

Aortic Dissection

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.

THE ONE-LINE TRUTH

A tear in the innermost layer of the aorta (the smooth lining) allows blood to split the wall into two channels—one true, one false—and that false channel can block the arteries feeding your brain, heart, kidneys, intestines, and legs.

The chain — how it unfolds

1. Sudden severe tearing or ripping chest pain radiates straight through to the back. — why: 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). — why: 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). — why: 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). — why: 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. — why: 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. — why: 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. — why: 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. — why: 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.

Spot it — what you see, 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.

Red flags — act now

- 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

Treat it — in this order

1. 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.

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

Your top priorities at the bedside

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. — why: 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. — why: 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. — why: 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.