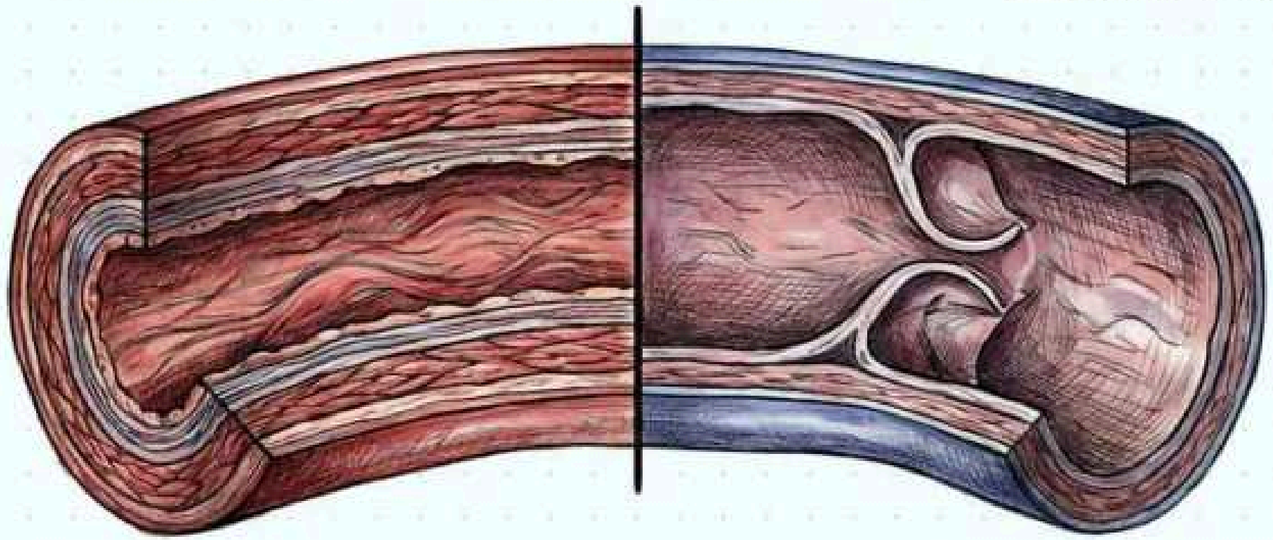
MED 525 B: Thrombosis & Anticoagulation

¶ 5.1

Thrombosis = Intravascular Blood Clot → Ischemia/Infarction/Embolization



Core Definition: Thrombosis is clot formation inside a blood vessel.

Clinical Impact: Leading cause of death and disability globally (underlies most heart attacks, strokes, and venous clots).

Arterial Thrombosis

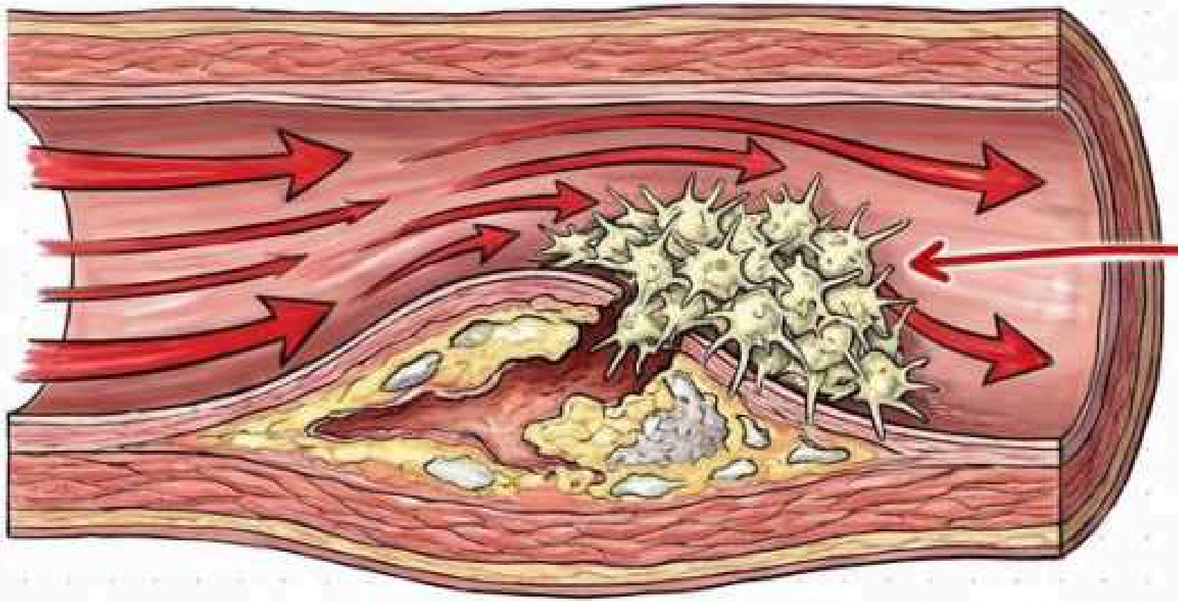
Venous Thrombosis

The Critical Split: Arterial and venous thrombosis are fundamentally different in both how they form and how they are prevented.

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¶ 5.2

The Mechanism: Usually forms on a ruptured atherosclerotic plaque.



High-Velocity Flow + Plaque Rupture → Platelet-Rich Clot (White Clot)

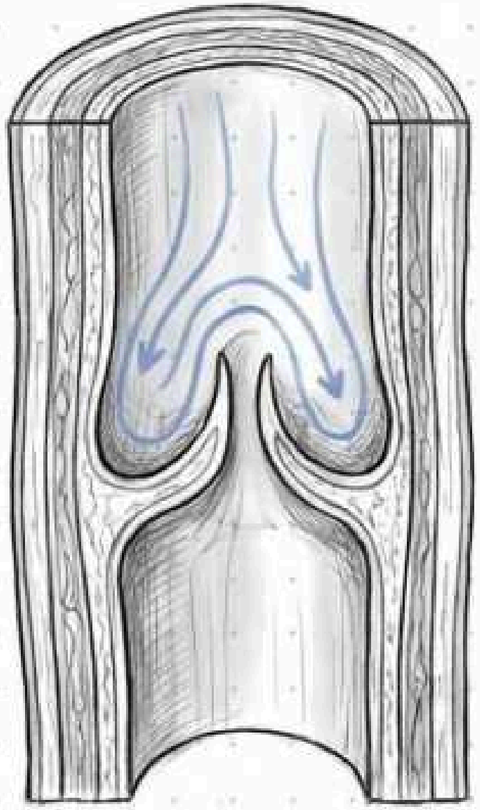
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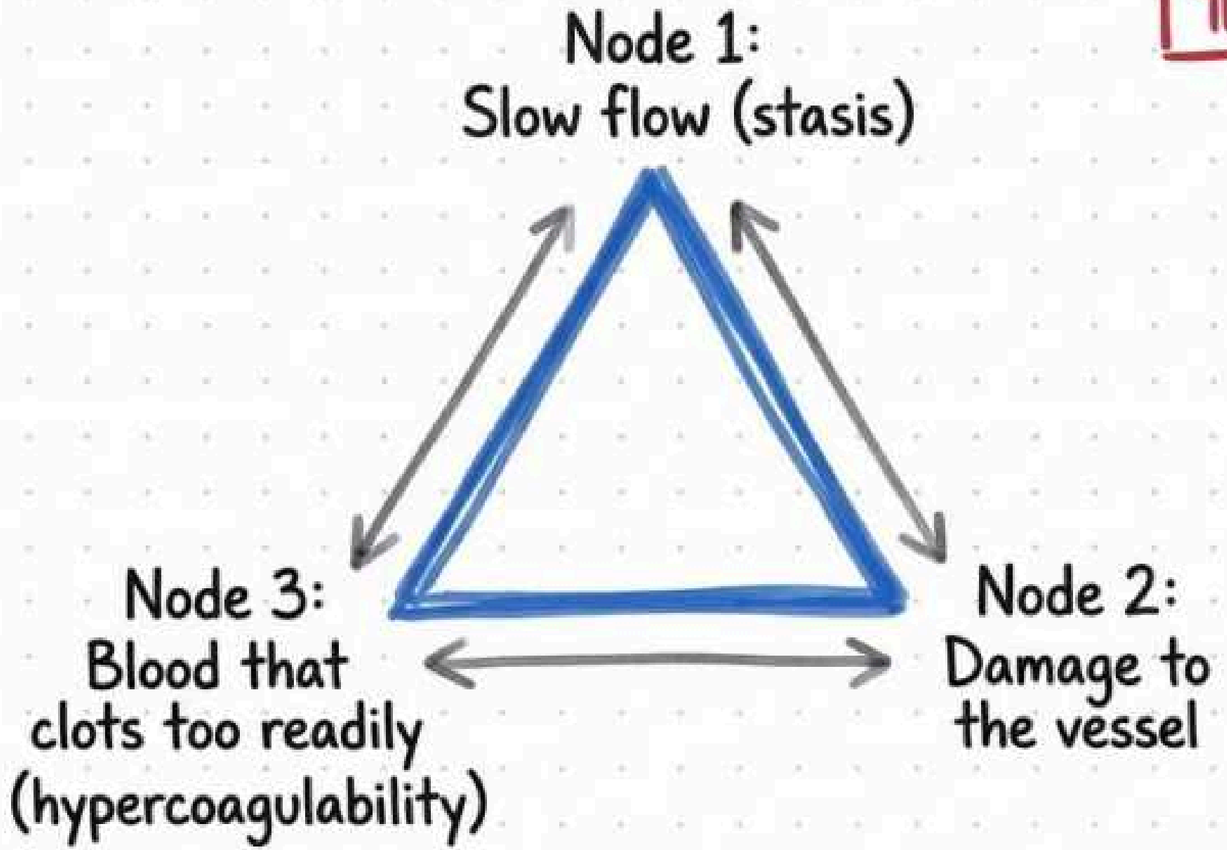
Pharmacological Intervention: $\text{Inhibition}_{\text{Platelet}}$ (e.g., Aspirin)

Clinical Goal: Antiplatelet drugs are the absolute mainstay of preventing heart attacks and strokes in patients with arterial disease.

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¶ 5.3





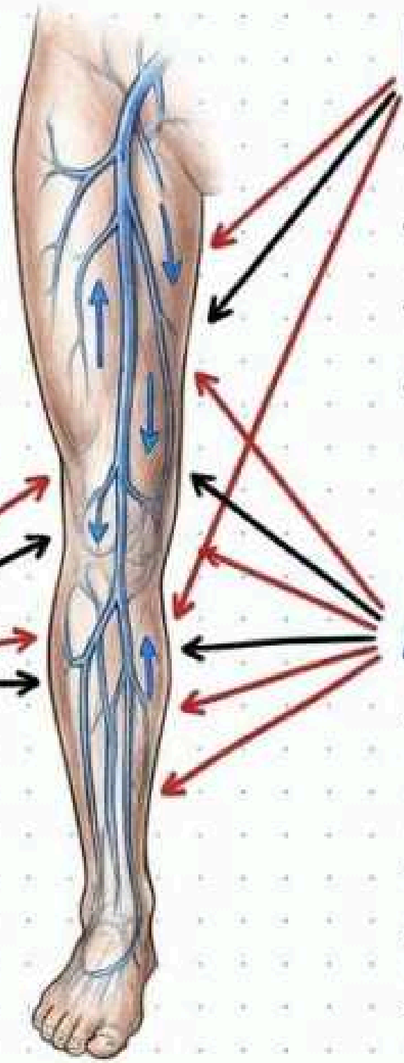
Slow Flow + Fibrin + Red Cells → Red Clot

Deep Vein Thrombosis (DVT) + Pulmonary Embolism (PE)
= Venous Thromboembolism (VTE)

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$VTE_{Risk} = \sum (Acquired\ Variables)$

¶ 5.4



High/Major Variables:

- Surgery (especially hip and knee)
- Cancer
- Immobility (long periods)
- Pregnancy & weeks after birth

Low/Minor Variable:

- Long-haul travel ($+\Delta risk_{small}$)

Additive/Systemic Variables:

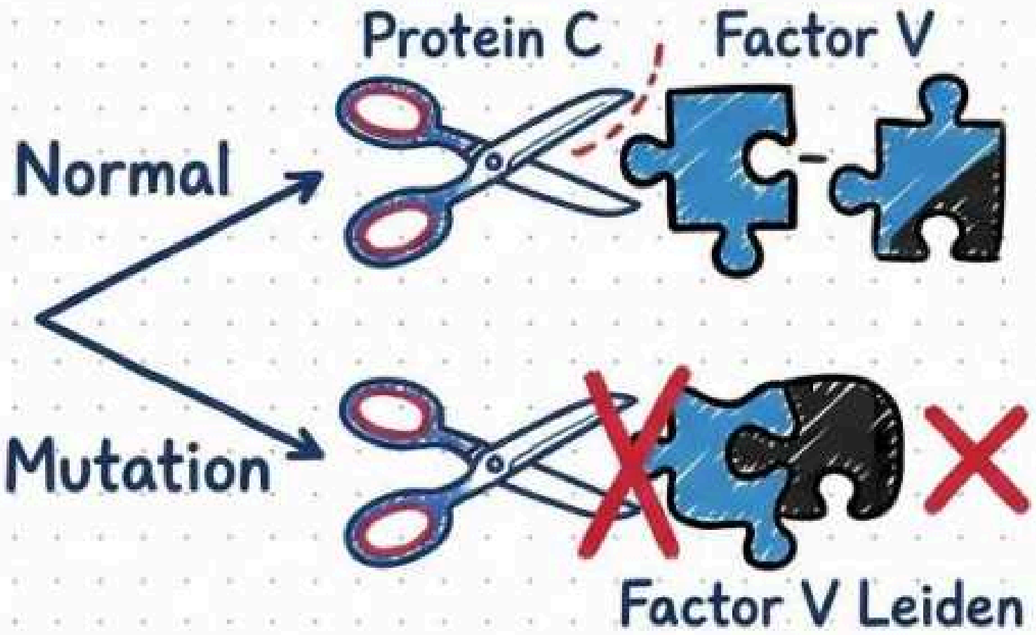
- Oestrogen (contraceptives, hormone therapy)
- Obesity
- Increasing age
- Previous clots

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¶ 5.5

Thrombophilia = An inherited tendency to clot.

Epidemiology: Factor V Leiden is the commonest form in people of European descent.



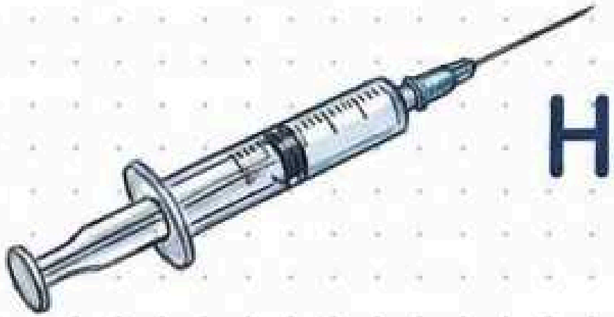
Factor $V_{Leiden} \neq$ Switched off by Protein C

Clinical Guideline: Testing for thrombophilia is useful only in selected cases. It rarely changes treatment and can cause needless worry.

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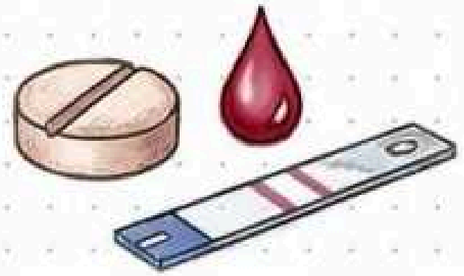
¶ 5.6

Core Principle: Anticoagulant drugs reduce the blood's ability to clot.



Heparin

- Discovered early 20th century.
- Acts within minutes.
- Mechanism: Boosts antithrombin.
- Delivery: Low-molecular-weight forms are given by injection.



Warfarin

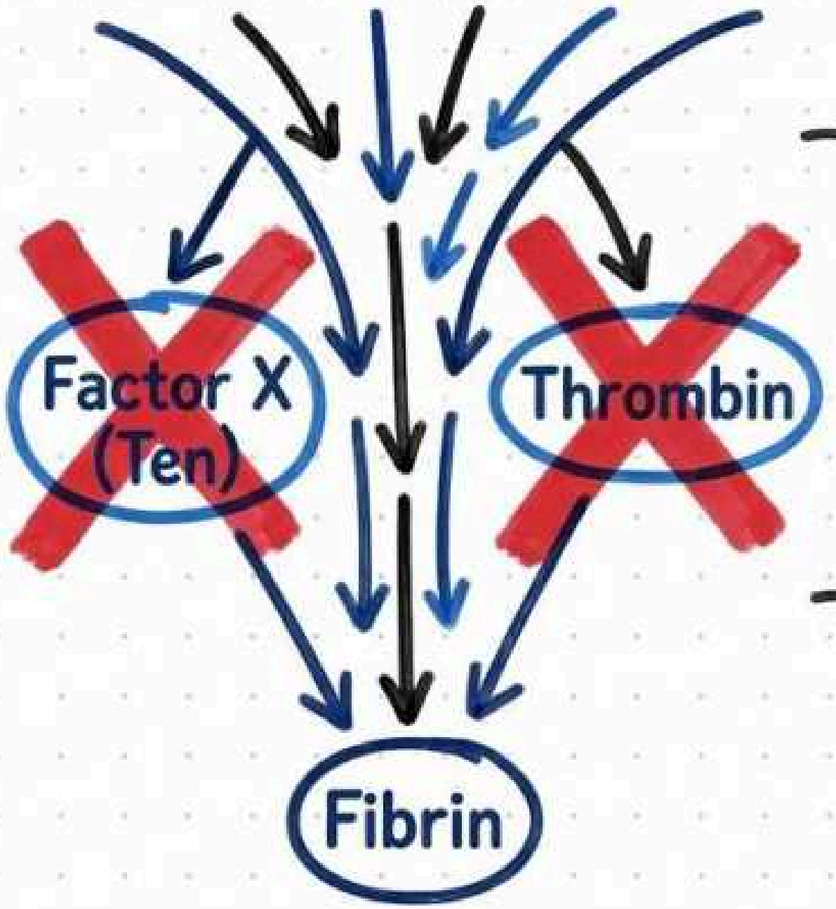
- Introduced in the 1950s.
- Takes days to work.
- Mechanism: Blocks Vitamin K.
- Monitoring: Requires regular blood tests of the INR.
- Vulnerabilities: Effect is altered by diet and many other drugs.

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The Modern Era: DOACs (introduced in the 2010s)

¶ 5.7

- Mechanism: Directly block specific clotting factors (Thrombin or Factor Ten).



The DOAC Advantage: Work quickly, require no routine blood monitoring, have fewer interactions.

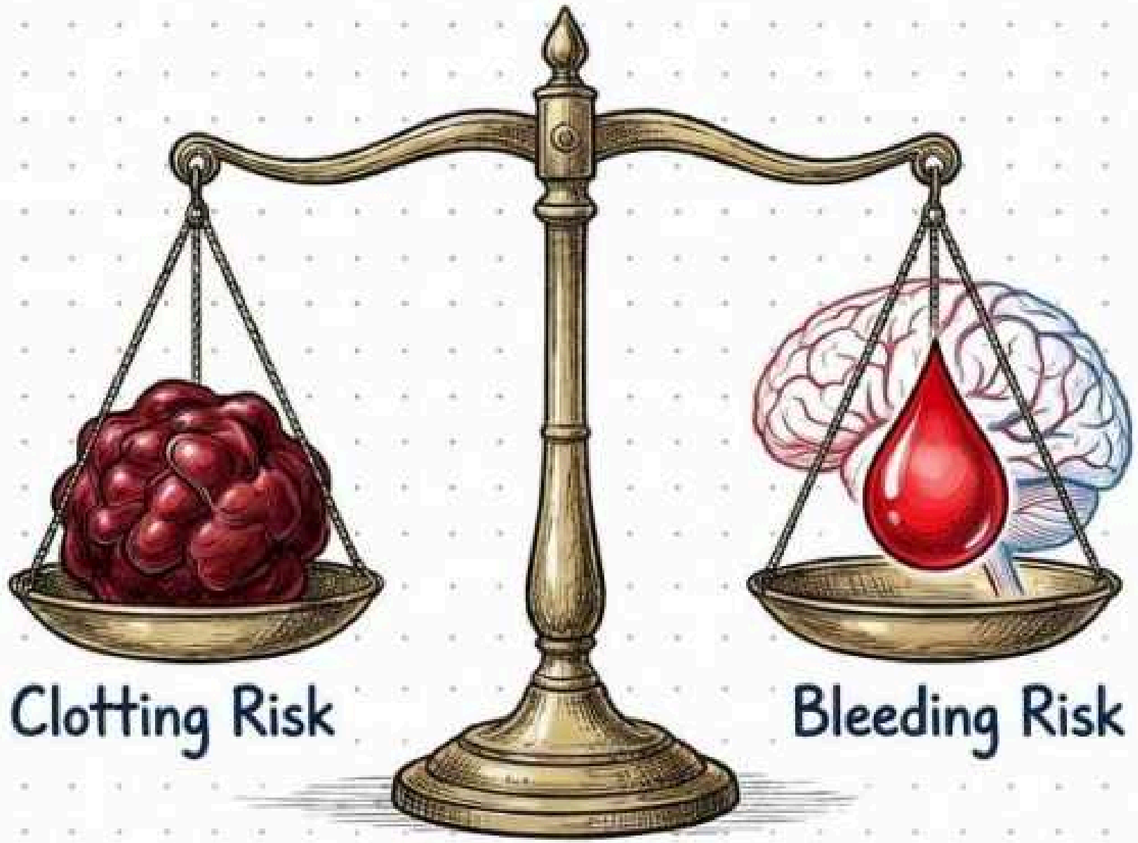
Clinical Shift: Have largely replaced warfarin for many uses.

The Exception: Warfarin remains essential for specific patients (e.g., those with mechanical heart valves).

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¶5.8

The Universal Risk: Every anticoagulant carries a risk of severe bleeding, including bleeding into the brain.



Treatment Decision = f(Clot Risk Score, Bleed Risk Score, Patient's Values)

Methodology: Balancing requires using established scoring systems for each risk (as previously described for atrial fibrillation in Med 521 B).

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¶ 5.9

Baseline Protocol: Treatment of VTE usually lasts at least three months.



Path A (Clear Temporary Cause / Provoked):
e.g., Post-surgery.
→ Treatment can stop at 3 months.

Path B (No Clear Cause / Unprovoked):
Risk of recurrence is higher.
→ Longer treatment may be advised.

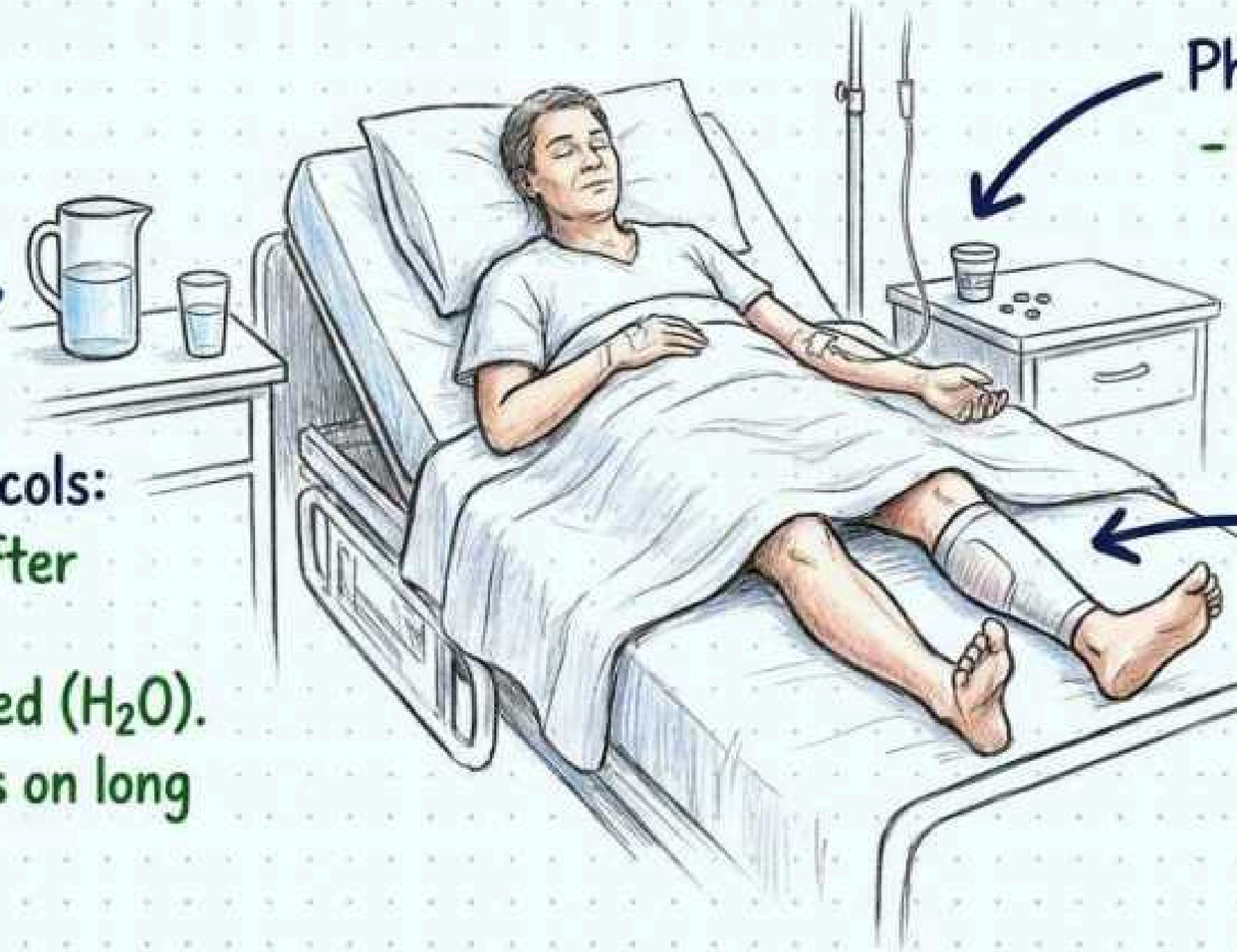
Safety Net: Antidotes now exist for several anticoagulants to reverse effects during serious bleeding events.

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¶ 5.10

The Standard of Care: Prevention is highly effective.

Every admitted patient gets a clotting risk assessment.



Pharmacological Intervention:

- Low-dose anticoagulants.

Behavioral Protocols:

- ☐ - Moving early after surgery.
- ☐ - Staying hydrated (H_2O).
- ☐ - Moving the legs on long journeys.

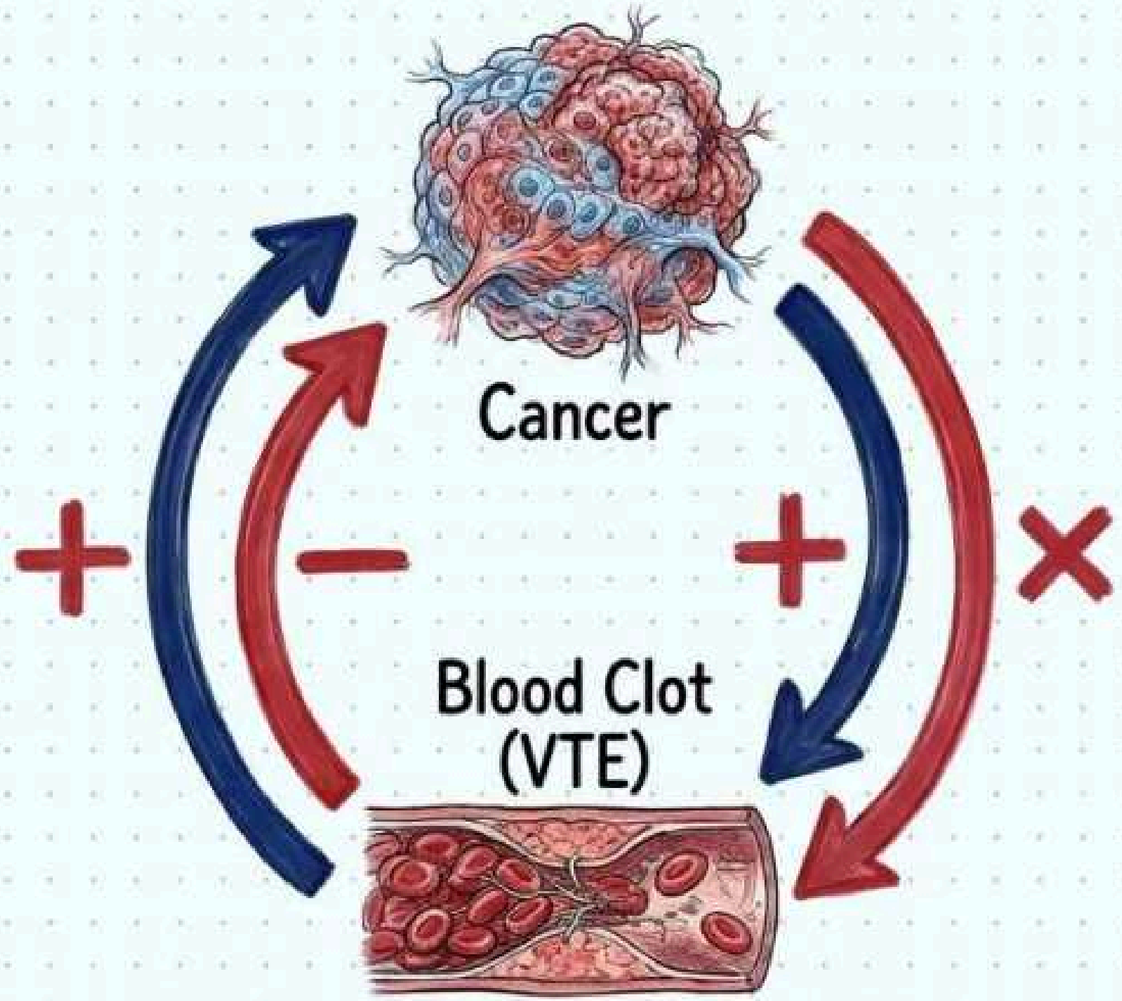
Mechanical Intervention:

- ☐ - Devices that physically squeeze the legs.

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¶ 5.11

The Baseline Link: Cancer raises the risk of clots considerably. (Note: A clot can occasionally be the first sign of an undiagnosed cancer).



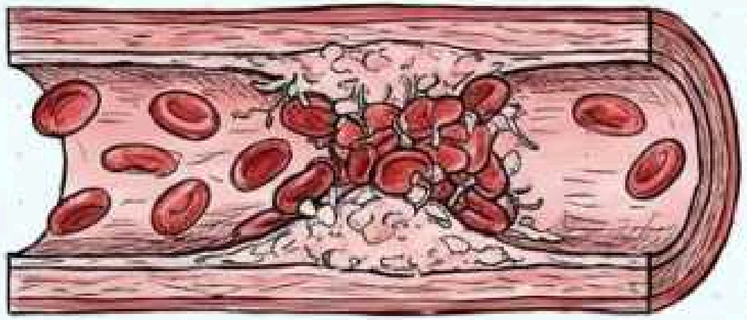
× The Multiplier Effect: Specific cancers + cancer treatments → further increased clotting.

The Ultimate Challenge: Managing these clots requires navigating a highly complex balance of an elevated clotting risk against an often increased bleeding risk.

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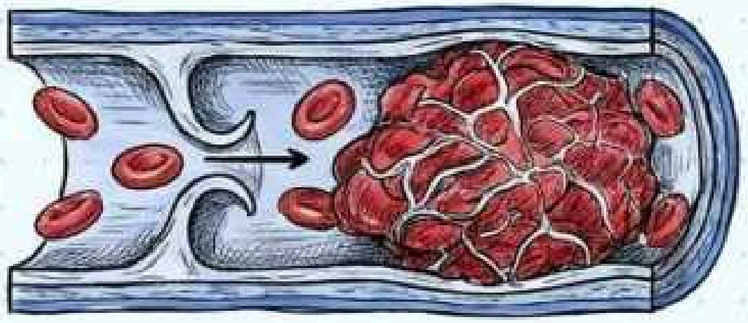
¶ 5.12

Arterial Thrombosis



Driven by platelets → Prevented mainly by Antiplatelet drugs.

Venous Thrombosis



Driven by the coagulation cascade → Prevented mainly by Anticoagulants.

The Golden Rule: Every clinical decision must weigh clotting against bleeding.

Coming Up Next: The final lecture will cover blood cancers, transfusion, and stem cell transplantation.

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