

VTE Prevention

Stop deadly clots before they start — score every patient's risk, then use blood thinners or leg pumps for anyone at medium or high risk.

THE ONE-LINE TRUTH

Hospital patients need their clot risk checked with validated scoring tools, then given medicine or compression devices to stop blood clots from forming.

The chain — how it unfolds

1. Patient admitted to hospital with immobility, surgery, or active cancer. — why: Bed rest stops the calf-muscle pump; surgery injures vessels and triggers inflammation; cancer cells release tissue factor — all three parts of Virchow's triad are now present, tipping the balance toward clot formation in deep leg veins.
2. Clotting cascade activates in pooled venous blood. — why: Stagnant blood concentrates activated clotting factors (thrombin, Factor Xa); injured endothelium exposes tissue factor that ignites the extrinsic pathway; hypercoagulable states (high estrogen, genetic mutations, inflammation) amplify the cascade — fibrin strands cross-link and trap red cells, forming a solid clot anchored to the vein wall.
3. A clot in a deep leg vein (Deep vein thrombosis) (DVT) grows silently in the calf or thigh. — why: Most DVTs cause no symptoms because deep veins have no sensory nerves; even large clots may go unnoticed unless they completely block venous return and cause leg swelling, warmth, and pain (present in fewer than half of cases).
4. Clot fragment breaks free and embolizes to the pulmonary arteries (PE). — why: Sudden movement, straining, or spontaneous detachment sends part of the clot through the right heart into the lung circulation, where it lodges in a pulmonary artery branch and blocks blood flow to that part of the lung.
5. Blocked lung segment cannot exchange oxygen (V/Q mismatch). — why: The obstructed artery means no blood reaches the alveoli in that zone, so oxygen breathed into those air sacs cannot enter the bloodstream — ventilation without blood flow (perfusion) drops total body oxygen saturation and raises dead-space ventilation.

6. Right ventricle strains against the blockage; large PE causes shock and cardiac arrest. — why: The right ventricle is a thin-walled, low-pressure pump not built to overcome sudden high resistance — a massive clot spikes pulmonary artery pressure, dilates and stuns the right ventricle (acute cor pulmonale), drops left-heart filling (obstructive shock), and can trigger fatal arrhythmia or pulseless electrical activity within minutes.
7. Prevention with pharmacologic (heparin, enoxaparin, fondaparinux) or mechanical (sequential compression devices) prevention (prophylaxis) stops the cascade before the clot ever forms. — why: Low-dose anticoagulants inactivate Factor Xa or thrombin, keeping the clotting cascade below the threshold for spontaneous clot formation; compression devices squeeze the legs rhythmically to mimic walking and prevent venous stasis — both strategies break Virchow's triad and drop VTE incidence by 50–70 percent in hospitalized patients, saving thousands of lives every year.

Spot it — what you see, and why

- Recent surgery (especially hip, knee, belly, or cancer surgery): Surgery triggers the blood's clotting system — the body thinks it needs to stop bleeding, so it tips toward making clots. At the same time, lying still afterward means blood pools and slows down in the leg veins, which lets clots form. These two forces together raise the odds of a clot by 10 to 100 times depending on the surgery.
- Being stuck in bed for three or more days, or not able to move around much (from illness, injury, or stroke): When your legs don't move, the calf muscles aren't squeezing the veins to push blood back up to the heart. Blood slows down and pools in the deep leg veins, which makes it easier for a clot to form — like how water sitting in a puddle gets scummy while a flowing stream stays clean.
- Active cancer or getting chemotherapy: Cancer cells release chemicals that activate the clotting system, and chemotherapy can damage the walls of blood vessels and lower platelet numbers while also making the remaining platelets stickier. Both changes tip the blood toward clotting. People with cancer are four to seven times more likely to get a blood clot.
- Past blood clot (DVT or PE) in your own history: A clot in the past means your clotting system is already tipped toward making clots — either because you inherited genes that make your blood stickier, or because a vein was damaged and never fully healed. Either way, the risk of another clot jumps to about 10 to 15 percent over the next few years without prevention.

- Pregnancy or just gave birth (especially in the first six weeks after delivery): Pregnancy hormones (especially estrogen and progesterone) make the blood clot faster to protect the mother from bleeding too much during delivery. The growing uterus also presses on the big veins in the belly, slowing blood flow from the legs. After delivery, clotting factors stay high for weeks while the body heals. Risk peaks in the first three weeks postpartum.
- Using birth-control pills or hormone replacement therapy (estrogen): Estrogen raises levels of clotting proteins made by the liver (like fibrinogen and factor VII) and lowers natural anticlotting proteins (like protein S). This shifts the balance toward clotting. Pills that combine estrogen and progestin raise clot risk about three to four times; the highest risk is in the first year of use.

Red flags — act now

- Sudden shortness of breath, fast breathing, or chest pain that gets worse with a deep breath (could mean a clot has broken off and traveled to the lungs — a pulmonary embolism (a clot lodging in a lung artery) — which can be deadly)
- One leg is swollen, painful, red, and warm compared to the other leg (sign of a a clot in a deep leg vein (deep vein thrombosis) or DVT forming right now)
- Coughing up blood or bloody mucus (can mean a clot in the lung has damaged the tissue)
- Fainting, feeling dizzy, or heart racing with shortness of breath (signs that a big clot in the lung is blocking blood flow and dropping blood pressure — life-threatening)

Treat it — in this order

1. Risk stratification at hospital admission using a validated scale (Padua Prediction Score for medical patients, Caprini Score for surgical patients) → Identifies which patients are at medium or high risk for clots and therefore need blood-thinning medication, versus low-risk patients who only need mechanical devices. This ensures you give the right prevention to the right person and avoid over-treating or under-treating.
2. Early mobilization (getting the patient out of bed and walking) and sequential compression devices (inflatable sleeves that squeeze the legs every few seconds) for all hospitalized patients → Squeezing the legs pushes blood out of the deep veins and back toward the heart, preventing it from pooling and clotting. Walking does the same thing naturally by using the calf muscles to pump blood upward.
3. Low-molecular-weight heparin (enoxaparin 40 mg injected under the skin once daily) or unfractionated heparin (5,000 units injected under the skin every 8 or 12 hours) for medium- or high-risk medical or surgical patients → Blocks factor Xa and thrombin (the final two steps in the clotting cascade), so the blood stays thinner and clots cannot form in the deep veins or travel to the lungs. This is the core of VTE prevention for at-risk patients.

4. Extended prevention (prophylaxis) with low-molecular-weight heparin or a direct oral anticoagulant (rivaroxaban 10 mg daily or apixaban 2.5 mg twice daily) for 14 to 35 days after major orthopedic surgery (hip or knee replacement) → Keeps clotting factors suppressed during the highest-risk window after surgery, when the body's clotting system is hyperactive from surgical trauma and the patient is still not fully mobile. This cuts the rate of late clots that form after hospital discharge.
5. Low-molecular-weight heparin or direct oral anticoagulant for hospitalized cancer patients; hold medication if active bleeding or platelet count below 50,000 → Cancer cells release tissue factor and microparticles that hyperactivate the clotting cascade. Blood thinners suppress this excessive clotting response and prevent DVT and PE while the cancer is being treated.

Your top priorities at the bedside

1. Complete VTE risk assessment using a validated tool like Padua Prediction Score or Caprini Score within four hours of hospital admission — why: Risk stratification (sorting patients by clot danger) identifies who needs blood thinners versus mechanical prevention only; missing high-risk patients leads to preventable clots that cause death or permanent lung damage
2. Apply sequential compression devices to both legs immediately for all patients unless they have acute DVT or severe peripheral artery disease — why: SCDs (leg squeeze machines) push blood out of leg veins every minute, preventing stasis (pooling) that triggers clot formation; they work within hours and have almost no bleeding risk
3. Assist patient out of bed and ambulate in hallway at least three times per day starting on day one of admission — why: Walking contracts calf muscles which pump blood back to the heart and prevent venous stasis; even two-minute walks reduce VTE risk by 30 percent compared to bedrest