

Aortic Dissection

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THE STORY IN ONE SENTENCE

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“Aortic dissection is a tear in the aortic wall that splits it into two channels. Without emergency treatment, death risk rises 1–2 percent for every hour of delay.”

Normal anatomy — the body's job

The aortic wall has three layers: the intima (smooth inner lining), media (muscular middle layer), and adventitia (tough outer coat). High-pressure pulsatile blood flow from the left ventricle (main pumping chamber) travels through the intact inner channel. Keep this picture in mind, because every unusual finding in dissection makes sense only as a change from this normal structure.

BODY'S JOB

If you do not know the normal three-layer wall and smooth one-channel flow first, the findings in dissection will seem random instead of connected.

1. Blood leaves the left ventricle through the aortic valve.
2. It enters the ascending aorta, a tube about as wide as a garden hose.
3. The wall has three layers stuck tightly together: intima (inner lining as smooth as wax paper), media (middle muscle that stretches with each pulse), and adventitia (outer coat as tough as canvas).
4. Blood flows in one channel down the center—picture a river in a canyon—and the wall stays solid.

5. Branch arteries peel off the aorta to feed the brain (carotid arteries), arms (subclavian arteries), heart muscle (coronary arteries), kidneys (renal arteries), intestines (mesenteric arteries), and legs (iliac arteries).
6. Each heartbeat sends a pressure wave (like a hammer blow) down the aorta; the elastic media absorbs the force and bounces back, smoothing the flow.
7. When all three layers stay glued together and the inner channel stays open, organs get steady blood flow and the body works normally.

What breaks

Usually one upstream event—a tear in the intima—explains every downstream finding together.

CLINICAL TERM

Chronic uncontrolled high blood pressure (hypertension) (high blood pressure over years) is the most common cause, weakening the media. High pressure is like a hammer hitting the wall thousands of times a day; eventually the muscle fibers fray. Connective-tissue diseases—Marfan syndrome, Ehlers-Danlos vascular type, Loeys-Dietz syndrome—produce abnormal collagen and elastin, making the wall fragile like tissue paper instead of canvas. A bicuspid aortic valve (a valve with two flaps instead of three) creates turbulent flow that slams against the ascending aorta, stressing the wall in one spot. Pregnancy increases stroke volume (the amount of blood pumped per beat) and aortic wall stress, especially in the third trimester and right after delivery. Blunt chest trauma (like a steering-wheel injury in a car crash) can tear the intima by sudden deceleration. Cocaine spikes blood pressure and heart rate acutely, raising wall stress in seconds. Certain genetic mutations (ACTA2, MYH11, TGFBR1, TGFBR2) weaken smooth-muscle cells in the media. Vasculitis (inflammation of blood vessels), especially giant cell arteritis, can weaken the aortic wall. Once the tear forms—the intimal flap peels back like wallpaper—each heartbeat's pressure wave drives blood into the media, splitting the layers and propagating the dissection forward and backward. Read every weird finding—arm blood-pressure difference, pulse deficit, stroke, heart attack (myocardial infarction) (heart attack), belly pain, limb starved blood flow (ischemia) (low blood flow to an arm or leg)—backward from this one broken thing upstream: the intimal tear and false lumen (abnormal second channel inside the wall).

How the body compensates

Vital signs may stay near normal while the body works hard to maintain blood flow (perfusion) (blood flow to organs). Your job is to recognize the trajectory toward crash before it happens.

1. The body raises systemic vascular resistance (constricts small arteries all over) and heart rate to keep blood pressure up and push blood through the compressed true lumen. This is the stress response: the brain detects falling organ blood flow (perfusion) and orders the sympathetic nervous system to squeeze vessels and speed the heart.
2. This compensation can hide how sick the patient is—the 'dangerous middle'—because blood pressure and oxygen saturation may look acceptable even as organs begin to suffer starved blood flow (ischemia) (not enough blood). A patient with a systolic pressure of 130 and a heart rate of 100 may still be heading toward rupture or organ failure.
3. The crash points to watch for are: aortic rupture (death in minutes from massive bleeding), cardiac tamponade (the heart squeezed by fluid around it) (blood fills the pericardial sac and stops the heart from filling—seen in type A dissection and recognized by Beck triad: jugular venous distention, muffled heart sounds, low blood pressure (hypotension)), acute severe aortic regurgitation (backward leak through the aortic valve causing pulmonary edema (fluid flooding the lungs) and shock), or malperfusion syndrome (stroke from blocked carotid artery, heart attack (myocardial infarction) from blocked coronary ostium, mesenteric starved blood flow (ischemia) from blocked superior mesenteric artery causing severe belly pain and bloody stool, acute kidney injury from blocked renal artery, or limb starved blood flow from blocked iliac or femoral artery causing a cold pale pulseless leg).
4. Type A dissection (involving the ascending aorta, which is any part from the valve to the innominate artery) has an in-hospital surgical mortality of about 15–25 percent even with emergency repair. The 2022 ACC/AHA Aortic Disease Guidelines recommend immediate surgery for type A.
5. Without surgery, type A dissection mortality is about 1–2 percent per hour in the first 48 hours, reaching roughly 50 percent by 48 hours and nearly 100 percent by two weeks. This is why every hour counts.
6. Spot the trajectory—rising troponin (sign of heart muscle damage), new neurologic deficit (sign of stroke or spinal-cord starved blood flow (ischemia)), dropping urine output (sign of kidney starved blood flow), worsening lactic acidosis (acid from oxygen-starved tissues) (sign of gut or muscle starved blood flow)—before the patient crashes, because once rupture or tamponade occurs the window to save the patient closes in minutes.
7. Trend vital signs, organ function, and imaging findings over time, not just one snapshot. A blood pressure that starts at 160/90 and drifts down to 110/70 over an hour is a red flag, even though 110/70 looks 'normal' on paper.

8. A stable-appearing patient whose dissection is propagating (seen on serial CT scans) or whose organs are beginning to fail (rising creatinine, new ST changes on ECG, new abdominal pain) is not truly stable. The goal is to catch the trajectory before the crash, not after.

Domino effect

1. Sudden severe tearing or ripping chest pain radiates straight through to the back. — High blood pressure or a fragile wall tears the intima (inner lining). Each heartbeat's pressure wave—like a hammer blow—drives blood into the media (middle layer), literally ripping the layers apart. This stretches and tears pain receptors in the adventitia (outer coat), producing the sensation of tearing or ripping. The pain radiates to the back because the descending aorta lies against the spine, and pain fibers from the chest and back share pathways in the spinal cord.
2. Blood pressure and pulse strength differ between the two arms (or between arms and legs). — As the false lumen expands, it compresses or shears off branch arteries at their origin—such as the subclavian artery (which feeds the arm) or the innominate artery (which splits into the right subclavian and right carotid). Less blood reaches one arm, so the pulse is weaker and the systolic pressure is lower—usually by 20 mm Hg or more. This is called a pulse deficit or blood-pressure differential, and it is a red flag for dissection.
3. End-organ starved blood flow (ischemia) (not enough blood to an organ) appears, depending on which branches the dissection blocks: stroke (carotid artery blocked), heart attack (myocardial infarction) or heart attack (coronary artery ostia blocked), severe abdominal pain (superior mesenteric artery blocked), acute kidney injury (renal artery blocked), or cold pulseless limb (iliac or femoral artery blocked). — The expanding false lumen, the intimal flap, or clot in the false lumen occludes (blocks) branch vessels at their origin, starving downstream organs of oxygen and glucose. Each symptom maps directly to the blocked artery: stroke means carotid or vertebral artery blocked; chest pain and ST elevation mean coronary ostium blocked; belly pain and bloody stool mean mesenteric artery blocked; rising creatinine and no urine mean renal artery blocked; cold pale leg with no pulse means iliac artery blocked.
4. Acute severe aortic regurgitation (backward leak through the aortic valve) produces sudden heart failure and pulmonary edema (fluid flooding the lungs) (fluid in the lungs). — In type A dissection (dissection involving the ascending aorta), the false lumen dilates the aortic root (the part of the aorta right above the valve). This pulls the valve leaflets apart so they cannot close properly. Blood floods backward into the left ventricle every beat—picture a door that will not latch. The ventricle overloads with volume, stretches, and cannot pump it all forward. Pressure backs up into the lungs, forcing fluid out of blood vessels into the air sacs (pulmonary swelling (edema)), and the patient becomes short of breath and may cough up pink frothy coughed-up mucus (sputum).

5. Cardiac tamponade (the heart squeezed by fluid around it) causes jugular venous distention (neck veins bulge), muffled heart sounds, and low blood pressure (hypotension) (low blood pressure)—this triad is called Beck triad. — If the dissection ruptures into the pericardial sac (the sac around the heart), blood fills the space between the sac and the heart. The sac does not stretch much, so even 100–200 mL of blood compresses the ventricles from the outside. The heart cannot fill with blood during diastole (the relaxation phase), so stroke volume (blood pumped per beat) drops. Cardiac output falls, blood pressure drops, and blood backs up into the veins, making the neck veins bulge. Heart sounds are muffled because the blood in the sac dampens the sound. This is a surgical emergency; without immediate pericardiocentesis (draining the sac with a needle) or surgery, the patient dies in minutes.
6. The body tries to compensate by raising heart rate, constricting peripheral vessels (raising systemic vascular resistance), and increasing sympathetic tone—but this higher pressure accelerates propagation of the tear. — Baroreceptors (pressure sensors in the carotid artery and aortic arch) detect falling organ blood flow (perfusion) and trigger the stress response: the brain orders the sympathetic nervous system to release adrenaline, speed the heart, and squeeze small arteries. This raises blood pressure temporarily and pushes blood through the compressed true lumen. But the higher systolic pressure and steeper pulse wave (dP/dt , the rate of pressure rise per beat) literally push the dissection farther along the aorta every heartbeat—like a zipper unzipping. This is why treatment must LOWER blood pressure and heart rate, even though the reflex is to raise them.
7. Without treatment, the dissection propagates, organs fail, and the aorta ruptures—death is nearly certain within hours to days. — The false lumen expands with every beat. More branches get blocked, more organs become blood-starved (ischemic). The adventitia (outer coat) stretches thinner and thinner until it tears, and blood bursts into the chest or belly. Once rupture occurs, the patient bleeds to death faster than a surgeon can open the chest. Even with immediate surgery, mortality is 15–25 percent for type A dissection. Without surgery, 50 percent are dead by 48 hours.
8. Trace it backward: the root cause is the intimal tear, which creates the false lumen, which blocks branches and may rupture—every symptom flows from that single upstream break. — Treatment must address the cause in reverse order. First, control heart rate and blood pressure (beta-blocker first to blunt dP/dt , then vasodilator like nitroprusside to lower systolic pressure to 100–120 mm Hg and heart rate to 60 beats per minute) to stop propagation. Then, surgically repair or cover the entry tear (open repair for type A, endovascular stent graft for complicated type B) to restore one-channel flow and prevent rupture. Every hour of delay raises the risk of death by 1–2 percent, so the mantra is: control pressure, image fast (CT angiography is the gold standard), and get the patient to the operating room or cath lab without delay.

Symptoms · what the patient feels, and why

- Sudden severe tearing or ripping chest pain, often shooting straight through to the back or between the shoulder blades: The inner lining of the aorta (the big artery leaving the heart) tears, and blood rushes into the wall of the artery, peeling the layers apart. This stretches pain nerves in the outer coat. Pain in the front chest means the tear is in the part of the aorta closest to the heart; pain between the shoulder blades means the tear is farther down in the chest.
- Blood pressure or pulse difference of 20 points or more between the two arms (or between an arm and a leg): The blood tunneling inside the artery wall squeezes or blocks a branch artery (like the one going to the arm). Less blood reaches that arm, so the pressure reading is lower on that side.
- Fainting (Syncope) (sudden fainting—you black out for a few seconds): Blood can fill the sac around the heart (tamponade), the heart valve can suddenly leak badly, or the aorta can rupture. Any of these drops the heart's pumping power so fast that the brain runs out of oxygen and you lose consciousness.
- One-sided weakness, trouble speaking, or sudden vision loss (stroke signs): The tear reaches into or pinches shut an artery that feeds the brain (carotid or vertebral artery). Without blood, that part of the brain stops working. Which side or function is lost depends on which artery is blocked.
- Sudden trouble breathing, crackling sounds in the lungs, and low oxygen (acute heart failure): The aortic valve starts leaking a lot of extra blood back into the heart's main pumping chamber every beat. The chamber can't handle the flood, so pressure backs up into the lungs and fluid leaks into the air sacs, making it hard to breathe.
- Severe constant belly pain, often with a high lactate level in the blood: The dissection blocks the arteries that feed the intestines (celiac or superior mesenteric artery). The gut runs out of oxygen and starts dying. Dying cells switch to a backup energy system that makes lactate, which piles up in the blood.
- Very little urine (less than half a milliliter per kilogram of body weight per hour) or a rising creatinine (kidney-injury marker): The renal arteries (kidney arteries) get blocked or overall blood flow drops, so the kidneys' filters don't get enough pressure to clean the blood and make urine. Waste products like creatinine build up.
- Cold, pale leg with no pulse (acute limb starved blood flow (ischemia)): The dissection travels down into or squeezes an artery to the leg (iliac or femoral artery), shutting off blood flow. Without oxygen, muscle and nerve tissue start to die within a few hours.
- Widened shadow in the middle of the chest on an X-ray (mediastinum wider than 8 centimeters): Blood filling the false tunnel inside the aorta wall—and any bleeding around the outside of the aorta—makes the aorta look fatter on the X-ray. This is a clue but not proof by itself.

- New high-pitched blowing heart murmur heard along the left edge of the breastbone during the resting phase of the heartbeat (aortic regurgitation): The dissection stretches the base of the aorta where the valve sits, so the valve flaps no longer seal tight. Blood leaks backward through the valve during the heart's resting phase, making the blowing sound.

Focused exam

- You measure blood pressure in both arms and find a difference greater than 20 mmHg systolic, or you feel pulses that are weaker or missing on one side compared to the other: A blood pressure difference between arms or unequal pulses means the dissection (tear inside the aorta wall) is blocking blood flow into one of the branch arteries that supply the arm, confirming that the aorta itself is damaged.
- You hear a new diastolic murmur (soft blowing sound during the resting phase of the heartbeat) best heard along the left sternal border: A new diastolic murmur in this setting tells you the dissection tore backward toward the aortic valve and made the valve leak blood backward into the heart during the resting phase, called aortic regurgitation—this can quickly overload the heart and cause failure.
- You check mental status, ask about chest pain that feels like a heart attack, check the belly for severe pain, and feel for cold or pulseless legs: These signs tell you if the dissection has blocked branch arteries feeding the brain, heart, intestines, or legs—knowing which organs are losing blood supply helps decide if surgery is needed right away or if medicine alone can work.
- You examine the neck veins for distention (bulging) and listen for muffled heart sounds: Bulging neck veins and muffled heart sounds mean blood is leaking into the sac around the heart (pericardial fluid collection (effusion)), squeezing the heart so it cannot fill—this is called tamponade and means the aorta may rupture any moment.
- You check pupils for size and symmetry, test arm and leg strength on both sides, and ask the patient to follow simple commands: Unequal pupils, one-sided weakness, or confusion mean the dissection has cut off blood to part of the brain (stroke) by blocking a carotid or vertebral artery—this changes how urgently surgery must happen and what the surgeon must repair.

Diagnostics · every test answers a question

- CT angiography of the chest, abdomen, and pelvis with intravenous contrast → Where is the tear in the aorta's inner lining, does it involve the ascending aorta (the first part leaving the heart, called Type A) or only the descending aorta (the part going down the chest and belly, called Type B), and are any branch vessels blocked by the false channel?

- Transthoracic echocardiography (ultrasound of the heart through the chest wall) or transesophageal echocardiography (ultrasound probe passed into the esophagus behind the heart) → Is the ascending aorta involved, is the aortic valve leaking backward, and is there fluid collecting around the heart that could squeeze it?
- D-dimer blood test → Is there evidence of abnormal clotting activity inside the blood vessels that raises suspicion for aortic dissection?
- Electrocardiogram (ECG or EKG) → Is there evidence of a heart attack caused by the dissection blocking a coronary artery, or is the chest pain coming from the aorta itself?
- Chest X-ray (posteroanterior and lateral views) → Is the mediastinum (the space in the middle of the chest containing the aorta and heart) widened, suggesting aortic pathology?
- Serum creatinine and blood urea nitrogen (BUN) → Are the kidneys losing function because the dissection is blocking one or both renal arteries?
- Serum lactate → Are the intestines or other organs switching to anaerobic metabolism because they are not receiving enough oxygen?

Treatment logic

- Intravenous esmolol or labetalol to reduce heart rate to 60 beats per minute or lower → Slowing the heart rate reduces the speed and force of each heartbeat's pressure wave traveling down the aorta—think of it like turning down the force of water hammering through a cracked pipe. This decreases mechanical stress on the torn inner lining and stops the tear from unzipping further with each heartbeat.
- Intravenous nicardipine, clevidipine, or nitroprusside (after beta-blocker is started) to lower systolic blood pressure to 100–120 mmHg → Lowering blood pressure reduces wall tension in the aorta—imagine lowering the water pressure in a cracked pipe so it does not burst. This limits shear stress on the torn lining and prevents the false channel from expanding. Always give a beta-blocker FIRST; giving a vasodilator alone can cause reflex a fast heart rate (tachycardia) (the heart speeds up in response to lower pressure), which worsens the dissection.
- Intravenous morphine or fentanyl for pain control → Controlling severe pain shuts down the body's stress response (sympathetic nervous system activation), which prevents surges of adrenaline and noradrenaline that would otherwise spike heart rate and blood pressure and make the dissection worse.

- Emergency open surgical repair (sternotomy and cardiopulmonary bypass) for Stanford Type A dissection (any involvement of the ascending aorta) → The surgeon removes the torn section of the ascending aorta and replaces it with a synthetic fabric tube (Dacron graft), then reconnects the graft to the remaining healthy aorta. This removes the entry tear, restores a single true channel for blood flow, prevents rupture into the pericardium (which causes instant death from tamponade), and can restore flow to blocked branch arteries. If the aortic valve is leaking badly, the surgeon repairs or replaces it during the same operation.
- Thoracic endovascular aortic repair (TEVAR) for complicated Type B dissection (descending aorta only) with malperfusion, uncontrolled pain, uncontrolled blood pressure, or enlarging aortic diameter → A vascular surgeon threads a stent-graft (a metal mesh tube covered in fabric) through the femoral artery in the groin up into the descending aorta and deploys it to cover the entry tear. The stent-graft redirects all blood flow into the true channel, which decompresses the false channel and can restore flow to blocked branch vessels. This prevents rupture and stops the dissection from extending. Uncomplicated Type B dissections (no malperfusion, pain controlled with medicine, blood pressure controlled) are managed with medicine alone and do NOT need TEVAR acutely.
- Long-term beta-blocker therapy (metoprolol, atenolol, or carvedilol) plus additional blood pressure medicines (ACE inhibitors such as lisinopril, or angiotensin receptor blockers such as losartan) targeting blood pressure less than 120/80 mmHg, with serial CT or MRI imaging every 3–6 months for the first year, then every 6–12 months → Chronic aggressive blood pressure control keeps mechanical stress on the aortic wall low, which prevents the false channel from slowly ballooning into an aneurysm and reduces risk of late rupture or a new dissection. Serial imaging catches complications early—if the aorta expands beyond 5.5 cm in the descending thoracic segment or 6 cm in the abdominal segment, the patient needs elective surgical or endovascular repair before rupture occurs.
- Avoid aortic valve replacement with a mechanical valve if the native valve can be repaired or if a tissue valve can be used instead, especially in patients with ongoing dissection in the descending aorta → Mechanical valves require lifelong warfarin (a blood thinner), which increases bleeding risk in patients with a false channel that could rupture. Repairing the native valve or using a tissue valve avoids the need for warfarin and reduces the risk of catastrophic bleeding if the aorta tears again.

Medications

Esmolol · Intravenous beta-blocker (short-acting)

Esmolol blocks tiny doorways on heart muscle cells called beta-receptors. When these doorways are blocked, the heart beats slower and with less force. This matters because every heartbeat pushes a wave of pressure through the aorta—like a hammer hitting the tear with

every beat. Slowing the heart down to under 60 beats per minute cuts the number of hammer-blows per minute and reduces the shearing force (the sideways tearing pressure) that tries to rip the torn wall further open.

1. The drug enters the bloodstream through an IV and travels to the heart.
2. It attaches to beta-1 receptors on heart cells, blocking adrenaline (the body's natural 'speed-up' signal) from binding.
3. With adrenaline blocked, the heart's pacemaker slows down—fewer electrical pulses fire per minute.
4. The ventricles contract less forcefully and less often.
5. Each heartbeat delivers a gentler pressure wave into the torn aorta, reducing the risk the tear will grow longer or deeper.

SAFETY WATCH

Can drop heart rate too low (bradycardia)—monitor every 5 minutes; esmolol wears off in about 9 minutes if stopped, so it is safe to adjust quickly. · Can lower blood pressure too much if given before heart rate is controlled—always reduce heart rate FIRST. · Avoid in severe asthma or certain heart-block conditions (the heart's electrical system is already too slow). · Watch for heart failure signs if the heart is already weak—beta-blockers reduce the heart's squeezing power.

Labetalol · Intravenous beta-blocker with mild alpha-blocking effect

Labetalol blocks two types of doorways on cells: beta-receptors (on the heart) and alpha-receptors (on small arteries). Blocking beta-receptors slows the heart and softens its squeeze. Blocking alpha-receptors relaxes the muscle rings around arteries, widening them slightly so blood flows with less pressure. Together, this lowers both heart rate and blood pressure in one shot, reducing the tearing force inside the aorta from both angles—fewer beats per minute and less pressure per beat.

1. Labetalol is given through an IV and spreads through the bloodstream.
2. It blocks beta-receptors on the heart, slowing the pulse and weakening each contraction.
3. It also blocks alpha-receptors on artery walls, telling the muscle rings to relax.
4. Arteries widen a little, so the same volume of blood flows at lower pressure.
5. The combination drops the hammering force on the torn aortic wall—both the rate of blows and the strength of each blow fall.

SAFETY WATCH

Can cause severe low blood pressure (hypotension) if given too fast—titrate the dose carefully and check blood pressure every 5–10 minutes. · Avoid in severe asthma, heart block, or severe heart failure. · Can mask the warning signs of low blood sugar in diabetic patients (because beta-blockers blunt the fast heartbeat and shakiness that usually signal low glucose). · Watch for dizziness or fainting when the patient tries to sit up—blood pressure may drop when standing.

Nicardipine · Intravenous calcium-channel blocker (vasodilator)

Nicardipine blocks tiny calcium gates on the muscle cells that wrap around arteries. Muscle cells need calcium to squeeze; without it, they relax. When artery walls relax, the vessel opens wider—like loosening a belt—and blood flows at lower pressure. This directly lowers systolic blood pressure (the peak pressure with each heartbeat) to the safe target of 100–120 mmHg, taking stress off the torn aortic wall. Critical rule: give nicardipine ONLY AFTER the heart rate is under control with a beta-blocker, because dropping pressure alone can make the heart beat faster and harder (reflex a fast heart rate (tachycardia)), which would increase shear force and worsen the tear.

1. Nicardipine is infused into a vein and travels to artery walls throughout the body.
2. It plugs the calcium channels, blocking calcium from entering muscle cells.
3. Without calcium, the muscle cells cannot contract; they stay relaxed.
4. Arteries widen, resistance to blood flow drops, and blood pressure falls.
5. The peak pressure hitting the torn aorta with each heartbeat is now lower, reducing the force trying to extend the tear.

SAFETY WATCH

NEVER give before heart rate is controlled—reflex a fast heart rate (tachycardia) will worsen shear stress and can propagate the tear. · Monitor blood pressure continuously (every 2–5 minutes)—nicardipine acts fast and can drop pressure too low. · Can cause headache, flushing, or swelling in the ankles (from dilated vessels leaking a little fluid). · Use cautiously in patients with severe aortic narrowing (stenosis) (a tight aortic valve) because dropping pressure too much can reduce blood flow to vital organs.

Sodium Nitroprusside · Intravenous nitrovasodilator

Nitroprusside releases nitric oxide inside blood-vessel walls. Nitric oxide is a natural signal that tells smooth muscle to relax. When both arteries and veins relax, blood pressure drops fast—arteries widen (less resistance) and veins pool more blood (less blood returning to the heart). This powerful, instant drop in systolic blood pressure takes mechanical stress off the torn

aorta. But nitroprusside can cause reflex a fast heart rate (tachycardia) (the heart speeds up to compensate for low pressure), so it must ALWAYS be paired with a beta-blocker given first. It is reserved for cases where nicardipine is not available or not working well enough.

1. Nitroprusside is given by continuous IV infusion.
2. Inside blood-vessel cells, the drug breaks down and releases nitric oxide (NO).
3. NO activates guanylate cyclase, which makes a molecule called cGMP.
4. cGMP tells the muscle fibers to relax.
5. Arteries widen, veins expand, pressure drops immediately, and the force on the dissected aorta falls.

SAFETY WATCH

MUST be given AFTER beta-blockade is established—never first-line alone. · Monitor blood pressure every 1–2 minutes during titration—drops pressure very fast. · Toxic if used longer than 48–72 hours at high doses: the drug breaks down into cyanide, then thiocyanate, which can poison the nervous system and cause confusion, seizures, or lactic acidosis (acid from oxygen-starved tissues). Check thiocyanate levels if infusion lasts beyond 48 hours. · Protect the IV bag from light—nitroprusside degrades when exposed to bright light, turning the solution brown and ineffective. · Use cautiously in kidney failure (thiocyanate builds up) and avoid in severe liver disease.

Morphine · Intravenous opioid analgesic

Morphine binds to opioid receptors in the brain and spinal cord—tiny docking stations that control pain signals and emotional distress. When morphine locks onto these receptors, it blocks pain messages from reaching the conscious brain and calms the fear and panic that come with severe chest or back pain. This matters in aortic dissection because pain and anxiety both trigger adrenaline release, which speeds up the heart and raises blood pressure—exactly what we are trying to avoid. By relieving pain, morphine indirectly helps keep heart rate and blood pressure from spiking, and it keeps the patient calm enough to cooperate with emergency treatment.

1. Morphine is injected into a vein and quickly crosses into the brain and spinal cord.
2. It attaches to opioid receptors on nerve cells that carry pain signals.
3. These receptors close the gate on pain messages—fewer signals make it up to the conscious brain.
4. The emotional centers in the brain (limbic system) also calm down, reducing fear and the adrenaline surge that comes with it.
5. With less pain and anxiety, the body's stress response quiets, heart rate stays lower, and blood pressure is easier to control.

SAFETY WATCH

Can slow breathing (respiratory depression)—watch respiratory rate and oxygen saturation, especially in elderly patients or those with lung disease. Have naloxone (opioid reversal drug) ready. · Can drop blood pressure if given as a large, fast bolus—give slowly in small doses (2–4 mg IV every 5–10 minutes) and titrate to pain relief. · Can cause nausea, vomiting, or severe itching. · Can cloud thinking or cause sedation—helpful for calming panic, but watch for over-sedation that makes it hard to assess the patient's mental status or detect worsening symptoms. · Use cautiously in patients with low blood pressure or shock—morphine can worsen low blood pressure (hypotension).

Metoprolol · Oral beta-blocker (long-term maintenance)

Metoprolol blocks beta-1 receptors on heart cells, just like esmolol or labetalol, but it is a pill taken by mouth for long-term daily use—not an emergency IV drug. After the acute dissection is stabilized (by IV drugs and often surgery), the patient must stay on a beta-blocker for life to keep heart rate low (goal under 60–70 beats per minute) and reduce the force of each heartbeat. This protects any remaining weak spots in the aorta and prevents new tears or expansion of the repaired area over months and years. Metoprolol is the workhorse for chronic control because it is inexpensive, well-studied, and easy to dose once or twice a day.

1. The patient swallows a metoprolol tablet; it is absorbed in the small intestine.
2. The drug enters the bloodstream and travels to the heart.
3. It blocks beta-1 receptors, preventing adrenaline from speeding up or strengthening the heartbeat.
4. Heart rate stays low and contraction force stays gentle, day in and day out.
5. Over months and years, the aorta is protected from repeated high-pressure waves, reducing the risk of re-dissection, aneurysm growth, or rupture.

SAFETY WATCH

Can cause fatigue, dizziness, or cold hands and feet (from slower circulation). · Can mask low blood sugar symptoms in diabetics. · Never stop suddenly—rebound a fast heart rate (tachycardia) and high blood pressure can occur, stressing the aorta and risking another event. Taper the dose slowly if discontinuation is needed. · Avoid in severe asthma, a slow heart rate (bradycardia), or advanced heart block. · Monitor heart rate and blood pressure at every follow-up visit; adjust dose to keep resting heart rate 60–70 bpm and systolic BP under 120 mmHg.

Losartan (or other ARB) / Enalapril (or other ACE inhibitor) · Angiotensin receptor blocker (ARB) or ACE inhibitor (oral, long-term)

These drugs block a hormone system called the renin-angiotensin system, which normally squeezes arteries and raises blood pressure. Losartan (an ARB) blocks the receptors where angiotensin II (a powerful vessel-constrictor) would attach; enalapril (an ACE inhibitor) stops angiotensin II from forming in the first place. Either way, arteries stay relaxed, blood pressure stays low, and the daily stress on the aorta is reduced. In patients with connective-tissue disorders (like Marfan or Loeys-Dietz syndrome), ARBs may also slow aortic-root enlargement by reducing inflammation and abnormal signaling in the vessel wall. These drugs are added to beta-blockers for long-term blood pressure control, with a goal systolic BP under 120 mmHg.

1. The patient takes the pill daily; it is absorbed from the gut.
2. ARB (losartan): travels to artery walls and kidneys, blocks angiotensin II receptors, so the squeezing signal never gets through—arteries stay open, blood pressure stays lower.
3. ACE inhibitor (enalapril): blocks the enzyme that converts angiotensin I into angiotensin II, so less of the squeezing hormone is made—same result, arteries relax, pressure drops.
4. Over weeks and months, steady lower blood pressure reduces mechanical strain on the aorta.
5. In genetic conditions, blocking angiotensin signaling may also reduce harmful remodeling of the aortic wall.

SAFETY WATCH

Can cause high potassium (hyperkalemia), especially if combined with potassium supplements, potassium-sparing diuretics, or in patients with kidney disease—check potassium and creatinine regularly. · ACE inhibitors can cause a dry, persistent cough (from buildup of bradykinin); switch to an ARB if this happens. · Rarely, ACE inhibitors cause severe tongue/throat swelling (angioedema)—stop immediately and switch to an ARB. · Avoid in pregnancy—both classes can harm the developing baby's kidneys and skull. · Can worsen kidney function if both kidney arteries are narrowed (renal artery narrowing (stenosis))—monitor creatinine after starting. · Can cause dizziness or lightheadedness from low blood pressure, especially when combined with beta-blockers; educate patients to stand up slowly.

Red flags

- Sudden severe tearing chest or back pain with a blood-pressure difference of 20 points or more between the arms
- Fainting, new one-sided weakness, trouble speaking, or stroke signs in someone with chest pain

- Sudden heart failure, very low blood pressure, or signs that fluid is squeezing the heart (muffled heart sounds, neck veins bulging, low blood pressure) along with chest pain
- Severe belly pain with a high lactate level or signs the intestines are dying
- Cold leg with no pulse, or sudden kidney injury, in someone with sudden chest pain

Nursing priorities

1. Obtain blood pressure readings in both arms and document the difference; notify the provider immediately if there is a 20 mmHg or greater difference between arms. — The false channel can compress or block the subclavian artery (the vessel feeding one arm), creating a pressure difference; this finding strongly suggests dissection and tells the surgical team which side of the aorta is involved, guiding the repair approach.
2. Place the patient on continuous cardiac monitoring and pulse oximetry; assess heart rate every 5 to 15 minutes and alert the provider if heart rate rises above 60 beats per minute after beta-blocker therapy starts. — Every heartbeat creates a pressure wave that pushes against the torn aorta and can extend the dissection further; keeping heart rate at 60 or below reduces this hydraulic stress (called dP/dT , or the force of each heartbeat) and prevents the tear from ripping longer.
3. Start two large-bore intravenous lines (18-gauge or larger) and prepare for intravenous esmolol or labetalol infusion; do not give any blood pressure medicine until the beta-blocker is running. — Beta-blockers must start first because if you lower blood pressure without slowing the heart, the heart will beat harder and faster to compensate, increasing the force on the torn aorta; this reflex a fast heart rate (tachycardia) (fast heart rate) can make the dissection extend and rupture.
4. Administer intravenous morphine or fentanyl as ordered for pain control; reassess pain level every 15 to 30 minutes using a 0-to-10 scale. — Severe pain triggers the sympathetic nervous system (the fight-or-flight response), which releases adrenaline and raises both heart rate and blood pressure; controlling pain reduces this stress response and helps keep the aorta stable while the team arranges surgery or imaging.
5. Keep the patient flat or with the head of bed elevated no more than 30 degrees; instruct the patient to avoid straining, coughing hard, or bearing down (Valsalva maneuver). — Straining, coughing, and bearing down all increase intra-thoracic pressure (pressure inside the chest), which translates directly to pressure on the aorta wall; even a small pressure spike can extend the dissection or cause the aorta to rupture into the chest cavity.

6. Monitor for signs of cardiac tamponade (the heart squeezed by fluid around it) every 15 minutes: muffled or distant heart sounds, jugular venous distension (bulging neck veins when sitting up 30 degrees), and narrowing pulse pressure (the gap between systolic and diastolic blood pressure shrinking to less than 25 mmHg). — Type A dissection can rupture through the aorta wall into the pericardial sac (the thin bag around the heart), filling it with blood; this squeezes the heart so it cannot fill with blood between beats, causing shock and cardiac arrest within minutes if not drained immediately.
7. Perform a focused neurologic assessment every 30 minutes: check arm and leg strength, speech clarity, facial symmetry, and pupil size and reaction; report any new weakness, slurred speech, or unequal pupils immediately. — The dissection can block the carotid arteries or the arteries feeding the spinal cord, causing stroke or paralysis; catching these changes early allows the surgical team to adjust the repair technique to restore blood flow to the brain and spinal cord before permanent damage occurs.
8. Prepare the patient and family for emergency surgery or transfer to a cardiac surgery center; explain that Type A dissections (involving the aorta near the heart) require open-heart surgery within hours, while Type B dissections (starting after the arch) are often managed with medicine first. — Type A dissections have a 1 to 2 percent mortality rate per hour without surgery because they can rupture, cause tamponade, or block coronary arteries; families need to understand the urgency so they can make informed decisions quickly, and early preparation reduces door-to-operating-room time.

Sources

- ESC Guidelines on the Diagnosis and Treatment of Aortic Diseases 2024
- ACC/AHA Guideline for the Diagnosis and Management of Aortic Disease 2022
- Society for Vascular Surgery / Society of Thoracic Surgeons Practice Guidelines
- Cochrane systematic reviews on aortic dissection management

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