Pulmonary Embolism

A plain-English guide for you and your family

WHAT IS HAPPENING

Your body has two sides to its heart. The right side takes used blood from your body and pumps it gently through your lungs to pick up fresh oxygen. That trip to the lungs should be an easy, low-pressure journey. But when a blood clot (usually from a deep vein in your leg or pelvis) breaks free and travels upstream, it gets stuck in the lung arteries and blocks the flow. Now the right side of your heart suddenly has to push against a wall instead of an open road. If the clot is big enough, the right heart swells, pumps weakly, and even squeezes into the left side's space. That means less blood gets out to your body. Your blood pressure drops. Your heart races to try to keep up. You feel short of breath because parts of your lungs are getting air but no blood flow to carry oxygen away — that air is wasted. Your oxygen level falls. None of these scary signs is the actual problem; each one is your body scrambling to survive while the clot chokes off flow. The real danger is that the right side of your heart can fail completely if the clot isn't cleared in time.

What you might feel

- Sudden shortness of breath, often with sharp chest pain when breathing in (pleuritic chest pain)
- Fast heart rate (tachycardia)
- Fast breathing (tachypnea)
- Low oxygen in the blood (hypoxemia)
- Coughing up blood (hemoptysis)
- Swelling or pain in one leg (often the calf or thigh)
- Feeling lightheaded or fainting (syncope)
- Very low blood pressure (hypotension) or near-cardiac arrest (massive PE)
- Signs of right heart strain on tests (elevated troponin or BNP, right ventricle looks enlarged on ultrasound) without low blood pressure (submassive PE)

Call the doctor right away if...

- Sudden collapse, very low blood pressure, or loss of consciousness (signs of massive PE that can stop the heart)
- Coughing up blood (may mean part of the lung is dying from lack of oxygen)
- Chest pain with severe shortness of breath that gets worse quickly (clot may be large or growing)
- Heart strain markers on tests (high troponin or BNP, right ventricle dysfunction on echo) even without low blood pressure (submassive PE needs close monitoring and may need clot-busting treatment)

What the care team will do

- Apply oxygen by nasal cannula or non-rebreather mask to keep SpO2 above 90%.
- Establish large-bore IV access (18-gauge or larger) and prepare for immediate anticoagulation with IV unfractionated heparin or subcutaneous LMWH (low-molecular-weight heparin).
- Place patient on continuous cardiac monitoring and measure vital signs every 15 minutes if unstable or every 1 hour if stable.
- Keep patient on strict bed rest with head of bed elevated 30 to 45 degrees and avoid Valsalva maneuvers (straining, bearing down).
- Draw blood for D-dimer (a clot breakdown product), troponin (a heart enzyme), BNP (brain natriuretic peptide, a heart-strain marker), and CBC (complete blood count) before starting anticoagulation if time allows.
- Assess for signs of active bleeding—check all IV sites, gums, urine color, and stool—and ask about recent surgeries or head trauma in the past 3 months.
- Notify provider immediately if systolic blood pressure drops below 90 mmHg or patient becomes confused, drenched in sweat (diaphoretic) (sweaty), or has chest pain.
- Measure bilateral calf circumference and inspect legs for unilateral swelling, warmth, redness (erythema) (redness), or cord-like veins.

Medicines you may be given — and why

Unfractionated heparin (UFH)

Heparin stops clots from growing bigger and prevents new clots from forming. It does NOT dissolve clots that are already there — it just freezes the problem in place so the body can slowly break down the existing clot on its own. Heparin works by turbocharging a natural brake called antithrombin III (a protein that puts the brakes on clotting). When heparin binds

to antithrombin, it makes antithrombin thousands of times faster at blocking thrombin (the enzyme that cements clots together) and factor Xa (an earlier step in the clotting chain). With those steps blocked, the blood stops trying to clot in the wrong places.

Low-molecular-weight heparin (LMWH): enoxaparin, dalteparin

LMWH is a refined, shorter version of regular heparin. It works the same way — turbocharging antithrombin III to block clotting enzymes — but it focuses more on factor Xa (an earlier step in the clotting chain) and less on thrombin. This makes the drug more predictable: you can dose it by weight, inject it under the skin once or twice a day, and usually skip daily blood tests. Like regular heparin, LMWH stops clots from growing and prevents new ones, but it does NOT dissolve clots already stuck in the lung.

Direct oral anticoagulants (DOACs): rivaroxaban, apixaban, edoxaban, dabigatran

DOACs are pills that directly block one specific clotting enzyme — either factor Xa (rivaroxaban, apixaban, edoxaban) or thrombin (dabigatran). Unlike heparin, which needs a helper protein (antithrombin), DOACs latch directly onto the target enzyme and shut it down. This stops new clots from forming and keeps the existing clot from growing. DOACs work fast (within hours), are dosed the same for everyone (no daily blood tests in most cases), and are taken by mouth. Like all anticoagulants, they do NOT dissolve clots — the body still has to clean up the blockage on its own.

Warfarin (Coumadin)

Warfarin blocks the liver from recycling vitamin K, a nutrient the liver needs to make several clotting factors (proteins II, VII, IX, and X). Without enough of these factors, the blood clots more slowly. Warfarin takes several days to work because the liver has to run out of the clotting factors it already made. Like other anticoagulants, warfarin stops clots from growing and prevents new ones, but it does NOT dissolve clots already stuck in the lung. Warfarin requires regular blood tests (INR) to keep the dose in the safe and effective range.

Alteplase (tPA, tissue plasminogen activator)

Alteplase is a powerful enzyme that actually breaks up clots. It activates plasminogen (an inactive protein floating in the blood) and turns it into plasmin (the body's natural clot-chewing enzyme). Plasmin chews through the fibrin mesh that holds the clot together, dissolving the blockage in the lung. This can save the life of a patient whose blood pressure is crashing or whose heart has stopped because the clot is so massive that the right side of the heart is failing. Alteplase works fast — within minutes to hours — but it comes with a high risk of serious bleeding because it dissolves clots everywhere in the body, including clots that are supposed to be there (like a healing wound or recent surgery site).

Catheter-directed thrombolysis or mechanical thrombectomy

This is a procedure, not a medicine you swallow or inject into a vein. A doctor threads a thin tube (catheter) through a vein in the leg or neck and guides it to the clot in the lung using X-ray. Once the catheter is in place, the doctor can either deliver a clot-busting drug (like alteplase) directly into the clot (catheter-directed thrombolysis) or use a special device to physically break up or vacuum out the clot (mechanical thrombectomy). This targets the clot more precisely than giving alteplase through a regular IV, so it may lower the risk of bleeding elsewhere in the body. It is used for patients with submassive PE (right heart strain but blood pressure is still okay) when the risk of full-dose systemic thrombolysis is too high, or when systemic thrombolysis has failed.

Inferior vena cava (IVC) filter

An IVC filter is a small metal cage placed inside the inferior vena cava (the large vein that carries blood from the lower body back to the heart). The filter acts like a net, catching any clots that break off from the leg veins before they can travel to the lungs. It does NOT dissolve clots, prevent clots from forming, or treat the PE that is already there — it only blocks new clots from reaching the lungs. IVC filters are used ONLY when anticoagulation (blood thinners) is absolutely contraindicated (for example, active bleeding or very recent surgery) and the patient is at high risk of another PE. Filters should be removed as soon as anticoagulation becomes safe again, because long-term filters can cause complications (the filter itself can trap clots, break, or migrate).

Where this information comes from

- ESC Guidelines for the Diagnosis and Management of Acute Pulmonary Embolism 2019
- CHEST Guideline on Antithrombotic Therapy for VTE Disease (CHEST 2021)
- AHA Scientific Statement on Pulmonary Embolism
- UpToDate Clinical Overview of Pulmonary Embolism (peer-reviewed synthesis)

Last reviewed: 2026-07-07