

DVT and PE

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THE STORY IN ONE SENTENCE

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“DVT and PE are two stops on the same dangerous road: a clot forms in a deep vein, then breaks off and blocks the lung—anticoagulate immediately and risk-stratify to prevent recurrence and death.”

Normal anatomy — the body's job

Normally, blood flows up from your legs through deep veins (large veins buried under muscle) back to your heart and lungs, and your body keeps blood liquid inside vessels but makes it clot quickly when a vessel is cut—this balance is called hemostasis.

BODY'S JOB

The deep veins in your legs carry used blood (low in oxygen, high in waste) back up to your heart. Muscle squeezes the veins when you walk, one-way valves keep blood from falling backward, and the blood stays liquid and smooth because the vein lining is healthy and releases chemicals that prevent clotting. Once blood reaches the right side of your heart, it is pumped into the lungs to pick up fresh oxygen, then returns to the left heart and out to the body again.

1. Blood in the leg drains from small veins into the deep veins (femoral, popliteal, iliac).
2. Calf muscle squeezes the veins like a pump with each step.
3. One-way valves open to let blood flow up and snap shut to stop it from sliding back down.
4. Blood climbs up through the pelvis into the inferior vena cava (the body's big return highway).

5. The inferior vena cava empties into the right atrium of the heart.
6. The right ventricle pumps blood into the pulmonary arteries, which branch into smaller and smaller vessels in both lungs.
7. Blood picks up oxygen in the lung capillaries, then flows back to the left heart and out to the body.

What breaks

When blood flow slows down, the vein wall gets damaged, or blood gets extra sticky (Virchow's triad), platelets and clotting proteins stick together inside the vein and form a solid clot that blocks the normal return flow of blood.

CLINICAL TERM

Venous thromboembolism (VTE) due to activation of the coagulation cascade in the setting of Virchow's triad: stasis (slow flow), endothelial injury (damage to the vein lining), and hypercoagulability (blood that clots too easily). The a blood clot (thrombus) (clot) forms in a deep vein—most often in the calf (distal DVT) or thigh/pelvis (proximal DVT)—and can embolize (break off) to the pulmonary circulation, causing PE.

How the body compensates

The body tries to keep oxygen delivery going by speeding up the heart, breathing faster, and recruiting other veins—but these fixes are temporary band-aids. Vital signs can look okay for a while, hiding how close the patient is to crashing, so you must watch trends and catch the tipping point before the right heart fails or oxygen drops too low.

1. {'what': 'Heart rate goes up (tachycardia).', 'why': 'The heart beats faster to push more blood per minute through the blocked lung or past the blocked leg vein, trying to keep oxygen delivery normal even though each heartbeat moves less blood forward.'}
2. {'what': 'Breathing gets faster and shallower (tachypnea).', 'why': 'The lungs try to pull in more air to make up for the lung areas that are blocked and cannot pick up oxygen—like trying to breathe through a smaller straw.'}
3. {'what': 'Other veins open up (collateral circulation).', 'why': 'Superficial veins near the skin dilate and take over some of the work, routing blood around the blocked deep vein—you might see surface veins bulging on the leg or chest.'}
4. {'what': 'Blood pressure may stay normal at first.', 'why': 'The body squeezes blood vessels elsewhere and pumps harder to keep pressure up—but this uses a lot of energy and cannot last.'}

5. {'what': 'Oxygen level may stay near-normal in small PE.', 'why': 'The unblocked parts of the lung work overtime, and the person breathes faster—so oxygen does not drop much until a large part of the lung is blocked.'}
6. {'what': 'Watch for the crash point: massive PE with shock, low blood pressure (hypotension), or cardiac arrest.', 'why': 'When more than 50% of the lung circulation is blocked, the right ventricle cannot pump hard enough, pressure backs up, the heart muscle runs out of oxygen, and blood pressure falls—this is obstructive shock and can be fatal within minutes.'}
7. {'what': 'Watch for paradoxical a traveling clot lodging in a vessel (embolism) in patients with a hole between heart chambers.', 'why': 'If there is a patent foramen ovale (PFO) or atrial septal defect, a clot can cross from the right heart to the left and travel to the brain (stroke) or other organs.'}
8. {'what': 'Watch for recurrent clots if the underlying cause is not fixed.', 'why': 'Without anticoagulation and risk-factor management, new clots keep forming—up to 50% recurrence risk over 10 years if untreated.'}
9. {'what': 'Look at trends, not snapshots.', 'why': '"A patient whose heart rate climbs from 90 to 120, whose oxygen drops from 95% to 90%, or whose blood pressure drifts down is decompensating—even if each number alone looks 'okay.'"}}

Domino effect

1. Virchow's triad is triggered: blood flow slows, the vein wall is injured, or blood gets stickier. — Long immobility (flight, surgery, bed rest), trauma, surgery, pregnancy, birth control pills, cancer, smoking, or inherited clotting disorders tip the balance so platelets and clotting proteins (fibrin) stick together instead of staying dissolved.
2. A solid clot (thrombus) forms inside a deep vein, usually in the calf or thigh. — Platelets clump on the damaged or slow-flow area, fibrin threads weave through them like a net, and red blood cells get trapped—forming a rubbery, solid plug that sticks to the vein wall and grows bigger over hours to days.
3. The clot blocks blood from draining out of the leg, causing unilateral swelling, pain, warmth, and redness. — Blood coming up from the foot hits the blockage and backs up, so pressure rises in the small veins and capillaries below the clot—fluid leaks out into the tissue (edema), the leg swells, feels heavy and tight, and may turn red or feel warm because blood is pooling and inflammatory chemicals are released.
4. A piece of the clot breaks loose (embolizes) and floats through the bloodstream. — Proximal clots (thigh or pelvis) are more likely to break off because blood flow is faster there; the fragment travels up the inferior vena cava, through the right atrium and right ventricle, and is pumped into the pulmonary arteries.

5. The clot gets stuck in a pulmonary artery branch—this is a pulmonary embolism (a clot lodging in a lung artery) (PE). — Pulmonary arteries branch and get narrower and narrower as they spread through the lungs; the clot wedges into a branch it cannot fit through, blocking blood flow to that section of lung tissue.
6. The blocked lung tissue cannot pick up oxygen (ventilation-blood flow (perfusion) mismatch), causing sudden shortness of breath, low oxygen (hypoxemia), fast breathing (tachypnea), and sometimes sharp chest pain (pleuritic pain). — Air still reaches the alveoli (tiny air sacs), but no blood flows past them to pick up the oxygen—so that part of the lung is wasted. The rest of the lung tries to compensate, and the person breathes faster and harder, but oxygen levels drop because too much lung is out of service. If the blocked lung tissue dies (pulmonary tissue death from blocked blood flow (infarction)), it releases inflammatory chemicals that irritate the pleura (lung lining), causing sharp chest pain that gets worse with deep breathing.
7. The right ventricle has to pump harder against the blocked pulmonary arteries, causing right heart strain, fast heart rate (tachycardia), elevated jugular venous pressure (JVP), and sometimes a new heart murmur or gallop. — The blockage raises pressure in the pulmonary arteries (pulmonary high blood pressure (hypertension)); the right ventricle, which is thin-walled and used to pumping against low pressure, suddenly faces high resistance—like trying to blow through a pinched straw. It dilates, struggles, and may fail. Troponin (a heart muscle injury marker) can rise, ECG may show right heart strain (S1Q3T3 pattern, right bundle branch block, or T-wave inversions in V1–V4), and the person's heart races to try to keep oxygen delivery going.
8. In massive PE, the right ventricle fails completely, causing obstructive shock: blood pressure drops (hypotension), the patient feels dizzy or faints (syncope), and may go into cardiac arrest. — When more than 50% of the pulmonary circulation is blocked, the right heart cannot pump enough blood forward into the lungs, so the left heart has nothing to pump out to the body—cardiac output crashes, blood pressure falls, organs (especially the brain) do not get enough oxygen, and the patient can die within minutes without emergency treatment (systemic thrombolysis, catheter embolectomy, or surgical embolectomy).

Symptoms · what the patient feels, and why

- Leg swelling (usually one leg): A blood clot in a deep vein blocks blood from flowing back to the heart, so fluid backs up and the leg gets puffy and tight.
- Leg pain or tenderness (often feels like a cramp in the calf): The clot irritates the vein wall and stretches it, and the backed-up blood presses on tissues, which triggers pain signals.
- Warmth and redness over the swollen area: The body sends extra blood and immune cells to the clot site to try to heal it, which makes the skin feel hot and look red.

- Sudden shortness of breath: If a piece of the clot breaks off and travels to the lungs (a PE), it blocks blood vessels in the lung so oxygen cannot get into the blood as easily.
- Sharp chest pain that gets worse when you breathe in deeply or cough: The blocked lung blood vessels cause the lung tissue to swell and the membrane around the lung (pleura) to get irritated, which hurts when the lung expands.
- Fast heart rate (tachycardia): The heart pumps faster to try to make up for the fact that less blood is getting oxygen in the blocked lung.
- Coughing up blood (hemoptysis): The clot damages small blood vessels in the lung tissue, so a little blood leaks into the air sacs and gets coughed out.
- Lightheadedness or fainting: A large clot in the lungs can drop blood pressure suddenly because the heart cannot pump enough oxygen-rich blood to the brain.

Focused exam

- One leg swollen, warm, red, and tender (not both legs): A blood clot stuck in a deep vein (DVT) blocks the blood from draining back to the heart, so fluid backs up below the clot and the leg swells. The one-sided swelling tells you it's a clot in one vein, not heart or kidney trouble (which would swell both legs).
- Calf is tender when you squeeze it, or pain behind the knee when you pull the foot up toward the shin: The calf veins are the most common spot for DVT to start. Tenderness or stretch pain points to a clot irritating that exact vein.
- Fast breathing (over 20 breaths per minute), fast heart rate (over 100), and oxygen level below 95% on room air: If a piece of the DVT clot broke loose and flew up to the lung (pulmonary a traveling clot lodging in a vessel (embolism) or PE), it blocks blood flow in the lung so oxygen can't get into the bloodstream. The heart speeds up and breathing speeds up trying to make up for the missing oxygen.
- Sharp chest pain that gets worse when you take a deep breath in: When a PE clot blocks a lung vessel, it irritates the thin lining around the lung (the pleura). Every time you breathe, that sore lining rubs and hurts.
- Fainting, blood pressure under 90 systolic, blue lips or fingernails: A massive PE means a huge clot has jammed so much of the lung that the right side of the heart can't push blood through. The whole body starts to shut down because not enough blood is moving.
- Measure around both calves with a tape measure; the swollen leg is at least 3 cm bigger around: DVT swelling is always one-sided because the clot only blocks one vein. Measuring both legs and finding a difference proves it's a clot, not swelling from heart failure or low protein (which would swell both legs equally).

Diagnostics · every test answers a question

- D-dimer blood test → Is the chance of a clot low enough that we can safely rule it out without imaging?
- Ultrasound (compression ultrasound) of the leg veins → Is there a clot stuck in a deep vein that we can see?
- CT angiogram (CTA) of the chest with contrast dye → Is there a clot blocking blood vessels inside the lung?
- Ventilation-blood flow (perfusion) scan (V/Q scan) if the patient can't have CT contrast (kidney disease, dye allergy) → Is air reaching parts of the lung that blood is NOT reaching (mismatch = clot)?
- Wells score (clinical decision rule: leg swelling, heart rate, recent surgery, prior clot, coughing up blood (hemoptysis), cancer) → What is the chance this patient has DVT or PE before we do any tests?
- Arterial blood gas (ABG) if PE is suspected and oxygen is low → How low is the oxygen in the bloodstream, and is the body trying to blow off CO₂ to fix it?
- Troponin and BNP (brain natriuretic peptide) if massive PE is suspected → Is the right side of the heart so overloaded that heart muscle is getting damaged?
- Echocardiogram (ultrasound of the heart) if massive PE with low blood pressure → Is the right side of the heart swollen and struggling because the clot is blocking so much lung?

Treatment logic

- Start anticoagulation immediately: DOAC (apixaban 10 mg twice daily × 7 days then 5 mg twice daily, or rivaroxaban 15 mg twice daily × 21 days then 20 mg once daily) OR low-molecular-weight heparin (enoxaparin 1 mg/kg twice daily) bridged to warfarin (INR goal 2–3) → Anticoagulation blocks clotting proteins (factor Xa or thrombin) so the clot stops growing. Your body's own enzymes slowly break down the clot over weeks to months. This fixes the upstream problem—stops the blood from clotting more while the vein or lung heals.
- Use low-molecular-weight heparin (LMWH, enoxaparin) instead of DOAC if the patient is pregnant, has active cancer, or has severe kidney disease (CrCl under 30) → LMWH is safe in pregnancy (DOACs cross the placenta and harm the baby). It also works better in cancer patients because cancer makes blood clot more aggressively. LMWH does not need dose adjustment by kidney function as much as some DOACs. Still blocks factor Xa to stop clot growth.

- Give thrombolysis (tissue plasminogen activator, tPA 100 mg IV over 2 hours, or catheter-directed tPA) if massive PE with shock (systolic BP under 90) or submassive PE with right heart strain on echo → tPA activates plasminogen, which dissolves the clot actively right now instead of waiting weeks. This unclogs the lung vessels fast so blood can flow and oxygen can get through. Buys time when the patient is about to die from right heart failure.
- Place an inferior vena cava (IVC) filter if the patient has a new clot but CANNOT take anticoagulation (active bleeding, recent brain surgery, severe bleeding disorder) → A metal basket sits in the big vein below the heart and catches clots before they reach the lung. Prevents PE when anticoagulation is too dangerous.
- Elevate the swollen leg above heart level, start walking as soon as it's safe (early mobilization), and fit the patient for compression stockings (30–40 mmHg) once swelling is controlled → Elevation and muscle movement (walking) help veins pump blood back to the heart against gravity, so fluid doesn't pool. Compression stockings squeeze the leg so veins can't stretch and leak fluid. Together they reduce pain and swelling.
- Give oxygen (nasal cannula or mask) to keep oxygen saturation above 90%, and monitor heart rate, blood pressure, and oxygen level continuously if PE is present → Oxygen keeps the body's cells alive while anticoagulation or tPA fixes the clot. Continuous monitoring tells you if the patient is getting worse (falling blood pressure, rising heart rate) and needs thrombolysis or ICU care.
- Continue anticoagulation for at least 3 months; extend indefinitely if the clot was unprovoked (no surgery, trauma, or temporary risk factor), recurrent, or the patient has active cancer or inherited clotting disorder (factor V Leiden, antiphospholipid syndrome) → Keeps the clotting system blocked long-term so the body can't form a new clot. Prevents recurrence—without anticoagulation, about 30% of unprovoked clots come back within 5 years.

Medications

Apixaban (Eliquis) or Rivaroxaban (Xarelto) · Direct oral anticoagulant (DOAC) - factor Xa inhibitor

These medicines block one specific step in the clotting chain called factor Xa (say 'ten-A'). Factor Xa is like a foreman that tells the body to build a clot. When you block it, the body can't finish making new clots, so the existing clot stops growing and your own cleanup crew (natural enzymes in the blood) can slowly dissolve it over weeks to months.

1. You swallow the pill; it enters your bloodstream from your intestine.
2. The drug finds and sticks to factor Xa enzymes floating in your blood.
3. Factor Xa can no longer turn prothrombin (an inactive protein) into thrombin (the active clot-builder).

4. Without thrombin, fibrinogen (a stringy protein) cannot become fibrin threads, so no new clot mesh forms.
5. The clot you already have stops growing, and your body's own plasmin enzyme slowly chews it apart day by day.

SAFETY WATCH

Bleeding risk: because clotting is blocked, any cut or internal injury bleeds longer. Watch for blood in urine, black tarry stools, or bruising easily. · Do NOT stop suddenly without your doctor saying so, or a new clot can form within days. · Avoid taking with strong drugs that change how your liver breaks down the DOAC (like certain antibiotics, antifungals, or seizure medicines); your doctor will check. · If you need emergency surgery, the drug wears off in about 24 hours (apixaban and rivaroxaban), but major bleeding may need a reversal agent.

Enoxaparin (Lovenox) or Dalteparin (Fragmin) · Low-molecular-weight heparin (LMWH)

These are injected under the skin (like an insulin shot) once or twice a day. They boost the power of a natural brake in your blood called antithrombin. Antithrombin normally slows down clotting enzymes, but LMWH makes it work 1,000 times faster, so clotting stops quickly. Because LMWH is predictable and does not need daily blood tests, it is the go-to choice for pregnant women (safe for baby), cancer patients (works even when cancer makes blood stickier), and anyone who cannot swallow pills.

1. You inject the medicine just under the skin of your belly or thigh.
2. It enters the small blood vessels and spreads through your bloodstream in minutes.
3. LMWH finds antithrombin (a protein that naturally slows clotting) and locks onto it.
4. The LMWH-antithrombin team now shuts down factor Xa and a little bit of thrombin much faster than antithrombin could alone.
5. Clotting slows down, the existing clot stops growing, and your body begins to dissolve it naturally.

SAFETY WATCH

Bleeding risk: same as DOACs—watch for unusual bruising, blood in urine or stool, or bleeding gums. · Injection-site bruising is common and not dangerous unless it spreads or hurts a lot. · Very rarely, LMWH can cause a dangerous side effect called heparin-induced a low platelet count (thrombocytopenia) (HIT), where your platelet count drops and clots form even more; doctors check your platelet count if you are on LMWH longer than 5–7 days. · Safe in pregnancy (does not cross the placenta to the baby), so it is the standard choice when pregnant. · If your kidneys are weak, the drug can build up; your doctor will adjust the dose or check a blood level.

Warfarin (Coumadin) · Vitamin K antagonist (oral anticoagulant)

Warfarin blocks your liver from using vitamin K to build several clotting factors (II, VII, IX, X). Without those factors, your blood clots much more slowly. It takes 3–5 days to see the full effect because the old clotting factors already in your blood have to wear out first. Warfarin is now used less often (DOACs are easier), but it is still the choice if you have a mechanical heart valve, severe kidney disease, or antiphospholipid syndrome.

1. You swallow the pill; it is absorbed in your intestine and travels to your liver.
2. Warfarin blocks an enzyme called VKORC1, which normally recharges vitamin K so it can be used again.
3. Without recharged vitamin K, your liver cannot finish building factors II, VII, IX, and X.
4. These half-made factors cannot help form clots, so clotting slows down.
5. The effect builds over several days as old, working factors are used up and not replaced.

SAFETY WATCH

Bleeding risk, especially if INR (a blood-test number that shows how thin your blood is) goes too high. Target is usually 2.0–3.0; above 4 is risky. · Foods high in vitamin K (kale, spinach, broccoli) can make warfarin less effective if you suddenly eat much more or less than usual; you do not have to avoid them, just keep amounts steady week to week. · Many antibiotics, antifungals, and even over-the-counter medicines change how warfarin works; always tell every doctor and pharmacist you are on it. · You need blood tests (INR) every 1–4 weeks to make sure the dose is right. · Do NOT take during pregnancy (can harm the baby); switch to LMWH if you become pregnant.

Alteplase (tPA, Activase) or Tenecteplase (TNKase) · Thrombolytic (clot-buster)

These medicines are only used in the emergency room or ICU when a clot is so big it is immediately life-threatening (massive PE with low blood pressure or right-heart strain). They

turn on plasminogen, a protein already in your blood, into plasmin, which is like scissors that cut the fibrin threads holding the clot together. The clot dissolves in hours instead of weeks, but bleeding risk is high, so doctors use it only when the benefit clearly outweighs the danger.

1. The drug is infused into a vein, usually over 15 minutes to 2 hours.
2. It travels through the bloodstream to the clot.
3. tPA binds to fibrin threads in the clot and activates plasminogen molecules stuck inside the clot.
4. Plasminogen becomes plasmin, an enzyme that chops fibrin into small pieces.
5. The clot breaks apart quickly, blood flow is restored, and the pieces are cleared by the liver and spleen.

SAFETY WATCH

High bleeding risk—about 1–3 in 100 patients have serious bleeding, including bleeding in the brain (intracranial bleeding (hemorrhage)). · Cannot be used if you had recent surgery (within 2–4 weeks), recent stroke, active bleeding anywhere, or very high blood pressure. · Used only in the ICU with close monitoring of blood pressure, heart rate, and bleeding signs. · After tPA, you continue on heparin or LMWH and then transition to a DOAC or warfarin for long-term prevention.

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

An IVC filter is a small cage placed inside the large vein (inferior vena cava) that carries blood from your legs back to your heart. It traps large clots before they can reach your lungs. It is only used when you absolutely cannot take blood thinners (for example, active bleeding in the brain or stomach) but you already have a clot in your leg. Most filters are temporary and should be removed as soon as it is safe to start anticoagulation, usually within weeks to months.

1. A doctor threads a thin tube (catheter) through a vein in your neck or groin up to the IVC.
2. The filter (folded inside the catheter) is pushed out and opens like an umbrella inside the vein.
3. Blood flows through the filter, but any clot bigger than a few millimeters gets caught in the wire struts.
4. The trapped clot either dissolves over time or sticks to the vein wall and is sealed off by scar tissue.

SAFETY WATCH

The filter itself can trigger clots on its struts if left in place long-term. · The vein where the catheter was inserted can get infected or form a clot. · Retrieval (removal) is recommended as soon as you can safely start blood thinners again, usually within 3–6 months. · Some filters are permanent, used only if you can never take anticoagulation (very rare). · Follow-up imaging and removal appointments are essential; do not skip them.

Red flags

- Sudden trouble breathing or very fast breathing
- Chest pain that feels sharp or stabbing, especially with a deep breath
- Coughing up blood
- Feeling faint, dizzy, or passing out
- Fast or irregular heartbeat with sweating or confusion

Nursing priorities

1. Check oxygen saturation with pulse oximeter and apply oxygen to keep SpO₂ above 90 percent if the patient has any shortness of breath or chest pain — PE blocks blood flow to part of the lung so that area cannot pick up oxygen; low oxygen levels mean the clot is large or the patient is decompensating (getting worse)
2. Start continuous pulse oximetry and cardiac monitoring to watch for arrhythmias (irregular heartbeat) and a fast heart rate (tachycardia) (fast heart rate) — A large PE strains the right side of the heart and can cause dangerous rhythms or right heart failure; a fast heart rate (tachycardia) over 100 is an early warning sign
3. Keep the patient on bed rest with the affected leg elevated above the level of the heart — Elevation uses gravity to reduce swelling and pain; bed rest prevents the clot from breaking loose and traveling to the lungs, though this is controversial and short-term only
4. Do not massage, rub, or apply heat to the swollen leg — Pressure or heat can dislodge the clot and send it to the lungs as a PE; the clot is fragile in the first few days
5. Measure and record the circumference of both calves and thighs at the same spot every shift — Serial measurements (repeated measures over time) show whether the clot is getting bigger despite treatment or whether swelling is going down with anticoagulation
6. Assess for signs of bleeding (gums, bruising, blood in urine or stool, nosebleeds) every four to eight hours after anticoagulation starts — Anticoagulants stop new clots but also increase bleeding risk; early detection of bleeding (hemorrhage) (bleeding) prevents life-threatening blood loss

7. Check baseline PT/INR, aPTT, CBC (complete blood count), and creatinine before giving the first dose of anticoagulant — Baseline labs show if the patient already has a bleeding disorder or low platelets (thrombocytopenia); creatinine checks kidney function because some anticoagulants are cleared by the kidneys and need dose adjustments
8. Teach the patient to report any sudden worsening of shortness of breath, new chest pain, dizziness, or confusion immediately — These symptoms mean a new PE or a massive PE (very large clot) that can cause shock (dangerously low blood pressure) or cardiac arrest within minutes

Sources

- American Society of Hematology (ASH) 2020 VTE guideline
- European Society of Cardiology (ESC) 2019 PE guideline
- American College of Chest Physicians (CHEST) Antithrombotic Therapy guideline
- NEJM Clinical Practice reviews on venous thromboembolism

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