

Syncope (fainting)

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THE STORY IN ONE SENTENCE

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“Fainting (Syncope) (fainting) — find out if it is reflex (triggered by nerves), orthostatic (standing up too fast), or cardiac (heart problem). An electrocardiogram and the patient's story are your main tools.”

Normal anatomy — the body's job

Your brain needs a steady river of oxygen-rich blood every single second to stay awake and working. Normally, your heart pumps blood out, your blood vessels squeeze or relax to keep pressure steady, and special sensors in your neck and chest tell your brain to adjust your heart rate and blood vessel tone when you stand, sit, or move around — so blood pressure stays high enough to feed your brain no matter what position you are in.

BODY'S JOB

Your heart is the pump, your blood vessels are the pipes, and your autonomic nervous system (the automatic control center in your brainstem) is the thermostat that keeps everything balanced. When you stand up, gravity pulls about a pint of blood down into your legs and belly. Within one or two heartbeats, sensors called baroreceptors (pressure detectors in the carotid arteries in your neck and the aorta near your heart) feel the pressure drop and send an alarm to your brainstem. Your brainstem instantly tells your heart to beat faster and your leg veins to squeeze tighter, pushing blood back up to your brain so pressure stays normal and you stay conscious. This happens automatically — you never have to think about it.

1. You stand up → gravity pulls blood into your legs and belly.
2. Baroreceptors in your neck arteries sense the pressure drop.
3. They send a signal to your brainstem (the automatic control room at the base of your brain).

4. Your brainstem fires two responses: speed up the heart and tighten the blood vessels in your legs.
5. Blood is pushed back up to your brain within two seconds.
6. Blood pressure at brain level stays normal, so you stay awake and clear-headed.

What breaks

Fainting (Syncope) happens when one of three systems fails: the reflex control (your vagus nerve overreacts and slams the brakes on your heart), the automatic pressure adjustment (your body cannot squeeze blood back up when you stand), or the heart itself (a rhythm problem or blockage stops it from pumping enough blood out).

CLINICAL TERM

The three main causes are reflex (neurally mediated) fainting (syncope), orthostatic low blood pressure (hypotension), and cardiac fainting. Reflex fainting includes vasovagal (emotional or pain trigger), situational (cough, micturition, defecation), and carotid sinus hypersensitivity. Orthostatic fainting is caused by volume depletion (dehydration, bleeding), autonomic failure (diabetes, Parkinson disease, aging), or medications (diuretics, alpha-blockers, vasodilators). Cardiac fainting is caused by arrhythmias (bradycardia from heart block or sick sinus syndrome, a fast heart rate (tachycardia) from ventricular a fast heart rate or supraventricular a fast heart rate) or structural heart disease (aortic narrowing (stenosis), hypertrophic cardiomyopathy, pulmonary embolism (a clot lodging in a lung artery), acute coronary syndrome). Each cause produces brief global cerebral poor blood flow (hypoperfusion) (not enough blood reaching the whole brain), leading to transient loss of consciousness with spontaneous full recovery.

How the body compensates

When blood flow to the brain starts to drop, the body fights back by speeding up the heart, squeezing blood vessels, and triggering you to lie down (by making you faint) — all to get blood back to the brain fast. These responses can keep you looking stable for a while, but they hide how close you are to a dangerous tipping point, especially in cardiac fainting (syncope).

1. {'what': 'Baroreceptors sense the pressure drop and fire signals to the brainstem.', 'why': 'This is the body's early warning system — it detects trouble before you feel dizzy.'}
2. {'what': 'The brainstem tells the heart to beat faster and blood vessels in the arms and legs to squeeze tight.', 'why': 'Faster heart rate and tighter vessels push more blood up to the brain; this can hold pressure steady for a few seconds to minutes.'}

3. {'what': 'You feel lightheaded, sweaty, or nauseous (in reflex fainting (syncope)).', 'why': 'The vagus nerve is firing; these warning symptoms are your body's last chance to make you sit or lie down before you faint.'}
4. {'what': 'If pressure keeps falling, you lose consciousness and fall down.', 'why': 'Falling flat is the body's emergency fix — when you lie horizontal, gravity stops pulling blood into your legs, and blood flows back to your brain immediately; this is why fainting (syncope) is brief and you wake up on your own.'}
5. {'what': 'Once lying flat, blood pressure recovers within seconds and consciousness returns fully.', 'why': 'The brain gets oxygen again; there is no lasting injury because the blackout was short.'}
6. {'what': 'In cardiac fainting (syncope), compensation may fail completely with no warning.', 'why': 'If the heart suddenly stops beating (asystole), beats chaotically (ventricular fibrillation), or cannot pump against a blockage, no amount of vessel squeezing can fix it — this is the dangerous crash point that can lead to sudden death.'}
7. {'what': 'Watch for red flags: fainting (syncope) during exercise, while lying down, with chest pain, with a family history of sudden death, or with an abnormal EKG.', 'why': 'These signs mean the heart itself is broken, not just the reflex control or blood volume; the next episode could be fatal, so urgent cardiology evaluation and monitoring are needed.'}

Domino effect

1. Blood flow to the brain drops below the minimum needed to stay conscious. — The brain uses one-fifth of the body's oxygen every minute and has no reserve; even three to five seconds of zero flow causes you to black out like flipping a light switch.
2. In reflex fainting (syncope), you get five to thirty seconds of warning (sweating, nausea, pale skin, tunnel vision). — The vagus nerve is ramping up slowly, dropping heart rate and blood vessel tone step by step; you feel the pressure falling before you actually faint.
3. In orthostatic fainting (syncope), you feel dizzy within seconds of standing, then faint if you do not sit down. — Gravity instantly pulls blood into your legs; if your body cannot squeeze it back up, brain pressure drops the moment you stand — lying back down fixes it right away.
4. In cardiac fainting (syncope), you lose consciousness suddenly with no warning, often during activity or even while sitting. — The heart's electrical system misfires or a blockage stops the pump completely; there is no slow ramp-down, just an instant loss of output — this is the most dangerous type because it can kill you if the rhythm does not restart.
5. You fall down, which makes you horizontal. — Lying flat is the body's emergency reset — gravity stops working against you, blood flows back to the brain, and consciousness returns within ten to thirty seconds.

6. You wake up fully with no confusion (unless you hit your head). — True fainting (syncope) is brief (under one minute) and the brain gets oxygen back before any cells die; if you wake up confused or it takes minutes to recover, it was probably a seizure or stroke, not fainting.
7. The doctor traces backward from your story and EKG to find which system broke. — Reflex fainting (syncope) has triggers and warnings; orthostatic fainting happens only when standing and stops when lying; cardiac fainting has no pattern, no warning, and often an abnormal EKG or heart history — finding the cause tells you if it is safe or life-threatening.
8. Treatment fixes the broken step: more fluids and salt for orthostatic, avoid triggers and learn to lie down for reflex, pacemaker or defibrillator or valve surgery for cardiac. — Each cause has a different fix; treating the wrong cause does not help and can be dangerous — that is why accurate diagnosis with EKG, orthostatic vitals, and sometimes a heart monitor or echocardiogram is essential.

Symptoms · what the patient feels, and why

- Sudden loss of consciousness: Blood pressure drops too low or the heart beats irregularly, so not enough oxygen-rich blood reaches the brain. The brain needs a steady supply to stay awake; when it drops even for a few seconds, you black out.
- Feeling lightheaded or dizzy just before fainting: The brain is starting to run low on blood and oxygen but hasn't completely shut down yet. You feel woozy, floaty, or like the room is spinning because the brain's balance and awareness centers are getting less fuel.
- Pale, clammy skin: The body diverts blood away from the skin to protect the heart and brain, so your skin looks washed-out and feels cold and sweaty. This is the body's emergency mode trying to keep vital organs fed.
- Nausea or stomach upset before passing out: The vagus nerve (a big nerve that connects the brain to the gut and heart) fires too hard during certain kinds of fainting, slowing the heart and upsetting the stomach at the same time.
- Blurred or tunnel vision: The eyes need a constant flow of oxygenated blood to see clearly. When blood pressure drops, the edges of your vision go gray or black first because the outer parts of the retina (the light-sensing layer in the back of the eye) lose blood supply before the center does.
- Fast recovery after lying flat: Lying down lets gravity help blood flow back to the brain without the heart having to pump uphill. Most people wake up within seconds to a minute once blood and oxygen return to normal levels in the brain.
- Chest pain or pounding heart before fainting: The heart may be beating dangerously fast, dangerously slow, or in a chaotic rhythm (arrhythmia, an irregular heartbeat) that can't pump blood effectively. Chest pain can mean the heart muscle itself isn't getting enough oxygen, a serious warning sign.

- Fainting during exercise or while lying down: Healthy fainting usually happens when standing because gravity pulls blood away from the brain. Fainting during hard exercise or when already flat suggests a dangerous heart problem—like a blocked valve, thickened heart muscle (hypertrophic cardiomyopathy), or a deadly rhythm—because the heart can't meet the body's oxygen demand even without gravity working against it.
- No warning signs before sudden collapse: A life-threatening heart rhythm (ventricular a fast heart rate (tachycardia) or ventricular fibrillation, chaotic electrical signals in the heart's lower chambers) can stop effective blood flow in an instant, so the brain shuts off before any lightheaded feeling builds. This is a medical emergency because the heart may not restart on its own.
- Jerking movements or confusion after waking up: Brief muscle twitches can happen in normal fainting when the brain is starved of oxygen for a few seconds, but prolonged shaking, biting the tongue, or staying confused for minutes afterward suggests a seizure instead of simple fainting (syncope).

Focused exam

- Heart rhythm on monitor or pulse check: Fainting (Syncope) from a heart problem—like an abnormal rhythm (too slow, too fast, or a pause), or a structural defect (such as a tight valve)—will show an irregular pulse, a very slow or very fast rate, or a long pause. Fainting from a nerve reflex or from standing up too quickly will show a normal, steady rhythm. The rhythm tells you which part of the system broke.
- Blood pressure lying down versus standing up (orthostatic vital signs): In orthostatic fainting (syncope) (fainting when you stand), standing drops the top number (systolic blood pressure) by 20 mmHg or more, or the bottom number (diastolic) by 10 mmHg or more, within three minutes. In reflex fainting, blood pressure stays stable lying and standing. This simple test separates the upstream causes.
- Warning signs before passing out (called a early-warning phase (prodrome)), like nausea, tunnel vision, warmth, sweating, or feeling clammy: A warning before fainting points to reflex fainting (syncope)—the body is signaling before it shuts down blood flow. Sudden fainting with no warning points to a heart rhythm problem, a structural heart defect, or orthostatic fainting. Whether or not there is a warning narrows down the cause.
- Trigger (like seeing blood, feeling pain, straining during a bowel movement, standing too long, strong emotion, changing position suddenly, or starting a new medicine): Reflex fainting (syncope) has a clear trigger—blood, pain, emotion, or straining. Orthostatic fainting follows standing up, losing fluid (vomiting, diarrhea, bleeding), or starting a blood-pressure-lowering drug. Heart-related fainting often has no trigger at all. The trigger points backward to what broke.

- How fast the person woke up and how they felt afterward: True fainting (syncope)—any of the three kinds (reflex, orthostatic, or heart-related)—has quick waking (seconds to a couple of minutes) and no confusion afterward. A seizure causes slower waking, confusion, and sleepiness (called a in the groggy phase after a seizure (postictal) state). This difference tells fainting apart from a mimic.
- Skin color and sweat during the event (pale and sweaty versus flushed or dry skin): Reflex fainting (syncope) shows pale skin and sweating—the body trying to keep blood flowing to the brain. Orthostatic fainting shows gradual paleness and cool skin as blood pools in the legs. Heart-related fainting may show pale or bluish skin, depending on how much oxygen is dropping. Skin color tells the body's defense story.

Diagnostics · every test answers a question

- Electrocardiogram (ECG or EKG) → Is the heart rhythm normal, or is there a pause, a fast rhythm, or a structural problem (like thick heart muscle or a past heart attack scar) that could cut off blood flow to the brain?
- Orthostatic vital signs (blood pressure and heart rate lying down, then standing for 1-3 minutes) → Does standing up drop blood pressure enough to starve the brain of blood?
- Complete blood count (CBC) → Is the person anemic (low red blood cells) or bleeding, which would lower the oxygen-carrying capacity and blood volume?
- Blood glucose (finger-stick or lab) → Is the blood sugar so low that the brain does not have enough fuel to stay awake?
- Pregnancy test (urine or blood hCG) in all women of childbearing age → Is the person pregnant, which changes blood volume and can cause orthostatic fainting (syncope) or unmask a heart problem?
- Echocardiogram (ultrasound of the heart) → Is there a structural heart problem—like a tight valve, thick heart muscle, or a weak pump—that blocks blood flow to the brain, especially during exertion?
- Holter monitor or event recorder (24-hour to 30-day heart rhythm recording) → Is there an intermittent rhythm problem that the short ECG in the office missed?
- Tilt-table test (lying flat, then tilted upright for up to 45 minutes while monitored) → Can we trigger a reflex fainting (syncope) episode in a safe setting to confirm the diagnosis when the history is unclear?

Treatment logic

- Sort the upstream break (reflex versus orthostatic versus heart-related) using history, physical exam, orthostatic vitals, and ECG → Identifying which system broke—the nervous system's reflex, the blood volume or blood-pressure-holding system, the heart's rhythm, or the heart's structure—tells you which treatment will actually work. Treating the wrong cause wastes time and can miss a life-threatening problem.
- For reflex (vasovagal) fainting (syncope): reassure the patient, teach physical counter-pressure maneuvers (crossing legs, squeezing hands together, tensing arm and leg muscles), and increase salt and fluid intake → Counter-pressure maneuvers squeeze blood back toward the brain and heart when warning signs start, stopping the faint before it happens. Adding salt (3-10 grams per day) and fluids (2-3 liters per day) expands blood volume so there is more blood to squeeze. These stop the reflex trigger before it drops blood pressure.
- For orthostatic fainting (syncope): stop or lower the dose of blood-pressure-lowering medicines, add compression stockings (waist-high, 15-20 mmHg or higher), increase salt and fluids, and consider fludrocortisone (0.1-0.2 mg daily) or midodrine (2.5-10 mg three times daily) if needed → Stopping offending drugs (like diuretics, alpha-blockers, or vasodilators) removes the brake on blood pressure. Compression stockings and salt restore blood volume and squeeze blood upward so standing does not drain blood from the brain. Fludrocortisone (a steroid that holds onto salt and water in the kidneys) and midodrine (a medicine that tightens blood vessels) rebuild the system that holds pressure upright.
- For heart rhythm fainting (syncope): admit to the hospital, place on continuous heart monitoring (telemetry), treat a slow heart rate (bradycardia) (slow heart rate) with a pacemaker, treat a fast heart rate (tachycardia) (dangerous fast rhythms like ventricular a fast heart rate) with an implantable cardioverter-defibrillator (ICD) or medicines (like beta-blockers or amiodarone), and correct electrolyte imbalances (low potassium, low magnesium) → A pacemaker keeps the heart from pausing or slowing too much. An ICD shocks the heart out of life-threatening fast rhythms. Medicines stabilize the rhythm. Fixing electrolytes (potassium above 4.0 mEq/L, magnesium above 2.0 mg/dL) prevents rhythm triggers. All of these restore steady blood flow to the brain.
- For structural heart fainting (syncope) (like aortic narrowing (stenosis) or hypertrophic cardiomyopathy): refer to cardiology, consider valve replacement (surgical or transcatheter aortic valve replacement, TAVR) for severe aortic narrowing, or septal reduction (surgery or alcohol ablation) for obstructive hypertrophic cardiomyopathy, and restrict vigorous exercise until treated → Replacing a tight valve or reducing thick heart muscle opens the path for blood to flow normally during exertion, preventing fainting (syncope) and sudden death. Exercise restriction prevents triggering fainting or a fatal rhythm before the defect is fixed.

- Enforce driving restrictions (per state law, typically 3-6 months after the last fainting (syncope) episode or until the cause is treated and controlled) and counsel on fall prevention (avoiding heights, swimming alone, or operating heavy machinery) → Fainting (Syncope) while driving, swimming, or on a ladder can kill the patient or others. Keeping the patient off the road and away from dangerous settings until the broken system is fixed prevents a crash or a fatal fall.
- Educate the patient and family to recognize warning signs (prodrome) and lie down immediately when they start → Lying down quickly moves blood back to the brain by gravity and stops the faint before it happens. This reduces injury from falling and gives the body time to recover.

Medications

Fludrocortisone · Mineralocorticoid (a hormone-like drug that tells the kidneys to hold onto salt and water)

Fludrocortisone acts like aldosterone, a natural hormone made by the adrenal glands (two small glands that sit on top of your kidneys). Aldosterone tells the kidneys to save salt and water instead of peeing them out. When your body holds more salt and water, blood volume goes up — like adding more fluid to a water balloon. A bigger blood volume means more blood stays in your arteries when you stand up, so blood pressure doesn't drop as far and your brain keeps getting the oxygen it needs. This drug is especially helpful for people whose fainting is caused by low blood volume or a nervous system that fails to tighten blood vessels when standing (a problem called orthostatic low blood pressure (hypotension)).

1. You swallow the pill once a day, usually in the morning.
2. Fludrocortisone travels through your bloodstream to the kidneys.
3. It binds to mineralocorticoid receptors inside the cells lining the kidney tubules (the tiny tubes where urine is formed).
4. Those receptors then tell the kidney cells to make more ENaC channels (tiny doors that pull sodium, or salt, back into the blood).
5. Water follows the salt automatically, so more water is reabsorbed too.
6. Over the next few days, your blood volume climbs.
7. When you stand up, the higher blood volume means less of a drop in pressure, so you are less likely to faint.

SAFETY WATCH

Can cause high blood pressure (hypertension) if it raises blood volume too much — your doctor will check your blood pressure at every visit. · Can cause low potassium (hypokalemia) because the kidney saves sodium but dumps potassium in exchange — you may need regular blood tests and potassium supplements or foods rich in potassium like bananas, oranges, and potatoes. · Can cause swelling in the ankles and feet (edema) because of the extra fluid in your body. · Can worsen heart failure if your heart is already weak and can't pump the extra fluid load — tell your doctor if you have any history of heart failure. · Not safe in pregnancy unless the benefits clearly outweigh the risks — talk to your doctor if you are pregnant or planning to become pregnant.

Midodrine · Alpha-1 adrenergic agonist (a drug that squeezes blood vessels by mimicking the 'tighten' signal from the sympathetic nervous system)

Midodrine is a prodrug — after you swallow it, your liver turns it into desglymidodrine, the active form. Desglymidodrine then acts like a mini burst of norepinephrine (the main chemical messenger that the sympathetic nervous system uses to tighten blood vessels). It binds to alpha-1 receptors on the smooth muscle wrapped around your arteries and veins and tells them to squeeze. When blood vessels squeeze, they get narrower, so blood pressure goes up — like pinching a garden hose raises the pressure inside. This helps people whose blood vessels don't tighten enough when they stand, which causes blood to pool in the legs and blood pressure to drop (orthostatic low blood pressure (hypotension)). By keeping vessels tighter, more blood returns to the heart and brain.

1. You swallow the pill, usually three times a day during waking hours (morning, midday, and late afternoon).
2. Your liver converts midodrine into desglymidodrine (the active molecule).
3. Desglymidodrine enters the bloodstream and travels to blood vessels all over your body.
4. It binds to alpha-1 adrenergic receptors on the smooth muscle cells in the walls of arteries and veins.
5. Those receptors trigger the muscle cells to contract (squeeze).
6. Blood vessels get narrower, so blood pressure rises.
7. When you stand up, the higher vascular tone (tighter vessels) means blood doesn't pool as much in your legs, and more blood reaches your brain.
8. The effect peaks about one hour after the dose and lasts two to three hours, which is why it is dosed several times a day.

SAFETY WATCH

Can cause high blood pressure when lying down (supine high blood pressure (hypertension)) because the vessels are squeezed even when you don't need them to be — do NOT take a dose within four hours of bedtime, and sleep with the head of your bed raised about 30 degrees (use a wedge or blocks under the bed legs). · Can cause goosebumps (piloerection) and tingling or itching of the scalp because it also acts on alpha receptors in the tiny muscles at the base of hairs in your skin. · Can cause trouble peeing (urinary retention) because it can tighten the smooth muscle at the bladder outlet — tell your doctor if you have an enlarged prostate or any bladder problems. · Avoid if you have severe coronary artery disease (blocked arteries feeding the heart), overactive thyroid (hyperthyroidism), or severe kidney failure. · Do NOT use if you have pheochromocytoma (a rare tumor of the adrenal gland that dumps bursts of adrenaline and norepinephrine into the blood). · Not recommended in pregnancy unless clearly needed — discuss with your doctor if you are pregnant or planning to become pregnant.

Pyridostigmine · Acetylcholinesterase inhibitor (a drug that boosts the parasympathetic nervous system's ability to keep blood vessels toned)

Pyridostigmine blocks the enzyme acetylcholinesterase, which normally breaks down acetylcholine (a messenger chemical used by the parasympathetic nervous system, the 'rest and digest' side of your automatic control system). By slowing that breakdown, acetylcholine builds up at nerve-to-nerve junctions in the autonomic nervous system. The extra acetylcholine helps strengthen the signal that keeps blood vessels slightly toned (gently squeezed) when you stand up. In people with mild autonomic failure (where the automatic nervous system is weak but not completely dead), this can improve blood vessel tone and reduce the drop in blood pressure. Think of it as turning up the volume on a weak radio signal.

1. You swallow the pill, usually two or three times a day.
2. Pyridostigmine is absorbed in your gut and travels through the bloodstream to nerve junctions throughout your body.
3. It binds to and blocks the enzyme acetylcholinesterase at autonomic ganglia.
4. Acetylcholine (the messenger) now hangs around longer instead of being chopped up immediately.
5. The extra acetylcholine activates nicotinic receptors at the ganglia, strengthening the nerve signal that keeps blood vessels toned.
6. When you stand up, your autonomic reflexes (the automatic adjustments that tighten vessels) work a little better, so blood pressure doesn't drop as much.
7. The effect builds over days to weeks; it is not an instant fix.

SAFETY WATCH

Can cause diarrhea, stomach cramps, nausea, and increased saliva or sweating because acetylcholine also acts on the gut and glands (the parasympathetic system controls digestion and secretions). · Can cause muscle twitching (fasciculations) if the dose is too high, because acetylcholine also acts at muscle junctions. · Avoid if you have asthma or chronic obstructive pulmonary disease (COPD) that is not well controlled, because acetylcholine can trigger bronchospasm (tightening of the airways in the lungs). · Avoid if you have a blockage in your intestines (bowel obstruction) or bladder (urinary obstruction). · Generally considered safer in pregnancy than midodrine or fludrocortisone, but still discuss with your doctor if you are pregnant or planning to become pregnant.

Beta-blockers (for example, metoprolol or atenolol) · Beta-adrenergic antagonists (drugs that block adrenaline and norepinephrine from speeding up the heart)

In a small subset of people with reflex (vasovagal) fainting (syncope) who have a very sensitive heart response — meaning their heart rate shoots up dramatically just before fainting — a beta-blocker can sometimes help by preventing that surge. The drug blocks beta-1 receptors in the heart, so adrenaline and norepinephrine (the sympathetic nervous system's main messenger chemicals) can't make the heart race as easily. By keeping the heart rate steadier, the idea is that the body is less likely to trigger the sudden vagal backlash (the big brake slam from the vagus nerve) that causes the faint. However, current guidelines (ESC 2018, AHA/ACC) note that beta-blockers do NOT help most people with vasovagal fainting and can even make orthostatic low blood pressure (hypotension) worse, so they are used very selectively and only after careful evaluation by a specialist.

1. You swallow the pill once or twice a day, depending on the specific beta-blocker (for example, metoprolol is often twice daily, atenolol once daily).
2. The drug enters the bloodstream and reaches the heart.
3. It binds to beta-1 adrenergic receptors on the cells of the SA node and AV node.
4. Those receptors are now blocked, so adrenaline and norepinephrine can't bind and speed up the heart.
5. Heart rate stays lower and more steady, even during stress or standing.
6. In theory, a steadier heart rate means less chance of triggering the reflex that causes a vagal surge and fainting — though this only works in a minority of carefully selected patients with a documented hyperadrenergic (high-adrenaline) response before fainting.

SAFETY WATCH

Can cause fatigue and make you feel sluggish because your heart can't speed up as easily during activity or stress. · Can lower blood pressure too much and worsen orthostatic low blood pressure (hypotension) — this is why they are used very cautiously and rarely in fainting (syncope) patients. · Can worsen asthma or COPD by allowing unopposed beta-2 blockade, which can tighten airways — use cardioselective beta-blockers (like metoprolol or atenolol) that mostly target the heart, not the lungs, and avoid in severe asthma. · Can mask the warning signs of low blood sugar (hypoglycemia) in people with diabetes, such as a racing heart and shakiness. · Do NOT stop suddenly — taper off slowly under your doctor's supervision to avoid rebound fast heart rate (tachycardia) or high blood pressure. · Generally avoided in pregnancy unless clearly needed; metoprolol is sometimes used in pregnancy for other conditions, but discuss with your doctor if you are pregnant or planning to become pregnant.

Red flags

- Chest pain, trouble breathing, or a pounding or skipping heartbeat before or after fainting
- Fainting during hard exercise, while lying flat, or with no warning at all
- Family history of sudden death before age 50 or known dangerous heart conditions
- Older age (over 60) with new fainting spells, especially if you have heart disease or diabetes
- Fainting that happens over and over in a short time, or staying confused and weak long after waking up

Nursing priorities

1. Place patient on continuous cardiac monitor and obtain a 12-lead ECG within 10 minutes — Arrhythmias cause 15 percent of fainting (syncope) and carry highest mortality risk; immediate ECG detects prolonged QT (risk for torsades arrhythmia), heart blocks (electrical conduction failure), and signs of starved blood flow (ischemia) (low oxygen to heart muscle)
2. Measure orthostatic vital signs: blood pressure and heart rate lying flat, then immediately after standing at 1 and 3 minutes — Orthostatic low blood pressure (hypotension) is defined as systolic drop of 20 mmHg or diastolic drop of 10 mmHg within 3 minutes of standing; identifies 15 percent of fainting (syncope) causes and guides medication adjustment and volume repletion

3. Establish IV access and check capillary refill, skin temperature, and mucous membranes for dehydration — Hypovolemia (low blood volume) from dehydration reduces venous return (blood coming back to heart) and causes both orthostatic and reflex fainting (syncope); IV access allows rapid fluid resuscitation if patient is unstable
4. Assess for injury from the fall, including head, spine, and hip examination, and ask about hitting head or loss of memory — Fainting (Syncope) causes unprotected falls because consciousness is lost before reflexes can catch the body; 30 percent sustain injuries including intracranial bleeding (bleeding in skull) and fractures that need immediate treatment
5. Review medication list for drugs that prolong QT interval, lower blood pressure, or slow heart rate, and hold next doses until provider reviews — Medications cause 10 percent of fainting (syncope); antihypertensives (blood pressure pills), diuretics (water pills), and QT-prolonging drugs (some antibiotics, antipsychotics) trigger orthostatic low blood pressure (hypotension) or arrhythmias
6. Keep patient supine with legs elevated if blood pressure is low, and do not let them stand unassisted until cleared — Gravity causes blood pooling in legs when upright; supine position with leg elevation increases venous return and cerebral blood flow (perfusion) pressure (blood flow to brain) by 20 percent, preventing repeat fainting (syncope)
7. Ask about and document red-flag features: fainting (syncope) during exertion, family history of sudden death, chest pain, or fainting while lying down — High-risk features predict cardiac fainting (syncope) with 5 percent risk of death or serious arrhythmia within 30 days; these patients need admission for telemetry monitoring (continuous heart rhythm watching) and echocardiogram (ultrasound of heart)
8. Teach patient to sit or lie down immediately if early-warning phase (prodrome) symptoms start, and avoid triggers like prolonged standing, heat, and dehydration — Recognizing early-warning phase (prodrome) allows 95 percent of vasovagal fainting (syncope) episodes to be aborted by sitting before cerebral poor blood flow (hypoperfusion) causes loss of consciousness; prevents trauma from falls

Sources

- ACC/AHA/HRS Guideline on the Evaluation and Management of Patients with Syncope, 2017
- American College of Emergency Physicians (ACEP) Clinical Policy on Syncope, 2023
- European Society of Cardiology (ESC) Guidelines for Syncope, 2018
- American Academy of Family Physicians (AAFP)

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