

Major Depressive Disorder and Suicide Risk

Keep the person alive first, then fix the brain chemistry and the life problems that fed it.

THE STORY IN ONE SENTENCE

Keep the person alive first, then fix the brain chemistry and the life problems that fed it.

“Major depression with suicide risk comes from broken mood circuits in the brain, plus access to deadly things, plus a life crisis that pushed the person past their limit.”

Normal anatomy — the body's job

A healthy brain keeps mood stable by balancing serotonin (a messenger that steadies mood and sleep), norepinephrine (a messenger that powers energy and focus), and dopamine (a messenger that fuels motivation and reward). The prefrontal cortex (the front of the brain that plans and controls impulses) works with the hippocampus (the memory center) and keeps the amygdala (the alarm center) calm unless real danger appears.

BODY'S JOB

These circuits let you feel hope, see a future, enjoy things, sleep well, think clearly, and stop yourself from acting on every bad feeling. When everything works, sadness comes and goes but never takes over your whole life.

1. Serotonin flows steadily from the raphe nuclei (clusters of nerve cells deep in the brainstem) to the prefrontal cortex, hippocampus, and amygdala, keeping mood level and sleep regular.
2. Norepinephrine flows from the locus coeruleus (another brainstem cluster) to the prefrontal cortex and limbic system, powering energy, attention, and the ability to care about things.
3. Dopamine flows from the ventral tegmental area (a reward center in the midbrain) to the prefrontal cortex and nucleus accumbens, making you feel pleasure and want to do things.
4. The prefrontal cortex reads the situation, plans ahead, and tells the amygdala 'this is not dangerous, stay calm'—so you don't spiral into panic or despair.

5. The hippocampus stores memories and helps you remember that bad times passed before, so this one will too.
6. The hypothalamic-pituitary-adrenal axis (the body's stress control system) releases cortisol (the main stress hormone) when needed, then shuts off once the danger is gone.

What breaks

Chronic stress, genes, trauma, medical illness, or drug use damage the prefrontal cortex, hippocampus, and amygdala. Serotonin, norepinephrine, and dopamine drop too low. The prefrontal cortex weakens, so the person can't plan, can't see a future, and can't stop dark thoughts. The amygdala fires nonstop, flooding the person with fear and hopelessness. The hippocampus shrinks, so the person forgets that pain ever ended before. Cortisol stays high and poisons brain cells. The person feels crushed, sees no way out, and starts thinking suicide is the only solution.

CLINICAL TERM

Major depressive disorder with suicidal ideation results from dysregulation of monoaminergic neurotransmission (serotonin, norepinephrine, dopamine deficiency), prefrontal-limbic circuit dysfunction, hippocampal atrophy, and hypothalamic-pituitary-adrenal axis hyperactivity. Genetic vulnerability, chronic psychosocial stress, and neuroinflammation converge to impair mood regulation, executive function, and stress termination. Suicidal ideation emerges when hopelessness, anhedonia, and impaired future-thinking combine with access to lethal means and acute psychosocial crisis.

How the body compensates

At first, the brain tries to fix low mood by pushing remaining serotonin and norepinephrine harder, and the person may hide their pain or seem to rally. The most dangerous time is when treatment starts and energy returns before hope does—because now the person has the strength to act on suicide plans—or right after hospital discharge, when support suddenly drops.

1. {'what': 'The brain tries to squeeze more serotonin and norepinephrine out of damaged circuits—so the person may have brief good days that make loved ones think things are improving.', 'why': 'Remaining nerve cells fire faster to make up for lost messengers, but this can't last and eventually burns out.'}
2. {'what': 'The person hides their pain, smiles, and pretends everything is fine (called 'masking')—so family and doctors miss the danger.', 'why': 'The person doesn't want to burden others, fears being locked up, or has already decided to die and wants to avoid interference.'}

3. {'what': "In the first 1-2 weeks of antidepressant treatment, energy and movement return before mood lifts—so the person finally has the strength to carry out a suicide plan they've been too exhausted to attempt.", 'why': 'Norepinephrine and dopamine rise faster than serotonin, so physical energy comes back while hopelessness remains.'}
4. {'what': 'In the first 30 days after leaving the hospital, suicide risk spikes—especially in the first week—because the person suddenly loses daily monitoring, faces the same life problems that triggered the crisis, and feels ashamed or alone.', 'why': 'Discharge removes the safety net before the brain has fully healed, and the person may feel abandoned or hopeless that even the hospital couldn't fix them.'}
5. {'what': "The person may suddenly seem calm or happy and give away belongings—this is often a sign they've decided to die and feel relieved the suffering will end soon.", 'why': 'Making a final plan brings a sense of control and peace, which looks like improvement but is actually the most dangerous moment.'}

Domino effect

1. Chronic stress, genes, trauma, or medical illness damage the prefrontal cortex (the brain's planning and brake center), hippocampus (the memory center), and amygdala (the alarm center). — High cortisol poisons brain cells, kills connections, and shrinks these structures. Genes can make serotonin receptors work poorly or reduce serotonin production from birth. Trauma or illness trigger inflammation that damages brain circuits.
2. Serotonin, norepinephrine, and dopamine drop too low. — Damaged circuits can't make enough of these messengers, or the receptors that receive them stop working well. Without these chemicals, the brain loses its ability to steady mood, power energy, or feel pleasure.
3. The person feels crushing sadness, loses interest in everything (anhedonia), can't sleep or sleeps all the time, can't eat or eats too much, feels exhausted, can't concentrate, feels worthless, and thinks about death. — Low serotonin → sadness, sleep chaos, appetite chaos. Low norepinephrine → exhaustion, brain fog. Low dopamine → anhedonia. Weak prefrontal cortex → can't see a future, can't control dark thoughts. Overactive amygdala → fear, hopelessness. Shrinking hippocampus → can't remember that pain ended before.
4. The person starts thinking 'I wish I were dead' (passive suicidal ideation), then 'I want to kill myself' (active suicidal ideation), then makes a plan for how to do it. — The broken prefrontal cortex can't imagine a future or stop catastrophic thinking. The overactive amygdala floods the person with unbearable emotional pain. Suicide thoughts appear as the brain's desperate, wrong solution to end suffering.

5. If the person has access to lethal means (guns, pills, heights, ropes) and a fresh crisis hits (breakup, job loss, humiliation, medical diagnosis), the risk of death in the next hours to days becomes very high. — Access to means makes the plan easy to carry out. A fresh crisis adds unbearable shame or hopelessness that pushes the person past their last bit of endurance. Impulsivity (from a damaged prefrontal cortex) means the person may act within minutes of deciding.
6. We intervene by removing or locking up all lethal means, admitting the person to the hospital if risk is high, starting an antidepressant (usually an SSRI like sertraline or escitalopram) to rebuild serotonin, and beginning psychotherapy (especially cognitive-behavioral therapy or dialectical behavior therapy) to teach new ways to handle pain and challenge hopeless thoughts. — Removing means buys time and prevents impulsive death. Hospitalization provides 24-hour monitoring during the highest-risk period. SSRIs increase serotonin in the synapse by blocking reuptake, which slowly rebuilds mood regulation over 4–8 weeks. Therapy rewires thought patterns and teaches skills the damaged prefrontal cortex can't generate on its own.
7. We watch closely in the first 1–2 weeks of treatment and the first 30 days after discharge, because energy may return before hope does, giving the person the strength to act on a plan—and we address life stressors (housing, job, relationships, trauma) because fixing brain chemistry alone won't work if the environment keeps reinjuring the person. — Suicide is never just biology and never just circumstance—it's both. Treat the brain and the life, or the person will keep cycling back into crisis.
8. With consistent treatment—medicine, therapy, safety planning, and life stability—serotonin, norepinephrine, and dopamine rise, the prefrontal cortex regrows connections, the amygdala calms, and the hippocampus heals. Suicidal thoughts fade, hope returns, and the person can see a future again. — The brain can heal if we give it time, chemistry support, new skills, and a safer environment. Recovery is real, but it takes weeks to months and requires the person to stay alive long enough for the circuits to rebuild.

Symptoms · what the patient feels, and why

- Persistent sad, empty, or hopeless mood lasting most of the day, nearly every day for at least two weeks: The brain's mood circuits—especially the prefrontal cortex (the thinking and planning area) and limbic system (the feeling center including the amygdala)—become less active, and serotonin (a brain messenger that steadies mood) and norepinephrine (a messenger that helps with energy and focus) drop too low, so the person feels stuck in sadness and can't see a way forward.

- Loss of interest or pleasure in nearly all activities, even things the person used to love: The reward pathway in the brain—especially the ventral striatum and nucleus accumbens (areas that make activities feel good)—stops responding normally because dopamine (the brain's main pleasure and motivation signal) is too low, so nothing feels worth doing anymore.
- Significant weight change (loss or gain of more than 5% in a month) or change in appetite nearly every day: Depression disrupts the hypothalamus (the brain's control tower for hunger, stress, and sleep), changing levels of cortisol (the body's main stress hormone) and other signals that tell the body when to eat, so the person either loses all interest in food or eats much more to try to feel better.
- Insomnia (trouble falling or staying asleep) or hypersomnia (sleeping too much) nearly every day: The brain's sleep-wake cycle, controlled by the suprachiasmatic nucleus in the hypothalamus and regulated by serotonin and melatonin (a sleep hormone), gets thrown off balance, so the person either cannot sleep at all or sleeps all the time but never feels rested.
- Psychomotor agitation (restless, can't sit still) or retardation (moving and thinking in slow motion) nearly every day, noticeable to others: Dopamine and norepinephrine imbalances affect the basal ganglia (deep brain structures that control movement) and motor cortex (the strip of brain that runs body movements), so the person either feels wound up and jittery or so slowed down that even simple actions feel like wading through thick mud.
- Fatigue or loss of energy nearly every day: Low serotonin, norepinephrine, and dopamine sap the brain's ability to generate drive and alertness, and chronic high cortisol from ongoing stress wears down the body like a battery running low, leaving the person exhausted even after rest.
- Feelings of worthlessness or excessive, inappropriate guilt nearly every day: The prefrontal cortex (which normally checks and balances thoughts) becomes underactive and can't stop the amygdala (the brain's alarm bell) from flooding the mind with harsh, self-blaming thoughts, so the person truly believes they are a burden or have failed beyond repair.
- Trouble thinking, concentrating, or making decisions nearly every day: The dorsolateral prefrontal cortex (the brain's executive office that handles focus, planning, and judgment) runs sluggishly when serotonin and norepinephrine are low, so even small choices feel overwhelming and the mind feels foggy.
- Recurrent thoughts of death, recurrent suicidal thoughts without a specific plan, or a suicide attempt or specific plan for committing suicide: Severe depression silences the prefrontal cortex's ability to see hope or alternatives, while pain and hopelessness feel unbearable; the person begins to believe that ending their life is the only way to stop the suffering, especially when serotonin (which also helps control impulsivity) is very low.

- Expressing a specific suicide plan, especially one that is detailed and uses highly lethal means (firearm, hanging, jumping from height): When someone has moved from vague thoughts to an actual plan—especially one that is quick and deadly—it means the prefrontal cortex's brakes on action have failed and the person is much closer to acting, making immediate intervention essential to save their life.

Focused exam

- Ask about thoughts of killing themselves using the C-SSRS (Columbia-Suicide Severity Rating Scale): Major depressive disorder with suicide risk means the person is in real danger right now. The C-SSRS tells you if they wish to die, if they have a plan to do it, and if they have things nearby they could use. This is the #1 exam for this exact condition.
- Ask: Do you have a plan? Do you have access to guns, pills, or other ways to hurt yourself? Have you tried before?: Major depression plus these three things together equals highest risk. The prefrontal cortex (the front of the brain that pumps the brakes on dangerous choices) is not working. Access to deadly things is what turns a wish to die into a death.
- Look for sad face, flat tone, slow speech, or person sitting still without moving much: These are the body signs of major depressive disorder. The brain circuits that run feelings and movement are stuck, so the person cannot express emotion or move the normal way.
- Ask: How long has this been going on? Did something bad happen right before this started?: Major depressive disorder comes from brain biology and life stress hitting at the same time. Both parts matter. A breakup or job loss can flip the switch in someone whose brain is already fragile.
- Check if they slept, ate, or got clean today. Ask if they can feel happy about anything.: Major depressive disorder makes the person lose interest in normal life and stop caring for their own body. This is called anhedonia—the joy switch is off. It happens because serotonin (a brain messenger that steadies mood) and dopamine (a brain messenger that creates pleasure) are running too low.
- Ask: Have you ever hurt yourself on purpose? Have you been in the hospital for this before?: Past suicide attempts or hospital stays mean the person's major depression has been severe before. They are at higher risk now because the brain pathways that lead to suicide have already been used.

Diagnostics · every test answers a question

- Urine drug screen (UDS) → Is the person using drugs that can look like depression or make real depression worse?
- Complete metabolic panel (CMP) → Are the body's salts, kidney, or liver broken in a way that causes sadness and low energy?

- Thyroid-stimulating hormone (TSH) → Is the thyroid running too slow and making the person look depressed?
- Complete blood count (CBC) → Is the person anemic (low on red blood cells) in a way that causes tiredness and low mood?
- Electrocardiogram (ECG) → Is the heart's electrical pattern safe before starting antidepressants that can stretch the QT interval?
- Vitamin B12 and folate levels → Is the person missing vitamins that the brain needs to make mood chemicals?

Treatment logic

- Suicide risk assessment using C-SSRS; remove access to means (firearms, medications, poisons) → Identifies imminent danger and stops the fastest paths to death. Buys time for the brain to heal.
- Hospitalization if active plan plus intent plus access to means → Creates a safe place where the person cannot act on suicidal thoughts while medication and monitoring start working.
- Start an SSRI (e.g. sertraline, escitalopram) or SNRI (e.g. venlafaxine, duloxetine) → SSRIs block the cleanup of serotonin (a brain messenger that steadies mood and sleep) so more stays in the gap between brain cells. SNRIs do the same for serotonin and norepinephrine (a brain messenger that powers energy and focus). This rebuilds the mood circuit in 4 to 6 weeks.
- Psychotherapy (CBT or interpersonal therapy) plus daily phone contact in first 2 weeks → CBT teaches the person to notice thought traps (like 'I am worthless' or 'nothing will get better') and replace them with real evidence. Interpersonal therapy helps rebuild relationships that were damaged by depression. Daily check-ins catch worsening early.
- ECT (electroconvulsive therapy) if depression is severe, treatment-resistant, or catatonic → Resets abnormal electrical patterns in the prefrontal cortex (the front of the brain that controls planning and emotion) and the limbic system (the brain's emotion center). This is the fastest treatment for life-threatening depression. Works in 1 to 2 weeks.
- IV ketamine or intranasal esketamine for acute suicidal thoughts → Ketamine blocks glutamate receptors (the brain's main 'go' signal) and can reduce suicidal urges within hours to days. Used when the person is in immediate danger and cannot wait 4 to 6 weeks for an SSRI to work.
- Coordinate housing, food security, substance-use treatment, sleep hygiene, and job support on the same treatment plan → Removes the constant stress signals that reinjure the stress-control circuit (the hypothalamus-pituitary-adrenal axis, which controls cortisol, the body's main stress hormone) every day. Makes medicine and therapy actually work.

Medications

Sertraline (Zoloft), Escitalopram (Lexapro), Fluoxetine (Prozac) · SSRI (Selective Serotonin Reuptake Inhibitor)

These medicines slow down the brain's clean-up crew that normally sweeps away serotonin (a messenger chemical that steadies mood, sleep, and thinking). By slowing the clean-up, more serotonin stays in the tiny gaps between brain cells, so the signals that lift mood and calm anxiety can keep going.

1. The medicine blocks the serotonin transporter protein (SERT), which normally sucks serotonin back into the sending nerve cell.
2. Serotonin builds up in the gap, so it keeps hitting the receiving cell's docking stations (receptors) again and again.
3. Over 2–4 weeks, the extra serotonin helps the brain grow new connections and calm down the overactive alarm circuits (the amygdala and the stress-hormone loop).

SAFETY WATCH

Increased suicide risk in people under 25 during the first 1–2 weeks (the FDA black-box warning): restlessness and energy may come back before hope does, so close follow-up is critical—call or see the patient within 1 week of starting. · Serotonin syndrome (too much serotonin) if combined with other serotonin drugs (e.g., another antidepressant, tramadol, St. John's wort): fever, confusion, muscle jerking, racing heart. Stop all serotonin drugs and call 911. · Sexual side effects (low desire, trouble with orgasm) in about half of people; talk openly so patients do not quit silently. · Bleeding risk (especially stomach or brain bleeds) if combined with NSAIDs (ibuprofen, aspirin) or blood thinners; use the lowest dose and watch for unusual bruising or black stools. · Low sodium (Hyponatremia) (low blood sodium) in older adults: confusion, weakness, falls. Check sodium if symptoms appear.

Venlafaxine (Effexor XR), Duloxetine (Cymbalta) · SNRI (Serotonin-Norepinephrine Reuptake Inhibitor)

These medicines block the clean-up of two messenger chemicals—serotonin (which steadies mood) and norepinephrine (which boosts energy and focus). More of both stay in the gaps between brain cells, so signals that lift mood and push back fatigue keep firing.

1. The medicine blocks both the serotonin transporter (SERT) and the norepinephrine transporter (NET), so both chemicals build up in the gap.
2. The extra norepinephrine helps with the low-energy, foggy-thinking side of depression; the extra serotonin helps with the sadness and anxiety.

3. Over 2–6 weeks, the brain's wiring rebalances and the overactive stress loop (hypothalamus-pituitary-adrenal axis) calms down.

SAFETY WATCH

Increased suicide risk in people under 25 in the first 1–2 weeks (black-box warning); see or call the patient within 1 week. · High blood pressure, especially at higher doses (norepinephrine tightens blood vessels); check BP before starting and every few months. · Serotonin syndrome if combined with other serotonin drugs; same signs as SSRIs (fever, confusion, muscle jerks). · Withdrawal symptoms (dizziness, brain zaps, nausea, anxiety) if stopped suddenly; taper slowly over weeks. · Sexual side effects, similar to SSRIs. · Sweating and dry mouth are common; warn patients so they do not quit.

Bupropion (Wellbutrin) · NDRI (Norepinephrine-Dopamine Reuptake Inhibitor)

This medicine blocks the clean-up of norepinephrine (which boosts energy and focus) and dopamine (which drives motivation and pleasure). More of both stay in the gaps between brain cells, so the signals that wake you up, sharpen your mind, and make things feel rewarding keep going.

1. The medicine blocks the norepinephrine transporter (NET) and the dopamine transporter (DAT), so both chemicals build up in the gap.
2. The extra dopamine helps bring back the feeling that life is worth living and that effort pays off; the extra norepinephrine fights fatigue and mental fog.
3. Bupropion does NOT boost serotonin, so it avoids some SSRI side effects (sexual problems, emotional blunting).

SAFETY WATCH

Seizure risk, especially above 450 mg/day or in people with a history of seizures, head injury, eating disorders (anorexia, bulimia), or heavy alcohol use. Do NOT use if seizure risk is high. · Increased suicide risk in people under 25 in the first weeks (black-box warning); close follow-up. · High blood pressure in some people; check BP. · Insomnia and jitteriness if taken late in the day; give the dose in the morning. · Do NOT combine with MAOIs (a rare older antidepressant class) or within 14 days of stopping one; dangerous reaction.

Mirtazapine (Remeron) · Tetracyclic antidepressant (works on norepinephrine and serotonin receptors)

This medicine blocks certain brake pedals (alpha-2 receptors) on the nerve cells that release norepinephrine and serotonin, so more of both chemicals pour out. It also blocks histamine

receptors (which makes you sleepy and hungry) and certain serotonin receptors that cause nausea and anxiety.

1. The medicine blocks alpha-2 receptors, which normally tell the nerve cell 'Stop making so much norepinephrine and serotonin.' With those brakes off, more of both chemicals flood the brain.
2. The medicine also blocks histamine receptors, so you feel sleepy and your appetite comes back (helpful if depression has stolen your sleep and weight).
3. By blocking the 5-HT₂ and 5-HT₃ serotonin receptors, it avoids the nausea and sexual side effects that SSRIs often cause.

SAFETY WATCH

Weight gain and increased appetite (sometimes a benefit if depression caused weight loss, but a problem if not wanted). · Sedation, especially at lower doses (oddly, higher doses can be less sedating); take at bedtime. · Increased suicide risk in people under 25 in the first weeks (black-box warning). · Rare but serious: agranulocytosis (dangerously low white blood cells, leaving you open to infection). If fever, sore throat, or mouth sores appear, stop the drug and check blood counts immediately. · Serotonin syndrome if combined with other serotonin drugs. · High cholesterol and triglycerides; check lipids if on long-term.

Esketamine (Spravato nasal spray) · NMDA receptor antagonist (blocks a specific glutamate receptor)

This medicine blocks NMDA receptors (docking stations for glutamate, the brain's main 'go' signal). Blocking these receptors in certain brain circuits triggers a burst of new connections (synapses) in the prefrontal cortex (the thinking and emotional-control center), which can rapidly lift mood and quiet suicidal thoughts—sometimes within hours to days.

1. Esketamine blocks NMDA receptors, which briefly disrupts the usual glutamate signaling.
2. This triggers a cascade: the brain releases more BDNF (brain-derived neurotrophic factor, a growth protein that helps nerve cells sprout new branches and connections).
3. Within hours to days, new synapses form, mood circuits reboot, and the grip of hopelessness and suicidal thinking can loosen.
4. The effect is fast but often temporary; repeat doses (twice a week, then weekly, then less often) and an oral antidepressant are usually needed to keep gains.

SAFETY WATCH

Dissociation (feeling detached from your body or reality) and blood pressure spikes during and right after the dose; patients must be monitored in a clinic for 2 hours and cannot drive home. · Sedation and dizziness; patients need a ride. · Abuse potential (ketamine is a controlled substance); esketamine is only given in certified clinics under observation. · Increased suicide risk monitoring still applies; esketamine does NOT eliminate risk, just reduces it faster in some people. · Bladder problems (pain, frequent urination) with long-term high-dose use (rare at prescribed nasal doses, but watch for it). · Not safe in pregnancy (can harm the fetus); use reliable birth control.

Electroconvulsive Therapy (ECT) · Neurostimulation procedure (not a drug, but a core medical treatment for severe depression)

A brief, controlled electrical pulse is sent through the brain (while the patient is asleep under anesthesia) to trigger a seizure lasting about 30–60 seconds. This seizure resets overactive and underactive brain circuits, boosts the release of BDNF (the growth protein that helps nerve cells rebuild connections), and rebalances neurotransmitters (serotonin, norepinephrine, dopamine). Mood often lifts after 6–12 treatments over 2–4 weeks.

1. Electrodes are placed on the scalp (either both sides or one side, depending on the protocol).
2. A brief electrical current (about 800 milliamps for less than 8 seconds) triggers a generalized seizure in the brain.
3. The seizure floods the brain with neurotransmitters, releases BDNF, and forces a hard reboot of mood circuits.
4. After the seizure stops (the patient is given a muscle relaxant so the body does not convulse), the brain begins to rewire over the next days and weeks.
5. Treatments are repeated 2–3 times per week until mood improves, then tapered to maintenance (monthly or less) if needed.

SAFETY WATCH

Memory problems: short-term memory loss (especially around the time of treatments) is common; long-term memory gaps (especially for events close to the treatment period) happen in some people and may be permanent. Unilateral (one-side) electrode placement causes less memory trouble than bilateral. · Anesthesia risks (rare complications like aspiration, heart rhythm changes, allergic reactions); an anesthesiologist monitors every session. · Confusion and headache right after waking up; these clear within hours. · Mania can be triggered in people with bipolar disorder; a mood stabilizer is often given alongside. · Increased suicide risk monitoring still applies—ECT reduces risk faster than medication, but safety planning and close follow-up remain critical. · Medical clearance needed: ECT is very safe, but people with recent heart attack, stroke, or brain mass need extra caution.

Red flags

- Any mention of a suicide plan, especially if the person has access to firearms, large amounts of pills, or other lethal means
- Recent suicide attempt or self-harm, or a history of past attempts (each prior attempt raises future risk)
- Severe hopelessness, saying things like 'everyone would be better off without me' or 'there's no point in going on'
- Sudden calm or improvement after a long depression—sometimes means the person has decided on suicide and feels relief that the pain will end
- Giving away prized possessions, writing goodbye notes, or putting affairs in order

Nursing priorities

1. Complete suicide risk assessment using the Columbia Suicide Severity Rating Scale (C-SSRS) within the first 15 minutes of contact, and remove all lethal means from the environment immediately — The first two weeks after starting an SSRI carry the highest suicide risk because the medication increases energy and motivation before it lifts mood, giving a depressed person the activation to act on suicidal thoughts; removing guns, pills, and poisons prevents impulsive action during this window
2. Establish one-to-one observation (a staff member within arm's reach at all times) if the patient endorses a plan plus intent, or use 15-minute safety checks if lower risk — Suicide attempts often happen impulsively within minutes of the decision; continuous observation interrupts the private moment needed to act and allows immediate intervention if the patient decompensates

3. Administer prescribed SSRI or SNRI at the same time every day, and educate the patient that the medication takes four to six weeks to work fully but side effects start immediately — Serotonin (a brain chemical that regulates mood) takes weeks to rebuild in the synapses (gaps between brain cells); explaining the delay prevents the patient from stopping the medication early when they feel nausea or headache but no mood improvement yet
4. Screen for akathisia (inner restlessness and inability to sit still) at every shift if the patient is on an SSRI, especially in the first two weeks — Akathisia is an SSRI side effect that feels unbearable and can worsen suicidal thoughts; it is often mistaken for anxiety but needs the SSRI dose lowered or a beta-blocker like propranolol added, not a higher antidepressant dose
5. Monitor weight, appetite, and sleep every day, and report any increase in insomnia or continued weight loss to the provider — Neurovegetative symptoms (physical signs of depression like sleep and appetite changes) are controlled by brainstem circuits that respond to medication; if these do not improve in two weeks, the current SSRI dose is too low or the wrong medication is being used
6. Facilitate referral to outpatient psychotherapy (CBT or interpersonal therapy) and schedule the first appointment before discharge or within 72 hours of starting treatment — Medication fixes the serotonin and norepinephrine imbalance, but therapy addresses the negative thought patterns and life stressors that triggered the depression; combination treatment cuts relapse risk in half compared to medication alone
7. Arrange daily phone or in-person contact for the first two weeks after the patient leaves your care, using a safety plan card that lists warning signs and crisis numbers — The highest suicide risk occurs in the first week after hospital discharge or treatment start, when the patient is still severely depressed but no longer under direct observation; daily contact creates accountability and catches deterioration before an attempt
8. Educate family members to lock up all firearms outside the home, store medications in a lockbox controlled by another adult, and avoid leaving the patient alone for long periods — Families often underestimate suicide risk or believe the patient will ask for help; 90% of people who survive a suicide attempt say the decision was made less than one hour before the act, so preventing access in that brief window saves lives

Sources

- APA Practice Guideline for the Treatment of Patients with Major Depressive Disorder (2010, updated 2020)
- U.S. Preventive Services Task Force (USPSTF) Depression Screening Recommendation 2023
- VA/DoD Clinical Practice Guideline for Assessment and Management of Patients at Risk for Suicide 2019

- The Columbia Lighthouse Project (Columbia Suicide Severity Rating Scale, C-SSRS)

Last reviewed: 2026-07-07