

Stable CAD and Chronic Angina

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THE STORY IN ONE SENTENCE

Stable coronary artery disease (CAD) means plaque has narrowed the heart's arteries, causing chest pain or pressure with exercise that stops when you rest — fix it with aspirin, a statin, blood-pressure control, and sometimes a procedure to open the artery.

“Plaque builds up in the heart's arteries, starving the muscle during effort — manage risk factors (aspirin, statin, BP control, lifestyle) and use medicines or procedures to restore supply so demand can be met without pain.”

Normal anatomy — the body's job

The heart is a muscular pump that works every second of your life, and it feeds itself through three main arteries (the right coronary artery, the left anterior descending artery, and the left circumflex artery) that branch off the aorta and wrap around the heart like a crown — delivering oxygen-rich blood to every squeeze.

BODY'S JOB

At rest, the coronary arteries deliver just enough oxygen and glucose to keep the heart muscle (myocardium) contracting smoothly. When you exercise, get stressed, or eat a big meal, your whole body needs more blood — so your heart rate and squeeze-strength go up. That means the heart itself needs more fuel. Healthy coronary arteries widen (dilate) instantly to meet that demand, doubling or tripling flow so supply always matches what the muscle is asking for. This balance — supply meeting demand — is what keeps the heart healthy and pain-free.

1. Oxygen-rich blood leaves the left ventricle (the heart's main pumping chamber) and enters the aorta (the body's biggest artery).

2. The very first branches off the aorta are the coronary arteries — they peel off just above the aortic valve and dive into the heart muscle itself.
3. These arteries split into smaller and smaller branches, reaching every fiber of the heart muscle with a network of tiny vessels (capillaries) where oxygen and glucose are handed off.
4. The heart muscle burns that fuel to contract, then squeezes used blood (now low in oxygen) into coronary veins, which drain into the right atrium and head back to the lungs to be recharged.
5. When you exercise, the sympathetic nervous system (your body's gas pedal) tells the heart to beat faster and harder, and the coronary arteries automatically widen to deliver the extra fuel — supply rises to match demand without you ever thinking about it.

What breaks

Fatty plaque builds up on the inside walls of the coronary arteries, narrowing the channel and blocking extra blood from reaching the heart muscle when demand goes up.

CLINICAL TERM

Atherosclerosis (plaque buildup made of cholesterol, calcium, inflammatory cells, and scar tissue) narrows the lumen (the open space inside) of one or more coronary arteries. At rest, enough blood can still flow through the narrowed artery to meet baseline demand. But during exertion, emotional stress, or a heavy meal — when the heart needs to pump harder and faster — the fixed obstruction prevents the artery from delivering the extra oxygen the myocardium is asking for, causing starved blood flow (ischemia) (not enough oxygen reaching the tissue) and triggering angina (chest pain or pressure).

How the body compensates

The body tries to keep the heart fed by growing tiny new vessels around the blockage (collateral circulation), slowing the heart rate with rest, and using stored energy — but these are temporary patches, not fixes. Vital signs and resting exams can look totally normal, which is why stable CAD is easy to miss until you ask about exercise or do a stress test.

1. At rest, the narrowed artery still delivers just enough oxygen, so the patient feels fine and vital signs (heart rate, blood pressure, oxygen saturation) are normal — this is the dangerous calm before the storm.
2. During exertion, the heart muscle downstream from the blockage runs low on oxygen (ischemia), triggering chest pain or pressure (angina) as a warning signal — the body is screaming 'stop moving, I can't keep up.'

3. If the patient stops and rests, demand drops, the pain goes away in minutes, and they feel fine again — this on-off pattern is the hallmark of stable angina and tells you the blockage is fixed but not yet complete.
4. Over weeks to months, the heart tries to grow tiny detour vessels (collateral arteries) around the blockage to deliver a little extra blood — this can delay symptoms but rarely fixes the problem completely.
5. If plaque keeps growing or a clot suddenly forms on top of it, the artery can close completely, cutting off all oxygen to that part of the heart — turning stable angina into an acute heart attack (myocardial tissue death from blocked blood flow (infarction)) with permanent muscle damage or sudden cardiac arrest if the rhythm stops.
6. The crash point to watch for: sudden worsening of chest pain (happens at rest or with less effort than before, lasts longer, does not respond to nitroglycerin) — called unstable angina — which means a clot is forming and a heart attack is minutes to hours away.
7. A patient who looks stable on the couch but gets chest pain walking one block is NOT stable — they are on a slow slide toward a heart attack, and the window to prevent permanent damage is closing.

Domino effect

1. Plaque (a mix of cholesterol, calcium, inflammatory cells, and scar tissue) builds up on the inside wall of a coronary artery, narrowing the channel and hardening the vessel so it cannot widen on demand. — Years of high LDL cholesterol (the 'bad' cholesterol), high blood pressure, smoking, diabetes, and inflammation damage the endothelium (the artery's inner lining), letting cholesterol particles burrow into the wall, triggering an immune response that walls off the mess with scar tissue and calcium — forming plaque that grows silently for decades.
2. At rest, enough blood squeezes through the narrowed artery to feed the heart muscle, so the patient feels fine and has no symptoms. — Resting demand is low — the heart is just ticking along — so even a 50-70 percent blockage can still deliver the trickle of oxygen and glucose the muscle needs to contract gently.
3. During exercise, stress, or a big meal, the sympathetic nervous system (the body's gas pedal) releases adrenaline (epinephrine), which tells the heart to beat faster and squeeze harder — demand for oxygen spikes. — The whole body needs more blood when you move or get stressed, so the heart has to pump more blood per minute — that means the heart muscle itself needs two or three times more fuel to keep up.

4. The narrowed coronary artery cannot widen or deliver extra blood, so the heart muscle downstream from the blockage runs low on oxygen (ischemia) — cells switch to burning fuel without oxygen (anaerobic metabolism), which makes lactic acid and hurts. — The plaque is like a clogged pipe — it is locked narrow and cannot stretch open, so supply stays flat even though demand is soaring. Starving muscle cells release pain signals (bradykinin, adenosine, hydrogen ions) that travel up sensory nerves to the spinal cord and brain, and you feel chest pain or pressure (angina), often radiating to the jaw, neck, shoulder, or left arm.
5. The patient stops exercising and rests (or takes a nitroglycerin pill under the tongue), demand drops back down, the trickle of blood is enough again, starved blood flow (ischemia) stops, lactic acid clears, and the pain goes away in 2–10 minutes. — Nitroglycerin releases nitric oxide, which relaxes veins and arteries throughout the body — this drops the workload on the heart (less blood coming back to fill it, lower blood pressure to pump against) so demand falls and the narrow artery can meet it again. Rest alone does the same thing by turning off adrenaline.
6. Without treatment, plaque keeps growing, the artery gets narrower, and chest pain starts happening with less and less effort — eventually even at rest — and a blood clot (thrombus) can suddenly form on top of a cracked plaque, blocking the artery completely. — Plaque is not stable — the cap of scar tissue can crack open (plaque rupture), exposing the gooey cholesterol core to flowing blood. Platelets (the blood's clotting cells) stick to the rupture, forming a clot that can seal off the artery in seconds, cutting all oxygen to the downstream muscle — turning stable angina into an acute heart attack (STEMI or NSTEMI) or triggering a deadly rhythm (ventricular fibrillation) and sudden cardiac arrest.
7. Treatment aims to restore the balance: medicines (aspirin to prevent clots, a statin to shrink plaque and calm inflammation, a beta-blocker or calcium-channel blocker to slow the heart and lower demand, an ACE inhibitor or ARB if you have high blood pressure or diabetes or heart failure) plus lifestyle changes (quit smoking, exercise daily, eat less saturated fat and trans fat, control blood sugar and blood pressure). — You cannot dissolve old plaque completely, but you can stop new plaque from growing, prevent clots from forming, and lower the heart's workload so supply can meet demand again without pain. Aspirin keeps platelets from sticking together. Statins lower LDL cholesterol and reduce inflammation in the artery wall. Beta-blockers slow the heart rate and block adrenaline, cutting demand. Lifestyle changes fix the root causes (high cholesterol, high blood pressure, insulin resistance, inflammation) so the disease does not get worse.

8. If medicines are not enough (chest pain keeps happening, or stress testing shows a large area of starved blood flow (ischemia), or the blockage is in a critical spot like the left main coronary artery), a cardiologist can open the artery with a balloon and prop it open with a metal stent (PCI) or a surgeon can sew in a bypass graft (CABG) to route blood around the clog. — Revascularization (restoring blood flow) fixes the supply line directly — new blood reaches the starving muscle, starved blood flow (ischemia) stops, chest pain goes away, and the risk of a future heart attack drops. The choice between PCI and CABG depends on how many arteries are blocked, where the blockages are, and whether you have diabetes or weak heart function — guidelines favor CABG for left main disease or three-vessel disease with diabetes, because long-term outcomes are better.

Symptoms · what the patient feels, and why

- Chest pressure or tightness that starts with effort and stops with rest: The heart muscle needs more oxygen when you exercise or get upset. In stable CAD, waxy plaque narrows the coronary arteries (the pipes feeding the heart). The narrowed pipe can deliver enough blood when you are calm, but when the heart works harder and needs more, the pipe cannot keep up — so the heart muscle gets hungry for oxygen (ischemia) and sends pain signals.
- Pain or heaviness that spreads to the left arm, jaw, neck, or back: The heart and these other body parts share the same nerve highways back to the spinal cord. When the heart sends a pain signal, the brain can get confused about exactly where it is coming from and feel it in the arm, jaw, or back instead (called referred pain).
- Shortness of breath during activity: When the heart muscle is starved for oxygen, it cannot squeeze as hard. A weaker squeeze means less blood is pushed forward to the lungs and body, so fluid can back up into the lungs (like a traffic jam) and you feel short of breath.
- Symptoms that go away within a few minutes of stopping the activity: When you rest, the heart does not need as much oxygen anymore. The narrowed artery can now keep up with the smaller demand, so the hunger (ischemia) stops and the pain melts away. This predictable on-off pattern is the hallmark of stable angina.
- Symptoms triggered the same way each time — for example, always after climbing two flights of stairs or walking uphill: The plaque narrows the artery by a fixed amount, so it takes roughly the same workload every time to push the heart past what that pipe can deliver. This steady threshold is why stable angina is so predictable.
- Relief within minutes of taking nitroglycerin under the tongue: Nitroglycerin releases nitric oxide, a signal that tells the smooth muscle around arteries to relax and widen. Wider coronary arteries let more blood through, and wider veins around the body mean less blood returns to the heart — both reduce the heart's workload and oxygen hunger, so the pain stops.

- A resting EKG that looks normal: At rest the narrowed artery delivers just enough oxygen, so the heart muscle is not blood-starved (ischemic) and the electrical signals look normal. The EKG only shows trouble when you stress the heart (exercise test) and push it past the artery's limit.
- Chest pain that happens at rest, lasts longer than usual, or comes on with less effort than before: These patterns suggest the plaque may have cracked or a clot is forming on top of it, suddenly shrinking the pipe even more. This is called unstable angina and means a full heart attack could be minutes to hours away.

Focused exam

- Chest pain or pressure that starts with exercise, climbing stairs, or strong emotion and goes away within 5–10 minutes of rest or after nitroglycerin under the tongue: This is the hallmark of stable angina — the heart muscle is not getting enough oxygen when it works hard, so it sends a pain signal. The pattern (pain with work, relief with rest) tells you the blockage is steady, not total.
- Normal heart sounds (S1, S2 clear, no extra sounds or murmurs) and clear lungs at rest: Stable CAD means the heart is still pumping well enough when the patient is sitting still — the exam will be quiet and normal between episodes because the muscle is not failing yet and fluid has not backed up into the lungs.
- Risk factors present: age > 55 in men or > 65 in women, current or past smoking, high blood pressure, high cholesterol, diabetes, or family history of early heart disease: These risk factors cause the plaque to build up over years. The more you see, the higher the chance the chest pain is coming from coronary artery disease and not something else like heartburn or muscle strain.
- Blood pressure $\geq 130/80$ mmHg or random glucose ≥ 140 mg/dL: High blood pressure makes the heart work harder (so it needs more oxygen while the arteries are already narrowed), and high blood sugar speeds up plaque growth. Both push stable CAD toward trouble if not controlled.
- Resting 12-lead EKG is normal or shows old changes (like Q waves or T-wave flattening) but no new signs of fresh injury: The resting EKG is often normal in stable CAD because the heart is getting just enough blood when the patient is still. Changes only appear during exercise or stress when demand outstrips supply.
- Patient describes the chest pain as pressure, squeezing, tightness, or a heavy weight — not sharp, stabbing, or burning: The quality of the pain tells you where it is coming from. Starved blood flow (Ischemia) (the heart muscle not getting enough blood) feels like pressure or tightness. Sharp or burning pain points more toward the esophagus, chest wall, or lungs.

Diagnostics · every test answers a question

- Lipid panel (total cholesterol, LDL, HDL, triglycerides) → How much bad cholesterol is floating in the blood and fueling new plaque growth?
- Hemoglobin A1c → Is blood sugar controlled, or is chronic high sugar accelerating plaque buildup and heart damage?
- Basic metabolic panel (sodium, potassium, creatinine, eGFR) → Are the kidneys still filtering well, and are electrolytes safe before starting medicines like ACE inhibitors or diuretics?
- Thyroid-stimulating hormone (TSH) → Could an overactive or underactive thyroid be mimicking or worsening the chest pain and fatigue?
- Exercise stress EKG (treadmill test) or pharmacologic stress test (if the patient cannot exercise) → Does the heart show starved blood flow (ischemia) (lack of blood flow) when we push the demand up with exercise or medicine?
- Coronary CT angiography (CCTA) or invasive coronary angiography → Where exactly is the blockage, how tight is it, and does it need a stent or bypass surgery?
- High-sensitivity troponin (if chest pain is new, changing, or not clearly stable) → Is there any fresh heart muscle damage happening right now, or is this truly stable chronic angina?

Treatment logic

- Aspirin 81 mg once daily (or clopidogrel 75 mg if aspirin allergy) → Stops platelets (tiny blood cells) from clumping together and forming a clot on top of the cracked plaque. This keeps a partial blockage from turning into a total blockage and causing a heart attack.
- High-intensity statin: atorvastatin 40–80 mg or rosuvastatin 20–40 mg once daily → Lowers LDL cholesterol to < 70 mg/dL (or < 55 mg/dL if very high risk), slows or stops new plaque growth, and can even shrink existing plaque a little by pulling cholesterol out of the artery wall. Also stabilizes plaque so it is less likely to crack and trigger a clot.
- Beta-blocker (e.g., metoprolol succinate 25–200 mg daily or carvedilol 3.125–25 mg twice daily) → Slows the heart rate and weakens each heartbeat, so the heart uses less oxygen during exercise. This pushes the threshold higher — the patient can do more before chest pain starts.
- Sublingual nitroglycerin 0.4 mg as needed for acute chest pain (patient keeps it in pocket), plus long-acting nitrate (e.g., isosorbide mononitrate 30–120 mg daily) if symptoms are frequent → Relaxes and widens the coronary arteries and veins, so more blood flows past the blockage and reaches the starving heart muscle. Stops chest pain within 1–5 minutes.

- ACE inhibitor (e.g., lisinopril 5–40 mg daily) or ARB (e.g., losartan 25–100 mg daily) if the patient also has high blood pressure, diabetes, reduced heart function ($EF < 40\%$), or prior heart attack → Lowers blood pressure by blocking angiotensin (a hormone that squeezes blood vessels), so the heart pumps against less resistance and needs less oxygen. Also protects the kidneys and slows heart remodeling (scarring and weakening) after a heart attack.
- Calcium channel blocker (e.g., amlodipine 5–10 mg daily or diltiazem 120–360 mg daily) if beta-blockers are not tolerated or if symptoms persist despite beta-blocker → Relaxes the smooth muscle in artery walls (including coronary arteries), so blood vessels widen and blood flows more easily. Also lowers blood pressure and heart workload.
- Coronary revascularization: percutaneous coronary intervention (PCI with stent placement) or coronary artery bypass grafting (CABG) if angina does not respond to maximum medicine, or if coronary angiography shows high-risk anatomy (e.g., left main narrowing (stenosis) $\geq 50\%$, three-vessel disease, or proximal LAD narrowing $\geq 70\%$) → Physically restores blood flow by opening the blocked artery (stent) or routing blood around the blockage (bypass graft). Chest pain goes away because the heart muscle gets the oxygen it needs even during exercise.

Medications

Aspirin 81 mg (low-dose baby aspirin) · Antiplatelet agent

Aspirin stops tiny blood cells called platelets from sticking together and forming clots inside the coronary arteries (the arteries that feed the heart muscle). When plaque is already built up in these arteries, even a small clot can plug the narrow opening and cause a heart attack. Aspirin keeps the blood flowing smoothly by blocking an enzyme in platelets called COX-1 (cyclooxygenase-1), which prevents the platelets from making thromboxane A₂, the sticky signal that tells platelets to clump.

1. Aspirin permanently blocks the COX-1 enzyme inside platelets.
2. Without COX-1, platelets cannot make thromboxane A₂, the chemical signal that tells platelets to stick together.
3. Platelets become less sticky and less likely to clump into a clot.
4. Blood flows more freely through narrowed coronary arteries.
5. Risk of sudden heart attack from a clot blocking a coronary artery drops.

SAFETY WATCH

Can cause stomach bleeding or ulcers—take with food if your stomach gets upset, and tell your doctor if you see black tarry stools or vomit that looks like coffee grounds. · Increases bleeding risk—tell your dentist and surgeon before any procedure. · Do not take if you have an active bleeding ulcer or severe bleeding disorder. · Avoid other NSAIDs like ibuprofen at the same time unless your doctor says it is safe.

High-intensity statin (atorvastatin 40-80 mg or rosuvastatin 20-40 mg) · HMG-CoA reductase inhibitor (statin)

Statins lower the amount of LDL cholesterol (the bad cholesterol that builds plaques) in your blood by blocking an enzyme in the liver called HMG-CoA reductase. This enzyme is the liver's cholesterol factory. When you block it, the liver makes much less cholesterol and pulls LDL cholesterol out of the blood to use instead. Lower LDL slows plaque growth, stabilizes existing plaques (makes them less likely to crack open), and cuts the risk of heart attack and stroke. The goal LDL for people with stable CAD is below 70 mg/dL, or below 55 mg/dL if your risk is very high.

1. Statin blocks the HMG-CoA reductase enzyme in liver cells.
2. The liver cannot make as much cholesterol anymore.
3. Liver cells grow more LDL receptors on their surface to grab LDL cholesterol from the blood.
4. LDL cholesterol in the blood drops.
5. Plaques grow more slowly, and existing plaques become more stable and less likely to rupture and cause a heart attack.

SAFETY WATCH

Can rarely cause muscle pain or weakness—tell your doctor right away if your muscles hurt or feel unusually weak. · Very rarely can cause rhabdomyolysis (muscle breakdown that can hurt the kidneys)—get emergency help if you have severe muscle pain, dark urine, or extreme weakness. · Can raise liver enzymes—your doctor will check blood tests before you start and occasionally afterward. · Avoid grapefruit juice with atorvastatin and some other statins, because it raises the drug level in your blood. · Tell your doctor if you are pregnant or planning pregnancy—statins are not safe during pregnancy.

ACE inhibitor (e.g., lisinopril, ramipril) or ARB (e.g., losartan, valsartan) · Angiotensin-converting enzyme inhibitor (ACE inhibitor) or Angiotensin II receptor blocker (ARB)

ACE inhibitors and ARBs both block the renin-angiotensin-aldosterone system (RAAS), the body's main blood pressure and fluid control system. Angiotensin II is a powerful chemical that tightens blood vessels and raises blood pressure. ACE inhibitors stop angiotensin II from being made by blocking the ACE enzyme. ARBs block the receptors where angiotensin II tries to work. Either way, blood vessels relax, blood pressure drops, and the heart does not have to pump as hard. These drugs also protect the heart muscle, kidneys, and blood vessel lining. They are especially important if you also have high blood pressure, diabetes, reduced heart pump function (ejection fraction below 40%), or a history of heart attack.

1. ACE inhibitor blocks the ACE enzyme, so angiotensin I cannot be turned into angiotensin II. ARB blocks the AT1 receptor, so angiotensin II cannot signal blood vessels to tighten.
2. Blood vessels relax and widen.
3. Blood pressure drops.
4. The heart pumps against lower resistance and does not have to work as hard.
5. Over time, this protects the heart muscle and blood vessel lining from further damage.

SAFETY WATCH

Can cause a dry, nagging cough with ACE inhibitors—if it bothers you, your doctor can switch you to an ARB. · Can rarely cause angioedema (sudden swelling of the face, lips, tongue, or throat)—get emergency help if you have trouble breathing or swallowing. · Can raise potassium levels—your doctor will check blood tests, and you may need to limit high-potassium foods like bananas and salt substitutes. · Can affect kidney function—blood tests will be checked before starting and periodically. · Do not take if you are pregnant or planning pregnancy—these drugs can harm a developing baby.

Beta-blocker (e.g., metoprolol, atenolol, carvedilol) · Beta-adrenergic receptor blocker

Beta-blockers are the first-choice medicine to prevent and control angina (chest pain). They block beta receptors in the heart, which are the landing spots for adrenaline (epinephrine) and noradrenaline (norepinephrine). When adrenaline hits these receptors, your heart beats faster and harder, which makes the heart muscle demand more oxygen. By blocking adrenaline's signal, beta-blockers slow the heart rate, lower blood pressure, and reduce how hard the heart squeezes. This cuts the heart's oxygen demand, so the narrowed coronary arteries can keep up and chest pain happens less often. Beta-blockers also protect against dangerous heart rhythms

and are especially important if you have had a heart attack or have reduced heart pump function.

1. Beta-blocker sits on beta-1 receptors in the heart and blocks adrenaline and noradrenaline from binding.
2. The SA node slows down, so the heart beats slower.
3. The heart muscle squeezes less forcefully.
4. Blood pressure drops because the heart pumps with less force.
5. The heart uses less oxygen with each beat, so narrowed coronary arteries can meet the demand and angina happens less often.

SAFETY WATCH

Can make you feel tired or slow you down—this usually improves after a few weeks. · Can cause dizziness or lightheadedness, especially when you stand up—stand slowly and sit down if you feel faint. · Can worsen asthma or COPD in some people—tell your doctor if you have lung disease; a cardioselective beta-blocker like metoprolol is usually safer. · Can mask the warning signs of low blood sugar if you have diabetes—check your blood sugar regularly. · Do not stop suddenly—your doctor will taper the dose slowly to avoid rebound chest pain or high blood pressure.

Calcium channel blocker (CCB) (e.g., amlodipine, diltiazem, verapamil) · Calcium channel blocker

Calcium channel blockers are a second-choice medicine for angina if a beta-blocker does not work well enough or causes side effects you cannot tolerate. They block calcium channels in heart muscle cells and the smooth muscle cells that line blood vessel walls. Calcium is the spark that makes these muscles squeeze. By blocking calcium from entering, CCBs relax the heart and widen blood vessels. This lowers blood pressure, reduces how hard the heart has to work, and increases blood flow through the coronary arteries. Some CCBs (like diltiazem and verapamil) also slow the heart rate, similar to beta-blockers. Others (like amlodipine) mainly relax blood vessels.

1. CCB blocks L-type calcium channels in the muscle cell membrane.
2. Calcium cannot flow into the cell.
3. Without calcium, the muscle cannot contract as strongly.
4. Blood vessels relax and widen, lowering blood pressure and increasing blood flow to the heart.
5. The heart muscle relaxes and uses less oxygen, so angina happens less often.

SAFETY WATCH

Can cause swelling in the ankles and feet, especially with amlodipine—elevating your legs can help. · Can cause flushing, headache, or dizziness. · Non-dihydropyridine CCBs (diltiazem, verapamil) can slow the heart too much or worsen heart failure—your doctor will monitor your heart rate and pump function. · Avoid grapefruit juice, which raises the drug level in your blood. · Do not use a non-dihydropyridine CCB and a beta-blocker together without close monitoring, because both slow the heart.

Long-acting nitrate (e.g., isosorbide mononitrate, isosorbide dinitrate) · Organic nitrate vasodilator

Long-acting nitrates relax and widen blood vessels, especially veins and the coronary arteries. They release nitric oxide (NO), a gas signal that tells smooth muscle in blood vessel walls to relax. This reduces the amount of blood returning to the heart (preload) and the pressure the heart pumps against (afterload), so the heart does less work and needs less oxygen. Widening the coronary arteries also improves blood flow to the heart muscle. Long-acting nitrates are taken once or twice a day to prevent angina, but they must be dosed with a nitrate-free interval (usually overnight) to avoid tolerance (where the drug stops working).

1. Nitrate is converted to nitric oxide (NO) in the blood vessel wall.
2. Nitric oxide activates an enzyme called guanylate cyclase inside smooth muscle cells.
3. This enzyme makes cyclic GMP (cGMP), a messenger that tells the muscle to relax.
4. Blood vessels widen, blood pressure drops, and the heart's workload decreases.
5. Coronary arteries also widen, improving blood flow to the heart muscle and reducing angina.

SAFETY WATCH

Can cause headaches, especially when you first start—this usually improves after a few days. · Can cause dizziness or fainting from low blood pressure—stand slowly and sit down if you feel faint. · Must be dosed with a nitrate-free interval (usually 10–14 hours overnight) to prevent tolerance. · Never take with PDE5 inhibitors (sildenafil/Viagra, tadalafil/Cialis, vardenafil/Levitra) for erectile dysfunction—this combination can cause life-threatening low blood pressure. · Tell emergency responders you take nitrates if you have chest pain, because it affects which medicines they can give you.

Ranolazine · Late sodium current inhibitor

Ranolazine is used for chronic angina that does not get better with standard medicines like beta-blockers, calcium channel blockers, or nitrates. It works by blocking late sodium channels in heart muscle cells. Normally, a little sodium keeps leaking into heart cells even after they

contract, and this extra sodium triggers extra calcium to come in. Too much calcium makes the heart muscle stiffer and raises pressure inside the heart, which squeezes the small blood vessels that feed the heart muscle and worsens angina. By blocking late sodium flow, ranolazine reduces calcium overload, relaxes the heart muscle, lowers pressure inside the heart chambers, and improves blood flow to the heart muscle. It does not slow the heart rate or lower blood pressure much, so it can be added to other angina medicines.

1. Ranolazine blocks late sodium channels in heart muscle cells.
2. Less sodium flows into the cell during relaxation (diastole).
3. Less sodium means less calcium is pulled in through the sodium-calcium exchanger.
4. Calcium overload in the cell drops.
5. The heart muscle relaxes better, pressure inside the heart falls, and small blood vessels in the heart wall open up.
6. Blood flow to the heart muscle improves, and angina happens less often.

SAFETY WATCH

Can cause dizziness, nausea, constipation, or headache. · Can prolong the QT interval on an ECG (a heart rhythm measurement)—your doctor will check an ECG before starting and avoid ranolazine if your QT is already long. · Avoid with strong CYP3A inhibitors (certain antibiotics, antifungals, HIV medicines) and grapefruit juice, which raise ranolazine levels. · Tell your doctor about all your medicines, because ranolazine interacts with many drugs.

Sublingual nitroglycerin (under-the-tongue quick spray or tablet) · Short-acting organic nitrate vasodilator

Sublingual nitroglycerin is a rescue medicine for sudden angina attacks. It works the same way as long-acting nitrates—it releases nitric oxide, which relaxes blood vessels—but it acts in 1–3 minutes because it absorbs directly into the bloodstream under your tongue. It widens the coronary arteries (improving blood flow to the heart muscle) and veins (reducing the amount of blood returning to the heart), so the heart's workload and oxygen demand drop quickly and chest pain goes away. You take one dose at the first sign of chest pain, wait 5 minutes, and if pain persists, take a second dose. If pain is still there after a third dose (15 minutes total), call 911—you may be having a heart attack.

1. Nitroglycerin dissolves under the tongue and enters the bloodstream in seconds.
2. It is converted to nitric oxide in blood vessel walls.
3. Nitric oxide activates guanylate cyclase, which makes cyclic GMP.
4. Blood vessels relax and widen rapidly.
5. Blood flow to the heart muscle improves, the heart's workload drops, and chest pain goes away within 1–3 minutes.

SAFETY WATCH

Can cause a sudden drop in blood pressure—sit or lie down before taking it to avoid fainting. · Common side effects are headache, flushing, and dizziness. · Tablets lose potency over time—replace them every 6 months and keep them in the original brown glass bottle, tightly closed. · Never take with PDE5 inhibitors (sildenafil/Viagra, tadalafil/Cialis, vardenafil/Levitra)—this combination can cause life-threatening low blood pressure. · If chest pain does not improve after 3 doses (one every 5 minutes), call 911 immediately.

Red flags

- Chest pain that starts at rest or wakes you from sleep
- Pain that lasts longer than 10–15 minutes or does not go away with rest or nitroglycerin
- New shortness of breath at rest, sweating, nausea, or feeling like you might pass out
- Pain that is suddenly much worse or comes on with far less effort than your usual pattern

Nursing priorities

1. Teach the patient to call 911 if chest pain lasts more than five minutes after taking one sublingual nitroglycerin tablet or if pain is worse than usual. — Prolonged chest pain or worsening pain means a heart attack may be starting. Heart attack (Myocardial infarction) (heart muscle death) happens when a clot blocks an artery completely. Early treatment within ninety minutes saves heart muscle and prevents death.
2. Check blood pressure at every visit and teach the patient to monitor blood pressure at home weekly. — High blood pressure makes the heart work harder and damages artery walls. A systolic pressure (top number) above one hundred thirty damages the heart over time. Blood pressure control with medicine prevents heart failure, heart attacks, and strokes.
3. Confirm the patient takes aspirin eighty-one milligrams and a high-dose statin every day and knows why these medicines prevent heart attacks. — Aspirin blocks platelets (clotting cells) from forming clots in narrowed arteries. Statins lower LDL cholesterol (bad cholesterol) and stabilize plaque so it does not rupture. Together these medicines cut heart attack risk by forty percent in patients with coronary artery disease.
4. Teach proper use of sublingual nitroglycerin: sit down, place one tablet under the tongue, wait five minutes, take a second tablet if pain continues, then call 911 if pain lasts after the second dose. — Nitroglycerin dilates (opens up) coronary arteries and brings more oxygen to the heart muscle. Sitting prevents falls because nitroglycerin lowers blood pressure suddenly. Three tablets over fifteen minutes is the limit before emergency care is needed.

5. Assess for signs of heart failure at each visit: ankle swelling, weight gain of more than three pounds in one day, shortness of breath when lying flat, and fatigue. — Coronary artery disease weakens the heart muscle over time. Heart failure (a weak pump) causes fluid to back up into the lungs and body. Early detection allows us to start ACE inhibitors and diuretics before the patient needs hospitalization.
6. Review the patient's daily activity level and teach that regular walking for thirty minutes five days a week improves blood flow and reduces chest pain. — Exercise training opens small collateral vessels (tiny backup arteries) that bring blood around blockages. Physical activity also lowers blood pressure, improves cholesterol, and strengthens the heart. Cardiac rehabilitation programs reduce death by twenty-five percent.
7. Screen for depression and anxiety at every visit using a simple two-question screen: feeling down or hopeless, and loss of interest in activities. — Depression doubles the risk of heart attack and death in patients with coronary disease. Stress hormones like cortisol and adrenaline raise blood pressure and make blood clot more easily. Treating depression with therapy or medicine improves heart outcomes.
8. Teach the patient to avoid sudden heavy lifting or shoveling snow because these activities spike blood pressure and oxygen demand quickly. — Isometric exercise (straining without moving, like lifting heavy objects) raises blood pressure suddenly and forces the heart to work much harder. Cold weather also constricts (narrows) arteries. Together these trigger angina and heart attacks in people with coronary disease.

Sources

- AHA/ACC 2023 Chronic Coronary Disease Guideline
- ESC 2019 Chronic Coronary Syndromes Guideline
- UpToDate
- NEJM

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