

Post-Traumatic Stress Disorder

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THE STORY IN ONE SENTENCE

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“Post-Traumatic Stress Disorder means your brain stays stuck in alarm mode even after the danger is gone.”

Normal anatomy — the body's job

A healthy brain keeps fear in check by balancing two main systems: the amygdala (the alarm center deep in the brain that flags danger) and the prefrontal cortex (the front part of the brain that weighs whether the alarm is real and tells the amygdala to calm down). When something scary happens, the amygdala fires, your body floods with adrenaline (a hormone that powers the fight-or-flight response), and after the danger passes, the prefrontal cortex steps in to say 'it is over, stand down.' The memory gets stored as a past event, not a current threat.

BODY'S JOB

The amygdala scans your surroundings every second for danger — like a smoke detector that never sleeps. The prefrontal cortex is like the person who checks whether it is real smoke or just burnt toast, then resets the alarm. Together, they keep you alert when you need to be and calm when you are safe. The hippocampus (a nearby brain structure that handles memory) time-stamps events so you know 'that happened then, not now.'

1. Scary event happens → amygdala fires → adrenaline and cortisol (the body's main stress hormone) flood the body → heart pounds, muscles tense, attention narrows to the threat.
2. Danger passes → prefrontal cortex checks the situation → sends calming signals to the amygdala → adrenaline and cortisol levels drop → body returns to baseline.
3. Hippocampus files the memory with a clear time stamp: 'That was then. I am safe now.' The memory becomes a story you can think about without reliving it.

What breaks

In PTSD, the trauma is so overwhelming that the prefrontal cortex cannot do its job in the moment. The amygdala stays locked in alarm mode, the hippocampus fails to time-stamp the memory as 'over,' and the trauma gets stored as if it is still happening. Every reminder reactivates the same fear circuit, so the memory feels real instead of past. Chronic stress also shrinks parts of the prefrontal cortex and hippocampus over time, making it even harder to calm the alarm or sort past from present.

CLINICAL TERM

The core defect in PTSD is a failure of fear extinction and memory consolidation. During or immediately after trauma, overwhelming stress impairs prefrontal cortex regulation of the amygdala and disrupts hippocampal encoding, so the traumatic memory is stored in a hyperaroused, fragmented, sensory-dominant form without a clear contextual time stamp. Subsequent reminders trigger amygdala reactivation as if the threat were current (re-experiencing). Chronic hyperarousal downregulates prefrontal inhibitory control and can cause structural changes (reduced hippocampal and prefrontal cortex volume on MRI in chronic PTSD). Dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis and altered levels of norepinephrine, cortisol, and serotonin perpetuate the cycle.

How the body compensates

At first, the body tries to protect you by avoiding reminders, numbing feelings, and staying on guard. These are survival tactics, not choices. But over time, avoidance shrinks your world, hyperarousal exhausts your body, and the thinking brain gets so tired it cannot fight back against dark thoughts. The system looks like it is holding together until a new stressor, re-traumatization, or isolation tips it over the edge.

1. Avoidance buys short-term relief: staying away from people, places, or thoughts that trigger the alarm keeps panic at bay for a while, but it also prevents the brain from learning that those reminders are not actually dangerous.
2. Emotional numbing protects against overwhelm: the brain dials down all feelings to avoid the painful ones, so you might feel detached, empty, or unable to enjoy things you used to love.
3. Hyperarousal keeps you 'ready': staying tense, scanning for danger, and sleeping lightly feel like they keep you safe, but they exhaust your body and brain, making it even harder to heal.
4. Negative beliefs become a mental shield: thinking 'I cannot trust anyone' or 'the world is dangerous' feels like it protects you from future hurt, but it isolates you and blocks recovery.

5. Big danger: re-traumatization during care or in daily life can deepen the circuit failure and worsen symptoms rapidly.
6. Suicide risk is real: PTSD roughly doubles the lifetime risk of suicide, especially when combined with depression, substance use, or isolation.
7. Substance use disorder (especially alcohol) is common and dangerous: people use alcohol or drugs to numb the alarm, but substances disrupt sleep, worsen depression, block therapy progress, and add their own medical and social harms.

Domino effect

1. Trauma overwhelms the brain's normal fear-processing system. — During the trauma, the amygdala (alarm center) fires at full strength, but the prefrontal cortex (thinking brain) is overwhelmed by the intensity, and the hippocampus (memory time-stamper) cannot file the event as 'over.' The memory gets stored in a raw, sensory, emotionally-charged form without a clear past-tense label.
2. The trauma memory stays stuck in the amygdala as a current threat. — Because the hippocampus did not time-stamp it properly, the brain treats the memory as happening now instead of then. Any reminder — a smell, a sound, a place, even a feeling — reactivates the amygdala as if the danger is back, so you relive the trauma instead of remembering it like a normal past event.
3. Intrusive re-experiencing: flashbacks, intrusive thoughts, and emotional flooding happen over and over. — Every reminder triggers the same fear circuit the trauma originally activated, so your heart pounds, your muscles tense, and you feel the same terror even though you are safe. The brain cannot tell the difference between the memory and the real thing.
4. Nightmares disrupt sleep and reinforce the fear loop. — During sleep, the brain tries to process and file away the stuck memory, but because the amygdala is still in overdrive, the processing comes out as nightmares. Waking up in terror reinforces the idea that you are not safe, and broken sleep prevents the brain from healing.
5. Avoidance of reminders becomes a central coping strategy. — The brain learns that anything connected to the trauma — people, places, conversations, even thoughts — triggers the alarm, so you avoid them to escape the panic. Avoidance gives short-term relief but prevents the brain from learning that those reminders are not actually dangerous, so the fear never fades.
6. Negative thoughts and beliefs take over. — Being stuck in alarm mode for weeks or months exhausts the prefrontal cortex, so it cannot challenge dark thoughts like 'I am broken,' 'it was my fault,' or 'no one is safe.' Those thoughts start to feel like facts instead of fear talking.

7. Hyperarousal keeps the body on constant high alert. — The amygdala keeps the body in fight-or-flight mode all day: muscles stay tight, your heart rate stays elevated, you jump at sudden sounds, and you scan every room for danger. This vigilance is exhausting and prevents relaxation even when you are objectively safe.
8. Sleep becomes fragmented and non-restorative. — Nightmares wake you up, hyperarousal keeps your body tense, and the alarm brain never fully shuts off. Without deep sleep, the prefrontal cortex cannot recover, memory consolidation fails, and the fear circuit stays stuck. Poor sleep also worsens mood, irritability, and the ability to cope during the day.
9. Every symptom — intrusive memories, avoidance, negative thinking, hyperarousal, broken sleep — is the body trying to survive with a fear system that is stuck in overdrive and a thinking brain that is too exhausted to reset it. — None of this is random or a character flaw. Each finding is the brain doing its best to protect you with a circuit that got jammed by trauma. Recovery requires helping the thinking brain come back online (through trauma-focused therapy like Cognitive Processing Therapy or Prolonged Exposure, which teach the brain to reprocess the memory safely) and sometimes medicine (like sertraline or paroxetine, SSRI antidepressants that raise serotonin and help regulate mood and fear) so the alarm can finally stand down and the memory can become a story from the past instead of a constant threat.

Symptoms · what the patient feels, and why

- Intrusive memories or nightmares of the trauma: The amygdala (the brain's alarm center) stays on high alert after the trauma and keeps replaying the scary event, even when you're safe. The hippocampus (the brain's memory organizer) has trouble filing the memory away as 'over,' so it pops up like it's happening again.
- Avoiding reminders of the trauma (people, places, conversations, or thoughts): Your brain tries to protect you from the pain by steering clear of anything that brings the trauma back. The prefrontal cortex (the front of the brain that makes plans) and the amygdala work together to shut down routes that trigger the fear.
- Feeling constantly on edge or jumpy (hyperarousal): The body's stress system—driven by the amygdala and fueled by cortisol (the main stress hormone) and norepinephrine (a fight-or-flight messenger)—stays stuck in 'danger mode.' Your nervous system thinks the threat is still there, so it keeps you ready to run or fight.
- Trouble sleeping or staying asleep: High levels of cortisol and norepinephrine keep your brain and body too alert to settle into deep sleep. Nightmares can also jolt you awake. The brain's sleep centers can't calm down when the alarm system won't turn off.

- Feeling numb, detached, or cut off from others: The brain tries to dull overwhelming feelings by turning down signals in the prefrontal cortex and limbic system (the emotion network). This blunts both bad and good feelings, so you may feel empty or far away even from people you care about.
- Negative thoughts about yourself or the world ('I'm broken,' 'nowhere is safe'): After trauma, the hippocampus and prefrontal cortex can overgeneralize danger and lose balanced perspective. Low serotonin (a brain messenger that steadies mood) also makes dark thoughts stick and crowd out hopeful ones.
- Sudden anger or irritability: The amygdala stays overactive and fires off strong reactions to small triggers. The prefrontal cortex (which normally pumps the brakes on big feelings) is weakened, so frustration or anger can flare up fast and feel hard to control.
- Thoughts of harming yourself or ending your life: PTSD can cause severe hopelessness, shame, and unbearable emotional pain. Low levels of serotonin and disrupted circuits in the prefrontal cortex make it harder to see a way forward, and suffering feels endless.

Focused exam

- Person startles easily when a door slams or phone rings: In Post-Traumatic Stress Disorder, the amygdala (the brain's alarm center that flags danger) stays stuck in high gear. The startle reflex gets turned up too high because the fear circuit never turns off, so even safe sounds feel like threats.
- Person avoids talking about the trauma or leaves the room when it comes up: Avoidance is a core sign of Post-Traumatic Stress Disorder. The hippocampus (the part of the brain that files memories) learned that thinking about the trauma feels dangerous, so it shuts the memory away to protect the person from reliving the pain.
- Nightmares about the traumatic event, multiple times per week: In Post-Traumatic Stress Disorder, the amygdala does not calm down at night. The brain replays the trauma instead of resting, because the fear circuit never got the signal that the danger is over.
- Flat mood, loss of interest in things they used to enjoy: Post-Traumatic Stress Disorder lowers serotonin and dopamine (brain messengers that control mood and pleasure). The brain stops signaling happiness the normal way, so everything feels gray and empty.
- Sleeping only 3 to 4 hours per night, tossing and turning: In Post-Traumatic Stress Disorder, norepinephrine (the brain's emergency alert chemical) stays high. Sleep cannot come when the brain thinks danger is near, so the person lies awake or wakes often.
- Trouble concentrating during a conversation; mind keeps drifting back to the trauma: Post-Traumatic Stress Disorder hijacks the prefrontal cortex (the front of the brain that focuses attention). The amygdala keeps pulling attention back to the scary memory instead of letting the person stay in the present, so focus breaks.

- Person says life is pointless and mentions wanting to be dead: Post-Traumatic Stress Disorder and suicide risk travel together. The combination of constant emotional pain, hopelessness, and low serotonin creates serious danger. This needs immediate safety planning and close follow-up.

Diagnostics · every test answers a question

- PTSD Checklist for DSM-5 (PCL-5) → Does the person meet the symptom criteria for Post-Traumatic Stress Disorder, and how severe are the symptoms?
- Clinician-Administered PTSD Scale (CAPS-5) → Is this truly Post-Traumatic Stress Disorder, and how bad is each symptom cluster (intrusion, avoidance, mood, arousal)?
- Patient Health Questionnaire-9 (PHQ-9) → Is depression also present, and is the person at risk for suicide?
- Generalized Anxiety Disorder-7 (GAD-7) → Is generalized anxiety disorder also present alongside Post-Traumatic Stress Disorder?
- Urine drug screen → Is the person using alcohol or other substances to numb the Post-Traumatic Stress Disorder symptoms?
- Thyroid-stimulating hormone (TSH) → Could thyroid disease be causing or worsening the sleep trouble, mood changes, or concentration problems?

Treatment logic

- Trauma-focused cognitive behavioral therapy (CBT) or Eye Movement Desensitization and Reprocessing (EMDR) for 12 to 16 weekly sessions → Rewires how the hippocampus stores the trauma memory and teaches the prefrontal cortex (the front of the brain that pumps the brakes on fear) to override the amygdala. The memory stops triggering the alarm system every time a reminder shows up, so the person can think about the event without reliving it.
- Sertraline 50 to 200 mg daily or paroxetine 20 to 60 mg daily (the two SSRIs approved by the FDA for Post-Traumatic Stress Disorder) → Boosts serotonin (a brain messenger that steadies mood and sleep) so the prefrontal cortex can put the brakes on the amygdala. The person can think clearer, sleep better, and stay less stuck in alarm mode. Start low and raise the dose slowly over 4 to 8 weeks.
- Prazosin 1 to 20 mg at bedtime for nightmares → Blocks norepinephrine (the brain's emergency alert chemical) during sleep so the amygdala stops replaying the trauma over and over at night. The person actually rests instead of waking in panic.

- Fix housing, ensure a steady safe place to sleep, connect to substance use treatment if needed, and schedule therapy at the same time each week → Removes the constant new stressors that keep the amygdala locked on high alert. Stable shelter, routine, and sobriety let the medicine and therapy actually work, because the brain can focus on healing instead of surviving the next crisis.
- Avoid benzodiazepines (like lorazepam, clonazepam, or alprazolam); do not prescribe them for Post-Traumatic Stress Disorder → Stops the trap where the person feels relief for 4 hours but their amygdala learns to fear the next moment without the pill, making Post-Traumatic Stress Disorder worse over time. Also prevents physical dependence and addiction.

Medications

Sertraline (Zoloft) · SSRI - selective serotonin reuptake inhibitor

Sertraline raises the level of serotonin (a brain messenger that steadies mood, sleep, and worry) by blocking the pump that normally sucks serotonin back out of the gap between nerve cells. With more serotonin staying in the gap longer, the brain gradually calms overactive fear circuits and steadies emotional responses to reminders of the trauma.

1. Sertraline blocks the serotonin reuptake transporter, a protein pump on the sending nerve cell that normally recycles serotonin out of the gap.
2. Serotonin stays in the gap longer and keeps activating receptors on the receiving nerve cell.
3. Over two to four weeks, the extra serotonin tells the amygdala to dial down its alarm volume and helps the prefrontal cortex regain control over fear responses.
4. Intrusive memories, nightmares, irritability, and the constant feeling of being on guard gradually ease.

SAFETY WATCH

Takes two to four weeks to start working; symptoms may feel worse in the first week. · Can cause nausea, loose stools, headache, trouble reaching orgasm, or feeling restless early on. · In people under 25, watch closely for new suicidal thoughts in the first few weeks. · Do NOT stop suddenly — taper slowly to avoid dizziness, zaps, and rebound anxiety. · Rarely can trigger serotonin syndrome (too much serotonin causing confusion, fast heart rate, sweating, muscle jerks) if combined with other serotonin drugs like tramadol or certain migraine medicines.

Paroxetine (Paxil) · SSRI - selective serotonin reuptake inhibitor

Paroxetine raises serotonin (a brain messenger that steadies mood and calms overactive fear) by blocking the pump that recycles serotonin back into nerve cells. More serotonin in the gap between brain cells gradually quiets the amygdala's alarm and helps the prefrontal cortex regain control over trauma responses.

1. Paroxetine blocks the serotonin reuptake pump on the sending nerve cell.
2. Serotonin stays in the gap longer and keeps activating receptors on the receiving cell.
3. Over two to four weeks, the extra serotonin tells overactive fear circuits to calm down.
4. Intrusive thoughts, irritability, hypervigilance (the constant feeling of being on guard), and avoidance gradually improve.

SAFETY WATCH

Takes two to four weeks to start working; some people feel more anxious or restless in week one. · Can cause dry mouth, constipation, weight gain, sweating, trouble reaching orgasm, or drowsiness. · In people under 25, watch closely for new or worse suicidal thoughts in the first month. · Has the worst withdrawal of any SSRI if stopped suddenly — must taper very slowly to avoid dizziness, brain zaps, irritability, and rebound anxiety. · Can interact with tamoxifen (a breast cancer drug) and reduce its effectiveness. · Rarely can trigger serotonin syndrome if combined with other serotonin-boosting medicines.

Prazosin (Minipress) · Alpha-1 blocker

Prazosin blocks alpha-1 receptors (docking sites for norepinephrine, the brain's main wake-up and alarm chemical) in the brain. Norepinephrine surges during REM sleep in PTSD and re-plays the trauma as vivid nightmares. By blocking these receptors, prazosin keeps the alarm chemical from hijacking dreams, so nightmares become less frequent and less terrifying.

1. Prazosin is taken at bedtime and crosses into the brain.
2. It blocks alpha-1 receptors, stopping norepinephrine from docking and firing up fear circuits during sleep.
3. The amygdala and locus coeruleus stay quieter during REM sleep.
4. Nightmares become less intense, less frequent, or stop entirely, and sleep quality improves.

SAFETY WATCH

Can cause dizziness or fainting when standing up, especially after the first dose — start with a low dose at bedtime and rise slowly in the morning. · May lower blood pressure too much if the person is already on blood pressure medicine. · Can cause stuffy nose, headache, or feeling tired during the day. · If nightmares return after stopping, they may come back just as strong (no taper usually needed, but nightmares often resume). · Does not work for everyone — about half of people get clear relief; the other half see little change.

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

Venlafaxine blocks the pumps that recycle both serotonin (a mood and sleep steadier) and norepinephrine (a focus and energy messenger) back into nerve cells. More of both chemicals stay in the gaps between brain cells, which calms overactive fear circuits and helps the prefrontal cortex regain control over trauma responses. It is used when an SSRI alone has not worked well enough.

1. Venlafaxine blocks both the serotonin reuptake pump and the norepinephrine reuptake pump.
2. Both chemicals stay in the gap longer and keep activating their receptors.
3. Over two to four weeks, the extra serotonin steadies mood and sleep, and the extra norepinephrine helps focus and motivation.
4. Intrusive thoughts, hypervigilance, irritability, and emotional numbness gradually improve.

SAFETY WATCH

Takes two to four weeks to start working. · Can raise blood pressure, especially at higher doses — check blood pressure before starting and a few weeks later. · Can cause nausea, dry mouth, sweating, trouble sleeping, or trouble reaching orgasm. · In people under 25, watch for new suicidal thoughts in the first month. · Withdrawal is often harsh if stopped suddenly — must taper slowly to avoid dizziness, brain zaps, irritability, and flu-like aches. · Rarely can trigger serotonin syndrome if combined with other serotonin drugs.

****NOT recommended: Benzodiazepines (lorazepam, clonazepam, alprazolam)**
· GABA-A receptor agonist (enhances the brain's main calming signal)**

Benzodiazepines boost GABA (the brain's main brake pedal) to quiet anxiety fast. However, in PTSD they interfere with the brain's natural process of learning that a trigger is no longer dangerous. They also cause dependence, worsen depression, raise the risk of overdose

(especially with opioids or alcohol), and make trauma therapy less effective. Current VA/DoD and APA guidelines say do NOT use them for PTSD.

1. Benzodiazepine docks at the GABA-A receptor and makes GABA's calming signal stronger.
2. Nerve cells fire less, and the person feels less anxious within 30 minutes.
3. The brain quickly gets used to this artificial calm and makes less GABA on its own.
4. Tolerance builds (the dose stops working), and stopping causes rebound anxiety, insomnia, and sometimes seizures.
5. The drug also blocks fear extinction (the process where the brain learns a trigger is safe now), so trauma therapy becomes harder.

SAFETY WATCH

High risk of dependence — the brain adapts within days to weeks, and stopping causes severe rebound anxiety, insomnia, tremor, and seizures. · Worsens depression and raises suicide risk in PTSD. · Sharply increases overdose death risk when combined with opioids or alcohol. · Impairs memory and learning, making trauma-focused therapy less effective. · Older adults face higher fall and confusion risk. · VA/DoD 2023 and APA 2017 guidelines explicitly recommend AGAINST benzodiazepines for PTSD.

Red flags

- Thoughts of suicide or a plan to harm yourself
- Harming yourself or taking dangerous risks on purpose
- Hearing voices or seeing things that aren't there (psychosis)
- Unable to care for yourself (not eating, drinking, or leaving bed for days)
- Severe panic attacks that feel like a heart attack or make you pass out

Nursing priorities

1. Ask directly about suicidal thoughts using clear words ('Are you thinking of killing yourself?') and assess for a specific plan, means, and timeline at every visit — The amygdala (fear center in the brain) stays overactive in Post-Traumatic Stress Disorder while the prefrontal cortex (decision-making area) shuts down, making impulsive self-harm more likely; direct questions reduce suicide risk by 30 percent because patients feel permission to share dangerous thoughts

2. Screen for alcohol and drug use with a validated tool like the AUDIT-C (Alcohol Use Disorders Identification Test) and connect patient to substance treatment before starting trauma therapy — Substances temporarily lower amygdala hyperactivity but prevent the brain from processing trauma memories; continuing use during therapy blocks neuroplasticity (the brain's ability to rewire itself) and drops therapy success rate from 70 percent to below 30 percent
3. Teach the patient one grounding technique (like 5-4-3-2-1: name five things you see, four you touch, three you hear, two you smell, one you taste) to use during flashbacks — During flashbacks the hippocampus (memory processor) confuses past and present, flooding the body with cortisol and adrenaline as if the trauma is happening now; grounding re-engages the prefrontal cortex to recognize safety in the present moment and stops the stress hormone surge
4. Verify the patient has a safe, stable place to sleep tonight and every night; if homeless, contact social work for emergency shelter placement before discharge — The brain cannot heal from trauma while facing ongoing danger; chronic homelessness keeps cortisol levels elevated 24 hours daily, shrinking the hippocampus and blocking memory processing needed for recovery; stable housing must come before therapy can work
5. Educate that Post-Traumatic Stress Disorder is a brain injury (changes in the amygdala, hippocampus, and prefrontal cortex visible on brain scans), not weakness or being 'crazy,' and recovery takes 3 to 6 months of weekly therapy — Shame and self-blame activate the same brain circuits as the original trauma, raising cortisol and worsening symptoms; understanding Post-Traumatic Stress Disorder as a medical condition reduces shame, increases treatment adherence by 40 percent, and lowers dropout rates from therapy
6. Review all current medications and ask specifically about nightmares; if nightmares occur more than twice weekly, notify provider to consider adding prazosin 1 mg at bedtime — Prazosin blocks alpha-1 receptors (adrenaline landing spots) in the brain that cause vivid trauma nightmares; nightmares fragment sleep architecture (the normal cycle of deep and REM sleep), preventing the brain from consolidating memories and keeping the patient in constant hyperarousal
7. Schedule the first therapy appointment before the patient leaves and give a written card with the date, time, therapist name, and phone number; confirm the patient has transportation — Avoidance is a core Post-Traumatic Stress Disorder symptom driven by an overactive amygdala that labels therapy itself as dangerous; no-show rates exceed 50 percent without concrete scheduling and barrier removal; early therapy start (within 2 weeks) doubles long-term recovery rates

8. Teach family members that anger outbursts and emotional numbness are symptoms of brain changes, not personal rejection, and that recovery is possible with treatment — Post-Traumatic Stress Disorder shrinks the prefrontal cortex (emotion regulation center) and enlarges the amygdala (fear and anger center), causing real loss of emotional control; family conflict raises the patient's cortisol levels and triples dropout from therapy; family understanding improves treatment completion by 35 percent

Sources

- VA/DoD Clinical Practice Guideline for PTSD 2023
- American Psychiatric Association DSM-5-TR 2022
- Cochrane Review on PTSD treatments
- National Center for PTSD (U.S. Department of Veterans Affairs)

Last reviewed: 2026-07-07