

Anxiety Disorder

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THE STORY IN ONE SENTENCE

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“Anxiety disorder—name which type it is first, because each type responds best to a different treatment.”

Normal anatomy — the body's job

The amygdala (the brain's alarm bell, buried deep near the center) watches for danger every second. When it spots a threat, it sends instant signals to the prefrontal cortex (the thinking and planning part behind your forehead), which decides 'Is this danger real?' and 'What should I do?' Then the autonomic nervous system (a network of nerves controlling heartbeat, breathing, sweating, and muscle tension without you thinking about it) kicks the body into high gear—heart rate climbs, breathing speeds up, and muscles tense—so you can fight the danger or run away. This system evolved over millions of years to keep humans alive.

BODY'S JOB

Normal anxiety protects you: you step off a curb and see a car swerving toward you, your amygdala fires in a fraction of a second, adrenaline (a hormone that speeds your heart and sharpens your focus) floods your blood, and you leap back onto the sidewalk. Once you are safe, cortisol (another stress hormone) levels drop, your heart slows back to normal, and the alarm shuts off. This on-off switch is healthy and keeps you ready for real danger without draining your energy all day.

1. Eyes or ears detect a potential threat (a snake on the trail, a loud crash, a stranger approaching fast) and send that information to the amygdala in milliseconds.

2. The amygdala fires and sends urgent signals to the prefrontal cortex ('Check this now!') and to the autonomic nervous system ('Get ready to move!').
3. The prefrontal cortex evaluates the information: 'Is this a real snake or a stick? Is that stranger dangerous or just jogging past me?' If the threat is real, the cortex plans a response (run, hide, call for help). If the threat is false, the cortex sends calming signals back to the amygdala ('Stand down, false alarm').
4. The autonomic nervous system has two branches: the sympathetic branch (gas pedal) releases adrenaline, raises heart rate and blood pressure, sends blood to big muscles, dilates pupils, and makes you sweat—preparing you to fight or flee. The parasympathetic branch (brake pedal) releases acetylcholine (a calming messenger), slows the heart, relaxes muscles, and turns digestion back on once danger passes.
5. In a healthy person, the amygdala and prefrontal cortex balance each other: the amygdala sounds the alarm, the cortex checks if it is real, and the nervous system revs up then calms down within minutes. The cycle completes, and you return to baseline—alert but not exhausted.

What breaks

In anxiety disorders, the amygdala becomes too sensitive (it fires at shadows and safe situations as if they were tigers) or the prefrontal cortex loses its ability to calm the amygdala down (the brake stops working), so the alarm stays on even when there is no danger. The result: your body stays in fight-or-flight mode hour after hour, day after day, and you feel terrified, exhausted, and trapped. Different anxiety disorders break the circuit in different places. Generalized anxiety disorder keeps baseline worry high all the time—you worry about work, health, money, and family every day for months, and you cannot turn it off even when everything is objectively fine. Panic disorder fires sudden, intense false alarms—your heart races, you cannot breathe, you feel like you are having a heart attack or dying, and the surge peaks in minutes then fades, but the fear of the next attack keeps you on edge. Social anxiety disorder misfires whenever other people might judge you—speaking in a meeting, eating in public, or making small talk feels unbearable, and you see every glance or facial expression as proof you are being evaluated and found lacking. Agoraphobia links fear to specific places where escape feels hard or help might not come—buses, bridges, crowds, wide-open parking lots, or even leaving your house alone—so you avoid those places and your safe zone shrinks. Specific phobia locks all the fear onto one trigger (heights, needles, dogs, blood, flying)—the fear is out of proportion to real danger, you know it does not make logical sense, but you cannot override it and you rearrange your life to avoid that one thing.

CLINICAL TERM

Anxiety disorders arise from dysregulation of the amygdala-prefrontal cortex circuit (the amygdala becomes hyperresponsive to perceived threats and the ventromedial prefrontal cortex fails to exert inhibitory control) combined with altered signaling of gamma-aminobutyric acid (GABA, the brain's main inhibitory neurotransmitter that calms neural activity) and serotonin (a monoamine neurotransmitter that modulates mood, arousal, and threat perception). Neuroimaging studies show increased amygdala activation and reduced prefrontal cortex engagement during fear processing in patients with anxiety disorders. Genetic, developmental (early-life stress, trauma), and environmental factors (chronic stress, substance use) contribute. DSM-5-TR defines each subtype by specific criteria. Generalized anxiety disorder (GAD): excessive anxiety and worry about multiple domains (work, health, finances, relationships) occurring more days than not for at least six months, plus three or more of six associated symptoms (restlessness, fatigue, difficulty concentrating, irritability, muscle tension, sleep disturbance) causing significant distress or functional impairment. Panic disorder: recurrent unexpected panic attacks (abrupt surges of intense fear or discomfort reaching a peak within minutes, with four or more of 13 somatic or cognitive symptoms such as palpitations, sweating, trembling, shortness of breath, chest pain, nausea, dizziness, derealization, fear of losing control, fear of dying) plus at least one month of persistent concern about additional attacks or maladaptive behavioral change (avoidance) related to the attacks. Social anxiety disorder (social phobia): marked fear or anxiety about one or more social situations in which the person is exposed to possible scrutiny by others (conversations, meeting unfamiliar people, being observed eating or drinking, performing in front of others), lasting at least six months, where the person fears they will act in a way or show anxiety symptoms that will be negatively evaluated, leading to avoidance or endurance with intense distress and significant impairment. Agoraphobia: marked fear or anxiety about two or more of five situations (using public transportation, being in open spaces, being in enclosed spaces, standing in line or being in a crowd, being outside the home alone) lasting at least six months, where the person fears escape might be difficult or help unavailable if panic-like or embarrassing symptoms occur, leading to avoidance or requiring a companion, causing significant distress or impairment. Specific phobia: marked fear or anxiety about a specific object or situation (flying, heights, animals, injections, seeing blood) lasting at least six months, where the phobic object or situation almost always provokes immediate fear, is actively avoided or endured with intense distress, and the fear is out of proportion to actual danger, causing significant impairment. Each subtype shows distinct neural substrates on functional MRI and differential response profiles to cognitive-behavioral therapy (CBT, a structured psychotherapy that identifies and restructures maladaptive thoughts and uses exposure to extinguish conditioned fear) versus selective serotonin reuptake inhibitors (SSRIs) or serotonin-norepinephrine reuptake inhibitors (SNRIs).

How the body compensates

Patients with anxiety disorders often hide their symptoms, avoid triggers entirely, or self-medicate with alcohol or benzodiazepines (sedative medicines that calm the nervous system quickly but carry high risk of tolerance and addiction), so on a brief clinic visit vital signs and affect may look deceptively stable. The real danger is missing suicide risk (especially when depression coexists), substance use disorder (which develops silently), or the tipping point where avoidance collapses work and relationships. Effective assessment requires asking about function—'Can you work? Leave the house? See friends?'—not just 'How anxious do you feel?'

1. {'label': 'Early avoidance gives false reassurance', 'detail': 'In the beginning, avoiding triggers keeps panic at bay—patients skip social events, stop driving on highways, quit jobs that require public speaking, or stay home instead of taking the bus—and they report 'I feel better' or 'I have not had a panic attack in weeks,' which masks the fact that their world is shrinking and functional capacity is declining.'}
2. {'label': 'The dangerous middle: stable appearance, mounting disability', 'detail': 'This is the phase clinicians often miss. The patient has narrowed life to a small safe zone (home, one trusted friend, online shopping instead of grocery stores) so they experience less acute distress day-to-day, but disability is building silently—lost income, strained relationships, social isolation, and loss of identity and purpose. The patient looks calm in the exam room because they have not confronted a trigger recently, but they are not recovering.'}
3. {'label': 'Watch for crash points: suicidal ideation, substance dependence, total withdrawal', 'detail': 'The complications that kill or permanently disable patients are not the anxiety symptoms themselves but the secondary consequences. Suicidal ideation emerges in up to 20% of patients with anxiety disorders, especially when comorbid major depressive disorder (present in 60 to 70% of cases) develops. Benzodiazepine or alcohol dependence develops insidiously as patients escalate doses to maintain relief, leading to tolerance (needing higher doses for the same effect), withdrawal symptoms (rebound anxiety, tremor, seizures), and overdose risk. Complete social withdrawal and inability to work signal severe impairment and predict long-term disability if not addressed urgently.'}
4. {'label': 'Screen every visit for depression, substance use, and suicidal thoughts', 'detail': 'Use validated tools: PHQ-9 (9-item Patient Health Questionnaire for depression, score 10 or higher indicates moderate depression requiring treatment), GAD-7 (7-item Generalized Anxiety Disorder scale, score 10 or higher indicates moderate anxiety), AUDIT-C (3-item alcohol screen, score 4 or higher in men or 3 or higher in women flags risky drinking), and DAST-10 (10-item Drug Abuse Screening Test). Always ask directly, 'In the past month, have you thought about hurting yourself or that you would be better off dead?'—suicidal ideation is the complication that kills, and asking does not increase risk (it increases detection and saves lives per multiple meta-analyses).'} }

5. {'label': 'Track function over time, not just symptom intensity', 'detail': "Ask concrete questions at every visit: 'Are you working or going to school? Can you leave the house alone? Are you seeing friends or family in person? Can you drive, take the bus, or go to the grocery store?' A patient who reports 'I only had two panic attacks this month instead of five' sounds improved, but if they have not left home in three weeks, they are getting worse—function is the outcome that matters for quality of life and long-term prognosis."}
6. {'label': 'Low-symptom days do not equal recovery if avoidance is widening', 'detail': "Patients may report feeling 'fine' because they have successfully avoided all triggers—this is compensation masking progression, not true improvement. Recovery means the patient can face previously feared situations (with or without mild anxiety) and function in work, relationships, and daily life. If the list of avoided situations grows longer ('I used to avoid planes, now I also avoid trains and long car rides'), the disorder is worsening even if panic attack frequency is stable."}
7. {'label': 'Benzodiazepine use is a red flag for inadequate treatment', 'detail': "Benzodiazepines (lorazepam, alprazolam, clonazepam, diazepam) provide rapid relief of acute anxiety (onset 30 to 60 minutes) by enhancing GABA (the brain's main calming messenger) activity, but they do not treat the underlying disorder, they cause tolerance within weeks (requiring dose escalation), they impair cognitive function and increase fall and motor vehicle accident risk, and they cause severe rebound anxiety and withdrawal (including life-threatening seizures) if stopped abruptly after prolonged use. Current guidelines (APA 2009, BAP 2014, NICE 2019) reserve benzodiazepines for short-term bridging (2 to 4 weeks maximum) while starting an SSRI or initiating CBT, or for acute crisis intervention—chronic benzodiazepine use is a marker that the patient is not receiving evidence-based first-line treatment (SSRI, SNRI, or CBT) and is at high risk for dependence and worsening long-term outcomes."}

Domino effect

1. Amygdala overactivity or prefrontal-cortex underactivity keeps the threat-detection system locked in the 'on' position even when no real danger is present. — The amygdala (the brain's alarm center deep in the middle of your brain) fires as if a predator is in the room—it floods the body with adrenaline (which makes the heart race, muscles tense, and breathing quicken) and cortisol (which raises blood sugar and blood pressure to fuel a fight-or-flight response). The prefrontal cortex (the front part of the brain behind your forehead that pumps the brakes on big feelings), which normally evaluates the situation and sends the 'all clear, calm down' signal, is not active enough to override the amygdala, so the alarm keeps ringing and the body stays in high alert. This mismatch—feeling terror and physical danger signals when sitting safely at home or work—is the core of every anxiety disorder.

2. Persistent hyperarousal (a state where the sympathetic nervous system stays switched on, keeping heart rate, blood pressure, muscle tension, and alertness high) makes every minute exhausting and unbearable. — Because the alarm never shuts off, the patient experiences chest tightness (the muscles between the ribs and the diaphragm stay contracted), shortness of breath or feeling like they cannot get enough air (from rapid shallow breathing or hyperventilation, which drops carbon dioxide in the blood and causes dizziness and tingling), heart pounding or racing (often 100 to 120 beats per minute at rest when it should be 60 to 80), sweating, trembling, nausea, and crushing fatigue (because running the stress response 24/7 drains energy reserves). These are not separate diseases—they are the cardiovascular, respiratory, and musculoskeletal systems responding to sustained adrenaline and cortisol as if the body were sprinting from danger all day long.
3. The patient starts avoiding triggers—places, people, activities, or situations that set off the alarm—because avoidance provides immediate relief from the unbearable physical and emotional distress. — Avoidance works instantly: if you fear crowds and you stay home, your heart rate drops, the chest tightness eases, and the terror fades within minutes. The brain learns 'staying home equals safety' and 'going into crowds equals danger,' which reinforces the false belief that crowds are truly dangerous (even though nothing bad actually happened there). This is negative reinforcement—the behavior (avoidance) is strengthened because it removes an unpleasant feeling (anxiety)—and it is one of the most powerful learning mechanisms in the brain, which is why avoidance becomes automatic and harder to break the longer it continues.
4. Avoidance spreads and the patient's world shrinks—they stop going to work, seeing friends, driving, shopping, or leaving home—until their functional capacity collapses. — The more situations a patient avoids, the more situations become triggers, because the brain never gets the chance to learn that those situations are actually safe (extinction learning cannot occur without exposure). The safe zone narrows: first they avoid highways, then all driving, then buses, then walking outside alone, until the only safe place is home (or one room in the home, or only when one specific person is present). This functional impairment—inability to work, maintain relationships, or perform daily activities like grocery shopping or attending medical appointments—is the hallmark that transforms symptoms into a disorder per DSM-5-TR criteria, which require that symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

5. Social isolation, loss of income, and loss of identity and purpose create a cascade of secondary consequences: depression, substance use, and suicidal ideation emerge as the patient loses hope and sees no way out. — Chronic anxiety and avoidance deplete serotonin (a brain messenger that steadies mood and sleep) and dopamine (a brain messenger that fuels motivation and the sense of reward), which leads to comorbid major depressive disorder in 60 to 70% of patients with anxiety disorders (per epidemiologic studies including the National Comorbidity Survey Replication). Depression adds hopelessness, anhedonia (loss of pleasure in activities that used to bring joy), low energy, and suicidal thoughts. Many patients self-medicate with alcohol (a central nervous system depressant that temporarily reduces anxiety but causes rebound anxiety, disrupts sleep, and leads to dependence) or benzodiazepines (which provide short-term relief but cause tolerance, cognitive impairment, and severe withdrawal). The combination of untreated anxiety, depression, social isolation, financial strain, and substance use raises suicide risk substantially above the general population—lifetime suicide attempt rates in people with anxiety disorders range from 10% to 20% depending on subtype and comorbidities, compared to 3 to 5% in the general population (per WHO and CDC data).
6. Physical health deteriorates from chronic stress and avoidance of medical care, compounding disability and increasing mortality risk. — Sustained high cortisol (the body's main stress hormone) and adrenaline levels damage the cardiovascular system over years: blood pressure stays elevated (contributing to high blood pressure (hypertension)), blood vessels become stiff and prone to atherosclerosis (plaque buildup narrowing arteries), and the heart works harder, increasing risk of heart attack (myocardial infarction) (heart attack, when a clot blocks blood flow to the heart muscle) and stroke (when a clot blocks blood flow to the brain). Chronic stress also impairs immune function (via cortisol's suppression of the body's infection-fighting responses), worsens metabolic health (insulin resistance, weight gain, type 2 diabetes), and disrupts sleep (leading to insomnia, which worsens anxiety and depression in a vicious cycle). Patients with severe agoraphobia or social anxiety avoid medical appointments, skip cancer screenings, delay treatment for acute illness, and have worse outcomes for chronic diseases like diabetes and heart disease because they cannot access care—avoidance of healthcare settings is a direct contributor to excess mortality in this population.

7. Without evidence-based treatment—cognitive-behavioral therapy (CBT, which uses exposure to extinguish conditioned fear and cognitive restructuring to challenge catastrophic thoughts) or pharmacotherapy (SSRIs or SNRIs, which restore serotonin signaling and reduce amygdala hyperactivity over 4 to 6 weeks)—anxiety disorders typically follow a chronic relapsing course, with most patients remaining impaired for years or decades. — Anxiety disorders do not resolve spontaneously in most cases. Naturalistic follow-up studies show that without treatment, only 20 to 30% of patients experience full remission over 10 years, and relapse rates are high even in those who improve. Avoidance and maladaptive cognitions become deeply ingrained through repetition (every time you avoid and feel relief, the neural pathway linking 'trigger leads to danger' and 'avoidance leads to safety' strengthens), making spontaneous recovery unlikely. However, both CBT and SSRIs or SNRIs are highly effective: meta-analyses show 50 to 60% response rates (defined as 50% or greater symptom reduction) with either modality, and combination therapy (CBT plus medication) achieves even higher rates in moderate to severe cases. Early intervention prevents the progression to severe functional impairment, comorbid depression, and substance use—this is why identifying the subtype and starting first-line treatment promptly is critical to changing the long-term trajectory and preventing years of disability.

Symptoms · what the patient feels, and why

- Excessive worry that is hard to control, happening more days than not for at least 6 months (generalized anxiety disorder): The amygdala (the brain's alarm center that flags danger) stays overactive, and the prefrontal cortex (the front part of the brain that pumps the brakes on fear) cannot calm it down. This keeps the brain stuck in threat-scanning mode, looping through 'what if' thoughts about health, money, work, or family. Worry becomes the default setting because the alarm never turns off.
- Sudden panic attacks that peak in under 10 minutes, with chest pain, racing heart, trouble breathing, dizziness, and fear of dying: The amygdala fires a false alarm and dumps adrenaline (a hormone that speeds up the body), triggering the sympathetic nervous system (the part that revs you up for danger). Your heart races, blood vessels squeeze tight (raising blood pressure), fast breathing blows off too much carbon dioxide (causing tingling and dizziness), and chest muscles clench. It feels exactly like a heart attack even though the heart is fine.
- Intense fear in social situations where others might judge you—like speaking in public, eating in front of people, or being watched (social anxiety disorder): The amygdala treats social attention as a threat, reading neutral faces as critical. This launches the same adrenaline flood as real danger, causing blushing, shaking, sweating, and a pounding heart whenever you feel observed. The brain mistakes social evaluation for actual danger.

- Avoiding places where you feel trapped or unable to escape—buses, crowds, malls, open spaces, or leaving home alone (agoraphobia): Past panic attacks in those places teach the brain to link the location with danger. Avoiding the place prevents panic (a reward called negative reinforcement), so the brain never learns the place is actually safe. Over time, fear spreads to more and more settings.
- Extreme fear of one specific thing—heights, needles, flying, animals, or blood (specific phobia): The amygdala stores a fear memory linking the trigger to danger (either learned from a bad experience or built in by evolution). Seeing or even thinking about the phobic object fires the full fight-or-flight response—adrenaline, racing heart, urge to run—even though the rational brain knows the fear is out of proportion.
- Restlessness, constant fatigue, trouble focusing, irritability, tight muscles, or broken sleep: Weeks of high adrenaline and cortisol (the body's main stress hormone) wear the body out. Muscles stay clenched (causing aches and tremor), the mind cannot focus because it is always scanning for threats, and sleep stays shallow because the nervous system cannot switch into the calm rest-and-digest mode needed for deep rest.
- Physical symptoms that mimic serious illness—chest pain, shortness of breath, dizziness, nausea, or diarrhea: Adrenaline and rapid breathing copy the symptoms of a heart attack or stroke. Many patients go to the emergency room fearing the worst. Full workup finds nothing structurally wrong because the symptoms are real but caused by the autonomic nervous system (the part that controls automatic body functions) stuck in overdrive, not by damaged organs.
- Thoughts of suicide or hurting yourself: Most people with anxiety disorders (60 to 70 percent) also have major depression. Social isolation from avoidance plus constant hopelessness drain serotonin and dopamine (brain messengers that steady mood and motivation). Substance use (common alongside anxiety) further lowers impulse control, sharply raising suicide risk.

Focused exam

- Patient trembling or sweating even when the room is cool: Anxiety disorder overactivates the sympathetic nervous system (the part of the body that controls the fight-or-flight response). The amygdala (the brain's alarm center that flags danger) falsely perceives threat and triggers the release of adrenaline (a stress hormone that speeds up the heart and sharpens alertness). This causes tremor and sweating even when no real danger exists.
- Heart racing (tachycardia, or heart rate above 100 beats per minute) or chest tightness without cardiac disease: Adrenaline makes the heart beat faster and harder to prepare the body to flee or fight. In anxiety disorder, this happens without real danger. Patients often fear they are having a heart attack, which worsens their anxiety and creates a self-feeding cycle.

- Patient avoiding places or situations—will not leave home, will not attend social events, will not ride elevators: Avoidance is a learned behavior driven by a faulty threat-detection system in the amygdala and prefrontal cortex (the front of the brain that pumps the brakes on big feelings). The patient avoids situations that trigger anxiety even though those situations pose no actual danger. Persistent avoidance is a core feature of anxiety disorder and signals the condition is interfering with daily life.
- Tight muscles, clenched jaw, or neck and shoulder pain: Chronic sympathetic activation causes muscles to stay contracted (held tight) as the body braces for a threat that never arrives. In anxiety disorder, this tension lasts for days to weeks and causes pain and fatigue.
- Patient reports trouble sleeping or racing thoughts at night: Hyperarousal (being on high alert all the time) persists even during rest because the brain's threat-detection system remains overactive. The patient anticipates danger and cannot achieve the relaxed state needed for sleep. This reflects chronic, uncontrolled worry typical of generalized anxiety disorder (GAD).
- Patient breathing fast and shallow or reports difficulty catching their breath: Fast breathing (Tachypnea) (rapid breathing) is part of the fight-or-flight response. Shallow breathing lowers carbon dioxide in the blood (hypocapnia), which causes lightheadedness and a sense of suffocation. This further activates the threat system, creating a loop that worsens anxiety.

Diagnostics · every test answers a question

- Thyroid-stimulating hormone (TSH) to rule out hyperthyroidism (overactive thyroid) → Does the patient have hyperthyroidism, which can mimic anxiety by increasing metabolic rate, heart rate, and sympathetic tone?
- Fingerstick blood glucose to rule out low blood sugar (hypoglycemia) (low blood sugar) → Is the patient hypoglycemic, which triggers adrenaline release and mimics anxiety symptoms?
- 12-lead electrocardiogram (ECG) and troponin if chest pain is present or patient is older or has cardiac risk factors → Does the patient have acute coronary syndrome (heart attack or unstable angina) or arrhythmia (abnormal heart rhythm) causing chest pain and a fast heart rate (tachycardia)?
- Urine drug screen if substance use is suspected → Is the patient using stimulants (cocaine, amphetamines) or withdrawing from alcohol or benzodiazepines, which can cause symptoms identical to anxiety?
- Complete blood count (CBC) to rule out anemia → Does the patient have anemia (low red blood cells), which can cause a fast heart rate (tachycardia), shortness of breath, and fatigue that mimic anxiety?

- Serum cortisol (morning level) if Cushing syndrome is suspected → Does the patient have excess cortisol production, which can cause anxiety, insomnia, and physical changes like weight gain and facial rounding?
- Electroencephalogram (EEG) if seizure activity is suspected → Could the patient be having temporal lobe seizures, which can cause sudden fear, strange sensations, and autonomic symptoms that mimic panic attacks?
- Caffeine intake history and review of medications and supplements → Is the patient consuming high amounts of caffeine or taking stimulants, decongestants, or supplements (like ephedra or guarana) that mimic anxiety?

Treatment logic

- Cognitive-behavioral therapy (CBT) or exposure therapy → CBT retrains the brain's threat-detection system by teaching patients to identify and challenge distorted thoughts (cognitive restructuring) and face feared situations in controlled, gradual steps (exposure). Repeated safe exposure without the feared outcome rewires neural circuits in the amygdala and prefrontal cortex, reducing the false alarm response.
- Selective serotonin reuptake inhibitor (SSRI) such as sertraline or escitalopram → SSRIs increase serotonin (a brain messenger that steadies mood and sleep) availability in brain synapses (connections between nerve cells), which dampens overactivity in the amygdala (brain's fear center) and prefrontal cortex (area that regulates emotion). This reduces the frequency and intensity of anxiety and panic symptoms. Full effect takes 4–6 weeks.
- Serotonin-norepinephrine reuptake inhibitor (SNRI) such as venlafaxine or duloxetine → SNRIs increase both serotonin (a brain messenger that steadies mood and sleep) and norepinephrine (a brain messenger that regulates alertness and stress) in brain synapses, which helps regulate the fear response and improves stress tolerance. They are especially helpful in generalized anxiety disorder (GAD) when SSRIs are not effective or not tolerated. Full effect takes 4–6 weeks.
- Benzodiazepine (such as lorazepam or alprazolam) only for acute panic or severe distress, short-term use only → Benzodiazepines enhance GABA (gamma-aminobutyric acid, the brain's main calming signal), which immediately suppresses the overactive threat-detection circuits and stops the panic response within 15–30 minutes.
- Beta-blocker (such as propranolol) for physical symptoms of panic or performance anxiety → Beta-blockers block beta-adrenergic receptors in the heart and blood vessels, preventing adrenaline from increasing heart rate and causing tremor. This reduces the physical symptoms of anxiety, making panic feel less intense and easier to control.

- Screen for and treat comorbid conditions: major depressive disorder, post-traumatic stress disorder (PTSD), obsessive-compulsive disorder (OCD), and substance use disorder (alcohol, cannabis, stimulants) → Treating depression, trauma, or substance use removes factors that maintain or worsen anxiety. For example, alcohol withdrawal and depression both increase sympathetic tone and dysregulate the hypothalamic-pituitary-adrenal (HPA) axis (the body's stress hormone system), perpetuating anxiety symptoms.
- Sleep hygiene, regular aerobic exercise, and reduce or eliminate caffeine intake → Adequate sleep and exercise lower baseline sympathetic nervous system activity and improve regulation of the hypothalamic-pituitary-adrenal (HPA) axis (the body's stress hormone system). Eliminating caffeine removes a stimulant that mimics and worsens anxiety symptoms by increasing heart rate and arousal.

Medications

Sertraline (Zoloft), Escitalopram (Lexapro), Fluoxetine (Prozac) · Selective Serotonin Reuptake Inhibitors (SSRIs)

SSRIs block a pump that sucks serotonin (a brain chemical that steadies mood and turns down worry) back into the nerve cell after it sends its message. By jamming that pump, more serotonin stays in the tiny gap between brain cells, letting the calm signal work longer. Over four to six weeks, this extra serotonin helps the brain rewire its alarm circuits—like giving a tangled fire alarm time to reset so it stops going off at every little thing.

1. A nerve cell releases serotonin into the tiny gap to carry a calm message to the next cell.
2. Normally, a pump on the sending cell quickly vacuums the serotonin back up, ending the signal.
3. The SSRI jams that pump so serotonin stays in the gap longer.
4. The receiving nerve cell gets a longer, stronger calm signal.
5. Over four to six weeks, the brain grows new connections that make calming signals easier and alarm signals harder to trigger.

SAFETY WATCH

Nausea, loose stools, and feeling wired or jittery in the first one to two weeks—these usually fade as your body adjusts. · Sexual side effects: less interest in sex or trouble reaching climax—happens in about 30 to 40 out of 100 people. · Activation (feeling more anxious or restless) in the first week—your doctor will start you at a low dose and raise it slowly. · Serotonin syndrome (too much serotonin) if you mix this with other drugs that boost serotonin—causes confusion, heavy sweating, fast heartbeat, and shivering; rare but serious. Tell your doctor about all your medicines. · Do NOT stop suddenly—taper slowly over weeks to avoid withdrawal (brain zaps like tiny electric jolts, dizziness, flu-like aches, crankiness). · Teens and young adults under 25 need close check-ins every one to two weeks for the first two months because SSRIs can rarely increase thoughts of self-harm before mood improves.

Venlafaxine (Effexor XR), Duloxetine (Cymbalta) · Serotonin-Norepinephrine Reuptake Inhibitors (SNRIs)

SNRIs block two pumps: one that recycles serotonin (a chemical that steadies mood and worry) and one that recycles norepinephrine (a chemical that boosts focus and energy). By jamming both pumps, more of each chemical stays active in the gaps between brain cells. This double boost helps people whose anxiety comes with low energy, poor focus, or tight, tense muscles—it calms worry while lifting drive.

1. A nerve cell releases serotonin and norepinephrine to send calm and alert messages to the next cell.
2. Normally, pumps on the sending cell vacuum both chemicals back up quickly, ending the signal.
3. The SNRI blocks both pumps, so serotonin and norepinephrine linger in the gap.
4. The receiving cell gets a longer calm signal and a gentle energy signal.
5. Over four to six weeks, the brain grows new pathways that make it easier to stay calm and focused under stress.

SAFETY WATCH

Nausea and dry mouth in the first week—taking it with food helps. · Rise in blood pressure at higher doses—your doctor should check your blood pressure every few months. · Sweating more than usual, especially at night. · Sexual side effects like SSRIs—less desire or delayed climax. · Withdrawal is worse than with SSRIs if you stop suddenly—must taper very slowly over weeks (brain zaps, dizziness, crankiness, flu-like aches). · Activation or jitteriness in the first few days—your doctor will start low and go slow. · Teens and young adults need close check-ins every one to two weeks for the first two months for any increase in thoughts of self-harm.

Lorazepam (Ativan), Alprazolam (Xanax), Clonazepam (Klonopin) · Benzodiazepines

Benzodiazepines (benzos) grab onto a special spot on the GABA-A receptor—a brake pedal built into nerve cells—and make that brake slam down much harder. GABA (the brain's main calming messenger) normally opens a small gate in the cell to let chloride ions (tiny negatively charged particles) flow in, making the cell less likely to fire. Benzos make that gate open wider and stay open longer, so excited, firing nerve cells shut down fast. Panic, muscle tension, and racing thoughts stop within 20 to 30 minutes. But the brain adapts within two to four weeks, needing more drug to get the same effect (tolerance), and stopping suddenly can trigger dangerous withdrawal like seizures and severe rebound anxiety.

1. GABA (the brain's brake chemical) lands on its receptor and opens a small gate in the nerve cell.
2. Chloride ions (negatively charged particles) flow into the cell through that gate.
3. The inside of the cell becomes more negative, making it harder for the cell to send an alarm signal.
4. A benzo sits next to the GABA receptor and makes the gate open wider and stay open longer.
5. The nerve cell becomes much quieter, fast—panic and muscle tension drop within 20 to 30 minutes.

SAFETY WATCH

Drowsiness, slowed thinking, and poor coordination—do not drive or operate machinery. · Memory problems, especially for events that happen while the drug is active (you may not remember things you did—called anterograde amnesia, like a blackout). · Tolerance (needing higher doses) develops in two to four weeks of daily use. · Physical dependence: stopping cold after weeks of daily use can cause life-threatening seizures, severe rebound anxiety, tremors, and confusion—you must taper very slowly under your doctor's watch. · High abuse potential—people with past drug or alcohol problems are at extra risk. · Older adults: increased fall risk, confusion, and worse memory—use lowest dose, shortest time possible. · Combining with alcohol, opioids, or other sedatives can slow or stop breathing—never mix. · Reserve for short-term rescue only (a few weeks maximum) or occasional use—not for daily long-term treatment.

Buspirone (BuSpar) · Azapirone (Serotonin 1A Partial Agonist)

Buspirone gently nudges serotonin 1A receptors in the brain—think of it as tapping the calm button instead of slamming it. It does NOT flood the system like SSRIs or sedate like benzos. Instead, it slowly shifts the balance so the amygdala (the brain's alarm center) gets more steady everything-is-okay signals from serotonin. It takes two to four weeks to work because the brain needs time to adjust how sensitive these receptors are and grow new calming pathways. There is NO tolerance, NO withdrawal, and NO abuse risk.

1. Buspirone lands on serotonin 1A receptors and gently activates them—not full blast, just a steady nudge.
2. On the sending cell, this nudge tells it to slow down serotonin release a bit (a calming feedback loop).
3. On the receiving cell, the nudge sends a gentle calm signal.
4. Over two to four weeks, the brain fine-tunes how sensitive these receptors are.
5. The result: the alarm center stops overreacting to everyday stress, and constant worry eases.

SAFETY WATCH

Dizziness and mild headache in the first few days—usually fades as your body adjusts. · Nausea—taking it with food helps. · Feeling jittery or restless briefly after each dose—rare. · No risk of tolerance, withdrawal, or abuse—safe to stop anytime without tapering. · Safe to use with SSRIs or SNRIs if your doctor prescribes both. · Does NOT impair driving or thinking once your body adjusts. · Less effective than SSRIs or cognitive behavioral therapy (talk therapy that teaches new ways to handle worry) for panic disorder or social anxiety—best used for generalized anxiety disorder.

Propranolol (Inderal), Atenolol (Tenormin) · Beta-Adrenergic Blockers (Beta Blockers)

Beta blockers sit on adrenaline receptors (also called beta receptors) in the heart, blood vessels, and lungs and block adrenaline (the fight-or-flight chemical, also called epinephrine) from landing. This stops the physical panic symptoms—racing heart, shaking hands, heavy sweating, trembling voice—but does NOT touch the worry thoughts in your brain. Think of it as unplugging the alarm bell so you do not hear it ring, even though the alarm is still going off inside. It works within 30 to 60 minutes and is perfect for performance anxiety (stage fright, test jitters, public speaking fear) when you need steady hands and a calm heartbeat but your mind is already prepared.

1. Stress triggers the adrenal glands (two small glands sitting on top of your kidneys) to dump adrenaline into your blood.
2. Adrenaline travels to the heart and tries to land on beta receptors there.
3. The beta blocker is already sitting in the receptor, blocking adrenaline from landing.
4. Without adrenaline's signal, the heart beats slower and with less force.
5. Blood pressure stays steady, hands stop shaking, voice stays calm—all within 30 to 60 minutes.

SAFETY WATCH

Slow heart rate (bradycardia)—check your pulse; do not use if your resting heart rate is under 60 or you feel dizzy. · Fatigue and feeling sluggish—common, especially at higher doses. · Cold hands and feet (less blood flow reaches your fingers and toes). · Can trigger wheezing or trouble breathing in people with asthma or COPD (chronic obstructive pulmonary disease, a lung disease that makes it hard to breathe)—avoid or use with extreme caution. · Can mask low blood sugar symptoms in people with diabetes—shakiness and fast heartbeat are hidden, so you may not notice low sugar until it is dangerously low. · Do NOT stop suddenly if you have taken it daily for weeks—your heart rate can rebound dangerously high; taper slowly under your doctor's watch. · Safe to take as needed 30 to 60 minutes before a stressful event like a speech, performance, or test.

Hydroxyzine (Vistaril, Atarax) · Antihistamine (H1 Receptor Antagonist)

Hydroxyzine blocks histamine receptors (called H1 receptors) in the brain. Histamine is a chemical that keeps you alert and awake. By blocking it, hydroxyzine causes drowsiness and a gentle calming effect within 30 to 60 minutes. It also has mild effects on other receptors that reduce nausea and loosen tight muscles. Because it does NOT work on GABA (like benzos do), there is NO tolerance, NO dependence, and NO abuse risk. It is used for short-term anxiety

relief or sleep, especially when benzos are too risky (people with a history of addiction or older adults).

1. Histamine normally lands on H1 receptors to keep the brain awake and alert.
2. Hydroxyzine sits on those receptors and blocks histamine from landing.
3. With less histamine signal, the brain's alertness drops, and drowsiness begins.
4. Tight muscles ease, and the jittery feeling fades.
5. Calming and sleepiness appear within 30 to 60 minutes.

SAFETY WATCH

Drowsiness—do not drive or operate machinery for several hours after taking it. · Dry mouth and dry eyes. · Rare heart rhythm change (prolonged QT interval, meaning the heart's electrical reset takes longer than normal) at high doses—avoid if you have heart rhythm problems or take other drugs that prolong QT. · Safe to stop anytime—no withdrawal or rebound. · Can be used occasionally for sleep or before events that make you anxious. · Less risky than benzos in older adults, but still causes drowsiness and raises fall risk.

Red flags

- Suicidal thoughts or a plan to harm yourself—screen at every visit; risk jumps when depression and substance use are also present
- New anxiety or panic starting after age 45—rule out overactive thyroid (hyperthyroidism), irregular heartbeat (cardiac arrhythmia), adrenal tumor (pheochromocytoma), or side effects from stimulants or steroids
- Substance use disorder or needing higher and higher doses of benzodiazepines—tolerance and rebound anxiety signal dependence; safe taper or addiction treatment is required
- Total social withdrawal or inability to work—severe functional loss predicts worse outcomes and may require intensive outpatient or inpatient psychiatric care
- Hallucinations or delusions (psychotic symptoms)—anxiety alone does not cause psychosis; consider schizophrenia, bipolar disorder, or drug-induced psychosis

Nursing priorities

1. Screen for suicide risk at every visit using direct questions about thoughts of self-harm, plans, and access to lethal means like guns or pills — Anxiety disorders double the risk of suicide, especially when depression or substance use is also present; early identification allows safety planning, removing lethal means, and psychiatric referral before a crisis occurs

2. Teach the patient to recognize early warning signs of panic attacks—like racing heart, sweating, or dizziness—and use grounding techniques such as slow breathing (breathe in for 4 counts, hold for 4, breathe out for 6) and naming 5 things they can see — Panic attacks trigger the sympathetic nervous system (fight-or-flight response), raising heart rate and breathing rate; slow breathing activates the parasympathetic nervous system (rest-and-digest), which lowers heart rate and stops the panic cycle within minutes
3. Monitor for benzodiazepine misuse by counting pills, asking about dose increases, and watching for signs of tolerance (needing more medicine for the same effect) or withdrawal (shaking, sweating, seizures when doses are missed) — Benzodiazepines like lorazepam and alprazolam bind to GABA receptors in the brain and cause physical dependence within 2 to 4 weeks; sudden停止 stopping can trigger life-threatening seizures, so tapers must be slow and supervised
4. Educate the patient that SSRIs like sertraline and SNRIs like venlafaxine take 4 to 6 weeks to reduce anxiety, and early side effects like nausea or jitteriness usually improve after the first week — SSRIs and SNRIs increase serotonin in the brain synapse (space between nerve cells) slowly over weeks by blocking reuptake pumps; knowing the timeline prevents patients from stopping too early and helps them tolerate temporary side effects
5. Refer the patient to cognitive-behavioral therapy (CBT) or exposure therapy as first-line treatment, especially for social anxiety disorder and specific phobias — CBT rewires thought patterns that trigger fear, and exposure therapy gradually desensitizes the amygdala (fear center in the brain) to anxiety triggers; both therapies have equal or better long-term results than medication alone and no side effects
6. Check thyroid function (TSH and free T4), electrolytes, blood sugar, and electrocardiogram (ECG) in patients over 45 or those with new anxiety, weight loss, tremor, or irregular heartbeat — Hyperthyroidism increases metabolism and releases excess thyroid hormone, mimicking anxiety with racing heart and tremor; low potassium, low blood sugar (hypoglycemia), and arrhythmias also cause anxiety-like symptoms but need different treatments
7. Teach the patient to limit caffeine to under 200 mg per day (about 2 cups of coffee) and avoid energy drinks, decongestants with pseudoephedrine, and recreational stimulants — Caffeine and stimulants block adenosine receptors in the brain, increasing alertness but also triggering the release of adrenaline (epinephrine), which raises heart rate and worsens panic and generalized anxiety symptoms
8. Document alcohol and substance use at every visit, and offer referral to addiction treatment if the patient is using substances to manage anxiety — Alcohol temporarily raises GABA (a calming brain chemical) but causes rebound anxiety and depression when it wears off; chronic use changes brain chemistry and makes anxiety disorders harder to treat without addressing both conditions together

Sources

- DSM-5-TR (Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision), American Psychiatric Association
- APA Clinical Practice Guideline for the Treatment of Anxiety Disorders (2023)
- Cochrane systematic reviews on CBT and pharmacotherapy for anxiety disorders
- U.S. Preventive Services Task Force (USPSTF) anxiety screening recommendation (2023)

Last reviewed: 2026-07-07