

Anxiety Panic

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THE STORY IN ONE SENTENCE

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“Panic attack is a false alarm from your brain's threat center; panic disorder means repeated attacks plus constant worry and avoidance that shrink your world.”

Normal anatomy — the body's job

Normally, your brain's amygdala (alarm center deep in the brain) detects threats and signals your body to prepare for danger—faster heart rate, quicker breathing, muscle tension. Your prefrontal cortex (thinking brain in the front of your skull) evaluates whether the threat is real and calms the alarm when it is not. This fear-then-calm cycle keeps you safe without constant panic.

BODY'S JOB

Understanding this normal alarm-and-brake system is essential, because panic disorder is what happens when the brakes fail and the alarm stays on.

1. Amygdala detects a threat (real or perceived) and sends signals to the hypothalamus (control center for automatic body functions).
2. Hypothalamus activates the sympathetic nervous system (fight-or-flight branch), releasing adrenaline and noradrenaline (stress hormones).
3. Heart rate and breathing speed up, pupils dilate (widen), blood flows to large muscles—your body is ready to run or fight.
4. Prefrontal cortex evaluates the situation: Is the threat real? If no, it sends inhibitory signals (calming messages) back to the amygdala.
5. Parasympathetic nervous system (rest-and-digest branch) reactivates, slowing heart rate and breathing, and the alarm turns off.

6. You return to baseline calm, ready to respond again if a new real threat appears.

What breaks

The amygdala fires an intense threat signal even when no danger exists, and the prefrontal cortex fails to shut down that alarm fast enough. Your body interprets normal sensations—like a skipped heartbeat or slight dizziness—as catastrophic danger, amplifying the alarm into a full panic attack.

CLINICAL TERM

Dysregulation (abnormal control) of the amygdala-prefrontal cortex circuit leads to excessive activation of the sympathetic nervous system (the fight-or-flight branch). Catecholamine release (stress hormones like adrenaline and noradrenaline flooding the bloodstream) causes a fast heart rate (tachycardia) (heart rate too fast), fast breathing (tachypnea) (breathing rate too fast), vasoconstriction (blood vessels narrowing), and heightened arousal. The patient misinterprets these physiologic sensations as evidence of imminent death or loss of control, creating a positive feedback loop (each symptom worsens fear, which worsens symptoms). Repeated attacks lead to anticipatory anxiety (constant worry about the next attack) and agoraphobia (avoidance of places or situations where escape feels difficult), which maintain the disorder.

How the body compensates

During a panic attack, vital signs may be only mildly elevated or even normal between episodes, masking the severity of distress. The danger is missing the progression to agoraphobia and functional impairment, or overlooking a true cardiac or pulmonary emergency on the first attack.

1. During an attack, heart rate and blood pressure rise, but often return to baseline within 20–30 minutes.
2. Between attacks, vital signs and physical exam are typically normal, which can lead clinicians to underestimate the patient's suffering and the risk of chronic avoidance behavior.
3. The key tipping point is development of agoraphobia: when fear of future attacks becomes so intense that the patient cannot leave home, work, or attend school.
4. On the first attack, clinicians must rule out heart attack (myocardial infarction) (heart attack), pulmonary embolism (a clot lodging in a lung artery) (blood clot in the lung), arrhythmia (abnormal heart rhythm), hyperthyroidism (overactive thyroid), and low blood sugar (hypoglycemia) (low blood sugar), because panic symptoms overlap with these emergencies.

5. Watch for worsening frequency of attacks, expanding avoidance, and new depressive symptoms, which signal the disorder is progressing and the window for effective early intervention is closing.
6. A patient who looks calm in the office but reports three attacks this week and has stopped driving is not stable—trajectory matters more than a single snapshot.

Domino effect

1. Amygdala activation without real threat — A trigger—often a normal body sensation like a skipped heartbeat, or a stressful thought—activates the amygdala (alarm center). Because the prefrontal cortex (thinking brain) does not inhibit it quickly, the amygdala signals danger and activates the sympathetic nervous system (fight-or-flight branch).
2. Catecholamine surge and autonomic arousal — The sympathetic nervous system releases adrenaline and noradrenaline (stress hormones) into the bloodstream, causing a fast heart rate (tachycardia) (heart rate too fast), fast breathing (tachypnea) (breathing rate too fast), sweating, tremor, and vasoconstriction (blood vessels narrowing).
3. Hyperventilation and respiratory alkalosis (blowing off too much carbon dioxide) — Rapid shallow breathing (hyperventilation) expels too much carbon dioxide, raising blood pH (respiratory too little acid in the blood (alkalosis), blood too basic). This shifts calcium binding and lowers ionized calcium, causing tingling in fingers, lips, and face and lightheadedness.
4. Chest pain and shortness of breath — Intercostal muscle tension (muscles between the ribs tightening), hyperventilation, and heightened awareness create chest tightness and a sensation of suffocation, mimicking cardiac starved blood flow (ischemia) (heart not getting enough blood).
5. Catastrophic misinterpretation — The patient interprets these sensations—racing heart, chest pain, dizziness—as proof of a heart attack or impending death, which amplifies fear and further activates the amygdala, creating a positive feedback loop (each symptom worsens fear, which worsens symptoms).
6. Peak intensity in 10 minutes, then gradual resolution — Catecholamine levels peak quickly but are metabolized (broken down) within 20–30 minutes. As hormones clear and the parasympathetic nervous system (rest-and-digest branch) reactivates, heart rate and breathing slow, and symptoms resolve, leaving the patient exhausted but physically unharmed.
7. Anticipatory anxiety and avoidance — After one or more attacks, the patient develops constant worry about the next attack and begins avoiding places or situations where escape feels hard (crowds, driving, public transit). This avoidance reinforces the fear and prevents the patient from learning that the sensations are not dangerous, perpetuating the disorder.

Symptoms · what the patient feels, and why

- **Palpitations or pounding heart:** Your body dumps epinephrine (the main stress hormone, also called adrenaline) into your blood. Epinephrine tells your heart to beat faster and squeeze harder. You feel your heart race or pound in your chest, even though nothing is actually wrong with your heart.
- **Sweating:** Your sympathetic nervous system (the body's fight-or-flight alarm) switches on. It tells your sweat glands to make watery sweat, as if you are about to run or fight. But you are not actually moving, so you just feel sweaty and clammy.
- **Trembling or shaking:** Epinephrine tells your muscles to tense up and get ready to move. This tension makes your hands, legs, or whole body shake. Your muscles are prepared for danger, even though there is no real danger.
- **Shortness of breath or feeling smothered:** You breathe too fast and too shallow (called hyperventilation). The muscles between your ribs also tighten. Together these make you feel like you cannot get enough air, even though your blood oxygen is normal or even high.
- **Chest pain or discomfort:** The muscles between your ribs (intercostal muscles) tighten. Fast breathing and paying very close attention to your body make your chest feel tight or sharp. It can feel like a heart attack, but your heart is not damaged.
- **Nausea or stomach pain:** Your body sends blood away from your stomach and intestines to your muscles. Your digestion slows down or stops. This causes nausea, cramping, or the urge to throw up or have a bowel movement.
- **Dizziness, lightheadedness, or feeling faint:** Breathing too fast blows off carbon dioxide from your blood. This makes your blood too basic (called respiratory alkalosis (blowing off too much carbon dioxide)). Your brain gets slightly less blood flow, and blood vessels also narrow. Together this makes you dizzy or feel like you will pass out, even though you almost never actually faint.
- **Tingling or numbness in fingers, face, or around the mouth:** When you hyperventilate, your blood becomes too basic. This changes how calcium sticks to proteins in your blood. The free calcium (the active kind) drops. Low free calcium makes your nerves fire more easily, causing tingling or numbness.
- **Feeling unreal or detached from yourself:** Extreme fear and fast breathing put your brain into a protective mode. The world feels unreal or dreamlike (called derealization), or you feel disconnected from your own body (called depersonalization). The prefrontal cortex (the front part of your brain that handles clear thinking) gets less active, and the amygdala (your brain's alarm center) takes over. Your brain does this to shield you from feeling overwhelmed.

- Fear of losing control, going crazy, or dying: The chest pain, shortness of breath, and dizziness feel so intense that you think you are having a heart attack, stroke, or mental breakdown. Your amygdala (the brain's fear center) is on high alert and sends alarm signals to the prefrontal cortex. Your mind catastrophizes (jumps to the worst conclusion), even though none of those things is actually happening.

Focused exam

- Heart rate above 100 beats per minute, regular rhythm, no murmur: Panic attacks trigger a fast heart rate (tachycardia) (a too-fast heart rate) through the body's automatic stress response, not from heart damage. The sympathetic nervous system (the body's gas pedal for fight-or-flight) dumps adrenaline into the bloodstream, which speeds the heart. The rhythm stays regular because the heart's electrical wiring still works normally. This pattern points to panic, not a broken heart valve or artery blockage.
- Blood pressure elevated during the attack, returns to baseline between attacks: Blood pressure spikes during panic because adrenaline squeezes blood vessels tight and speeds the heart. When the attack ends, the parasympathetic nervous system (the body's brake pedal that calms everything down) takes over, and pressure drops back to normal. This on-off pattern indicates panic disorder, not chronic high blood pressure.
- Breathing fast and shallow, patient reports feeling like they cannot get enough air: The stress response drives hyperventilation (breathing too fast), which blows off too much carbon dioxide from the blood. Low carbon dioxide pinches brain blood vessels, causing dizziness and tingling in the hands and face. The patient feels like they are suffocating, but oxygen saturation (the percent of oxygen the blood is carrying) stays normal—usually 95% or higher—because the lungs are moving air just fine.
- Chest tightness or pressure, no tenderness when you press on the chest wall: Chest discomfort in panic comes from tight intercostal muscles (the muscles between the ribs) and heightened awareness of the heartbeat, not from the heart muscle starving for oxygen. No chest-wall tenderness when you press and a normal heart tracing (ECG) make panic disorder the likely answer.
- Patient describes feeling detached from self or that surroundings seem unreal, but knows who they are, where they are, and what day it is: Depersonalization (feeling detached from yourself) and derealization (feeling the world is not real) happen when the prefrontal cortex (the front of the brain that reasons and makes sense of the world) gets flooded by signals from the amygdala (the brain's alarm center). The patient still knows who and where they are, which rules out delirium (acute confusion from infection, low oxygen, or drugs), psychosis (lost contact with reality), or stroke (sudden brain injury from blocked blood flow).

- Shaking and sweating during the attack, skin warm and dry between episodes: Tremor and sweating result from the sympathetic nervous system dumping adrenaline. Between attacks, the autonomic controls return to normal. This on-off pattern is classic for panic disorder, not an overactive thyroid, infection with fever, or low blood sugar.

Diagnostics · every test answers a question

- Electrocardiogram (ECG) → Is the heart's electrical activity normal, or are there signs of heart attack, dangerous rhythm, or heart strain?
- Cardiac troponin I or T → Is heart muscle breaking down right now, indicating a heart attack?
- Thyroid-stimulating hormone (TSH) → Is the thyroid gland making too much hormone, which can mimic panic attacks?
- Basic metabolic panel (sodium, potassium, glucose, creatinine) → Are electrolytes and blood sugar at safe levels, or is an imbalance causing the symptoms?
- Urine drug screen → Are stimulants or withdrawal from sedatives causing the symptoms?
- D-dimer (if chest pain, shortness of breath, and risk factors for clot present) → Is there a blood clot in the lung (pulmonary a traveling clot lodging in a vessel (embolism))?
- Chest radiograph (X-ray) → Is there a collapsed lung, fluid in the lungs, or infection causing the breathing trouble?
- Oxygen saturation by pulse oximetry → Is the blood carrying enough oxygen?

Treatment logic

- Controlled breathing technique: slow to 6 breaths per minute (inhale through nose for 4 seconds, exhale through mouth for 6 seconds) during acute attack → Reduces hyperventilation, which raises carbon dioxide back toward normal and reverses respiratory alkalosis (blowing off too much carbon dioxide) (blood pH too high from low carbon dioxide). This stops dizziness and tingling. Activates the parasympathetic nervous system (the body's calming brake), which lowers heart rate and tells the amygdala (the brain's alarm center) that no real danger exists.
- Selective serotonin reuptake inhibitor (SSRI) such as sertraline 25 to 50 mg daily, paroxetine 10 to 20 mg daily, or escitalopram 5 to 10 mg daily, started low and increased gradually over 2 to 4 weeks → Increases serotonin (a brain messenger chemical that regulates mood and anxiety) in the prefrontal cortex and quiets the amygdala over 4 to 6 weeks. This corrects the brain-chemistry imbalance that allows spontaneous panic attacks to happen. The American Psychiatric Association lists SSRIs as first-line medication for panic disorder (APA Practice Guideline for Panic Disorder, 2009).

- Cognitive-behavioral therapy (CBT) with interoceptive exposure: patient deliberately triggers panic sensations (rapid breathing, spinning to create dizziness) in a safe setting, then practices staying calm and reinterpreting the sensations as harmless → Teaches the patient to reinterpret body sensations (racing heart, fast breathing) as not dangerous. Repeated exposure without running away extinguishes (erases through learning) the fear response. The amygdala learns that these body sensations do not predict death or disaster. The American Psychiatric Association lists CBT with exposure as first-line talk therapy for panic disorder (APA Practice Guideline for Panic Disorder, 2009).
- Benzodiazepine such as lorazepam 0.5 to 1 mg or alprazolam 0.25 to 0.5 mg as needed for severe attacks, or short-term bridge (1 to 4 weeks) while waiting for SSRI to start working → Enhances GABA (gamma-aminobutyric acid, the brain's main calming chemical) activity, which rapidly reduces central nervous system arousal and stops the panic attack within 20 to 30 minutes. Provides immediate relief when distress is unbearable.
- Psychoeducation: explain to patient that panic attacks are not dangerous, cannot cause heart attack or death, and will always pass within 20 to 30 minutes → Reduces catastrophic misinterpretation of body sensations (thinking 'I am dying' when heart races). When the patient understands the physiology (how the sympathetic nervous system works and why the body triggers fight-or-flight), fear of the next attack decreases. This breaks the fear-of-fear cycle that perpetuates panic disorder.
- Prevention of avoidance and agoraphobia: encourage continued engagement in daily activities, use of public spaces, and travel even when anxious → Prevents conditioned avoidance, which maintains and worsens panic disorder over time. Early return to feared situations provides real-life practice and prevents the spiral into agoraphobia (fear of places where escape might be hard or embarrassing), which develops in about one-third of untreated panic disorder patients.

Medications

Selective Serotonin Reuptake Inhibitors (SSRIs) · Antidepressant—serotonin reuptake inhibitor

Panic attacks happen when the brain's alarm center (the amygdala) fires even though there is no real danger. It floods the body with adrenaline (the fight-or-flight chemical), making the heart race, hands shake, and breath come fast. Over time, the person starts avoiding places where escape feels hard (like crowded stores or bridges)—this is called agoraphobia. SSRIs work by keeping more serotonin (a calming brain messenger) in the tiny gaps between nerve cells in the fear center and the thinking part of the brain (prefrontal cortex). More serotonin quiets the overactive alarm system and helps the brain stop firing panic signals. It takes 4 to 6

weeks for the brain circuits to reset and for panic attacks to become less frequent and less intense.

1. Serotonin is released from one nerve cell into the gap (synapse) to send a calming signal to the next nerve cell.
2. Normally, serotonin is quickly sucked back into the first nerve cell by a pump (the serotonin reuptake transporter).
3. The SSRI blocks that pump, so serotonin stays in the gap longer and keeps sending the calming signal.
4. Over 4 to 6 weeks, the extra serotonin remodels the fear circuits: the amygdala becomes less jumpy, and the prefrontal cortex gets better at shutting down false alarms.
5. Panic attacks happen less often, feel less scary, and the person regains confidence to handle everyday situations.

SAFETY WATCH

Start low and go slow: Begin with a low dose (sertraline 25 mg, escitalopram 5–10 mg, paroxetine 10 mg) to reduce early jitteriness and anxiety spikes, then increase every 1 to 2 weeks to the target dose (sertraline 50–200 mg, escitalopram 10–20 mg, paroxetine 20–60 mg). · First 2 weeks: Watch for activation syndrome—increased anxiety, trouble sleeping, restlessness, or agitation. Reassure the patient this is temporary and means the drug is starting to work; it usually fades by week 2 to 3. · Screen for bipolar disorder before starting: SSRIs can trigger mania (severe high mood, racing thoughts, risky behavior) in people with undiagnosed bipolar disorder. Ask about prior manic or hypomanic episodes. · Serotonin syndrome: Rare but dangerous if SSRIs are combined with other serotonin-boosting drugs (tramadol, triptans, MAO inhibitors, St. John's wort). Symptoms include confusion, high fever, fast heart rate, rigid muscles, and shaking. Stop all serotonin drugs and get emergency care immediately. · Bleeding risk: SSRIs slightly increase bleeding risk (nosebleeds, bruising, stomach bleeding), especially if combined with NSAIDs (ibuprofen, naproxen), aspirin, or blood thinners. Consider a proton pump inhibitor (PPI) if the patient takes daily NSAIDs. · Sexual side effects: Common (low libido, trouble reaching orgasm, erectile dysfunction). Discuss openly; if bothersome, consider switching to bupropion or mirtazapine, which have lower sexual side effects. · Pregnancy: Paroxetine carries the highest risk of first-trimester heart defects in the baby (FDA category D). Sertraline has the most safety data in pregnancy and is preferred if medication is needed. Weigh the mother's panic severity (which can harm both mother and baby) against medication risks with shared decision-making. · Stopping the drug: Never stop suddenly. Taper slowly over 2 to 6 months to avoid withdrawal symptoms (dizziness, electric 'brain zaps,' nausea, flu-like achiness, return of anxiety). · Suicide risk: In people under 25, SSRIs slightly increase suicidal thoughts in the first few weeks. Monitor closely; see the patient weekly for the first month. The overall benefit in reducing panic and depression far outweighs this risk, but early follow-up is critical.

Cognitive-Behavioral Therapy with Interoceptive and Situational Exposure · Psychotherapy—behavioral and cognitive intervention

Panic disorder involves two problems: a hypersensitive internal alarm (fearing body sensations like a racing heart) and external avoidance (avoiding places where escape feels hard). CBT with exposure retrains the brain by repeatedly showing it that the feared sensations and situations are not dangerous. Interoceptive exposure intentionally triggers panic-like sensations (spinning to feel dizzy, breathing through a straw to feel short of breath) in a controlled setting, proving to the amygdala that these sensations do not lead to death or loss of control. Situational exposure involves gradually entering feared places (starting with easiest, building to hardest) while staying long enough for the fear to peak and then naturally fall. Over

many trials, the amygdala learns the situation is safe, the fear response weakens, and the person regains their life.

1. **Psychoeducation:** Teach the patient what panic is (a false alarm, not a heart attack), how adrenaline causes the symptoms (racing heart, shaking, dizziness), and that panic peaks in 10 minutes and always ends without harm. Knowledge reduces catastrophic thinking.
2. **Cognitive restructuring:** Identify catastrophic thoughts ('I'm having a heart attack,' 'I'll faint and everyone will stare,' 'I'm going crazy') and challenge them with evidence ('My heart has been checked and is healthy,' 'Panic makes you feel faint but almost never causes actual fainting because blood pressure goes UP, not down,' 'Panic is not psychosis').
3. **Interoceptive exposure:** Deliberately trigger panic-like sensations—spin in a chair to induce dizziness, hyperventilate for 1 minute to feel lightheaded, run in place to make the heart race. Stay with the sensation without escape until the fear peaks and falls (usually 3–10 minutes). Repeat daily. The amygdala learns these sensations are not dangerous, and fear of the sensations (the core of panic disorder) weakens.
4. **Situational exposure hierarchy:** List feared situations from easiest to hardest (e.g., standing in a store doorway → walking one aisle → staying 10 minutes → going during a busy time → going alone). Start with the easiest. Enter the situation, stay until anxiety peaks and begins to drop (do NOT leave while anxiety is rising—that teaches escape works and strengthens the fear). Repeat the same step until it feels boring, then move to the next harder step.
5. **Remove safety behaviors:** Safety behaviors (always carrying a phone, only going with a trusted person, sitting near exits, carrying 'just in case' medication) prevent full learning that the situation is safe. Gradually drop these crutches during exposure so the brain learns 'I am safe on my own, without escape plans.'
6. **Relapse prevention:** Practice exposure even after panic stops, especially during stress or life changes, to keep the fear circuits weak and prevent return of avoidance.

SAFETY WATCH

Medical clearance: Before starting interoceptive exposure (exercises that raise heart rate and breathing), ensure the patient does not have unstable heart disease, uncontrolled high blood pressure, or severe asthma, as these exercises could trigger a medical event. Check with the primary care doctor if there is any cardiac history. · Pacing: If exposure feels overwhelming and the patient repeatedly escapes or refuses, slow down. Break the step into smaller pieces or add temporary support (therapist present, trusted person nearby) and fade it gradually. Pushing too hard too fast can worsen avoidance. · Suicidal ideation: If panic disorder occurs with severe depression, assess suicide risk before starting exposure. Severely depressed patients may need medication and safety planning first. · Contraindication in active psychosis or mania: CBT exposure requires reality testing and impulse control. Defer exposure if the patient is actively manic or psychotic; stabilize with medication first.

Benzodiazepines (short-term or rescue use only) · Anxiolytic—GABA-A receptor positive allosteric modulator

Benzodiazepines work by turbocharging GABA (the brain's main 'brake pedal' chemical). GABA slows down nerve activity, especially in the amygdala (fear center) and cortex (thinking brain). Benzodiazepines make GABA work stronger and faster at its receptor (like turning up the volume on a calming signal), which very quickly quiets racing thoughts, stops the heart from pounding, and stops the feeling of impending doom. They work within 20 to 60 minutes—much faster than SSRIs—which is why they are used for acute panic attacks. However, the brain adapts within 2 to 4 weeks, needing higher doses to feel the same effect (tolerance), and stopping suddenly can cause dangerous rebound anxiety and seizures (dependence). For this reason, benzodiazepines are used only short-term (2 to 4 weeks max) while waiting for an SSRI to start working, or as rare 'rescue' medication for severe attacks.

1. GABA (the calming neurotransmitter) binds to GABA-A receptors on nerve cells, opening a channel that lets chloride ions flow into the cell.
2. Chloride makes the inside of the nerve cell more negative (hyperpolarized), which makes it much harder for the cell to fire.
3. The benzodiazepine sits on the GABA-A receptor at a different spot and changes the receptor's shape, making GABA bind more tightly and open the chloride channel wider and longer.
4. The result: nerve activity in the fear and arousal centers drops rapidly, the flood of adrenaline stops, the heart slows, breathing calms, and the sense of terror fades within 20 to 60 minutes.

SAFETY WATCH

Short-term use only: Use for 2 to 4 weeks maximum, or only as needed for rare, severe attacks (not daily). The brain develops tolerance (needing higher doses) and physical dependence (withdrawal symptoms if stopped) within 2 to 4 weeks of daily use. ·

Withdrawal seizures: Stopping benzodiazepines suddenly after regular use (especially high doses or long-term use) can cause life-threatening withdrawal seizures, severe rebound anxiety, tremor, confusion, and (rarely) delirium. Always taper slowly—reduce dose by 10 to 25% every 1 to 2 weeks, sometimes over many months for long-term users. ·

Respiratory depression: Benzodiazepines slow breathing. When combined with opioids (pain pills), alcohol, or other sedatives, they can cause dangerously slow breathing, unconsciousness, and death. Never combine with opioids unless no other option exists and the patient is closely monitored. ·

Cognitive impairment: Benzodiazepines cause drowsiness, slow reaction time, and memory problems (especially forming new memories). Do not drive, operate machinery, or make important decisions while taking them. Use with extreme caution in older adults, who are at high risk for falls, hip fractures, confusion, and worsening dementia. ·

Substance use disorder: Avoid in patients with a history of alcohol or drug addiction, as benzodiazepines have high abuse and dependence potential. ·

Pregnancy and breastfeeding: Benzodiazepines cross the placenta and are found in breast milk. First-trimester use may (data are mixed) increase risk of cleft lip/palate. Near delivery, they can cause floppy baby syndrome (low muscle tone, difficulty feeding, breathing problems). Use only if benefit clearly outweighs risk, and use the lowest dose for the shortest time. ·

Paradoxical reactions: Rarely, benzodiazepines cause the opposite effect—agitation, aggression, disinhibition (loss of impulse control)—especially in children, older adults, or people with certain psychiatric conditions. Stop the drug immediately if this occurs. ·

Examples and dosing: Lorazepam 0.5–1 mg as needed (works in 30–60 minutes, lasts 6–8 hours). Clonazepam 0.25–0.5 mg as needed or twice daily (works in 20–60 minutes, lasts 8–12 hours; longer half-life makes tapering easier but also raises dependence risk with daily use). Alprazolam 0.25–0.5 mg as needed (works in 15–30 minutes, lasts 4–6 hours; very fast onset and short duration increase abuse and rebound anxiety risk—generally avoid for panic disorder).

Serotonin-Norepinephrine Reuptake Inhibitors (SNRIs) · Antidepressant—serotonin and norepinephrine reuptake inhibitor

SNRIs work like SSRIs but block the reuptake (re-absorption) of both serotonin and norepinephrine (another brain chemical involved in alertness and stress response). By keeping both chemicals in the synapse (gap between nerve cells) longer, SNRIs calm the overactive fear circuits in the amygdala and prefrontal cortex and also reduce the physical 'revved up' feeling driven by norepinephrine in the brainstem. SNRIs are a good alternative if a patient does not

respond to or cannot tolerate an SSRI. Like SSRIs, they take 4 to 6 weeks to reset the brain's fear system.

1. Serotonin and norepinephrine are released into the synapse to send calming and regulating signals to the next nerve.
2. Normally, pumps (serotonin and norepinephrine transporters) quickly pull them back into the first nerve cell.
3. The SNRI blocks both pumps, so serotonin and norepinephrine stay in the gap longer.
4. Over 4 to 6 weeks, the sustained higher levels remodel fear and arousal circuits: the amygdala fires less, the prefrontal cortex regulates emotions better, and the brainstem stops sending excessive 'danger' signals to the body.
5. Panic attacks become less frequent and less intense, and physical symptoms (racing heart, sweating, tremor) lessen.

SAFETY WATCH

Blood pressure: SNRIs (especially venlafaxine at higher doses) can raise blood pressure because norepinephrine tightens blood vessels. Check blood pressure before starting and recheck after dose increases. If blood pressure rises significantly, lower the dose or switch to an SSRI. · Activation syndrome and suicide risk: Same as SSRIs—monitor closely in the first 2 weeks for increased anxiety, agitation, or suicidal thoughts, especially in patients under 25. Start low, go slow. · Serotonin syndrome: Same risk as SSRIs when combined with other serotonergic drugs (tramadol, triptans, MAO inhibitors). Watch for confusion, high fever, muscle rigidity, tremor. · Bleeding risk: Slightly increased, especially with NSAIDs, aspirin, or blood thinners. · Withdrawal: Venlafaxine has a very short half-life and causes severe withdrawal symptoms (intense dizziness, brain zaps, irritability) if a dose is missed or stopped suddenly. Taper very slowly over 2 to 6 months. Duloxetine has a longer half-life and is somewhat easier to taper. · Pregnancy: Limited data; generally prefer SSRIs (especially sertraline) as first choice in pregnancy. Use SNRIs only if SSRIs have failed and benefit justifies potential risk. · Examples and dosing: Venlafaxine extended-release 37.5–75 mg daily, increase to 150–225 mg daily (higher doses needed for anxiety than depression). Duloxetine 30 mg daily, increase to 60 mg daily (also FDA-approved for generalized anxiety disorder; less studied in panic disorder specifically but used off-label with good results).

Red flags

- Chest pain with abnormal ECG (heart tracing), high troponin (a blood test that shows heart muscle damage), or risk factors for a real heart attack (such as diabetes, high blood pressure, smoking, or family history)

- Sudden shortness of breath with low oxygen (hypoxia), unequal breath sounds on exam, or risk for a blood clot in the lung (pulmonary a traveling clot lodging in a vessel (embolism), especially after surgery, long flight, or leg swelling)
- First panic attack ever in someone over 45 with no history of anxiety—must first rule out heart disease, thyroid problems, or medication or drug causes
- Thoughts of suicide or a plan to hurt yourself, especially if panic disorder happens together with major depression or alcohol or drug use
- Cannot leave home, lost job, or life is severely limited by fear of having another attack (called agoraphobia)—needs urgent referral to a psychiatrist or therapist

Nursing priorities

1. Place patient on continuous pulse oximetry (oxygen level monitor) and cardiac monitor, obtain 12-lead ECG within 10 minutes of arrival, and notify provider immediately if chest pain is present. — Chest pain is the most common symptom shared by both panic attack and heart attack (myocardial tissue death from blocked blood flow (infarction)), so life-threatening cardiac causes must be ruled out first before diagnosing panic disorder.
2. Assess oxygen saturation and apply 2 to 4 liters of oxygen by nasal cannula if SpO2 (oxygen level) is below 94 percent. — Hyperventilation (rapid breathing) during panic causes the patient to feel short of breath, but true low oxygen (hypoxia) (low oxygen) signals pulmonary embolism (a clot lodging in a lung artery), pneumothorax (collapsed lung), or heart failure instead of panic.
3. Move patient to a quiet, low-stimulation room with dimmed lights and minimal staff traffic. — The amygdala (fear center in the brain) is overactive during panic attacks, and reducing sensory input helps calm the sympathetic nervous system (fight-or-flight response) that drives the racing heart and sweating.
4. Sit with the patient and coach slow breathing at 6 breaths per minute: breathe in through the nose for 4 seconds, hold for 2 seconds, breathe out through the mouth for 6 seconds. — Slow controlled breathing activates the vagus nerve (main nerve of the parasympathetic calming system), lowers carbon dioxide levels that dropped from hyperventilation, and interrupts the panic cycle within 3 to 5 minutes.
5. Obtain fingerstick blood glucose and administer 15 grams of fast-acting carbohydrate if glucose is below 70 mg/dL. — Low blood sugar (Hypoglycemia) (low blood sugar) causes trembling, sweating, racing heart, and confusion identical to panic attack symptoms but requires glucose replacement, not anxiety treatment.
6. Ask patient to name five things they can see, four they can touch, three they can hear, two they can smell, and one they can taste. — This grounding technique (5-4-3-2-1 method) shifts brain activity from the amygdala (fear center) to the prefrontal cortex (thinking center), breaking the dissociation (feeling detached or unreal) that worsens panic.

7. Avoid giving benzodiazepines like lorazepam or alprazolam during the first episode until ECG, troponin, and chest X-ray results are reviewed by the provider. — Benzodiazepines lower blood pressure and mask symptoms, which delays diagnosis of heart attack or pulmonary embolism (a clot lodging in a lung artery) and creates dependence (addiction risk) if used regularly for panic disorder.
8. Educate patient that panic symptoms peak in 10 minutes and resolve in 20 to 30 minutes, even without medication, because the body cannot maintain fight-or-flight hormone levels longer than that. — Fear of the panic attack itself (fear of fear) creates a cycle where anticipatory anxiety triggers more attacks, but understanding the time-limited physiology reduces this conditioned fear response.

Sources

- DSM-5-TR (Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision)
- APA Practice Guideline for the Treatment of Patients with Panic Disorder, 2009
- Cochrane Systematic Review: Psychological therapies for panic disorder with or without agoraphobia
- AHA/ACC guidelines for evaluation of chest pain and acute coronary syndromes

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