours, it keeps the brain's opioid system stable all day, preventing the roller coaster of highs and crashes that comes with street opioids.

1. Methadone binds to and fully activates mu opioid receptors, delivering a steady, complete signal.
2. This steady signal satisfies the brain's opioid need, stopping withdrawal symptoms (shaking, sweating, pain, panic) and quieting cravings.
3. Because it lasts much longer than heroin or fentanyl, one daily dose keeps the receptors occupied all day, preventing the peaks and valleys that drive repeated drug-seeking.
4. The stable receptor activation also blocks the euphoria from other opioids — if someone uses heroin while on methadone, they feel little or no high because the receptors are already occupied.

5. Over months to years, this stability lets the brain begin to heal and re-regulate its reward and stress systems.

SAFETY WATCH

Methadone must be dispensed daily at a certified opioid treatment program (OTP) clinic, not picked up at a regular pharmacy, to ensure safe dosing and prevent diversion. · Early in treatment, methadone can slow breathing and cause overdose, especially in the first two weeks before the body adjusts. Dose is started low and raised slowly (usually 5–10 mg every few days) under close monitoring. · Methadone can cause a dangerous heart rhythm problem (prolonged QT interval) that may lead to sudden death. An EKG is recommended before starting and periodically during treatment, especially at higher doses or if the person takes other QT-prolonging drugs. · Mixing methadone with benzodiazepines, alcohol, or other sedatives sharply raises overdose risk. · Methadone causes constipation, sweating, sexual side effects, and weight gain in many people. · Many common medicines (antidepressants, antibiotics, antifungals, HIV drugs) change how the liver breaks down methadone, which can make the dose too high or too low. Always review all medications together. · Pregnancy is not a reason to stop methadone. It is safe and preferred for pregnant people with opioid use disorder. The newborn will need monitoring for neonatal opioid withdrawal syndrome, but this is far safer than uncontrolled opioid use during pregnancy. · Stopping methadone suddenly causes severe withdrawal. Any dose change or stop must be done slowly and under medical supervision.

Naltrexone (Vivitrol monthly injection, ReVia oral tablet) · Opioid antagonist (blocker)

Naltrexone sits on the mu opioid receptors without turning them on at all — it's like a blank key stuck in the lock. This completely blocks any opioid (heroin, fentanyl, oxycodone) from working. If someone tries to use opioids while on naltrexone, they feel nothing — no high, no relief. Over time, because using brings no reward, the brain's craving and drug-seeking behavior weakens.

1. Naltrexone binds tightly to mu opioid receptors but does not activate them.
2. This blocks any opioid drug from attaching and working — the receptor is occupied but silent.
3. Without any receptor signal, opioids produce no euphoria, no pain relief, and no reward.
4. Because the brain's reward system gets no feedback from opioid use, cravings gradually lessen in many people.
5. The monthly injection (Vivitrol) keeps receptors blocked for about 28 days; the daily pill works for 24 hours.

SAFETY WATCH

The person **MUST** be completely opioid-free for 7–10 days (sometimes longer for long-acting opioids like methadone or buprenorphine) before starting naltrexone. Starting it too soon causes precipitated withdrawal — sudden, intense, and dangerous withdrawal symptoms. A urine drug test and sometimes a naloxone challenge test are used to confirm it is safe to start. · Naltrexone offers no protection if someone relapses and uses opioids. In fact, because tolerance has dropped, the same dose that was once safe can now cause overdose. People who stop naltrexone are at very high overdose risk. · The monthly injection can cause pain, swelling, or hard lumps at the injection site. · Naltrexone can cause nausea, headache, fatigue, and trouble sleeping, especially in the first few weeks. · High doses can harm the liver. Liver function tests should be checked before starting and monitored during treatment. It should not be used in people with severe liver disease. · Naltrexone will block the effect of any opioid painkiller, so if the person needs surgery or has a serious injury, pain control becomes difficult. They should carry a medical alert card and tell all providers they are on naltrexone. · Adherence is harder with daily pills; many people do better with the monthly injection because it removes the daily decision.

Naloxone (Narcan nasal spray, injectable) · Opioid antagonist (overdose rescue)

Naloxone is an emergency antidote that instantly kicks opioids off the mu opioid receptors and blocks them from reattaching. In an overdose, opioids slow breathing so much that the person stops getting oxygen. Naloxone rapidly reverses this, restarting breathing within 2–3 minutes. It does not treat opioid use disorder itself — it only buys time to get emergency help.

1. Naloxone binds very tightly to mu opioid receptors and shoves off any opioid sitting there.
2. It blocks the receptors so the opioid cannot reattach.
3. This instantly removes the opioid's effect on the brainstem's breathing center, and breathing resumes.
4. Naloxone works in 2–5 minutes when sprayed in the nose or injected into muscle, faster if given into a vein.
5. It wears off in 30–90 minutes. Because many street opioids (especially fentanyl) last longer, the person can slip back into overdose after naloxone fades, so emergency medical help (call 911) is always required.

SAFETY WATCH

Naloxone will immediately cause withdrawal symptoms (shaking, sweating, vomiting, agitation, pain) in anyone who is opioid-dependent. This is uncomfortable but not dangerous, and it is far better than death from overdose. · After giving naloxone, always call 911. The person needs medical evaluation and monitoring because naloxone wears off and the overdose can return. · More than one dose may be needed, especially with fentanyl or carfentanyl. If the person does not start breathing after 2–3 minutes, give a second dose. · Naloxone should be given to every person with opioid use disorder and their family or friends as a take-home rescue kit. Many states allow pharmacies to dispense it without a prescription. · Store naloxone at room temperature away from light. Check the expiration date and replace when needed. · Naloxone nasal spray is easier for non-medical people to use than injectable forms.

Red flags

- Slow, shallow breathing or pauses in breathing (sign of overdose—call 911 and give naloxone if available)
- Cannot be woken up, lips or fingernails turning blue (not enough oxygen—life-threatening)
- Chest pain, fever, or new heart murmur (possible endocarditis, a dangerous heart-valve infection from injecting)
- Severe confusion, seizure, or sudden collapse (possible overdose, stroke, or brain infection)
- Injecting alone or using unknown pills or powder (high risk of accidental overdose, especially with fentanyl)

Nursing priorities

1. Assess airway, breathing, and circulation every 15 minutes if the patient recently used opioids or shows any sedation, and keep naloxone 0.4 mg IV or 4 mg nasal spray at the bedside ready to give immediately. — Opioids bind to mu receptors in the brainstem and suppress the medulla respiratory center, causing slow shallow breathing, pauses in breathing (apnea) (pauses in breathing), low oxygen (hypoxia) (low oxygen), respiratory arrest, and death—naloxone (an opioid antagonist that blocks mu receptors) reverses this within 2 to 3 minutes and saves lives.

2. Give the patient and at least one family member or friend naloxone nasal spray (4 mg, two-dose pack) or auto-injector and teach step-by-step how to recognize overdose (cannot wake the person, slow or no breathing, blue lips) and how to give naloxone, call 911, perform rescue breathing, and stay until help arrives. — Most overdose deaths happen when the person is alone or bystanders do not know how to respond—naloxone is safe (no harm if given to someone not overdosing), works fast, and can be given by anyone, and teaching multiple people increases the chance someone will be present and able to reverse the next overdose.
3. Screen for withdrawal symptoms using the Clinical Opiate Withdrawal Scale (COWS score), checking for sweating, tremor, yawning, runny nose, dilated pupils, gooseflesh, vomiting, diarrhea, and restlessness, and document the score before starting buprenorphine. — Buprenorphine is a partial agonist at mu receptors—if you give it while full-agonist opioids (like fentanyl or heroin) are still attached to receptors, buprenorphine kicks them off and triggers precipitated withdrawal (sudden severe cramping, vomiting, diarrhea, panic), so the patient must be in mild to moderate withdrawal (COWS score 8 or higher) before the first dose.
4. Check for signs of injection-site infection (redness, warmth, pus, swelling) and systemic infection (fever over 100.4°F or 38°C, chills, new heart murmur on listening with a stethoscope (auscultation)), and notify the provider immediately if present because these may signal endocarditis or sepsis. — Injecting opioids introduces skin bacteria (especially *Staphylococcus aureus* and *Streptococcus*) into the bloodstream, and bacteria stick to heart valves causing endocarditis—vegetations (clumps of bacteria and clot on the valve) break off and travel as septic traveling clots (emboli) to lungs, brain, kidneys, and spleen, causing stroke, abscess, and multi-organ failure if untreated.
5. Offer and draw blood for hepatitis C antibody, hepatitis B surface antigen, HIV antigen/antibody test, and complete blood count (CBC) with the patient's informed consent, explaining that injection drug use carries high risk for these infections and early treatment prevents liver failure and death. — Up to 80 percent of people who inject drugs have hepatitis C (a virus that damages the liver and causes cirrhosis and liver cancer), and rates of hepatitis B and HIV are also much higher than the general population—direct-acting antiviral drugs cure hepatitis C in 8 to 12 weeks, antiretroviral therapy controls HIV, and hepatitis B vaccine prevents infection.

6. Build trust by using nonjudgmental language (say 'person with opioid use disorder' not 'addict,' say 'substance use' not 'abuse'), listen without interrupting, validate the patient's feelings, and explain that opioid use disorder is a chronic medical brain disease, not a moral failure. — Stigma and shame from healthcare workers cause patients to hide drug use, skip follow-up appointments, leave against medical advice, and avoid the emergency department during overdose—trauma-informed respectful care increases treatment retention, and patients who stay in medication-assisted treatment for at least 6 months have much lower relapse and death rates.
7. Coordinate same-day or next-day referral to an addiction-medicine provider, opioid treatment program, or buprenorphine prescriber before discharge, give the patient a written appointment card with date and address, and arrange transportation if needed. — The highest-risk time for overdose death is the first four weeks after hospital discharge because tolerance drops during admission (when opioid use stops or decreases) and many patients relapse to previous doses that are now lethal—starting medication-assisted treatment in the hospital and ensuring follow-up within 72 hours cuts mortality by 50 percent.
8. Assess and document pain level, chronic pain conditions, and psychiatric diagnoses (depression, anxiety, PTSD, bipolar disorder), because untreated pain and mental illness drive continued opioid use and require integrated treatment with non-opioid pain management and psychiatric medication or therapy. — Over half of people with opioid use disorder have co-occurring chronic pain or mental health conditions—opioids temporarily relieve emotional pain, flashbacks, and panic by increasing dopamine and calming the amygdala (the brain's fear center), so treating the underlying condition with antidepressants, mood stabilizers, trauma therapy, physical therapy, and non-opioid analgesics (like gabapentin, NSAIDs, or nerve blocks) reduces opioid cravings and improves long-term recovery.

Sources

- ASAM National Practice Guideline for the Treatment of Opioid Use Disorder (2020)
- SAMHSA TIP 63: Medications for Opioid Use Disorder
- DSM-5-TR (American Psychiatric Association)
- CDC Clinical Practice Guideline for Prescribing Opioids for Pain (2022)

Last reviewed: 2026-07-07