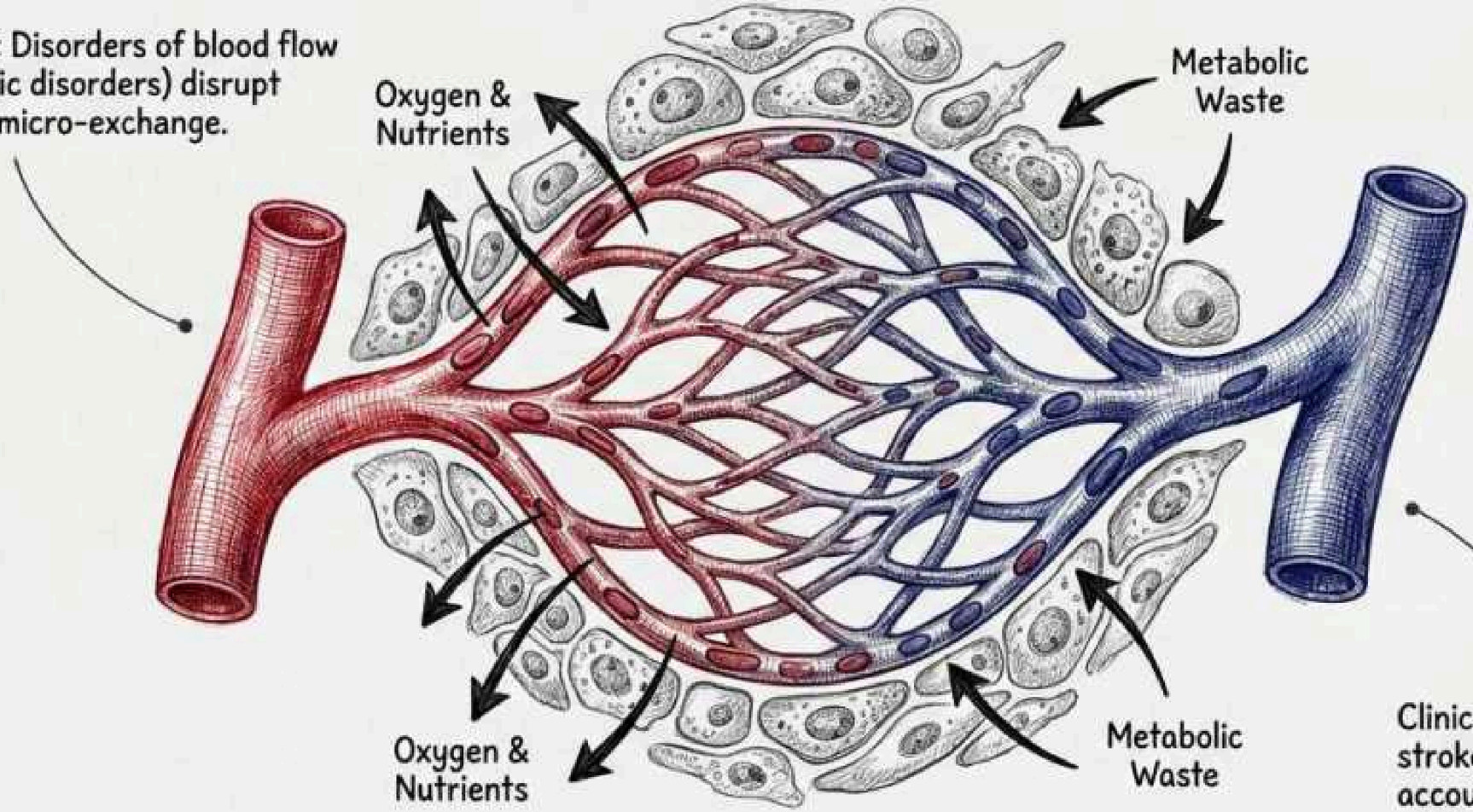
Every tissue depends on continuous perfusion for survival

¶ 3.1

Core Concept: Disorders of blood flow (haemodynamic disorders) disrupt this essential micro-exchange.



Clinical Impact: Heart attacks, strokes, and pulmonary clots account for a massive share of global mortality.

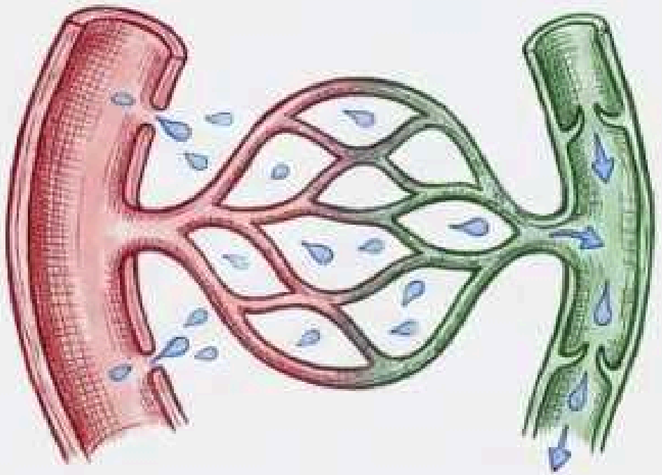
Fluid imbalance leads to tissue swelling and oedema

¶ 3.2

$$J_v = K_f([P_c - P_i] - \sigma[\pi_c - \pi_i])$$

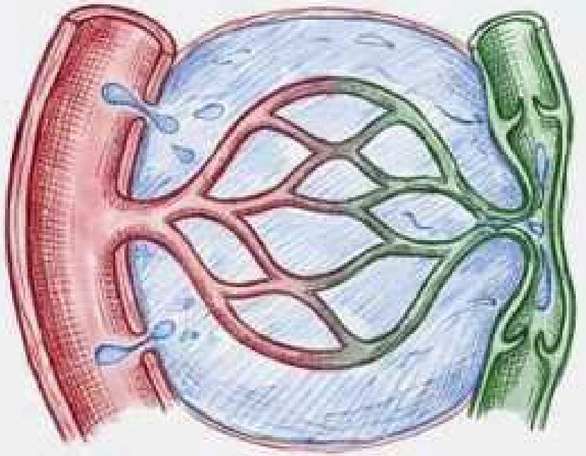
(Net fluid movement = Filtration forces - Absorption forces)

Normal



Definition: Oedema is the accumulation of fluid in tissues.

Oedema



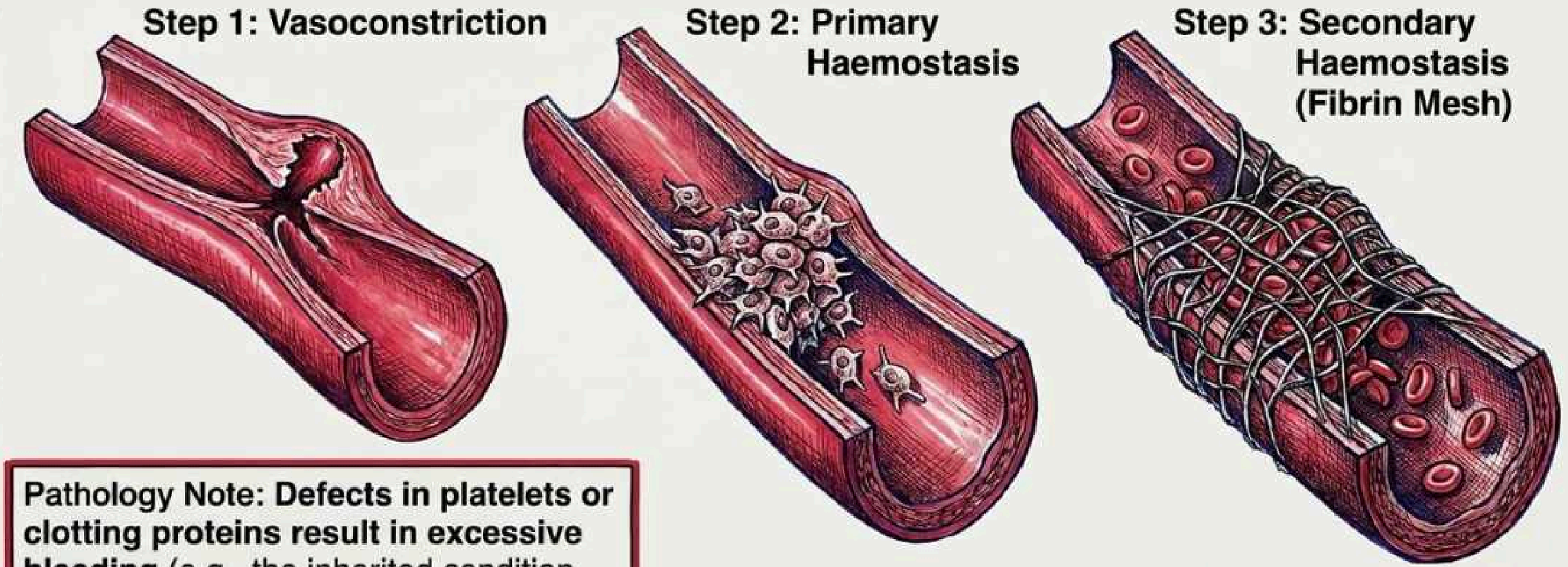
Four Triggers:

1. ↑ Venous pressure (e.g., heart failure)
2. ↓ Blood protein (e.g., liver/kidney disease)
3. Blocked lymphatics
4. Leaky vessels (inflammation)

Red Flag Warning: Pulmonary oedema = fluid drowning the lungs = severe heart failure.

The body stops haemorrhage through a three-step haemostasis cascade

¶ 3.3

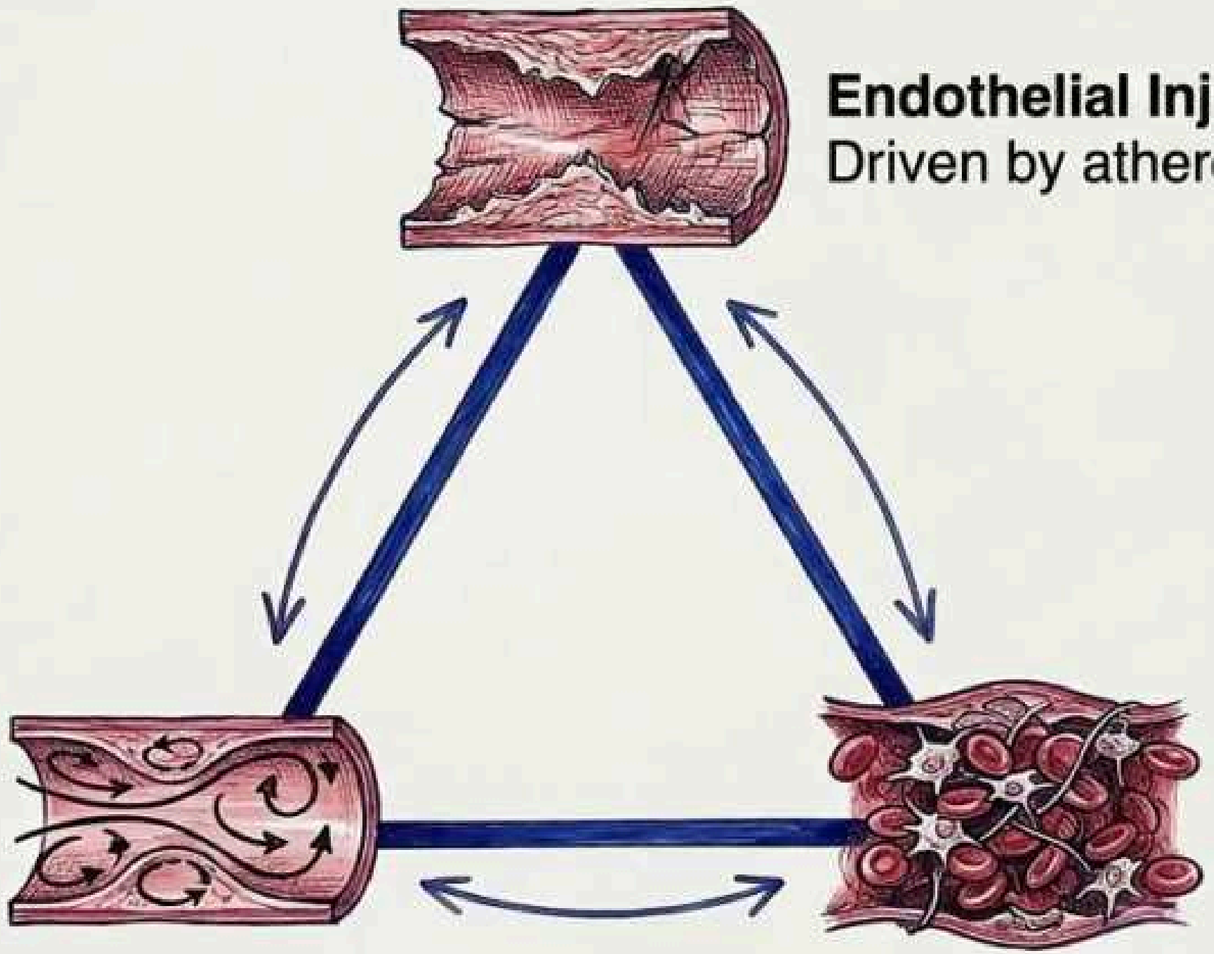


Pathology Note: Defects in platelets or clotting proteins result in excessive bleeding (e.g., the inherited condition Haemophilia from Medicine 503 B).

Inappropriate clotting is driven by Virchow's Triad

¶ 3.4

Core Definition: Thrombosis = formation of a solid mass of blood (thrombus) inside a living vessel or heart.



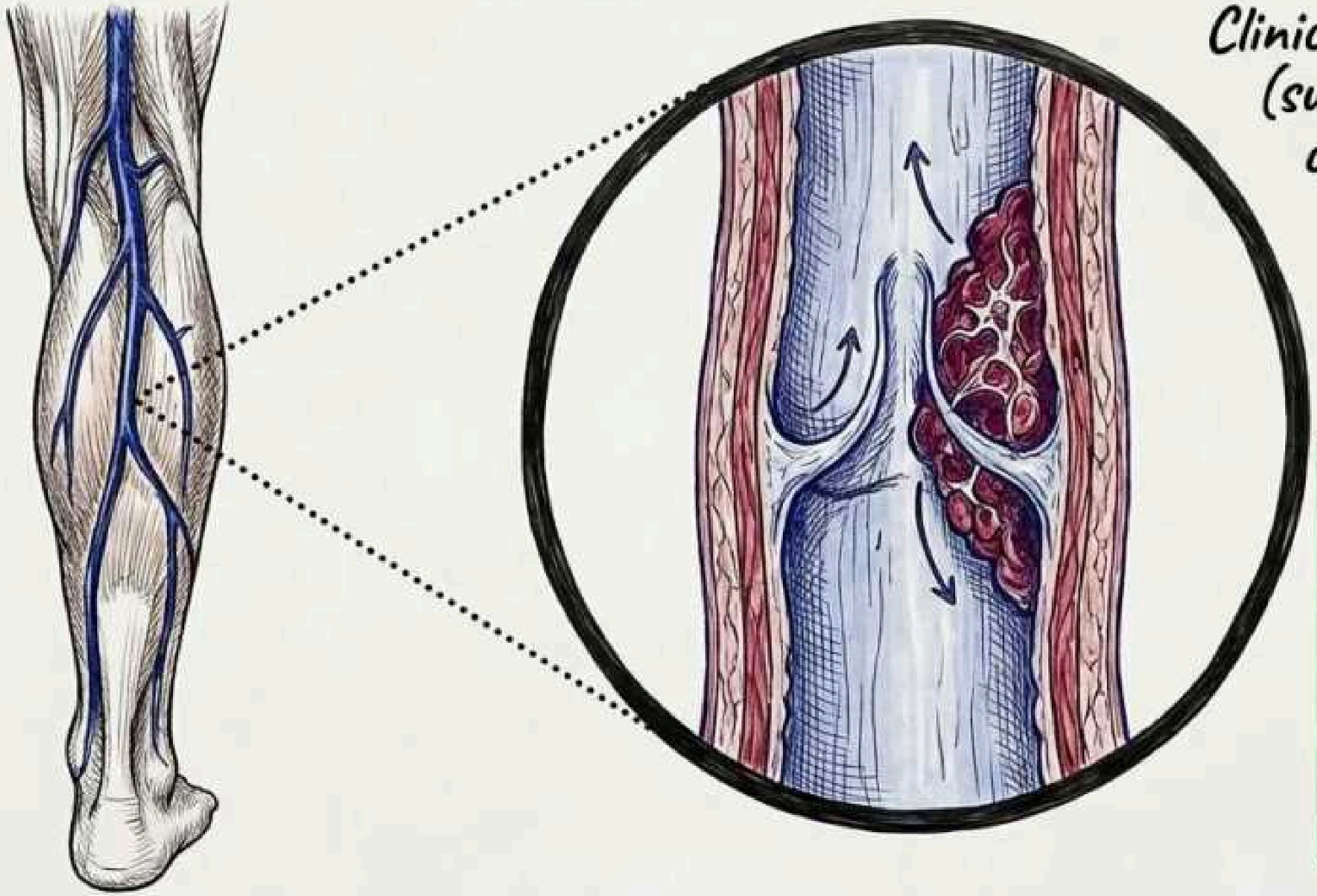
Endothelial Injury:
Driven by atherosclerosis.

Abnormal Flow:
Stagnant or turbulent movement.

Hypercoagulability:
Inherited, or caused by pregnancy, cancers, hormones, and surgery.

Deep vein thrombosis perfectly illustrates the elements of the triad

¶ 3.5



Clinical Catalyst: Immobility (surgery, long flights) causes stagnant flow. Surgery or illness simultaneously induces hypercoagulability.

Clinical Management:

- Early patient movement
- Compression stockings
- Anticoagulant medicines

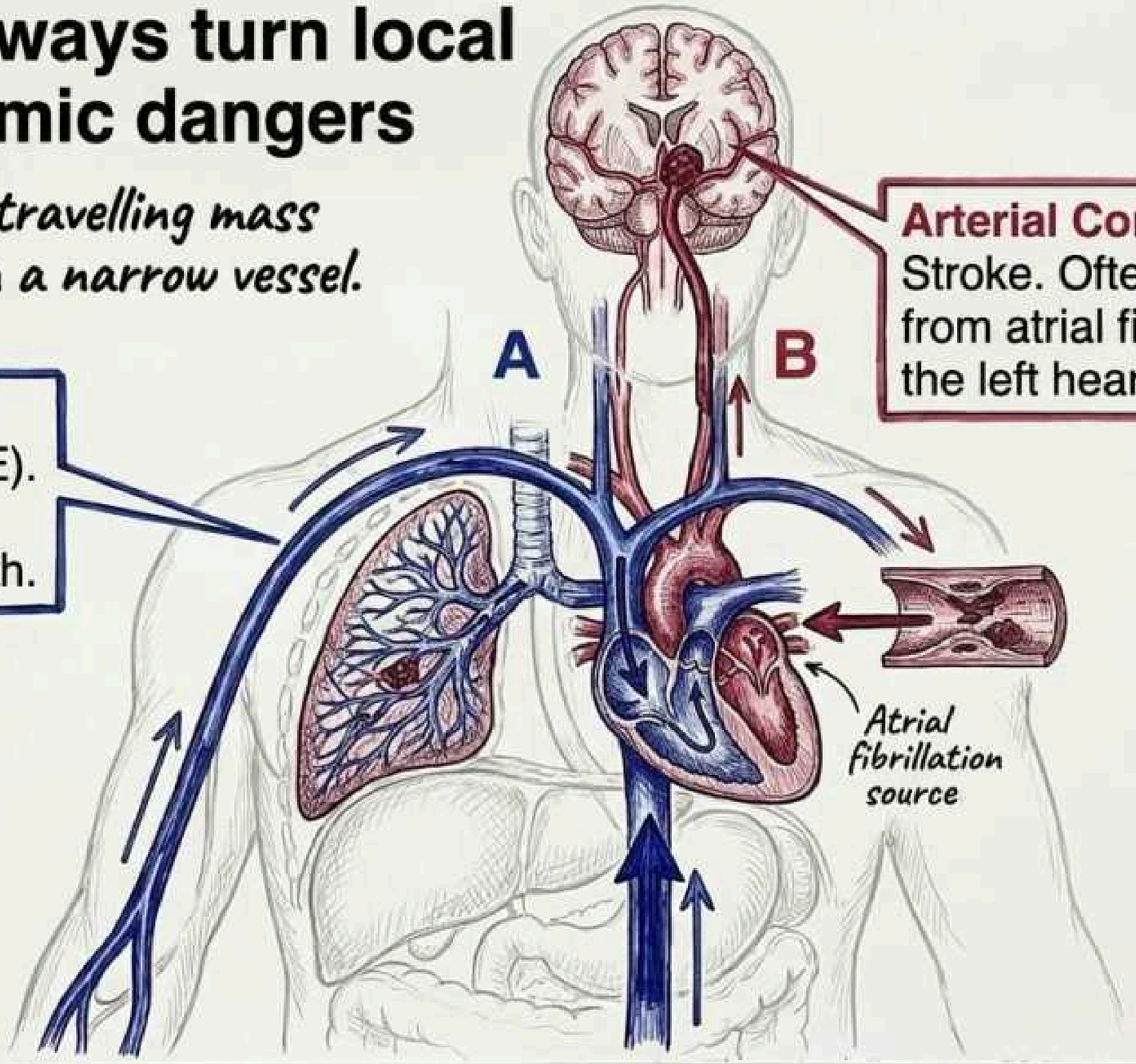
Embolism pathways turn local clots into systemic dangers

¶ 3.6

Definition: Embolus = a travelling mass that lodges in a narrow vessel.

Venous Consequence:
Pulmonary Embolism (PE). Sudden breathlessness, chest pain, potential death.

Arterial Consequence:
Stroke. Often originates from atrial fibrillation in the left heart.

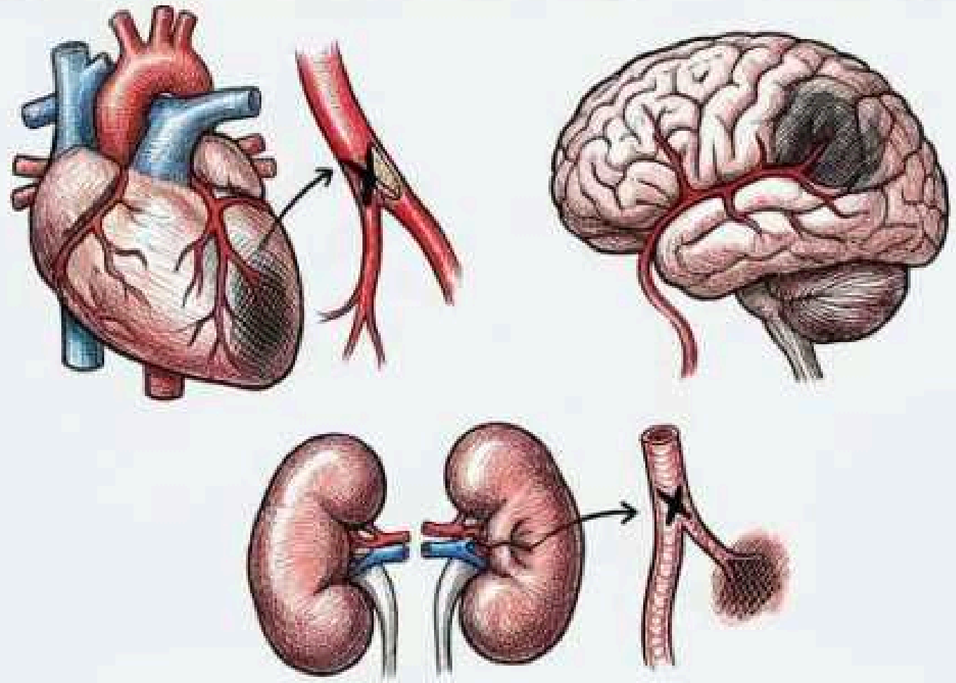


Infarction vulnerability depends on the organ's vascular architecture

¶ 3.7

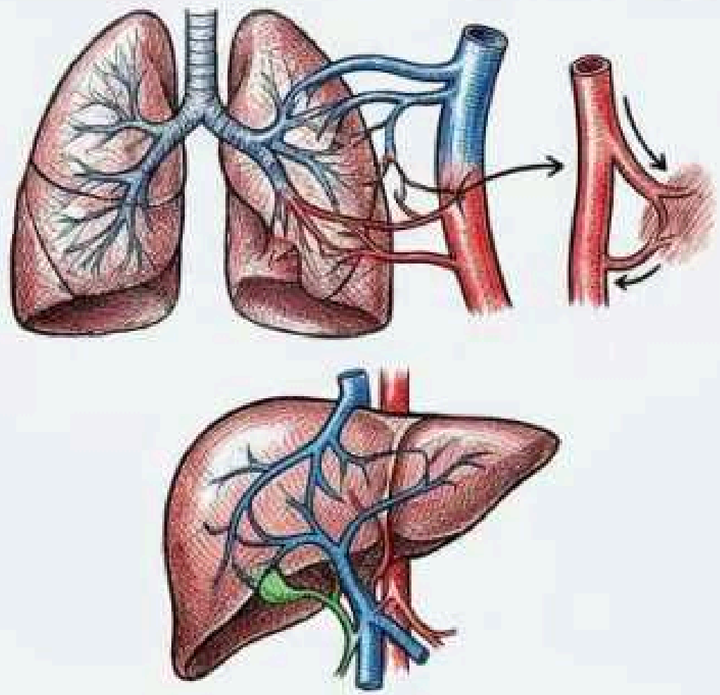
Core Definition: Infarct = Area of tissue death due to blocked blood supply (e.g., Myocardial infarction, Stroke).

Highly Vulnerable: Single/End-Arterial Supply



Severe lack of backup perfusion makes them highly susceptible to fatal infarcts.

Protected: Dual Blood Supply

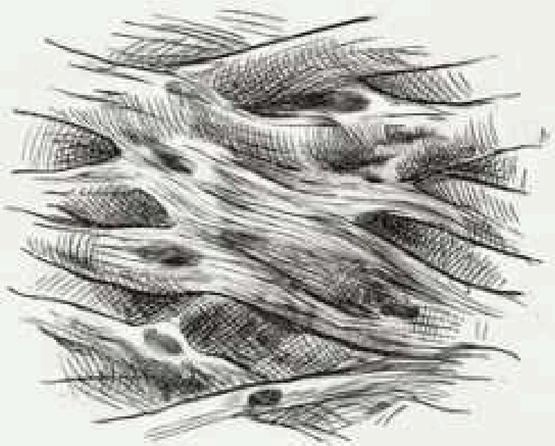


Alternative collateral blood pathways protect the tissue from immediate death upon single blockage.

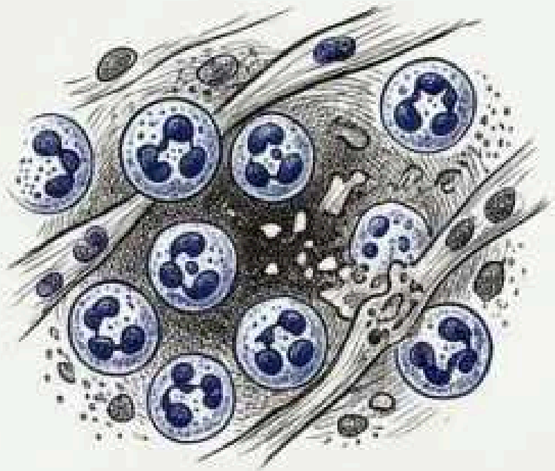
An infarct evolves through predictable cellular stages over time

¶ 3.8

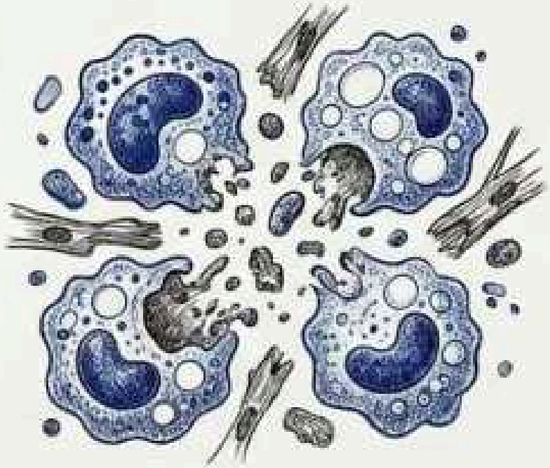
Day 1 | Days 2–4 | Days 5–10 | Weeks Later



Coagulative necrosis (dead muscle, from lecture 1).

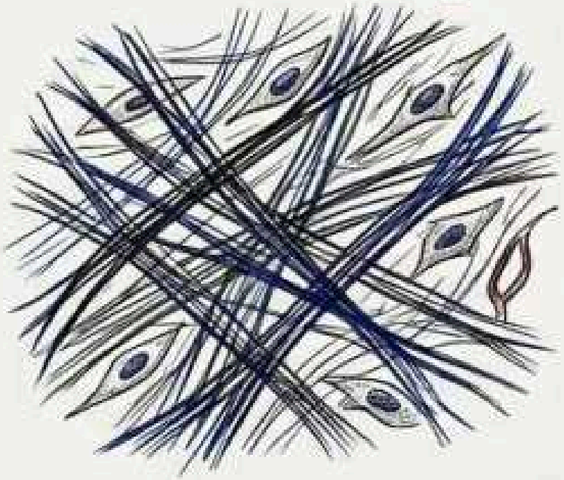


Neutrophil invasion.



Macrophage clearance.

Critical Warning: Tissue is exceptionally soft and weak—high risk of rupture!



Granulation tissue transitions into a firm fibrous scar.

Shock represents a systemic collapse of tissue perfusion

¶ 3.9

Core Definition: Systemic circulation failure; cells are starved of oxygen leading to rapid organ failure.

$$BP = CO \times SVR$$

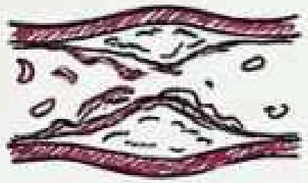
(Blood Pressure = Cardiac Output × Systemic Vascular Resistance)

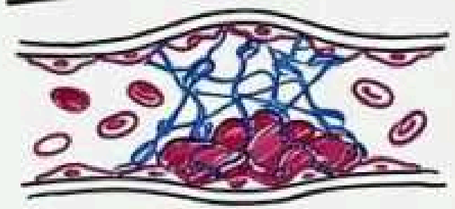
Cardiogenic	Hypovolaemic	Distributive
Heart fails as a pump (e.g., large heart attack).	Severe fluid loss (bleeding, dehydration).	Massive vasodilation (sepsis, anaphylaxis).
↓	↓	↓
Mathematically drops the CO	Mathematically drops the CO	Mathematically drops the SVR

Haemodynamic disorders form a continuous, lethal chain reaction

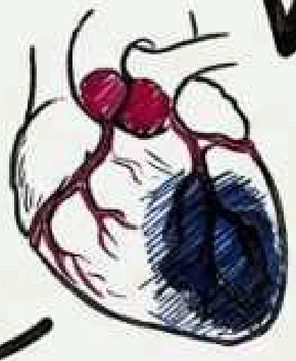
¶ 3.10

Step 1: Atherosclerosis damages the artery wall.

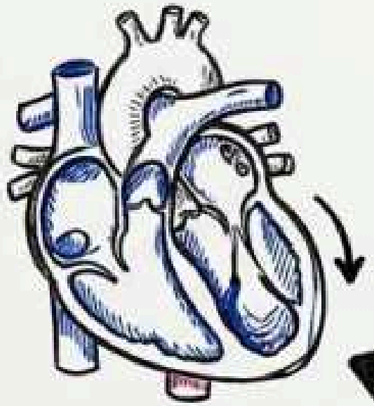




Step 2: Endothelial damage encourages Thrombosis.



Step 3: The thrombus blocks the artery, causing an Infarction (tissue death).



Step 4: Massive myocardial infarction leads directly to Cardiogenic Shock.

Instructor Note:

Following these chains of cause and effect is the essence of pathological reasoning.

Lecture 6 will explore this exact chain in deep detail.

Clinical application of thrombosis and embolism principles

¶ 3.11



The Scenario: A patient undergoes a long operation and spends several days in bed.

Question 1: Why are they at risk for deep vein thrombosis?

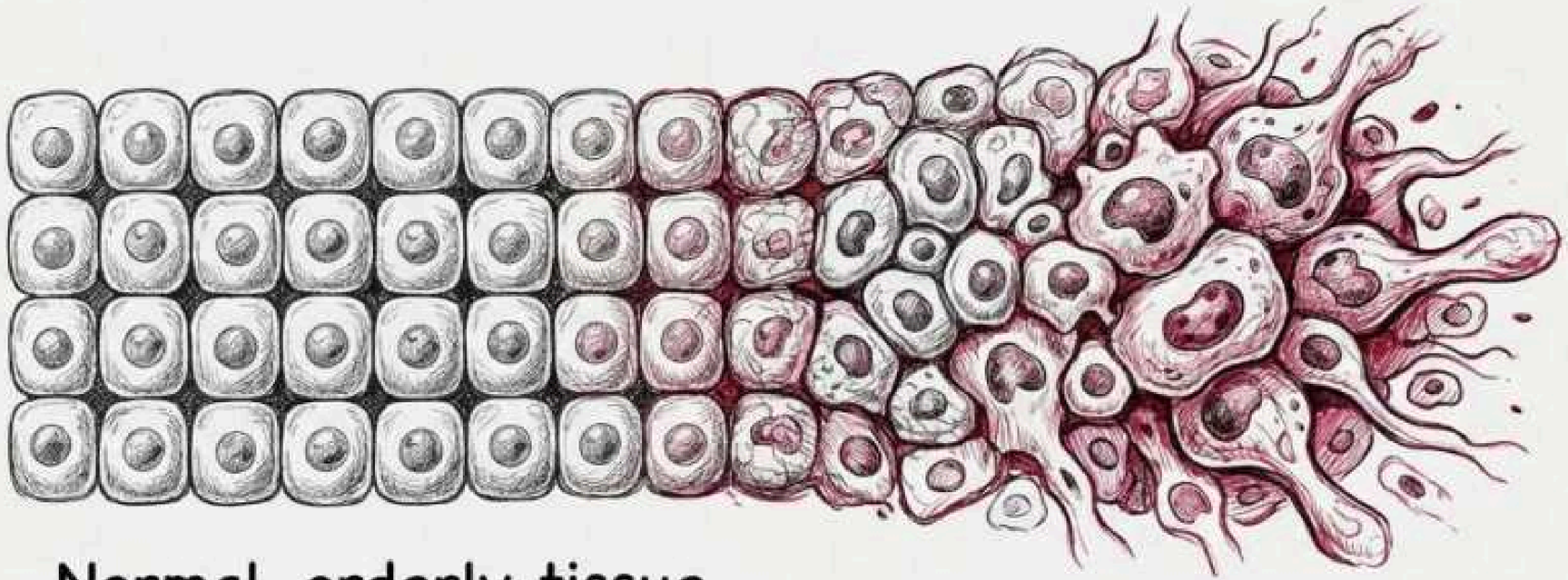
Answer 1: Virchow's Triad. Bed rest = stagnant flow. Surgery = hypercoagulable state.

Question 2: Why could that clot become dangerous elsewhere in the body?

Answer 2: Embolism. The clot can break off, travel through the right heart, and lodge in the lungs causing a fatal Pulmonary Embolism.

Pathology eventually shifts from flow disorders to abnormal growth

¶ 3.12



Normal, orderly tissue

Neoplasia (Cancer)

Looking Ahead: Lecture 4 will focus on Neoplasia.
Core Focus: The abnormal growth of tissue, marking the shift into the pathology of cancer.