

Chest Pain and ACS Triage

A plain-English guide for you and your family

WHAT IS HAPPENING

Your heart is a muscle that beats about 100,000 times every day. To do that work, it needs a steady supply of oxygen-rich blood. Three coronary arteries wrap around the outside of the heart and deliver that oxygen. As long as supply matches demand—what the heart needs versus what the arteries bring—everything runs smoothly. But chest pain happens when that balance breaks. Sometimes a coronary artery gets blocked by a blood clot that forms on top of a ruptured cholesterol plaque, cutting off oxygen to part of the heart muscle (that is a heart attack). Sometimes the arteries squeeze tight in a spasm, or a big artery like the aorta tears open inside its wall, or a clot travels up from a leg vein and lands in the lung. Sometimes the heart is fine but it is being asked to do too much—like when your heart rate shoots to 180 or your blood pressure spikes to 220. The chest pain you feel is your heart muscle screaming that it is not getting enough fuel. That pain often feels like pressure, tightness, or a heavy weight on your chest. It can spread to your jaw, neck, shoulders, or left arm because those areas share the same nerve highways. Your body knows something is wrong, so it floods you with adrenaline—you start sweating, feel sick to your stomach, and get a sense of dread. On an ECG (a test that records the heart's electrical signals), doctors see changes that show which wall of the heart is starving. A blood test called troponin goes up because dying heart cells leak that protein into your bloodstream. Every one of these signs points back to the same upstream problem: the heart is not getting what it needs. The faster a team finds and fixes that break—whether it is opening a blocked artery, stopping a tear, or dissolving a clot—the more heart muscle they can save and the better your chance of going home healthy.

What you might feel

- Pressure, squeezing, or heaviness in the center of the chest (often brought on by activity, relieved by rest)
- Pain or discomfort spreading to the left arm, jaw, neck, back, or stomach
- Sweating (cold, clammy skin) without obvious reason
- Shortness of breath (with or without chest discomfort)
- Nausea or vomiting

- Light-headedness or feeling faint
- Sudden, severe 'ripping' or 'tearing' pain radiating to the back
- Sharp, stabbing chest pain that worsens with deep breathing or lying flat, plus a scratchy sound on exam
- Sudden sharp pain on one side, trouble breathing, and absent breath sounds on that side
- Sharp pain with breathing, low oxygen on the monitor, fast heart rate, and sometimes coughing up blood

Call the doctor right away if...

- Chest pain with sweating, shortness of breath, or feeling faint—these suggest the heart is in serious trouble right now.
- New irregular heartbeat or very slow or very fast pulse—the heart's electrical system may be failing.
- Blood pressure dropping or signs of shock (cool, clammy skin; confusion; weak pulse)—the heart is not pumping enough blood to keep the body alive.
- Sudden tearing pain to the back—the aorta may be ripping open, a true surgical emergency.
- Active bleeding or very high bleeding risk in someone who needs blood thinners—treatment must be carefully balanced to save the heart without causing dangerous bleeding.

What the care team will do

- Treat every chest-pain arrival as an emergency: ECG in hand within 10 minutes.
- Never leave a 'normal-looking' chest-pain patient in the waiting room.
- Take blood pressure in both arms if dissection is possible.
- IV, monitor, and aspirin per protocol once dissection and bleeding are not suspected.
- Draw the first troponin at arrival and repeat per protocol.
- Reassess pain after every treatment, and repeat the ECG if the pain changes.

Medicines you may be given — and why

Aspirin

Aspirin stops platelets (tiny blood cells that make clots) from sticking together. It does this by blocking an enzyme called COX-1, which platelets need to make a sticky chemical called thromboxane A2. Without that chemical, platelets cannot clump. This keeps a clot in the blocked heart artery from growing bigger.

P2Y12 inhibitor (clopidogrel, ticagrelor, or prasugrel)

These drugs block a different pathway platelets use to stick together. Platelets have a receptor called P2Y12 that responds to a chemical signal (ADP) telling them to clump. By blocking that receptor, the drug stops platelets from getting the 'stick together' message. Combined with aspirin (which blocks a separate pathway), this dual blockade keeps clots from growing in the narrowed heart artery.

Heparin (unfractionated) or enoxaparin (low-molecular-weight heparin)

Heparin works with a natural protein in your blood called antithrombin. Antithrombin normally slows down clotting, but it works slowly. Heparin turbocharges it—making antithrombin 1,000 times faster at shutting down thrombin (the enzyme that weaves the clot's mesh) and factor Xa (an earlier enzyme in the clotting chain). This stops new clot threads from forming and keeps the existing clot from growing.

Nitroglycerin

Nitroglycerin releases nitric oxide inside the walls of blood vessels. Nitric oxide is a natural signal that tells smooth muscle in vessel walls to relax. When the muscle relaxes, veins widen (reducing the amount of blood returning to the heart, so the heart does less work) and coronary arteries widen slightly (improving blood flow to the heart muscle). Both effects lower the oxygen demand of the heart and can ease chest pain.

Morphine

Morphine binds to opioid receptors in the brain and spinal cord, blocking pain signals and changing how the brain interprets pain. It also reduces anxiety and can slightly widen blood vessels, lowering the heart's workload. In acute coronary syndrome, morphine is used for severe chest pain that nitroglycerin has not relieved—it helps the patient feel less distress while the team works to open the artery.

Statin (atorvastatin or rosuvastatin, high-intensity)

Statins block an enzyme in the liver (HMG-CoA reductase) that makes cholesterol. With less cholesterol being made, the liver pulls more cholesterol out of the blood to use for its needs, lowering blood cholesterol levels. Over weeks and months, this shrinks cholesterol deposits in artery walls, stabilizes plaques (making them less likely to rupture), and reduces inflammation. In acute coronary syndrome, starting a high-dose statin right away has been shown to improve outcomes even before cholesterol drops—likely because statins also calm inflammation in the plaque.

Beta-blocker (metoprolol or carvedilol)

Beta-blockers block receptors (called beta-1 receptors) on heart muscle cells. Normally, adrenaline and related stress hormones bind to these receptors and make the heart beat faster and harder. By blocking the receptors, beta-blockers slow the heart rate and reduce how forcefully the heart squeezes. Both effects lower the heart's oxygen demand, which protects struggling heart muscle when blood flow is reduced. Beta-blockers also reduce the risk of dangerous heart rhythms (like ventricular fibrillation) after a heart attack.

ACE inhibitor (lisinopril, enalapril) or ARB (losartan, valsartan)

ACE inhibitors block an enzyme (ACE) that makes angiotensin II, a hormone that squeezes blood vessels tight and tells the kidneys to hold onto salt and water. By blocking this enzyme, blood vessels relax and blood pressure falls, easing the heart's workload. ARBs block the receptor where angiotensin II works, achieving a similar effect. After a heart attack—especially if the heart's pumping strength is reduced—these drugs prevent harmful remodeling (scarring and enlargement) of the heart, reduce the risk of heart failure, and improve long-term survival.

Fibrinolytic (alteplase, tenecteplase, or reteplase)

Fibrinolytics are enzymes (or enzyme activators) that break up clots by turning on the body's natural clot-dissolving system. They activate plasminogen (a protein in blood) into plasmin, which chews up fibrin threads holding the clot together. The clot dissolves, and blood flow is restored to the blocked coronary artery. Fibrinolytics are used in STEMI when a cardiac catheterization lab is not available within 120 minutes—they are a time-sensitive alternative to restore blood flow and save heart muscle.

Esmolol or labetalol (for aortic dissection)

In aortic dissection, the wall of the aorta (the body's largest artery) has torn, creating a false channel where blood is flowing under high pressure. Every heartbeat generates a pressure wave (called dP/dt) that can rip the tear further. Beta-blockers slow the heart rate and reduce the force of each heartbeat, lowering that pressure wave. Esmolol works within seconds and wears off in minutes (easy to control with IV infusion). Labetalol also blocks alpha receptors, which helps lower blood pressure at the same time. The goal is to bring heart rate below 60 and systolic blood pressure to 100–120 mmHg to stop the tear from extending.

Nitroprusside or nicardipine (for aortic dissection)

Once heart rate and contractility are controlled with a beta-blocker, blood pressure is lowered further with a vasodilator. Nitroprusside releases nitric oxide, which relaxes smooth muscle in artery walls, widening vessels and dropping blood pressure rapidly. Nicardipine blocks calcium channels in vascular smooth muscle, preventing contraction and also lowering blood pressure. The key rule in aortic dissection is: NEVER give a vasodilator before a beta-blocker—if you

drop blood pressure without slowing the heart first, the heart will speed up reflexively, increasing dP/dt and making the dissection worse.

Where this information comes from

- AHA/ACC 2021 Chest Pain Guideline
- AHA/ACC 2023 Acute Coronary Syndrome Guideline
- European Society of Cardiology 2020 ACS Guideline
- JAMA 2022 Chest Pain Evaluation Review

Last reviewed: 2026-07-07