Pulmonary Embolism

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> **THE STORY IN ONE SENTENCE**
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> A blood clot has traveled from somewhere else in the body and is now stuck in the pulmonary arteries (the blood vessels that feed the lungs).

“Pulmonary Embolism (a clot lodging in a lung artery) — a clot is blocking blood flow through the lungs.”

Normal anatomy — the body's job

Used blood flows back from your body into the right atrium (the top-right chamber of your heart). It drops down into the right ventricle (the bottom-right pumping chamber). The right ventricle squeezes gently and pushes that blood through the pulmonary arteries into your lungs, where it picks up oxygen. The right ventricle is built to pump against low pressure — like pushing a swing, not lifting a truck. It does not have much muscle reserve, so if the pressure suddenly spikes, it can fail quickly.

> **BODY'S JOB**
>
> The right ventricle's job is to move used blood smoothly through the lungs at low pressure so oxygen can hop aboard. When this pump suddenly hits a blockage, everything downstream starts to break.

1. Used blood returns from your body into the right atrium.
2. Blood flows down into the right ventricle.
3. The right ventricle pumps blood through the pulmonary arteries into the lungs.
4. In the lungs, oxygen jumps into the blood and carbon dioxide jumps out.
5. Fresh blood returns to the left side of the heart and is pumped out to your body.
6. The whole loop depends on smooth, low-pressure flow through the lungs.

What breaks

A blood clot that formed somewhere else (usually a deep leg vein) breaks off, travels through your veins, and gets stuck in the lung arteries. The clot physically blocks blood flow and also triggers the lung blood vessels to squeeze tight. Now the right ventricle has to pump against sky-high pressure. If the clot is large, the right ventricle swells, pumps poorly, and bulges into the left ventricle's space. That crushes how much blood the left side can fill and pump out to your body.

> **CLINICAL TERM**
> A blood clot (Thrombus) formation and embolization with right-ventricular afterload crisis

How the body compensates

At first, your body can hide how bad things are by speeding up your heart and tightening your blood vessels to keep your blood pressure from crashing. Vital signs may look okay even while the right ventricle is drowning. That window is dangerous because it lets the team think you are stable when you are actually one step from collapse. The cliff edge is right-ventricular failure with dropping blood pressure.

1. {'what': "The sympathetic nervous system (your body's fight-or-flight wiring) floods the bloodstream with epinephrine and norepinephrine (adrenaline hormones).", 'why': 'Baroreceptors (pressure sensors) in your blood vessels detect falling cardiac output and signal the brainstem to hit the gas pedal and keep blood pressure from crashing.'}
2. {'what': 'Heart rate shoots up (tachycardia).', 'why': "Adrenaline makes the sinoatrial node (the heart's natural pacemaker in the right atrium) fire faster to pump more blood per minute even though each beat is weaker."}
3. {'what': 'Blood vessels in your skin, gut, and muscles squeeze tight (peripheral vasoconstriction).', 'why': 'Norepinephrine tells smooth muscle in those vessel walls to clamp down and redirect blood to your brain and heart — the parts you cannot live without.'}
4. {'what': 'Breathing rate climbs (tachypnea).', 'why': 'Chemoreceptors (oxygen and carbon-dioxide sensors) in the carotid arteries and brainstem detect low oxygen and high dead space, signaling the respiratory center in the medulla to speed up breathing and try to pull in more oxygen.'}
5. {'what': 'For a while, blood pressure holds steady.', 'why': 'Fast heart rate plus tight blood vessels can temporarily mask how little blood is actually being pumped out — this is the dangerous middle where things look okay but are not.'}

6. {'what': 'The crash point: right-ventricular failure with progressive low blood pressure (hypotension) (falling blood pressure).', 'why': 'Once the right ventricle is overstretched and starved of oxygen, it stops pumping effectively. Cardiac output collapses and no amount of adrenaline can fix a mechanical traffic jam.'}
7. {'what': 'Giving IV fluids can make things worse, not better.', 'why': 'Pouring more volume into an already swollen, failing right ventricle overfills it further, crushing the left ventricle even more and dropping cardiac output.'}
8. {'what': 'Norepinephrine (a vasopressor drip) helps maintain coronary blood flow (perfusion) pressure (blood flow to the heart muscle itself).', 'why': 'The right ventricle needs oxygen to survive the crisis. Norepinephrine raises blood pressure just enough to keep blood flowing through the coronary arteries that feed the right-ventricular muscle.'}
9. {'what': 'A patient who becomes with low blood pressure (hypotensive) after PE diagnosis needs an immediate decision about thrombolysis (clot-busting medication) or embolectomy (surgical or catheter clot removal).', 'why': 'Once blood pressure crashes, the window to reverse the blockade and save the right ventricle is minutes to hours — not tomorrow.'}

Domino effect

1. A clot blocks one or more pulmonary arteries. — The clot (usually from a leg or pelvic vein) travels to the lungs and wedges into a branch that is too narrow to pass, cutting off blood flow to that lung tissue.
2. Pulmonary vascular resistance spikes. — The physical clot plus reflex squeezing of downstream lung vessels (from serotonin and thromboxane released by the clot) creates a sudden high-pressure wall the right ventricle must push against.
3. The right ventricle swells and pumps weakly. — The thin-walled right ventricle has little muscle reserve, so when afterload skyrockets, the chamber overstretches, loses squeeze power, and begins to fail.
4. The swollen right ventricle bulges into the left ventricle's space. — The heart sits in a stiff sac (pericardium) with no room to expand outward, so the bloated right side pushes through the interventricular septum and crushes the left ventricle.
5. Cardiac output (blood pumped to the body) drops and blood pressure falls. — The squashed left ventricle cannot fill properly, so even though it tries to squeeze, very little blood gets out to the rest of the body each beat.
6. Heart rate shoots up (tachycardia). — Baroreceptors (pressure sensors) in the aorta and carotid arteries detect the drop in blood pressure and signal the brainstem to flood the body with epinephrine, making the sinoatrial node (heart's pacemaker) fire faster to try to keep total flow up.

7. Low oxygen in the blood (Hypoxemia) (low oxygen in the blood) develops. — Blocked lung vessels cannot carry blood past the air sacs to pick up oxygen, and the failing right heart worsens ventilation-blood flow (perfusion) mismatch (some lung gets air but no blood, some gets blood but collapses), so oxygen levels plummet.
8. Dead space (lung areas with air but no blood flow) shoots up. — Air reaches alveoli (air sacs) downstream from the clot, but because no blood flows there, that ventilation is completely wasted — oxygen cannot jump into blood that is not there.
9. The patient feels severely short of breath (dyspnea) and breathes fast (tachypnea). — Chemoreceptors in the carotid bodies and medulla detect low oxygen and high carbon dioxide from dead space and poor gas exchange, triggering the brainstem respiratory center to speed up breathing in a desperate attempt to pull in more oxygen and blow off CO_2.
10. If the clot burden is massive and the right ventricle fails completely, the patient can arrest (heart stops) or die within minutes. — Once the right ventricle can no longer push any blood forward, the left side has nothing to pump, systemic blood flow (perfusion) ceases, and vital organs (especially the brain) shut down from lack of oxygen.

Symptoms · what the patient feels, and why

- Sudden shortness of breath, often with sharp chest pain when breathing in (pleuritic chest pain): A blood clot gets stuck in a lung artery and blocks blood flow to part of the lung. That lung tissue still tries to work but can't get the blood it needs, so breathing feels hard and painful — like trying to fill a tire with a kinked hose.
- Fast heart rate (tachycardia): The heart beats faster to try to push blood past the clot and keep oxygen moving to the body. It's working overtime, like a pump speeding up when a pipe is blocked downstream.
- Fast breathing (tachypnea): The lungs sense low oxygen and try to catch up by breathing faster. Your brain's breathing control center (the medulla) tells you to take more breaths per minute, trying to make up for the blocked lung section.
- Low oxygen in the blood (hypoxemia): The clot blocks blood from picking up oxygen in that part of the lung. Even though you're breathing, not enough oxygen gets into the bloodstream — like air going into a balloon that has a sealed-off section.
- Coughing up blood (hemoptysis): The blocked lung tissue can bleed because it isn't getting enough oxygen (a small 'lung attack' called pulmonary tissue death from blocked blood flow (infarction)). Blood leaks into the air sacs and gets coughed up.
- Swelling or pain in one leg (often the calf or thigh): The clot usually starts in a deep vein in the leg (deep vein clot formation (thrombosis)). Before it breaks off and travels to the lung, it can cause swelling, warmth, and aching in that leg.

- Feeling lightheaded or fainting (syncope): A big clot can suddenly drop blood pressure because the right side of the heart can't pump blood forward through the blocked lung artery. Less blood reaches the brain, and you feel faint or pass out.
- Very low blood pressure (hypotension) or near-cardiac arrest (massive PE): When a huge clot blocks the main lung arteries, the right ventricle (the heart chamber that pumps blood to the lungs) can't push blood through. Pressure backs up, the heart strains and may fail, and blood pressure crashes — a life-threatening emergency.
- Signs of right heart strain on tests (elevated troponin or BNP, right ventricle looks enlarged on ultrasound) without low blood pressure (submassive PE): A medium-large clot forces the right ventricle to work much harder, stretching and straining it. Blood tests pick up leaked heart enzymes (troponin, BNP) and imaging shows the right side swelling, even before blood pressure drops.

Focused exam

- Sudden a fast heart rate (tachycardia) (heart rate over 100) with a clear trigger — recent surgery, long flight, or leg swelling: The right ventricle (the heart's right pumping chamber) is struggling to push blood through a clot-blocked lung. The heart speeds up trying to keep blood pressure and oxygen delivery alive.
- Fast breathing (Tachypnea) (breathing faster than 20 times per minute) that does not match the fever or pain level: Dead space (lung areas with air but no blood flow) tricks the body into breathing faster to chase enough oxygen. This mismatch is a red flag for pulmonary embolism (a clot lodging in a lung artery).
- Low oxygen in the blood (Hypoxemia) (oxygen saturation below 94% on room air) without crackles, wheezing, or airway disease: Pulmonary embolism (a clot lodging in a lung artery) breaks the match between breathing in and blood flow picking up oxygen. The mismatch causes low oxygen even when the lungs sound clear.
- Pleuritic chest pain (sharp, stabs worse with deep breath) in a patient with leg immobilization or recent surgery: When a clot blocks a lung vessel, that piece of lung stops getting blood and becomes irritated. It hurts when you breathe. This pain is a classic clue.
- Unilateral leg swelling, warmth, or calf tenderness (sign of a clot in a deep leg vein (deep vein thrombosis)): Pulmonary embolism (a clot lodging in a lung artery) usually starts upstream in a leg vein. Finding a clot in the leg proves the source and raises confidence in the diagnosis.
- Signs of right-heart strain — loud P2 (second heart sound), elevated jugular venous pressure (neck veins full), or right-heart heave on the chest wall: The right ventricle is pumping against a suddenly high load from the clot. The heart dilates and struggles. These are signs the right side is starting to fail.

Diagnostics · every test answers a question

- Wells score or revised Geneva score for pretest probability → What is the chance this patient has pulmonary embolism (a clot lodging in a lung artery) before we order imaging or labs?
- D-dimer (whole blood or ELISA) → Can we safely rule out pulmonary embolism (a clot lodging in a lung artery) without radiation imaging in a low-risk patient?
- CT pulmonary angiography (CTPA) with IV contrast → Is there a clot inside the pulmonary arteries?
- Ventilation-blood flow (perfusion) (V/Q) scan → Is there a mismatch between air reaching the lungs and blood flowing through them (used when contrast CT is unsafe due to kidney failure or allergy)?
- Transthoracic echocardiogram (bedside ultrasound of the heart) → Is the right ventricle dilated or struggling, signaling that the clot is large enough to strain the heart?
- Arterial blood gas (ABG) → How low is the oxygen and how much is the body compensating by breathing faster?
- Troponin and BNP (brain natriuretic peptide) → Is the right ventricle so strained that heart muscle is being damaged?
- Compression ultrasound of the legs (duplex Doppler) → Is there a clot in the leg veins that could have traveled to the lungs?

Treatment logic

- Anticoagulation with unfractionated heparin IV bolus and infusion, or low-molecular-weight heparin (enoxaparin) subcutaneous, or fondaparinux subcutaneous — start immediately upon clinical suspicion before imaging if delay is expected and bleeding risk is acceptable → Stops the clot from getting bigger and prevents new clots from forming. Buys time for the body's own fibrinolytic system (natural clot dissolvers) to slowly break down the a traveling clot (embolus) over days to weeks.
- Systemic thrombolysis (alteplase 100 mg IV over 2 hours, or alternative regimen per protocol) for massive pulmonary embolism (a clot lodging in a lung artery) with sustained low blood pressure (hypotension) (systolic BP < 90 mmHg for ≥ 15 minutes) or cardiac arrest → Actively dissolves the clot lodged in the pulmonary arteries within hours, dropping pulmonary vascular resistance so the right ventricle is no longer crushed by back-pressure. Restores cardiac output and can be life-saving.
- Supplemental oxygen by nasal cannula or non-rebreather mask to target SpO2 ≥ 90% → Increases the oxygen content of blood flowing through the working parts of the lung, so tissues get more oxygen even though some lung areas are blocked and cannot participate in gas exchange.

- Catheter-directed thrombolysis or mechanical thrombectomy (suction embolectomy, rheolytic fragmentation) for submassive pulmonary embolism (a clot lodging in a lung artery) with right ventricular dysfunction and elevated troponin or BNP but no shock → Delivers clot-busting drug or physically removes clot directly at the site inside the pulmonary artery, lowering right-heart strain faster than anticoagulation alone while using lower thrombolytic doses than systemic therapy — this lowers systemic bleeding risk compared to full-dose IV alteplase.
- Transition to oral anticoagulation (apixaban, rivaroxaban, edoxaban, dabigatran, or warfarin) and continue for at least 3 months after a provoked pulmonary embolism (a clot lodging in a lung artery) (with a clear temporary trigger like surgery or immobilization), or indefinitely for unprovoked pulmonary embolism (no clear trigger) or recurrent events, weighing bleeding risk → Prevents recurrent venous thromboembolism and protects against another clot reaching the lungs. Lowers the 1-year recurrence rate from about 10% down to 1–2%.
- Inferior vena cava (IVC) filter placement — only when absolute contraindication to anticoagulation exists (active major bleeding, recent brain or spinal surgery, severe a low platelet count (thrombocytopenia)) or recurrent a traveling clot lodging in a vessel (embolism) despite therapeutic anticoagulation → Traps clots traveling from the leg veins before they reach the heart and lungs, providing a mechanical safety net when you cannot use blood thinners.
- Hemodynamic support with IV fluids (500–1000 mL crystalloid bolus cautiously if with low blood pressure (hypotensive) and no signs of volume overload) and vasopressors (norepinephrine preferred) for massive pulmonary embolism (a clot lodging in a lung artery) with shock → Maintains systemic blood pressure and coronary blood flow (perfusion) pressure to keep the right ventricle fed with oxygen while you prepare definitive clot removal (thrombolysis or embolectomy). Gentle fluid bolus may increase right ventricular preload enough to improve cardiac output without overloading the failing right heart.

Medications

Unfractionated heparin (UFH) · Injectable anticoagulant (fast-acting blood thinner)

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.

1. You give heparin through an IV so it reaches the blood instantly.
2. Heparin finds and sticks to antithrombin III, a natural clotting-brake protein floating in the blood.
3. Once bound, heparin makes antithrombin work much faster — it rapidly shuts down thrombin (the clot-cement maker) and factor Xa (an earlier clot-building enzyme).
4. With those enzymes blocked, the clotting cascade stops. No new clot forms and the existing clot stops growing.
5. The body's own cleanup crew (plasmin and other enzymes) slowly chips away at the clot over days to weeks.

SAFETY WATCH

Bleeding: Any cut, nosebleed, or internal bleed can be harder to stop. Watch for blood in urine, stool, or vomit, unusual bruising, or severe headache. · Heparin-induced a low platelet count (thrombocytopenia) (HIT): In rare cases, heparin triggers the immune system to attack platelets (the blood's clotting cells), which paradoxically causes dangerous clots. Check platelet counts every 2–3 days. · Needs frequent blood tests (aPTT) to make sure the dose is just right — too little won't protect, too much increases bleeding risk. · Must be given by IV or injection under the skin; cannot be swallowed as a pill.

Low-molecular-weight heparin (LMWH): enoxaparin, dalteparin · Injectable anticoagulant (blood thinner)

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.

1. You inject LMWH under the skin (usually in the belly).
2. The drug is absorbed into the bloodstream over a few hours, giving steady protection all day.
3. LMWH binds to antithrombin III and supercharges it, mostly targeting factor Xa (a key clotting enzyme early in the cascade).
4. With factor Xa blocked, the clotting chain stops before it can build thrombin and cement a clot.

5. The existing clot stops growing, new clots are prevented, and the body slowly clears the blockage on its own.

SAFETY WATCH

Bleeding: Same risk as regular heparin — watch for blood in urine, stool, unusual bruising, or severe headache. · Lower risk of HIT (heparin-induced a low platelet count (thrombocytopenia)) than unfractionated heparin, but still possible. Monitor platelets. · Use with caution if kidneys are not working well — LMWH is cleared by the kidneys, and buildup increases bleeding risk. · Must be given by injection; cannot be taken as a pill.

Direct oral anticoagulants (DOACs): rivaroxaban, apixaban, edoxaban, dabigatran · Oral anticoagulant (blood-thinner pill)

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.

1. You swallow a DOAC pill; it is absorbed in the intestine and enters the bloodstream.
2. The drug travels through the blood and finds its target enzyme: factor Xa (rivaroxaban, apixaban, edoxaban) or thrombin (dabigatran).
3. The DOAC molecule plugs the active site of the enzyme, blocking it from doing its job in the clotting cascade.
4. With the enzyme shut down, the blood stops trying to clot in the wrong places. The existing clot stops growing, and new clots are prevented.
5. The body's natural cleanup system slowly breaks down the clot over weeks.

SAFETY WATCH

Bleeding: Same risk as other blood thinners — watch for blood in urine, stool, unusual bruising, severe headache, or gum bleeding. · Use with caution if kidneys are not working well — some DOACs (especially dabigatran and edoxaban) are cleared by the kidneys, and buildup increases bleeding risk. · Some DOACs interact with other medications (especially drugs that affect liver enzymes); check with your doctor or pharmacist. · If serious bleeding happens, reversal agents exist for some DOACs (idarucizumab for dabigatran; andexanet alfa for factor Xa inhibitors), but they are expensive and not always immediately available. · Must be taken every day as prescribed — missing doses leaves you unprotected.

Warfarin (Coumadin) · Oral anticoagulant (vitamin K antagonist blood thinner)

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.

1. You swallow a warfarin pill; it is absorbed in the intestine and travels to the liver.
2. Warfarin blocks an enzyme in the liver called vitamin K epoxide reductase (VKORC1), which recycles vitamin K.
3. Without recycled vitamin K, the liver cannot make enough of clotting factors II, VII, IX, and X.
4. Over several days, the levels of these clotting factors drop, and the blood clots more slowly.
5. The existing clot stops growing, new clots are prevented, and the body slowly clears the blockage on its own.

SAFETY WATCH

Bleeding: Same risk as other blood thinners. Watch for blood in urine, stool, unusual bruising, severe headache, or bleeding gums. · Requires regular blood tests (INR) — usually weekly at first, then monthly once stable — to make sure the dose is just right. Too little won't protect; too much increases bleeding risk. · Many foods (especially leafy greens high in vitamin K) and medications interact with warfarin and can throw the INR off. Keep vitamin K intake consistent day to day. · Takes several days to start working and several days to wear off, making it slower to adjust than newer blood thinners. · If serious bleeding happens, vitamin K or clotting-factor concentrates can reverse warfarin, but reversal takes hours.

Alteplase (tPA, tissue plasminogen activator) · Thrombolytic (clot-busting drug)

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).

1. Alteplase is given through an IV so it reaches the blood instantly.
2. The drug finds plasminogen (an inactive clot-chewing enzyme) floating in the blood and stuck in the clot.
3. Alteplase activates plasminogen, turning it into plasmin (the active clot-chewing enzyme).
4. Plasmin breaks apart the fibrin mesh (the protein net that holds the clot together), dissolving the clot piece by piece.
5. Blood flow through the lung is restored, oxygen levels rise, and the pressure on the right side of the heart drops.

SAFETY WATCH

Major bleeding: High risk of life-threatening bleeding, especially in the brain (hemorrhagic stroke), stomach, or anywhere there has been recent surgery or injury. Carefully screen patients before use. · Only used for massive PE (low blood pressure or cardiac arrest) or selected submassive PE (right heart strain without low blood pressure) when the benefit outweighs the bleeding risk. · Contraindicated (must NOT be used) if the patient has had recent surgery, stroke, head injury, or active bleeding. · Given as a one-time IV infusion over 2 hours (or faster in cardiac arrest). · After alteplase, patients need continuous anticoagulation (heparin or another blood thinner) to prevent new clots.

Catheter-directed thrombolysis or mechanical thrombectomy · Interventional procedure (not a pill or injection)

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.

1. The patient goes to a procedure room (catheter lab). Imaging (ultrasound or X-ray) guides the doctor.
2. A thin catheter is inserted into a large vein (usually in the groin or neck) and threaded up to the clot in the lung.
3. For catheter-directed thrombolysis: A small dose of clot-busting drug (alteplase) is delivered right into the clot through tiny holes in the catheter. The drug soaks into the clot and activates plasmin, which chews the clot apart from the inside.
4. For mechanical thrombectomy: A device (suction catheter, rotating blade, or fragmenting tool) physically breaks up or vacuums out the clot.
5. Blood flow through the lung is restored, oxygen levels rise, and pressure on the right heart drops.
6. The patient still needs anticoagulation (blood thinner) afterward to prevent new clots.

SAFETY WATCH

Bleeding: Lower risk than full-dose systemic thrombolysis, but still possible — especially at the catheter insertion site or if the catheter punctures a blood vessel. · Requires a specialized team and equipment (interventional radiology or cardiology), so not available at every hospital. · Complications from the catheter itself: blood vessel injury, infection at the insertion site, or rarely, the catheter dislodging part of the clot and sending it elsewhere. · After the procedure, patients need continuous anticoagulation (heparin or another blood thinner) to prevent new clots.

Inferior vena cava (IVC) filter · Implantable device (not a medication)

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).

1. The patient goes to a procedure room (interventional radiology). A thin catheter is inserted into a vein (usually in the neck or groin).
2. The collapsed filter is threaded through the catheter and positioned in the inferior vena cava, just below where the kidneys drain.
3. The filter is released and expands, anchoring itself to the vein wall.
4. Blood flows through the filter normally, but if a clot breaks off from a leg vein, the filter catches it before it can reach the heart and lungs.
5. The clot trapped in the filter is slowly broken down by the body's cleanup system.
6. When anticoagulation becomes safe again, the filter should be removed (if it is a retrievable type) to avoid long-term complications.

SAFETY WATCH

Filter complications: The filter itself can trap clots and cause leg swelling or pain (deep vein clot formation (thrombosis) at the filter site). Rarely, the filter can break, tilt, or migrate to the heart or lung. · Infection or bleeding at the insertion site. · Retrievable filters should be removed as soon as anticoagulation is safe (usually within weeks to months). Leaving a filter in long-term increases complication risk. · Only used when anticoagulation is absolutely contraindicated and the risk of another PE is very high. NOT a routine treatment.

Red flags

- 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)

Nursing priorities

1. Apply oxygen by nasal cannula or non-rebreather mask to keep SpO2 above 90%. — The clot blocks blood flow to lung tissue, so oxygen cannot transfer into the bloodstream. Supplemental oxygen increases the amount available for gas exchange in the unblocked lung areas and prevents tissue low oxygen (hypoxia) (low oxygen in organs).
2. 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). — Anticoagulation stops the clot from growing larger and prevents new clots from forming. Starting treatment within the first hour reduces mortality (death rate) by preventing clot extension and recurrent traveling clots (emboli) (new clots breaking off).
3. Place patient on continuous cardiac monitoring and measure vital signs every 15 minutes if unstable or every 1 hour if stable. — PE can cause sudden right ventricle (RV) failure because the clot increases pressure in the pulmonary arteries. A fast heart rate (Tachycardia) (heart rate over 100), low blood pressure (hypotension) (systolic BP under 90), or new arrhythmias (irregular heartbeats) signal decompensation (worsening) requiring thrombolysis or ICU transfer.

4. Keep patient on strict bed rest with head of bed elevated 30 to 45 degrees and avoid Valsalva maneuvers (straining, bearing down). — Movement or straining increases venous return (blood flowing back to the heart) and can dislodge more clots from the legs. The semi-upright position reduces work of breathing and improves ventilation (air movement) in the lung bases.
5. 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. — Elevated troponononin and BNP indicate right ventricle strain from the clot blocking blood flow. These biomarkers (blood test markers) identify submassive PE patients who may need advanced therapies like catheter-directed thrombolysis. D-dimer helps rule out PE when low but is not specific.
6. 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. — Anticoagulation increases bleeding risk. Identifying contraindications (reasons not to use blood thinners) like recent brain surgery, GI bleed (stomach or intestine bleeding), or bleeding-driven (hemorrhagic) stroke prevents life-threatening bleeding (hemorrhage) (severe bleeding) from treatment.
7. Notify provider immediately if systolic blood pressure drops below 90 mmHg or patient becomes confused, drenched in sweat (diaphoretic) (sweaty), or has chest pain. — These signs indicate massive PE with obstructive shock (the clot blocks so much blood flow that the heart cannot pump effectively). The patient needs emergent thrombolysis with alteplase (a clot-dissolving drug) within 30 minutes to restore pulmonary blood flow and prevent cardiac arrest.
8. Measure bilateral calf circumference and inspect legs for unilateral swelling, warmth, redness (erythema) (redness), or cord-like veins. — Finding DVT (deep vein clot formation (thrombosis)) confirms the clot source and supports the PE diagnosis. A difference of more than 2 cm between calves suggests DVT. This also guides patient education about compression stockings and mobilization after anticoagulation starts.

Sources

- 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)

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