

# PTSD and Trauma Symptoms

Trauma-focused CBT, EMDR, and prolonged exposure are the first-line treatments that work best.

## THE STORY IN ONE SENTENCE

Trauma-focused CBT, EMDR, and prolonged exposure are the first-line treatments that work best.

“PTSD locks fear memories in the present so the brain's alarm center (amygdala) keeps firing even when the danger is long gone.”

## Normal anatomy — the body's job

The healthy brain runs a three-part safety system: the amygdala (alarm bell) detects danger and triggers the fear response, the hippocampus (memory librarian) time-stamps events and files them as 'past,' and the prefrontal cortex (calm-down center) talks to the amygdala to turn off the alarm when the danger is over. Neurotransmitters like serotonin, GABA (the brain's main calming signal), and norepinephrine (the wake-up and alert chemical) keep this system balanced so you can react to real threats but relax when safe.

## BODY'S JOB

This system lets you respond to danger—heart racing, muscles tensing, focus narrowing—then return to calm once the threat passes. The hippocampus makes sure scary memories are labeled 'that happened before' so you do not relive them as if they are happening now. The prefrontal cortex pumps the brakes on the amygdala, telling it the coast is clear.

1. Danger arrives → the amygdala (alarm center deep in the brain) fires instantly, triggering the body's fight-or-flight response: adrenaline surges, heart pounds, muscles tense.
2. The hippocampus (memory center next to the amygdala) records the event and stamps it with time and place, filing it as 'past' so you remember it without reliving it.
3. When the danger ends → the prefrontal cortex (front of the brain, behind your forehead) sends calming signals to the amygdala, turning off the alarm.
4. GABA and serotonin rise, norepinephrine falls, and the body returns to rest mode: heart slows, muscles relax, sleep returns.

5. The memory stays, but it feels like history—not a current threat.

## What breaks

---

When trauma is too intense or lasts too long, the hippocampus gets damaged by stress hormones (especially cortisol, the body's main stress chemical), so it cannot time-stamp the memory as 'past.' The amygdala gets overactive and keeps firing as if the danger is still happening. The prefrontal cortex weakens under chronic stress, so it loses its ability to calm the amygdala down. The result: the alarm never turns off, and old fear memories feel like they are happening right now.

### CLINICAL TERM

PTSD involves amygdala hyperreactivity (the alarm center fires too easily), hippocampal dysfunction (memories are not consolidated or time-stamped, so they intrude as flashbacks), and prefrontal cortex hypoactivity (reduced top-down inhibition of the fear response). Chronic elevation of cortisol and norepinephrine, with low serotonin and GABA, keeps the sympathetic nervous system stuck in overdrive. This is not a personality flaw—it is a measurable change in brain circuits caused by overwhelming stress.

## How the body compensates

---

At first, the person may look fine because they avoid anything that triggers the alarm—they stay away from reminders, numb their feelings, or use alcohol or drugs to quiet the anxiety. But avoidance actually strengthens the brain's belief that the trauma is still dangerous, so the alarm grows louder, mood darkens, and the window of safe space shrinks. The dangerous moment is when the person seems functional but is quietly burning out inside, at high risk for suicide, substance use disorder, or a second trauma that shatters the last bit of control.

1. The person avoids trauma reminders—places, people, conversations, even thoughts—to keep the alarm quiet. Avoidance buys temporary relief but teaches the brain the danger is still real.
2. Numbing sets in: emotions flatten, joy disappears, and the person feels disconnected from loved ones and from their own body.
3. Hyperarousal becomes the new normal: the body stays tense, sleep is broken by nightmares, and the person is always scanning for danger (hypervigilance).
4. Negative thoughts take over: guilt ('It was my fault'), shame ('I am broken'), and hopelessness ('I will never get better') replace hope.
5. Substance use may begin as self-medication—alcohol or drugs quiet the alarm temporarily but worsen sleep, mood, and brain function, and raise the risk of addiction.

6. Big danger: re-traumatization during care (being restrained, touched without warning, or dismissed) can deepen PTSD. Suicide risk is real, especially when hopelessness peaks and support is absent.

## Domino effect

---

1. Trauma overwhelms the brain's fear system. — The amygdala (alarm center) fires so intensely during the trauma that the hippocampus (memory librarian) cannot process the event normally—it gets stored as fragments (sights, sounds, smells, body sensations) without a clear time stamp or narrative.
2. The hippocampus shrinks under chronic stress hormones. — Cortisol (the body's main stress hormone) floods the brain during and after trauma; high cortisol damages hippocampus cells, so it loses the ability to file memories as 'past'—they intrude as vivid, present-tense flashbacks.
3. The prefrontal cortex weakens and loses control over the amygdala. — Chronic stress and high cortisol also weaken the prefrontal cortex (the calm-down center at the front of the brain), so it cannot send the 'all clear' signal to the amygdala—the alarm keeps ringing even when the person is safe.
4. Intrusions, nightmares, and flashbacks crash in without warning. — Because the hippocampus cannot label the memory as 'over,' the amygdala treats any reminder—a sound, a smell, a place—as if the trauma is happening right now, flooding the body with adrenaline and norepinephrine (the wake-up and panic chemicals).
5. Avoidance becomes the main coping strategy. — The brain learns that facing reminders triggers unbearable panic, so it pushes the person to avoid anything connected to the trauma—but avoidance prevents the brain from learning the danger is over, so the fear grows stronger.
6. Negative thoughts and mood changes take hold. — Avoidance and numbing cut the person off from positive experiences and relationships; low serotonin (the mood-steadying chemical) and high cortisol drive guilt, shame, hopelessness, and emotional flatness.
7. The body stays locked in red-alert mode (hyperarousal). — The sympathetic nervous system (fight-or-flight system) never gets the signal to stand down, so norepinephrine stays high—muscles stay tense, sleep is broken, startle reflex is hair-trigger, and the person scans constantly for danger (hypervigilance).
8. Every symptom is the brain trying to survive a danger that is over. — Intrusions, avoidance, dark thoughts, and hyperarousal are not random—they are the brain and body working perfectly to protect against a threat that is no longer there. The system is not broken; it is stuck in the past, and therapy helps it catch up to the present.

## Symptoms · what the patient feels, and why

---

- **Flashbacks or intrusive memories:** The amygdala (the brain's alarm center) stays hyper-alert after trauma and keeps replaying the danger signal even when the event is over, so the memory bursts in unwanted—like the alarm keeps ringing even though the fire is out.
- **Nightmares or sleep problems:** High cortisol (the body's main stress hormone) and an overactive amygdala disrupt normal sleep cycles and trigger scary dreams, because the brain is stuck in threat mode and cannot fully relax at night.
- **Avoiding people, places, or things that remind you of the trauma:** The brain tries to protect you by steering clear of anything that could re-trigger the fear alarm, so you skip places or conversations that feel connected to the bad event—like ducking away from a hot stove after you got burned.
- **Feeling numb, detached, or unable to enjoy things:** The prefrontal cortex (the front part of the brain that manages feelings) gets overwhelmed and turns down emotional volume to stop the flood; serotonin (a brain messenger that steadies mood) also drops, so you feel flat or disconnected from people and hobbies you used to love.
- **Being jumpy, easily startled, or always on edge:** The amygdala stays in high-alert mode and the body pumps extra adrenaline and cortisol, so your nervous system reacts to small sounds or movements as if they were real danger—like a smoke detector that goes off every time you toast bread.
- **Anger outbursts or irritability:** Chronic stress keeps cortisol and norepinephrine (a brain signal for fight-or-flight) high, and the prefrontal cortex that normally pumps the brakes on big feelings is weakened, so frustration boils over fast.
- **Trouble concentrating or remembering things:** High cortisol shrinks connections in the hippocampus (the brain's memory and learning center), and constant hypervigilance uses up mental energy, leaving less brain power for focus and everyday tasks.
- **Thoughts of hurting yourself or ending your life:** Severe PTSD can cause very low serotonin and dopamine (brain messengers that support hope and reward), plus hopelessness from ongoing suffering, which can lead to suicidal thinking—this is a medical emergency that needs immediate care.

## Focused exam

---

- **Hypervigilance — eyes darting around the room, jumps hard at a sudden noise or door slam:** The amygdala (the brain's alarm center that flags danger) is stuck on high alert, as if the threat is still happening right now. The person's nervous system cannot turn off its threat-scanner, so every small sound feels like danger.

- Avoidance — refuses to talk about the trauma, skips places or people that trigger memories: The hippocampus (the brain's memory-filing system) never marked the trauma as 'done and over.' Because the memory feels like it is still happening, the brain runs away from anything that reminds it of the event to avoid reliving the terror.
- Intrusive memories or flashbacks — suddenly feels like the trauma is happening again, right now: The trauma memory is not stored as a finished story from the past. Instead, it replays as if it is live — the person does not remember the event, they relive it. This means the memory circuit broke and cannot label the event as 'old news.'
- Negative mood or guilt — 'It was my fault,' 'I deserved it,' or 'I should have died instead': The prefrontal cortex (the front of the brain that helps you think clearly and put on the brakes) goes quiet when the amygdala screams too loud. The person cannot think straight about what happened, so blame and dark thoughts stick hard.
- Sleep trouble — nightmares about the trauma, or waking up in a panic and staying awake: During sleep, the brain is supposed to sort and file memories. In PTSD, the trauma memory gets stuck on repeat instead. The amygdala keeps the threat-alarm ringing, so the body refuses to relax enough to sleep.
- Body signs of being wound up — resting heart rate over 90, tight muscles, clenched jaw: The sympathetic nervous system (the part that flips on 'run or fight' mode) is stuck in the ON position all day. The body cannot relax because the brain keeps signaling that danger is near, flooding the body with adrenaline and cortisol (the main stress hormone).
- Using alcohol, drugs, or self-harm to cope: When the fear becomes unbearable, the person tries to quiet it with substances or pain. Alcohol and drugs numb the pain for a little while, but they stop the brain from healing the broken memory circuit and raise the risk of overdose or suicide.

## Diagnostics · every test answers a question

---

- PTSD Checklist for DSM-5 (PCL-5) or Clinician-Administered PTSD Scale (CAPS-5) → Does the person meet the full picture for PTSD — trauma exposure, intrusions, avoidance, negative mood, and hyperarousal lasting more than one month and causing real problems at work or home?
- PHQ-9 (depression screen) → Is depression happening at the same time as PTSD, or is the low mood only part of the PTSD itself?
- GAD-7 (anxiety screen) → Is there a separate generalized anxiety disorder on top of PTSD, or is the worry only from hypervigilance?
- Columbia-Suicide Severity Rating Scale (C-SSRS) → Is the person thinking about suicide or planning to hurt themselves?

- Toxicology screen (urine drug test) → Is the person using substances to self-medicate the PTSD symptoms?
- Thyroid-stimulating hormone (TSH) → Could a thyroid problem be causing or worsening the anxiety, sleep trouble, or mood symptoms?

## Treatment logic

---

- Trauma-focused cognitive behavioral therapy (TF-CBT) or Eye Movement Desensitization and Reprocessing (EMDR) → Rewires the broken fear memory by helping the person process the trauma safely, bit by bit, instead of avoiding it. This lets the prefrontal cortex (the brain's thinking and braking center) talk to the amygdala (the alarm center) again and finally mark the trauma as 'done and over.'
- SSRI antidepressant — sertraline or paroxetine (FDA-approved for PTSD) → Boosts serotonin (a brain messenger that steadies mood and puts the brakes on fear) so the prefrontal cortex gets stronger chemical fuel to calm down the amygdala's alarm. Makes therapy stick better and lowers intrusions and hyperarousal.
- Prazosin (an alpha-blocker) for nightmares → Blocks norepinephrine (adrenaline's cousin) at night so the body stops jolting awake in fight-or-flight mode. Better sleep means the brain can heal and file memories during rest.
- Safety, housing stability, and regular therapy access → A safe bed, a steady place to live, and scheduled therapy appointments tell the nervous system that the danger is actually over. The body can only learn that lesson if it feels safe enough to listen and the brain can predict that help is coming.
- Avoid benzodiazepines (like lorazepam or clonazepam) for PTSD → Benzos boost GABA (the brain's main calming signal) and numb fear fast, but they stop the brain from processing the trauma memory. They also raise the risk of dependence, overdose when mixed with alcohol or opioids, and make PTSD worse over time.
- Consider MDMA-assisted therapy in FDA-approved research settings if standard therapy fails → MDMA (used only under close medical supervision in trials) softens the fear response just enough so the person can talk about the trauma without being flooded by terror. This can help therapy work when nothing else has.

## Medications

---

### **Sertraline (Zoloft) · SSRI (selective serotonin reuptake inhibitor)**

Sertraline raises serotonin—a brain messenger that steadies mood, sleep, and fear signals—by blocking the pump that normally sucks it back into the nerve ending. Over weeks, more serotonin sits in the gap between nerves, which slowly calms the amygdala (the brain's alarm

center that flags danger) and strengthens the prefrontal cortex (the front of the brain that pumps the brakes on big feelings).

1. You swallow the pill; it enters your bloodstream and crosses into the brain.
2. Sertraline plugs the serotonin transporter—the protein that normally vacuum-cleans serotonin out of the synapse—so serotonin levels rise in the gap.
3. Over two to six weeks, higher serotonin levels help the amygdala quiet down its constant danger alerts and help the prefrontal cortex regain control over fear and flashback circuits.
4. Intrusive thoughts, nightmares, and hypervigilance (being on constant lookout for threats) begin to ease; mood and sleep improve.

#### **SAFETY WATCH**

Nausea, headache, trouble sleeping, or feeling jittery in the first one to two weeks—usually fades as your body adjusts. · Sexual side effects (lower interest or delayed climax) in up to half of people; talk to your clinician if this bothers you—sometimes switching doses or adding a different medicine helps. · Small risk of worsening suicidal thoughts in people under age 25 during the first few weeks—watch mood closely and call your clinician right away if dark thoughts spike. · Do NOT stop suddenly—taper slowly under clinician guidance to avoid dizziness, brain zaps (brief electric-shock feelings in the head), or mood swings. · Do NOT combine with MAOIs (an older class of antidepressants) or tramadol without clinician approval—dangerous serotonin buildup can happen.

### **Paroxetine (Paxil) · SSRI (selective serotonin reuptake inhibitor)**

Paroxetine raises serotonin—a brain messenger that steadies mood, sleep, and fear signals—by blocking the pump that normally sucks it back into the nerve ending. Over weeks, more serotonin sits in the gap between nerves, which slowly calms the amygdala (the brain's alarm center) and strengthens the prefrontal cortex (the part that puts the brakes on big feelings).

1. You swallow the pill; it enters your bloodstream and crosses into the brain.
2. Paroxetine plugs the serotonin transporter—the protein that normally vacuum-cleans serotonin out of the synapse—so serotonin levels rise in the gap.
3. Over two to six weeks, higher serotonin levels help the amygdala quiet its constant danger alerts and help the prefrontal cortex regain control over fear and flashback circuits.
4. Intrusive thoughts, nightmares, and hypervigilance (being on constant lookout for threats) begin to ease; mood and sleep improve.

**SAFETY WATCH**

Nausea, dry mouth, drowsiness, or weight gain—common in the first few weeks. · Sexual side effects (lower interest or delayed climax) in up to half of people; talk to your clinician if this bothers you. · Paroxetine has a shorter half-life than other SSRIs, so stopping suddenly can cause more intense withdrawal symptoms (dizziness, brain zaps, irritability)—always taper slowly under clinician guidance. · Small risk of worsening suicidal thoughts in people under age 25 during the first few weeks—watch mood closely and call your clinician right away if dark thoughts spike. · Do NOT combine with MAOIs or tramadol without clinician approval—dangerous serotonin buildup can happen. · Use caution in pregnancy—paroxetine carries a small increased risk of heart defects in the baby during the first trimester; your clinician will help weigh risks and benefits.

**Prazosin (Minipress) · Alpha-1 adrenergic blocker**

Prazosin blocks alpha-1 receptors—docking ports for norepinephrine (the brain's fight-or-flight chemical)—in the brain and blood vessels. By blocking these ports, prazosin turns down the surge of norepinephrine that triggers nightmares and night sweats, so you sleep through the night without terrifying dreams jolting you awake.

1. You take prazosin about one to two hours before bed; it enters your bloodstream and crosses into the brain.
2. Prazosin plugs the alpha-1 receptors so norepinephrine cannot dock and send its fight-or-flight alarm.
3. With the norepinephrine alarm muffled, the brainstem stays calmer during REM sleep (the dream stage), so nightmares are less intense and less frequent.
4. You wake less often in terror, sweat less at night, and feel more rested in the morning.

**SAFETY WATCH**

Dizziness or feeling lightheaded when you stand up, especially the first few nights—prazosin lowers blood pressure, so stand slowly and sit on the edge of the bed before getting up. · Start at a low dose (usually one milligram at bedtime) and increase slowly to find the dose that stops nightmares without making you too dizzy. · Take it only at night—taking it during the day can cause fatigue and low blood pressure when you are active. · If you miss a dose, do NOT double up the next night—just resume the usual dose. · Tell your clinician if you have very low blood pressure, heart failure, or are taking other blood-pressure drugs—prazosin can stack with them and drop pressure too low.

**Venlafaxine (Effexor XR) · SNRI (serotonin-norepinephrine reuptake inhibitor)**

Venlafaxine raises both serotonin (a brain messenger that steadies mood and sleep) and norepinephrine (a messenger that sharpens focus and energy) by blocking the pumps that normally suck them back into the nerve ending. Over weeks, more of both messengers sit in the gap between nerves, which calms the amygdala's alarm and strengthens the prefrontal cortex's control over fear and flashback circuits.

1. You swallow the extended-release capsule; it dissolves slowly and enters your bloodstream, then crosses into the brain.
2. Venlafaxine plugs both the serotonin and norepinephrine transporters so both messengers stay in the synapse longer.
3. Over two to six weeks, higher levels of serotonin and norepinephrine help the amygdala quiet its constant danger alerts and help the prefrontal cortex regain control over fear responses.
4. Intrusive thoughts, hypervigilance, emotional numbness, and low energy begin to ease.

**SAFETY WATCH**

Nausea, headache, sweating, or feeling jittery in the first one to two weeks—usually fades as your body adjusts. · Can raise blood pressure slightly at higher doses—your clinician will check your pressure regularly. · Sexual side effects (lower interest or delayed climax) are common; talk to your clinician if this bothers you. · Small risk of worsening suicidal thoughts in people under age 25 during the first few weeks—watch mood closely and call your clinician right away if dark thoughts spike. · Do NOT stop suddenly—taper slowly under clinician guidance to avoid dizziness, brain zaps, irritability, or flu-like feelings. · Do NOT combine with MAOIs or tramadol without clinician approval—dangerous serotonin buildup can happen.

**Fluoxetine (Prozac) · SSRI (selective serotonin reuptake inhibitor)**

Fluoxetine raises serotonin—a brain messenger that steadies mood, sleep, and fear signals—by blocking the pump that normally sucks it back into the nerve ending. Over weeks, more serotonin sits in the gap between nerves, which slowly calms the amygdala (the brain's alarm center) and strengthens the prefrontal cortex (the part that puts the brakes on big feelings).

1. You swallow the pill; it enters your bloodstream and crosses into the brain.
2. Fluoxetine plugs the serotonin transporter—the protein that normally vacuum-cleans serotonin out of the synapse—so serotonin levels rise in the gap.
3. Over two to six weeks, higher serotonin levels help the amygdala quiet its constant danger alerts and help the prefrontal cortex regain control over fear and flashback circuits.
4. Intrusive thoughts, nightmares, and hypervigilance begin to ease; mood and sleep improve.

**SAFETY WATCH**

Nausea, headache, trouble sleeping, or feeling jittery in the first one to two weeks—usually fades as your body adjusts. · Sexual side effects (lower interest or delayed climax) in up to half of people; talk to your clinician if this bothers you. · Small risk of worsening suicidal thoughts in people under age 25 during the first few weeks—watch mood closely and call your clinician right away if dark thoughts spike. · Fluoxetine has a very long half-life (stays in your body for weeks), so withdrawal symptoms are usually milder when you stop, but it also means drug interactions can linger—tell any new clinician you are on fluoxetine. · Do NOT combine with MAOIs or tramadol without clinician approval—dangerous serotonin buildup can happen.

## Red flags

---

- Thoughts of suicide or plans to hurt yourself
- Severe dissociation (feeling completely disconnected from your body or reality)
- Psychotic symptoms like hearing voices or seeing things that are not there
- Inability to care for yourself (not eating, bathing, or getting out of bed)
- Violent behavior toward others

## Nursing priorities

---

1. Complete suicide risk assessment using a validated tool like the Columbia Suicide Severity Rating Scale and create a written safety plan with the patient — The amygdala (fear center) and prefrontal cortex (decision-making area) are dysregulated in PTSD, increasing impulsive self-harm risk. PTSD patients have 4 to 6 times higher suicide attempt rates than the general population.
2. Establish a calm, predictable environment with controlled lighting, minimal noise, and clear explanations before any physical touch or procedures — The hyperarousal state in PTSD keeps the sympathetic nervous system (fight-or-flight response) chronically activated. Sudden movements, loud noises, or unexpected touch can trigger the amygdala to respond as if the trauma is happening again, causing flashbacks or panic.
3. Screen for traumatic brain injury history, especially in patients with combat or assault trauma, using questions about loss of consciousness or confusion after head impact — TBI (traumatic brain injury) and PTSD share overlapping symptoms like irritability, sleep problems, and concentration difficulties but need different treatments. The hippocampus (memory center) may be damaged by both physical injury and chronic cortisol (stress hormone) exposure.

4. Assess current housing stability, food security, and safety from ongoing abuse or violence before focusing on trauma therapy — The hypothalamic-pituitary-adrenal axis (stress response system) cannot reset when a person faces ongoing threats. Active danger keeps cortisol levels elevated, making trauma processing ineffective and potentially retraumatizing.
5. Review all current medications and flag benzodiazepines like lorazepam or clonazepam for gradual discontinuation under provider supervision — Benzodiazepines interfere with fear extinction learning in the prefrontal cortex, making exposure therapy less effective. VA/DoD guidelines show benzodiazepines worsen PTSD outcomes and increase substance dependence risk.
6. Document trauma nightmares separately from general insomnia, including frequency and content, to guide prazosin dosing — Prazosin blocks alpha-1 adrenergic receptors in the brain, reducing norepinephrine (stress chemical) surges during REM sleep. Typical dosing starts at 1 milligram at bedtime and increases based on nightmare frequency, often reaching 5 to 15 milligrams for effect.
7. Connect the patient with trauma-focused therapy resources and verify they understand PTSD is treatable with specific evidence-based therapies, not just medication — Neuroplasticity (the brain's ability to rewire) allows trauma-focused CBT and EMDR to reduce amygdala hyperactivity and strengthen prefrontal cortex control. Studies show 60 to 80 percent of patients improve significantly with these therapies, but many believe PTSD is permanent.
8. Monitor blood pressure before and after prazosin doses, especially the first dose and any dose increases, as orthostatic low blood pressure (hypotension) can cause falls — Prazosin lowers blood pressure by blocking alpha-1 receptors in blood vessels. A drop of more than 20 millimeters mercury systolic when standing increases fall risk, especially in older adults or patients taking other blood pressure medications.

## Sources

---

- VA/DoD Clinical Practice Guideline for PTSD 2023
- American Psychiatric Association (DSM-5-TR)
- National Institute of Mental Health (NIMH)
- Cochrane Review on Psychological Therapies for PTSD

Last reviewed: 2026-07-07