

Stable CAD and Chronic Angina

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.

THE ONE-LINE TRUTH

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.

The chain — how it unfolds

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. — why: 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. — why: 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. — why: 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. — why: 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. — why: 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. — why: 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). — why: 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. — why: 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.

Spot it — what you see, 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.

Red flags — act now

- 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

Treat it — in this order

1. 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.
2. 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.
3. 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.
4. 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.
5. 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.

Your top priorities at the bedside

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. — why: 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. — why: 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. — why: 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.