

Aortic Dissection

A plain-English guide for you and your family

WHAT IS HAPPENING

Your aorta is the main blood vessel leaving your heart. Its wall has three layers: a smooth inner lining (intima), a muscular middle layer (media), and a tough outer coat (adventitia). Normally, blood flows smoothly through the center. In an aortic dissection, a tear forms in the inner lining. With every heartbeat, the pressure forces blood into the wall itself, splitting the layers apart. This creates a second false channel that can expand and block the branches that feed your brain, heart, kidneys, intestines, and legs. The most common cause is years of high blood pressure that was not controlled; over time, high pressure weakens the wall. Diseases that affect your body's connective tissue—like Marfan syndrome or the vascular type of Ehlers-Danlos syndrome—make the wall fragile. A bicuspid aortic valve (a valve with two flaps instead of three), pregnancy, a hard blow to the chest, and cocaine use also raise your risk. The hallmark is sudden, severe chest pain that feels like tearing or ripping and often shoots straight through to your back. Your pulse and blood pressure may be different in your two arms. Depending on which arteries get blocked, you might have a stroke, a heart attack, severe belly pain, kidney failure, or a cold pale leg. Dissections near the heart (type A) need emergency surgery. Dissections farther away (type B) are often managed first with medicines that lower blood pressure and heart rate, then surgery if problems appear. Every hour without treatment raises the chance of rupture, shock, or organ death—so if you feel sudden tearing chest or back pain, call 911 right away.

What you might feel

- Sudden severe tearing or ripping chest pain, often shooting straight through to the back or between the shoulder blades
- Blood pressure or pulse difference of 20 points or more between the two arms (or between an arm and a leg)
- Fainting (Syncope) (sudden fainting—you black out for a few seconds)
- One-sided weakness, trouble speaking, or sudden vision loss (stroke signs)
- Sudden trouble breathing, crackling sounds in the lungs, and low oxygen (acute heart failure)

- Severe constant belly pain, often with a high lactate level 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)
- Cold, pale leg with no pulse (acute limb starved blood flow (ischemia))
- Widened shadow in the middle of the chest on an X-ray (mediastinum wider than 8 centimeters)
- New high-pitched blowing heart murmur heard along the left edge of the breastbone during the resting phase of the heartbeat (aortic regurgitation)

Call the doctor right away if...

- 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

What the care team will do

- 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.
- 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.
- 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.
- Administer intravenous morphine or fentanyl as ordered for pain control; reassess pain level every 15 to 30 minutes using a 0-to-10 scale.
- 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).
- 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).

- 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.
- 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.

Medicines you may be given — and why

Esmolol

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.

Labetalol

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.

Nicardipine

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.

Sodium Nitroprusside

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.

Morphine

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.

Metoprolol

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.

Losartan (or other ARB) / Enalapril (or other ACE inhibitor)

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.

Where this information comes from

- 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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