

Acute Coronary Syndrome

A clot blocks a heart artery, and heart muscle starts dying because it cannot get oxygen.

THE STORY IN ONE SENTENCE

A clot blocks a heart artery, and heart muscle starts dying because it cannot get oxygen.

“Acute Coronary Syndrome means heart muscle is starving for oxygen right now because a blood clot has partly or completely blocked one of the arteries that feeds it.”

Normal anatomy — the body's job

Coronary arteries deliver oxygen-rich blood to heart muscle. The heart uses more oxygen per ounce of tissue than almost any other organ because it beats nonstop, about one hundred thousand times a day. Even at rest, the heart pulls nearly all the oxygen out of the blood passing through. When the heart needs more oxygen during exercise or stress, the only way to get it is to widen the coronary arteries and push more blood through. Keep that picture in mind, because every abnormal finding in Acute Coronary Syndrome is a change from this normal state. When a coronary artery gets blocked by a cracked cholesterol patch and a clot, that is the upstream break we are tracing.

BODY'S JOB

If you understand the normal job first, the abnormal findings make sense instead of feeling like random facts to memorize.

1. Oxygen-rich blood leaves the left ventricle (the main pumping chamber) through the aorta (the body's biggest artery).
2. Immediately as blood exits, the first two branches off the aorta are the left coronary artery and the right coronary artery.
3. The left coronary artery splits into two branches: the left anterior descending artery, which feeds the front wall and the septum (the wall between the two pumping chambers), and the circumflex artery, which feeds the side and back walls.
4. The right coronary artery feeds the bottom wall, also called the inferior wall, and usually the right ventricle.

5. Blood flows through these arteries into smaller and smaller branches, like a tree branching into twigs, until it reaches capillaries (the tiniest vessels) inside the heart muscle.
6. In the capillaries, oxygen leaves the blood and enters muscle cells through diffusion (moving from high concentration to low), where it fuels energy production inside mitochondria (the cell's power plants).
7. The heart muscle squeezes about seventy times per minute at rest, and each squeeze requires energy in the form of ATP (a molecule cells burn like gasoline).
8. Because the heart extracts almost all the oxygen on the first pass, any drop in blood flow immediately starves the muscle. There is no reserve to draw from.

What breaks

One broken thing—a cholesterol plaque that cracks open and triggers a clot inside a coronary artery—explains all the clinical findings together.

CLINICAL TERM

An atherosclerotic plaque (a buildup of cholesterol, fat, calcium, and inflammatory cells in the artery wall) becomes unstable. The fibrous cap (the thin layer covering the plaque) ruptures and exposes the soft, fatty core to the bloodstream. Platelets (tiny clotting cells) recognize the exposed material as an injury and immediately stick together. A blood clot (thrombus) (a blood clot) forms on top of the ruptured plaque. The clot partly or completely blocks the artery. Heart muscle downstream of the blockage becomes blood-starved (ischemic) (not enough blood is reaching the tissue to feed it). Within twenty to forty minutes, blood-starved muscle stops contracting normally. If blood flow is not restored, muscle cells die, called tissue death from blocked blood flow (infarction), within two to six hours. That break does not stay local. The downstream signals you will actually see at the bedside—chest pain, ST-segment changes on the EKG, rising troponin, weak heart squeeze on ultrasound—all point backward to this one broken thing upstream.

How the body compensates

Vital signs can look stable while the body compensates. Your job is to recognize the patient is on the edge of a crash before it happens.

1. The sympathetic nervous system (your fight-or-flight system) releases epinephrine (adrenaline) and norepinephrine from the adrenal glands and nerve endings to keep blood pressure and heart rate up, so vital signs may look near normal early on. This is the body's attempt to squeeze harder and faster to make up for weak heart muscle.

2. That is the dangerous middle, because it can fool the team into missing how unstable the patient really is. Blood pressure might be one hundred over seventy, heart rate ninety, and the patient looks okay on paper, but inside, the heart is struggling.
3. The crash points to watch for are life-threatening arrhythmias (abnormal heart rhythms): ventricular fibrillation, where the ventricles quiver chaotically instead of pumping; ventricular a fast heart rate (tachycardia), where the ventricles race so fast they cannot fill with blood between beats; and complete heart block, especially in inferior or right ventricular infarctions, where the electrical pathway between the atria and ventricles is cut off and the ventricles beat too slowly to keep the body alive.
4. Mechanical complications usually happen three to seven days after the tissue death from blocked blood flow (infarction): papillary muscle rupture causes sudden severe mitral regurgitation, where the valve between the left atrium and left ventricle fails and blood shoots backward into the lungs; ventricular free-wall rupture, where the heart wall tears open and blood floods into the pericardial sac around the heart, causing cardiac tamponade (the heart squeezed by fluid around it) (a compression that stops the heart from filling); and ventricular septal defect, where a hole punches through the wall between the two ventricles so oxygen-poor and oxygen-rich blood mix, and the heart has to work much harder.
5. Cardiogenic shock happens when the heart is too weak to pump enough blood to keep the body alive. Blood pressure drops below ninety systolic, skin becomes cool and clammy from poor circulation, urine output falls below thirty milliliters per hour because the kidneys are not getting enough blood, and the patient becomes confused as the brain does not get enough oxygen. Lactate rises because tissues switch to anaerobic metabolism (making energy without oxygen, which produces lactic acid).
6. Listen for new heart murmurs—they signal valve problems or septal rupture. A harsh holosystolic murmur (a sound that lasts the whole time the heart squeezes) at the left sternal border suggests ventricular septal defect. A new blowing murmur at the apex (the bottom tip of the heart) suggests mitral regurgitation from papillary muscle rupture or starved blood flow (ischemia).
7. Watch for sudden drops in blood pressure, worsening shortness of breath, cool clammy skin, and signs of poor cardiac output: weak pulses, confusion, agitation, low urine output, mottled skin.

8. Right ventricular infarctions are tricky. The right ventricle pumps blood to the lungs. When it fails, it needs higher filling pressure (more blood return) to squeeze effectively. Standard heart failure treatments—nitrates such as nitroglycerin, which open veins and drop filling pressure, or diuretics such as furosemide (water pills that reduce blood volume)—cause the blood pressure to crash. Right ventricular infarctions need IV normal saline boluses to keep filling pressure up. Look for the triad: low blood pressure (hypotension) (low blood pressure), elevated jugular venous pressure with visible neck veins, and clear lungs on exam because the left side is not the problem.
9. Watch for the crash point before it happens, because once compensation fails, the window to save muscle and life narrows fast. Early intubation, mechanical circulatory support such as an intra-aortic balloon pump or Impella device, and vasopressors such as norepinephrine may be needed.
10. Look at trends—how the patient is changing over minutes to hours—not just one snapshot in time. A heart rate climbing from eighty to one hundred ten, blood pressure drifting from one twenty systolic to one hundred, oxygen saturation dropping from ninety-eight percent to ninety-four percent, respiratory rate rising from sixteen to twenty-four—those are early warnings that compensation is starting to fail.

Domino effect

1. Atherosclerotic plaque inside a coronary artery ruptures, and the fibrous cap covering it cracks open. — Atherosclerotic plaque is a buildup of cholesterol, fat, calcium, and inflammatory cells such as macrophages (immune cells that eat debris and release signals) in the artery wall. The plaque has a soft, fatty core covered by a thin fibrous cap made of collagen and smooth muscle cells. Chronic inflammation weakens the cap. Enzymes called matrix metalloproteinases (proteins that break down collagen), released by macrophages, digest the collagen. Mechanical stress from blood pressure pulsing against the plaque, especially at branch points where flow is turbulent (rough and swirling), causes the cap to crack or tear. The soft, fatty core inside is suddenly exposed to the bloodstream.
2. Platelets stick to the exposed plaque and form a blood clot (thrombus) (a blood clot). — Platelets are tiny cell fragments that float in the bloodstream and seal injuries. When the fibrous cap ruptures, collagen, tissue factor, and von Willebrand factor (proteins that signal damage) from the plaque core are exposed. Platelets have receptors, including GP Ib and GP IIb/IIIa (docking sites on the platelet surface), that recognize these proteins as danger signals. Platelets immediately stick to the exposed material, activate, change shape, and release chemicals such as ADP and thromboxane A₂ (signals that recruit more platelets). A clot builds on top of the ruptured plaque. The clotting cascade (a series of enzyme reactions) is also triggered, and fibrin strands weave through the platelet plug to strengthen it.

3. The a blood clot (thrombus) partly or completely blocks the coronary artery, and blood flow downstream drops or stops. — The clot grows rapidly and narrows or closes the artery. If the blockage is complete, oxygen-rich blood cannot reach the heart muscle supplied by that artery. If the blockage is partial, some blood still trickles through or arrives through tiny collateral vessels (backup routes that connect neighboring coronary arteries). These collaterals develop slowly over time in people with chronic narrowing, so someone with old blockages may tolerate a new clot better than someone with previously clean arteries.
4. Heart muscle downstream becomes blood-starved (ischemic) (starved of oxygen). — Heart muscle cells need oxygen to make ATP (the energy molecule that powers every cell function). Without oxygen, mitochondria (the cell's power plants) cannot run the electron transport chain (the main ATP-producing pathway). Cells switch to anaerobic glycolysis (making energy without oxygen), but this process is far less efficient, producing only two ATP per glucose molecule instead of thirty-six, and it generates lactic acid as a byproduct. Lactic acid builds up and makes the environment acidic. Within seconds, ATP levels drop, and the muscle starts to fail.
5. Blood-starved (Ischemic) muscle cannot contract normally, and that wall segment stops squeezing. — Muscle contraction requires ATP. During contraction, myosin heads (tiny motors inside muscle fibers) pull on actin filaments (protein threads that slide past each other), and each pull burns one ATP molecule. When ATP runs out, myosin heads cannot detach and reset, so the muscle fibers cannot shorten and squeeze. That wall segment moves weakly, called hypokinesis, or not at all, called akinesis. On an echocardiogram (an ultrasound of the heart), you can see the injured wall segment sit still while the rest of the heart squeezes. This reduces stroke volume (the amount of blood pumped with each beat), and cardiac output drops.
6. Electrical signals in the blood-starved (ischemic) zone become abnormal. — Normal heart rhythm depends on sodium, potassium, and calcium ions flowing in and out of cells in a precise pattern. Ion pumps, especially the sodium-potassium ATPase pump (a pump that uses ATP to move ions), require ATP to maintain concentration gradients (the difference in ion levels inside versus outside the cell). When ATP runs out, the pumps fail. Potassium leaks out of cells, and sodium and calcium flood in. The resting membrane potential (the electrical charge across the cell membrane) changes from its normal negative eighty-five millivolts to a less negative value. Cells depolarize (fire their electrical signal) and repolarize (reset for the next beat) in abnormal ways. The injured zone becomes electrically unstable, and dangerous arrhythmias such as ventricular a fast heart rate (tachycardia) and ventricular fibrillation can start.

7. ST-segment changes appear on the EKG. — The ST segment on an EKG represents the moment between ventricular depolarization (when the ventricles contract) and repolarization (when they reset). Starved blood flow (Ischemia) creates an abnormal electrical current, called an injury current, between the injured zone and the healthy zone during this moment. If the injury goes through the full thickness of the heart wall, called transmural starved blood flow, the ST segment lifts up, called ST elevation, in the EKG leads facing that wall. If the injury affects mainly the subendocardium (the inner layer), the ST segment drops down, called ST depression, or the T wave flips upside down, called T-wave inversion. The pattern and location of ST changes tell you which coronary artery is blocked: ST elevation in leads II, III, and aVF means the right coronary artery and the inferior wall; ST elevation in V1 through V4 means the left anterior descending artery and the front wall; ST elevation in I, aVL, V5, and V6 means the circumflex artery and the side or back wall.
8. Cardiac troponin leaks into the bloodstream. — Troponin is a protein inside heart muscle cells that regulates contraction by controlling calcium binding to actin and myosin. The troponin complex has three parts: troponin C binds calcium, troponin I inhibits contraction, and troponin T binds the complex to tropomyosin (another regulatory protein). When muscle cells die or their membranes break down from severe starved blood flow (ischemia), troponin spills out into the bloodstream, like grain spilling from a torn sack. Troponin I and troponin T are specific to heart muscle, so a rise in blood troponin means heart muscle is dying. Troponin is more specific and sensitive than older markers such as CK-MB. Troponin levels rise within two to six hours after symptom onset, peak at twelve to twenty-four hours, and stay elevated for one to two weeks. The first troponin test may be normal if drawn too early, within the first hour or two. That is why current guidelines recommend repeating troponin at three to six hours if the first is normal but suspicion is high. High-sensitivity troponin assays (newer tests approved in the United States and widely used in Europe) detect much smaller amounts and rise faster, sometimes within one to two hours, and can rule out heart attack (myocardial infarction) more quickly using zero-hour and one-hour or two-hour protocols.

9. Chest pain or discomfort occurs. — Blood-starved (Ischemic) muscle releases chemicals, including adenosine, bradykinin, hydrogen ions from lactic acid buildup, potassium ions, and prostaglandins (inflammatory signals). These chemicals activate cardiac pain nerves, mainly C fibers (slow-conducting nerve fibers that carry dull, aching, pressure-like pain signals). The nerves send signals up the spinal cord through the dorsal horn (the back part of the spinal cord) to the thalamus (the brain's relay station) and then to the somatosensory cortex (the brain region that senses touch and pain), where pain is perceived. The brain perceives the signal as chest pressure, squeezing, heaviness, or tightness, often described as an elephant sitting on the chest. The pain may radiate to the jaw, neck, shoulder, left arm, or even the upper abdomen because those areas share nerve pathways, called dermatomes, with the heart. Not everyone feels typical chest pain—women, older adults, and people with diabetes are more likely to have atypical symptoms such as shortness of breath, nausea, fatigue, or just feeling unwell.
10. The sympathetic nervous system activates, releasing epinephrine and norepinephrine, causing sweating, nausea, and anxiety. — The blood-starved (ischemic) heart signals distress through vagal afferents (sensory nerve fibers that travel in the vagus nerve) to the brainstem, which activates the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis (the body's main stress response system). The adrenal glands release epinephrine (adrenaline) and norepinephrine into the bloodstream. These stress hormones cause sweating, especially cold clammy sweats, by stimulating sweat glands, increase heart rate and blood pressure to try to maintain cardiac output, and trigger nausea by slowing gut motility (movement of food through the intestines). Patients often describe a sense of impending doom, an overwhelming feeling that something is terribly wrong.
11. Inflammatory cells arrive at the site of dead muscle and begin cleaning up debris. — Within hours after tissue death from blocked blood flow (infarction), neutrophils (a type of white blood cell that eats bacteria and debris) migrate into the dead tissue, attracted by inflammatory signals such as interleukin-1, tumor tissue death (necrosis) factor, and complement fragments (immune system alarm signals). Neutrophils release enzymes that break down dead cells. After a few days, macrophages (another type of white blood cell) arrive and continue the cleanup by engulfing cellular debris through phagocytosis (eating and digesting debris). This inflammatory phase weakens the heart wall temporarily, raising the risk of mechanical complications such as free-wall rupture, typically three to seven days after the tissue death from blocked blood flow.

- 12.** Fibroblasts move in and lay down scar tissue over weeks to months. — After dead tissue is cleared, fibroblasts (cells that make collagen) migrate into the area and start building scar tissue. They lay down a dense network of collagen fibers that is strong and prevents the heart wall from tearing, but scar tissue does not contract like muscle. The more muscle that dies, the weaker the heart becomes. Over weeks to months, the heart compensates by enlarging, called ventricular remodeling, to maintain stroke volume, but this remodeling eventually leads to heart failure. ACE inhibitors such as lisinopril and beta-blockers such as metoprolol reduce harmful remodeling and improve survival by blocking the renin-angiotensin-aldosterone system (a hormone system that raises blood pressure and makes the heart work harder) and reducing wall stress.

Symptoms · what the patient feels, and why

- Chest discomfort—pressure, squeezing, heaviness, or tightness: When heart muscle does not get enough blood (ischemia, meaning the tissue is starving), the dying cells release chemicals like adenosine and bradykinin. These chemicals wake up pain nerves in the heart wall that send signals up the spinal cord to the brain. The brain reads those signals as deep pressure or squeezing, not a sharp stab.
- Pain spreading to the left arm, jaw, neck, back, or upper belly: Pain nerves from the heart and nerves from your arm, jaw, and belly all plug into the same spots in the spinal cord (they share the same nerve root level). So when the heart nerve fires, the brain sometimes guesses the pain is coming from your arm or jaw instead. This mix-up is called referred pain.
- Sweating (diaphoresis): When the heart is in trouble, your body's alarm system (the sympathetic nervous system, the branch that runs fight-or-flight) switches on. That system tells sweat glands all over your body to turn on, just like when you are scared or running hard.
- Nausea or vomiting: Pain nerves from the heart wake up the vagus nerve (a big nerve that runs from the brainstem down to the stomach and heart). The vagus nerve sends a signal to the vomit center in the brainstem, making you feel sick or throw up. This happens especially when the bottom wall of the heart (inferior wall) is hurt.
- Shortness of breath (dyspnea): When heart muscle is starving, it cannot squeeze well. The left ventricle (the main pumping chamber on the left side) cannot push all the blood out, so blood backs up into the lungs. Fluid leaks from the blood vessels into the air sacs (pulmonary congestion), making it hard to breathe.
- Lightheadedness or fainting (syncope): If the heart cannot pump enough blood forward (low cardiac output, meaning not enough blood is leaving the heart) or if the heart starts beating in a dangerous way (arrhythmia), blood pressure drops. The brain does not get enough oxygen and you feel dizzy or pass out.

- Feeling the heart race or beat irregularly (palpitations): Starving heart muscle messes up the electrical signals that tell the heart when to beat. The heart may fire extra beats, race too fast (tachycardia), or skip beats. You feel this as fluttering, pounding, or a racing feeling in your chest.
- Atypical presentation—just feeling tired, weak, or short of breath without any chest pain (especially in women, older adults, and people with diabetes): High blood sugar over time damages nerves (diabetic neuropathy), so pain signals from the heart may never reach the brain. Older adults and women sometimes have different pain thresholds or show symptoms like tiredness and trouble breathing instead of classic chest pain.

Focused exam

- Crushing chest pressure or squeezing that spreads to the arm, jaw, neck, or back: When a coronary artery (a pipe feeding oxygen-rich blood to the heart muscle) gets clogged by a clot, the starving muscle sends pain signals through nerves that the arm, jaw, and neck also share—so the brain feels the ache in those spots even though the real problem is in the heart itself. This spreading pattern is the single biggest clue that the heart is in immediate danger and needs help within minutes.
- Cold, clammy sweat breaking out all over the skin, often with waves of nausea: The autonomic nervous system (the body's automatic control center for heart rate, breathing, and digestion) senses the heart crisis and dumps epinephrine (the main fight-or-flight hormone, also called adrenaline) and norepinephrine (another stress hormone) into the blood. These chemicals switch on sweat glands everywhere and trigger the stomach to feel queasy. Sudden drenching sweat plus chest pain is a red flag for a heart attack happening right now.
- New heart murmur (a whooshing sound when listening with a stethoscope) or crackling sounds in the lungs called crackles in the lungs (rales): Dying heart muscle can tear the thin strings called chordae tendineae that anchor a valve leaflet, letting the leaflet flap backward and causing a murmur. Or the weakened pump cannot keep up with the blood coming in, so blood backs up into the pulmonary veins (vessels from the lungs to the heart) and leaks fluid into the alveoli (tiny air sacs)—you hear that fluid as crackles. Both mean a dangerous complication is unfolding right now.
- Blood pressure drops below 90 systolic, or signs of shock—pale cold skin, confusion, weak thready pulse that is hard to feel: The damaged left ventricle (the heart's main pumping chamber on the lower left) cannot squeeze hard enough to push blood forward to the brain, kidneys, and body. When organs do not get enough oxygen-rich blood, the person slips into cardiogenic shock (shock caused by pump failure)—a life-threatening emergency that can spiral into organ failure and death within hours if not reversed.

- Irregular heartbeat, or heart rate slower than 50 beats per minute or faster than 120 beats per minute: Oxygen-starved heart muscle scrambles the electrical signals that normally travel from the sinoatrial node (the heart's natural pacemaker in the right atrium) through the conduction pathways. Dangerous rhythms like ventricular a fast heart rate (tachycardia) (very fast rhythm starting in the ventricles) or ventricular fibrillation (chaotic quivering with no effective pump) can spiral into sudden cardiac arrest within minutes.
- Shortness of breath, or the person sitting bolt upright and leaning forward to catch their breath: When the left ventricle cannot pump efficiently, blood backs up into the pulmonary veins and floods the alveoli with fluid—this is called pulmonary edema (fluid flooding the lungs) (fluid in the lungs). The person literally drowns in their own fluid and cannot get air in and out, a sign of acute heart failure complicating the heart attack.

Diagnostics · every test answers a question

- 12-lead electrocardiogram (EKG or ECG) → Is there ST-segment elevation or depression showing that a coronary artery is blocked or severely narrowed right now?
- High-sensitivity troponin I or troponin T (blood test) → Is heart muscle actually dying right now and leaking its inside proteins into the bloodstream?
- Complete blood count (CBC) → Is there enough hemoglobin to carry oxygen to the struggling heart, or is active bleeding making things worse?
- Basic metabolic panel (BMP) with creatinine and electrolytes—sodium, potassium, chloride, bicarbonate, glucose → Are the kidneys working well enough to safely clear contrast dye used in heart catheterization, and are potassium and sodium in the safe zone?
- Chest X-ray (portable anterior-posterior view if the person is too unstable to stand) → Is fluid backing up into the lungs because the heart is failing, or is there another cause of chest pain like a collapsed lung or torn aorta?
- Transthoracic echocardiogram (bedside ultrasound of the heart) → Which part of the heart wall is not moving, how weak is the pump overall, and are there mechanical complications like a torn valve or hole in the wall between chambers?
- Coronary angiography (cardiac catheterization with dye injection) → Exactly where is the blockage, how severe is the narrowing, and can it be opened with a balloon and stent immediately?
- B-type natriuretic peptide (BNP) or N-terminal pro-BNP (NT-proBNP) → Is the heart muscle stretched and under pressure from volume overload or pump failure?

Treatment logic

- Aspirin 162–325 mg chewed immediately (non-enteric-coated tablet) → Irreversibly blocks cyclooxygenase-1 (COX-1, the enzyme inside platelets that makes clotting signals) so platelets cannot produce thromboxane A₂ (the chemical messenger that tells platelets to stick together). Chewing gets the drug absorbed through the lining of the mouth into the bloodstream within 5 minutes instead of waiting 30–60 minutes for a swallowed pill to dissolve in the stomach. Cuts 30-day death rate by about 23% even before any other treatment starts.
- Second antiplatelet drug—ticagrelor 180 mg loading dose, prasugrel 60 mg loading dose, or clopidogrel 600 mg loading dose (dual antiplatelet therapy, called DAPT) → Blocks the P₂Y₁₂ receptor on the platelet surface, a completely different pathway from aspirin—this stops ADP from activating platelets. Using two drugs hits platelet clumping from two angles at once, like locking two separate doors. Keeps the artery open after it has been unblocked by balloon and stent or clot-busting drugs, and cuts the chance of another heart attack or stent clot formation (thrombosis) (new clot forming on the stent) by 20–30%.
- Anticoagulation—unfractionated heparin IV bolus (60–70 units per kg body weight, max 5000 units) plus continuous infusion, or enoxaparin 1 mg per kg subcutaneous injection every 12 hours, or bivalirudin continuous infusion → Heparin amplifies antithrombin III (a natural brake on clotting) to shut down the coagulation cascade—the chain reaction that weaves fibrin strands into a stable mesh holding the clot together. Bivalirudin directly inhibits thrombin itself. Both prevent new clot from forming on top of the existing a blood clot (thrombus), inside stents, or on catheters threaded through arteries during PCI.
- Urgent percutaneous coronary intervention (PCI)—door-to-balloon time under 90 minutes for STEMI, within 24 hours for high-risk NSTEMI (GRACE score >140, refractory starved blood flow (ischemia), hemodynamic instability, or life-threatening arrhythmia) → A guide catheter threaded from the radial artery at the wrist (or femoral artery in the groin) delivers a deflated balloon to the blockage under live X-ray guidance. Inflating the balloon crushes the a blood clot (thrombus) and plaque against the artery wall and cracks open the fibrous cap, then a drug-eluting stent (a tiny mesh tube coated with medication that prevents scar tissue from growing back) is deployed to scaffold the artery open. Immediately restores forward blood flow and salvages blood-starved (ischemic) myocardium before irreversible tissue death (necrosis) (cell death) happens.

- Fibrinolytic therapy (alteplase, tenecteplase, or reteplase IV) if PCI cannot be performed within 120 minutes of first medical contact and symptom onset is within 12 hours → Converts plasminogen (an inactive enzyme floating in the blood) into plasmin (the active enzyme that chews up fibrin), chemically dissolving the clot mesh. Restores TIMI 3 flow (normal brisk flow with dye filling the whole artery) in about 50–60% of patients. Most effective when given within 3 hours of symptom onset—every 30-minute delay cuts the benefit and more muscle dies.
- Beta-blocker (metoprolol tartrate 25–50 mg by mouth every 6–12 hours, carvedilol, or bisoprolol) started within 24 hours if the person is hemodynamically stable → Blocks beta-1 adrenergic receptors on cardiomyocytes (heart muscle cells), which lowers heart rate and contractility (how hard the heart squeezes). This decreases myocardial oxygen demand—the amount of oxygen the injured muscle needs—while it heals and forms scar. Reduces the risk of ventricular arrhythmias, another episode of starved blood flow (ischemia), and reinfarction by about 20–25%. Long-term use over months to years reduces death rate.
- High-intensity statin (atorvastatin 80 mg or rosuvastatin 40 mg by mouth once daily) started immediately in the hospital, regardless of baseline LDL cholesterol level → Inhibits HMG-CoA reductase (the enzyme in liver cells that makes cholesterol), lowering LDL cholesterol by 50% or more. Stabilizes the fibrous cap on atherosclerotic plaques so they are less likely to rupture again, reduces vascular inflammation driven by inflammatory cytokines, and improves endothelial function (how well the artery lining works). Cuts the chance of another heart attack and cardiovascular death by 25–30% over 1–2 years.
- ACE inhibitor (lisinopril starting at 2.5–5 mg daily and titrated up, enalapril, or ramipril) or ARB (losartan, valsartan) if ACE inhibitor causes intolerable cough—started within 24 hours if LVEF $\leq$ 40%, anterior wall MI, clinical heart failure, high blood pressure (hypertension), or diabetes → ACE inhibitor blocks angiotensin-converting enzyme so the body cannot make angiotensin II (a powerful hormone that squeezes vessels and raises blood pressure). ARB blocks the angiotensin II receptor directly. Both lower afterload (the pressure the left ventricle has to pump against), prevent pathologic remodeling (progressive thinning, stretching, and dilation of the ventricle that leads to heart failure), and decrease aldosterone release (a hormone that makes the kidneys hold onto salt and water). Reduces death rate by 20–25% in people with reduced ejection fraction or heart failure after MI.

Medications

Aspirin · Antiplatelet (cyclooxygenase inhibitor)

Aspirin stops tiny blood cells called platelets from sticking together. Platelets usually help seal cuts, but when plaque (a fatty bump inside a heart artery) cracks open, platelets rush over and glue themselves into a clot that can completely block blood flow to the heart. Aspirin permanently shuts off an enzyme called cyclooxygenase-1 (COX-1) inside each platelet. Without

that enzyme, platelets cannot make thromboxane A₂ (the sticky chemical signal that makes them clump). This keeps the dangerous clot from growing bigger and choking off the artery.

1. Plaque inside a coronary artery cracks or ruptures, exposing sticky collagen and tissue factor underneath
2. Platelets rush to the crack and start sticking to the exposed material and to each other
3. Aspirin permanently turns off the cyclooxygenase-1 (COX-1) enzyme inside each platelet for the platelet's entire 7–10 day life
4. Without COX-1, platelets cannot make thromboxane A₂, the chemical that signals other platelets to clump
5. Platelets become much less sticky, so the clot grows more slowly or stops growing entirely
6. More blood can keep flowing through the narrowed artery to feed starving heart muscle

SAFETY WATCH

Bleeding risk goes up—watch for black, tarry stools (bleeding in the stomach or intestines), vomit that looks like coffee grounds, unusually heavy periods, or large bruises that appear without injury · Do NOT give if the patient has active bleeding anywhere (stomach ulcer, recent bleeding stroke, bleeding in the brain) · Can irritate the stomach lining and cause pain, heartburn, or nausea; chewing the first dose helps it absorb through the mouth and work within 5 minutes instead of 30 · Rarely causes severe allergic reaction (hives, throat or tongue swelling, trouble breathing)—ask about aspirin or NSAID allergy before giving the first dose · Even low-dose aspirin raises bleeding risk, so tell the patient to let any doctor or dentist know they are on it before procedures

Ticagrelor, Prasugrel, or Clopidogrel (P2Y₁₂ inhibitors) · Antiplatelet (P2Y₁₂ receptor blocker)

These drugs block a second sticky pathway on platelets. Even with aspirin on board, platelets can still clump tightly if they receive a signal through a receptor called P2Y₁₂ (a lock on the platelet's surface). When a chemical called ADP (adenosine diphosphate) fits into that lock, the platelet gets much stickier and recruits more platelets to the clot. P2Y₁₂ inhibitors plug the lock so ADP cannot activate it. Using aspirin plus a P2Y₁₂ inhibitor together (called dual antiplatelet therapy) shuts down platelet clumping through two separate doors, cutting the chance that the clot will grow back or a new one will form.

1. When plaque ruptures, damaged cells and activated platelets release ADP into the bloodstream
2. ADP normally binds to the P2Y₁₂ receptor on platelets, which makes them change shape, get very sticky, and amplify the clotting signal to other platelets

3. The P2Y₁₂ inhibitor molecule locks onto the receptor first, so ADP cannot attach and send its signal
4. Without the ADP signal, platelets stay calmer and much less likely to stick to each other or release more clotting chemicals
5. Combined with aspirin (which blocks the thromboxane pathway), nearly all the major platelet activation routes are shut down
6. The clot grows much more slowly or stops growing, keeping the artery open enough for blood to flow

SAFETY WATCH

Bleeding risk is higher than with aspirin alone—watch for any unusual bleeding (gums, nose, cuts that won't stop, blood in urine or stool) or new large bruises · Prasugrel should NOT be used if the patient has ever had a stroke or TIA (mini-stroke), or if body weight is under 60 kg (132 pounds), because bleeding risk is dangerously high in those groups · Ticagrelor causes shortness of breath (dyspnea) in about 10–15% of patients, usually mild and harmless; it goes away on its own in most cases, but warn the patient it may happen so they don't panic · Clopidogrel must be activated by liver enzymes (CYP2C19); some people have gene variants that make those enzymes work poorly, so clopidogrel doesn't protect them well—ticagrelor and prasugrel do not have this problem and work in everyone · Do NOT stop these drugs early (especially in the first year after a stent is placed) without talking to the cardiologist—stopping too soon lets clots form again on the stent or at the plaque site, causing another heart attack · Tell the patient to let any doctor or dentist know they are on a P2Y₁₂ inhibitor before any procedure, because bleeding risk is higher

Heparin, Enoxaparin (low-molecular-weight heparin), or Bivalirudin · Anticoagulant (clotting-cascade inhibitor)

Anticoagulants stop clots from growing by blocking the proteins in the blood that weave a tight mesh around the platelet clump. Think of a clot as a net: platelets are the knots, and clotting proteins (especially thrombin and Factor Xa) are the threads that tie everything together into a solid plug. Heparin and enoxaparin boost a natural brake in the blood called antithrombin III, which shuts down thrombin and Factor Xa. Bivalirudin grabs thrombin directly and blocks its active site. Either way, the protein mesh cannot form, so the clot stays soft and small instead of turning into a hard plug that completely blocks the artery.

1. When plaque ruptures, the clotting cascade starts—a chain reaction where one clotting protein activates the next in line
2. Factor Xa and thrombin are key proteins late in the cascade; thrombin is the most powerful one because it cuts fibrinogen (a dissolved protein floating in blood) into fibrin threads

3. Fibrin threads weave through and around the platelet clump, turning it into a hard, stable clot that can completely block the artery
4. Heparin and enoxaparin bind to antithrombin III and make it 1000 times more powerful; supercharged antithrombin then locks onto Factor Xa and thrombin and inactivates them
5. Bivalirudin binds directly to thrombin's active site and blocks it from cutting fibrinogen
6. With thrombin and Factor Xa shut down, fibrinogen stays dissolved and no fibrin net forms
7. The clot remains soft and small, and blood can still squeeze past while the patient is rushed to the catheterization lab to open the artery

SAFETY WATCH

Major bleeding risk—watch for blood in urine (pink or red urine), black or tarry stools (intestinal bleeding), large bruises, bleeding gums, or any bleeding that will not stop with pressure · Heparin can rarely cause a dangerous reaction called HIT (heparin-induced a low platelet count (thrombocytopenia)), usually 5–10 days after starting; the platelet count drops and clots form paradoxically—check platelet count every 2–3 days if heparin is used longer than 4 days; switch to bivalirudin if HIT develops · Enoxaparin dose must be cut in half or avoided if the kidneys are not working well (creatinine clearance below 30 mL/min), because the drug builds up in the blood and causes severe bleeding · Bivalirudin has much lower HIT risk and is often the first choice during heart catheterization · Heparin can be reversed quickly with protamine sulfate if serious bleeding happens; enoxaparin reversal with protamine is only partial; bivalirudin wears off on its own within about 25 minutes (very short half-life) so you just stop the drip

Metoprolol, Carvedilol, Atenolol, or other Beta-Blocker · Beta-adrenergic receptor blocker

Beta-blockers slow the heart rate and lower blood pressure, which reduces how hard the heart has to work. During a heart attack, part of the heart muscle is not getting enough oxygen because a coronary artery is blocked or narrowed. If the heart keeps racing and squeezing hard (driven by adrenaline flooding the body during the stress of a heart attack), the starving muscle runs out of oxygen even faster and more cells die. Beta-blockers sit on receptors in the heart where adrenaline normally docks and block adrenaline from attaching. This calms the heart down—it beats slower and squeezes less forcefully—so the oxygen-starved muscle can survive on the reduced blood supply while doctors work to open the artery.

1. Adrenaline (epinephrine) and noradrenaline (norepinephrine)—the body's fight-or-flight hormones—flood the bloodstream during a heart attack
2. These hormones lock onto beta-1 receptors on heart muscle cells and the SA node (the heart's natural pacemaker)

3. Beta-blocker molecules sit in those receptors like a key stuck in a lock, blocking adrenaline and noradrenaline from attaching
4. The SA node fires more slowly, so heart rate drops (fewer beats per minute means the heart uses less oxygen every minute)
5. Each heartbeat is weaker because the heart muscle contracts with less force (lower contractility)
6. Blood pressure drops because the heart is not pumping as hard and because beta-blockers reduce renin release from the kidneys (less angiotensin II and aldosterone)
7. The blood-starved (ischemic) (oxygen-starved) heart muscle can survive longer on the reduced oxygen supply, and less muscle dies while waiting for the artery to be opened

SAFETY WATCH

Do NOT give if the patient is in cardiogenic shock (blood pressure dangerously low, heart too weak to pump enough blood to the organs) or has severe acute heart failure with fluid backing up into the lungs right now—beta-blockers will make the pump even weaker and can kill the patient · Do NOT give if heart rate is already very slow (below 50–60 beats per minute) or if the heart's electrical system is blocked (second- or third-degree AV block without a pacemaker) · Use very carefully in people with asthma or COPD—non-selective beta-blockers (propranolol, carvedilol) can tighten airways by blocking beta-2 receptors in the lungs; choose a cardioselective beta-1 blocker (metoprolol, atenolol, bisoprolol) if you must use one, but even those can cause problems at high doses · Beta-blockers can mask the warning signs of low blood sugar (hypoglycemia) in diabetics—normally the heart races and you get shaky when sugar drops, but beta-blockers block that signal; warn diabetic patients to check blood sugar more often · Start at a low dose and increase slowly over days to weeks; do NOT stop suddenly (especially after long-term use) because rebound a fast heart rate (tachycardia) (fast heart rate) and high blood pressure can trigger another cardiac event or dangerous arrhythmia · Can cause fatigue, dizziness, cold hands and feet (less blood flow to extremities), and sexual dysfunction in some patients

Atorvastatin, Rosuvastatin, or other High-Intensity Statin · HMG-CoA reductase inhibitor (statin)

Statins lower cholesterol in the blood AND stabilize the cracked plaque inside arteries so it is much less likely to rupture again. The liver makes most of the body's cholesterol using an enzyme called HMG-CoA reductase (the rate-limiting enzyme in cholesterol production). Statins block that enzyme, so the liver makes far less cholesterol. When blood cholesterol drops, the liver pulls LDL cholesterol (the 'bad' kind) out of the bloodstream and out of artery plaques to meet its needs. Over weeks to months, the fatty core of the plaque shrinks. Statins also reduce inflammation inside the plaque (lowering C-reactive protein and other inflammatory markers), which makes the fibrous cap over the plaque thicker and tougher. A

stable plaque with a thick cap is much less likely to crack open and trigger another heart attack.

1. The liver normally makes cholesterol using a multi-step pathway; HMG-CoA reductase is the rate-limiting (slowest, controlling) enzyme in that pathway
2. Statin molecules look like HMG-CoA (the enzyme's natural substrate) and bind to the enzyme's active site, blocking it
3. Cholesterol production inside liver cells drops sharply
4. Liver cells respond by putting more LDL receptors on their surface to grab LDL cholesterol from the blood to meet their needs
5. Blood LDL cholesterol level falls by 40–55% with high-intensity statins (atorvastatin 40–80 mg or rosuvastatin 20–40 mg)
6. With less LDL in the blood, less cholesterol gets deposited into artery plaques, and cholesterol is slowly pulled out of existing plaques
7. Statins also reduce inflammatory signals (like interleukin-6, tumor tissue death (necrosis) factor, and C-reactive protein) inside plaques, which calms down immune cells (macrophages) that make plaques unstable
8. The fibrous cap over the plaque thickens and becomes less likely to tear, and the soft, rupture-prone lipid core shrinks
9. Risk of another rupture and another acute coronary syndrome drops by about 25–30% within weeks to months

SAFETY WATCH

Muscle pain or weakness (myalgia) happens in about 10–15% of patients; rarely (less than 0.1%), severe muscle breakdown (rhabdomyolysis) can occur, releasing muscle protein (myoglobin) into the blood and damaging the kidneys—stop the statin immediately and check creatine kinase (CK) if severe muscle pain, dark urine, or weakness develops · Liver enzyme levels (ALT, AST) can rise mildly; check them before starting and monitor during treatment, but mild elevations (less than 3 times the upper limit of normal) are usually safe and do NOT require stopping the statin unless symptoms (jaundice, severe fatigue) develop · Small increase in new-onset diabetes (about 0.2% absolute risk increase per year), especially in people who are already prediabetic or have metabolic syndrome; the benefit of preventing heart attack and stroke far outweighs this risk · Do NOT combine with certain drugs that block statin breakdown: macrolide antibiotics (clarithromycin, erythromycin), azole antifungals (itraconazole, ketoconazole), HIV protease inhibitors, or gemfibrozil (a fibrate)—these raise statin blood levels dangerously high and increase rhabdomyolysis risk · Grapefruit and grapefruit juice block the enzyme (CYP3A4) that breaks down some statins (atorvastatin, simvastatin, lovastatin), raising blood levels—one glass is usually safe, but avoid drinking large amounts daily; rosuvastatin, pravastatin, and pitavastatin are NOT affected by grapefruit · Rarely causes memory problems or confusion; usually reversible when the drug is stopped, but the link is weak and statins do NOT increase dementia risk long-term

Nitroglycerin · Nitrate vasodilator

Nitroglycerin relaxes and widens blood vessels, especially veins, which reduces how hard the heart has to work. When veins relax, they hold more blood (like widening a hose), so less blood returns to the heart (this is called lowering preload). With less blood filling the heart chambers, the heart does not have to stretch and squeeze as hard, which lowers oxygen demand. Nitroglycerin also relaxes arteries slightly, lowering the pressure the heart has to pump against (called afterload). Both effects together reduce the heart's workload.

Nitroglycerin may also widen coronary arteries (especially if spasm is part of the problem) and improve blood flow to blood-starved (ischemic) areas. It works by releasing nitric oxide inside vessel walls—nitric oxide is a powerful signal that tells smooth muscle in the vessel wall to relax.

1. Nitroglycerin enters the bloodstream (absorbed under the tongue, through the skin from a patch, or given by IV)
2. Inside the smooth muscle cells of vessel walls, nitroglycerin is converted into nitric oxide (NO)
3. Nitric oxide activates an enzyme called guanylate cyclase, which makes a molecule called cyclic GMP (cGMP)

4. Cyclic GMP tells smooth muscle cells to relax by lowering calcium levels inside the cells
5. Veins dilate (widen) significantly, so blood pools in the body's veins instead of returning quickly to the heart
6. With less blood returning to the heart, the heart chambers do not fill as much (lower preload), so each beat requires less stretch and less oxygen
7. Arteries also relax slightly, lowering the pressure the left ventricle has to pump against (lower afterload), which reduces oxygen demand further
8. If coronary artery spasm is contributing to reduced flow, nitroglycerin may relax the spasm and improve oxygen supply to the heart muscle
9. The balance tips: oxygen demand drops significantly, oxygen supply may improve a little, and chest pain (angina) eases within 1–3 minutes

SAFETY WATCH

Do NOT give if systolic blood pressure is below 90 mmHg or if the patient is already with low blood pressure (hypotensive)—nitroglycerin will drop pressure further and can cause shock · Do NOT give if the patient took a phosphodiesterase-5 inhibitor for erectile dysfunction (sildenafil [Viagra], tadalafil [Cialis], vardenafil [Levitra]) in the last 24–48 hours (48 hours for tadalafil because it lasts longer)—the combination causes severe, life-threatening blood pressure drop because both drugs raise nitric oxide or cGMP levels · Do NOT give in right ventricular tissue death from blocked blood flow (infarction) (usually seen with inferior wall STEMI, often with ST elevation in lead V4R) or if jugular veins are distended and the patient has signs of right heart failure—the right ventricle depends heavily on preload to pump blood into the lungs, and dropping preload will collapse cardiac output and blood pressure · Common side effects: severe pounding headache (because blood vessels in the head and face dilate), dizziness or lightheadedness (from blood pressure drop), facial flushing, and nausea · Tolerance (reduced effect) develops quickly if nitroglycerin is used continuously without a break—after about 24 hours of constant IV infusion or wearing a patch all day and night, the drug stops working well; give a nitrate-free period (at least 10–12 hours off per day) to restore effectiveness · Always check blood pressure before giving each dose (especially sublingual tablets or spray); hold the dose if systolic BP is below 100–110 mmHg or if the patient feels dizzy or lightheaded · Sublingual tablets can cause a brief burning sensation under the tongue (that is how you know the tablet is active); spray works the same way and does not expire as quickly as tablets

Alteplase, Tenecteplase, or Reteplase (fibrinolytics / thrombolytics) · Fibrinolytic (clot-busting drug)

Fibrinolytics break up the clot that is blocking the coronary artery by activating the body's natural clot-dissolving system. When a clot forms, fibrin threads weave through it like a net, holding it together. The body has an enzyme called plasmin that cuts fibrin threads and dissolves clots, but plasmin is normally stored in an inactive form called plasminogen. Fibrinolytics convert plasminogen into active plasmin right at the clot, so plasmin chews through the fibrin net and the clot falls apart. Blood flow through the artery resumes, and oxygen reaches the starving heart muscle before too many cells die. This only works if given very early—ideally within 12 hours of symptom onset, but the earlier the better (greatest benefit in the first 2–3 hours).

1. A clot has blocked or nearly blocked a coronary artery, cutting off blood flow to part of the heart muscle
2. Fibrinolytic drug is injected into a vein (IV) and travels through the bloodstream to the clot
3. The drug binds to plasminogen molecules that are stuck to fibrin threads in the clot
4. It converts plasminogen into plasmin, the enzyme that cuts fibrin
5. Plasmin chews through the fibrin net, breaking the clot into smaller pieces that wash away in the bloodstream
6. The artery opens back up, blood flow resumes, and oxygen delivery to the heart muscle is restored
7. If given early enough (within 1–3 hours), most of the heart muscle can be saved

SAFETY WATCH

Major bleeding risk is the biggest danger—fibrinolytics dissolve clots everywhere in the body, not just in the heart, so any recent wound, ulcer, or fragile blood vessel can start bleeding heavily; watch for bleeding at IV sites, gums, urine (red or pink), stools (black or bloody), vomit (bright red or coffee-ground), or sudden severe headache (possible brain bleed) · Do NOT give if the patient has had any stroke ever (ischemic or bleeding-driven (hemorrhagic)), brain tumor, or head injury in the past 3 months, or any surgery or major trauma in the past 2–4 weeks—bleeding risk is too high · Do NOT give if blood pressure is dangerously high (systolic above 180–185 mmHg or diastolic above 110 mmHg) because the risk of bleeding in the brain (hemorrhagic stroke) goes up sharply · Do NOT give if the patient is on blood thinners (warfarin with INR above 1.7) or has a known bleeding disorder (hemophilia, severe liver disease with impaired blood clotting (coagulopathy)) · Do NOT give if the patient had CPR with chest compressions for more than 10 minutes or has suspected aortic dissection (tearing of the main artery)—clot-busting can cause catastrophic bleeding in those situations · Intracranial bleeding (hemorrhage) (bleeding in the brain) happens in about 0.5–1% of patients treated with fibrinolytics and is often fatal; risk is higher in older patients (over 75 years), smaller body size, women, and those with high blood pressure · Allergic reactions (rash, hives, low blood pressure, trouble breathing) can happen, especially with streptokinase (an older fibrinolytic derived from bacteria); tenecteplase and alteplase are less allergenic because they are human-based · Reperfusion arrhythmias (sudden abnormal heart rhythms, including ventricular a fast heart rate (tachycardia) or even ventricular fibrillation) can occur when blood flow suddenly returns to blood-starved (ischemic) tissue; have a defibrillator ready

Enalapril, Lisinopril, Ramipril, or other ACE Inhibitor (or ARB if ACE inhibitor not tolerated) · Angiotensin-converting enzyme (ACE) inhibitor

ACE inhibitors block an enzyme that makes a powerful hormone called angiotensin II, which squeezes blood vessels tight and raises blood pressure. After a heart attack, angiotensin II also triggers harmful remodeling—the heart muscle stretches, thins out, and weakens over weeks to months, raising the risk of heart failure. ACE inhibitors block the enzyme (ACE) that converts angiotensin I into angiotensin II, so angiotensin II levels drop. Blood vessels relax (lowering blood pressure and reducing how hard the heart has to pump), and the harmful remodeling is slowed or prevented. ACE inhibitors also raise levels of bradykinin (a helpful molecule that widens vessels and protects the heart), though this can cause a dry cough in some people. Started early after a heart attack (within 24 hours if the patient is stable), ACE inhibitors reduce the risk of heart failure and death.

1. After a heart attack, damaged heart muscle releases signals that activate the renin-angiotensin-aldosterone system (RAAS)

2. Renin (from the kidneys) converts angiotensinogen (made by the liver) into angiotensin I
3. ACE (mostly in the lungs and blood vessels) converts angiotensin I into angiotensin II
4. Angiotensin II constricts blood vessels (raising blood pressure), tells the adrenal glands to release aldosterone (which makes the kidneys hold onto salt and water), and directly triggers fibrosis and remodeling in the heart
5. ACE inhibitor blocks the ACE enzyme, so angiotensin I cannot be converted into angiotensin II
6. Angiotensin II levels fall, blood vessels relax (lowering afterload), and the kidneys release more salt and water (lowering preload)
7. Harmful remodeling (scar expansion, chamber dilation, wall thinning) slows or stops, preserving heart function
8. Risk of progressive heart failure and death drops by about 20–25% over months to years

SAFETY WATCH

Do NOT give if the patient has ever had angioedema (sudden severe swelling of the lips, tongue, or throat) from an ACE inhibitor before—it can happen again and block the airway, which is life-threatening · Do NOT give in pregnancy (especially second and third trimesters)—ACE inhibitors cause severe birth defects, kidney failure in the fetus, and fetal death; switch to another drug if a woman becomes pregnant · Do NOT give if potassium is already high (hyperkalemia, potassium above 5.5 mEq/L) or if severe kidney failure is present (creatinine above 3 mg/dL or GFR below 30)—ACE inhibitors raise potassium by blocking aldosterone, and high potassium can cause dangerous heart rhythms · Watch for dry, persistent cough (happens in 10–15% of patients) caused by bradykinin buildup—if intolerable, switch to an ARB (angiotensin receptor blocker like losartan or valsartan), which does not raise bradykinin · Check kidney function (creatinine, GFR) and potassium 1–2 weeks after starting or increasing dose—creatinine may rise slightly (up to 30% is acceptable), but if it jumps sharply or potassium climbs above 5.5, hold or reduce the dose · Can cause dizziness or lightheadedness (from blood pressure drop), especially when standing up; start with a low dose and increase slowly · Do NOT combine with potassium supplements, potassium-sparing diuretics (spironolactone, amiloride), or NSAIDs (ibuprofen, naproxen) without close monitoring—these raise potassium even further and increase kidney injury risk

Morphine · Opioid analgesic

Morphine relieves severe chest pain and reduces anxiety during a heart attack. Pain and anxiety both trigger the release of adrenaline and noradrenaline, which make the heart beat faster and harder, raising oxygen demand in muscle that is already starving. Morphine binds to opioid receptors (mostly mu receptors) in the brain and spinal cord, blocking pain signals and

producing a calming effect. It also causes veins to relax and dilate (venodilation), which reduces preload (less blood returning to the heart) and lowers the heart's workload slightly. By easing pain and anxiety, morphine helps bring heart rate and blood pressure down, which can reduce oxygen demand and limit further muscle damage.

1. Severe chest pain from starved blood flow (ischemia) (lack of oxygen) sends pain signals up nerves to the spinal cord and brain
2. Pain and fear trigger the sympathetic nervous system to release adrenaline and noradrenaline, making the heart race and blood pressure climb
3. Morphine is given by IV and quickly crosses into the brain and spinal cord
4. It binds to mu-opioid receptors, blocking pain signals and producing euphoria or calm
5. Pain intensity drops sharply, anxiety eases, and the patient relaxes
6. With less pain and less anxiety, sympathetic nervous system activity falls, so heart rate and blood pressure come down
7. Morphine also causes veins to dilate, reducing the amount of blood returning to the heart (lower preload), which lowers the heart's workload
8. The net effect is less oxygen demand, though morphine does NOT treat the underlying blockage

SAFETY WATCH

Can cause respiratory depression (slow, shallow breathing) at high doses, especially in older adults or if combined with other sedatives (benzodiazepines, alcohol); monitor breathing rate and oxygen saturation closely · Can cause nausea and vomiting in many patients; have an antiemetic (like ondansetron) ready · Lowers blood pressure by dilating veins—check blood pressure before and after each dose; do NOT give if systolic BP is below 90 mmHg or the patient is already with low blood pressure (hypotensive) · Can cause severe itching (histamine release) in some patients, though this is usually not dangerous · Risk of physical dependence and addiction with repeated use, though this is not a concern in the acute setting for a single event · Morphine may delay absorption of oral P2Y12 inhibitors (ticagrelor, prasugrel) because it slows gut motility; some guidelines now recommend using morphine sparingly and only if nitroglycerin and reassurance do not control pain · Reverse severe respiratory depression with naloxone (Narcan) if needed

Red flags

- ST-segment elevation on the EKG—this means a coronary artery is completely blocked (STEMI, ST-elevation heart attack (myocardial infarction)) and the patient needs the artery opened within 90 minutes with a balloon and stent (percutaneous coronary intervention, or PCI) or clot-busting medicine (fibrinolytic therapy)

- Cardiogenic shock—cool clammy skin, blood pressure very low, confusion, almost no urine output—means the heart is failing and cannot pump enough blood to keep the body alive
- New heart murmur heard on exam—suggests a papillary muscle tore (causing the mitral valve to leak, called acute mitral regurgitation) or a hole opened between the pumping chambers (ventricular septal defect, or VSD); both are life-threatening mechanical problems
- Ventricular arrhythmias on the monitor—ventricular a fast heart rate (tachycardia) (V-tach, fast dangerous rhythm from the lower chambers) or ventricular fibrillation (V-fib, heart just quivers and stops pumping)—can stop the heart and cause sudden cardiac arrest
- Right ventricular tissue death from blocked blood flow (infarction) signs—low blood pressure, neck veins bulging (elevated jugular venous pressure, or JVP), lungs clear on exam—needs IV fluids, NOT water pills (diuretics) or nitroglycerin, which will drop pressure even more

Nursing priorities

1. Get a 12-lead ECG within 10 minutes of arrival. — A STEMI is diagnosed on the ECG, and every minute of delay is heart muscle dying.
2. Put the patient on a heart monitor and get IV access right away. — Deadly rhythms like ventricular fibrillation are most likely in the first hours of a heart attack.
3. Give chewed aspirin as ordered — once dissection or bleeding is not suspected. — Chewing gets it working fast. Aspirin stops platelets from making the clot bigger.
4. Draw the first troponin now and repeat it on schedule. — One normal troponin never rules out a heart attack. The trend over hours tells the real story.
5. Treat pain and give nitroglycerin as ordered — hold it if the blood pressure is low, a right-sided MI is suspected, or erection pills were taken. — Nitro relaxes vessels and drops preload. In the wrong patient it can crash the pressure.
6. Give oxygen only if the oxygen level is below about 90%. — Extra oxygen when it is not needed can actually add to heart damage.
7. Keep the patient on bed rest and NPO (nothing to eat or drink) until the plan is clear. — Activity makes the heart work harder, and they may need to go to the cath lab soon.
8. Watch closely for change: new or worse pain, rhythm changes, falling blood pressure, or a new murmur. — These are signs the heart attack is growing or a mechanical complication (like a torn valve muscle) is starting.

Sources

- AHA/ACC Guideline for the Management of Patients With Non-ST-Elevation Acute Coronary Syndromes (2023)
- AHA/ACC STEMI Guideline (2023)
- ESC Guidelines for the Management of Acute Coronary Syndromes (2023)
- American College of Cardiology Clinical Consensus on High-Sensitivity Cardiac Troponin (2021)

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