

Reactions of Blood Vessels in Acute Inflammation

Vasodilation • permeability • exudation • stasis

An integrated vascular mechanism of acute inflammation

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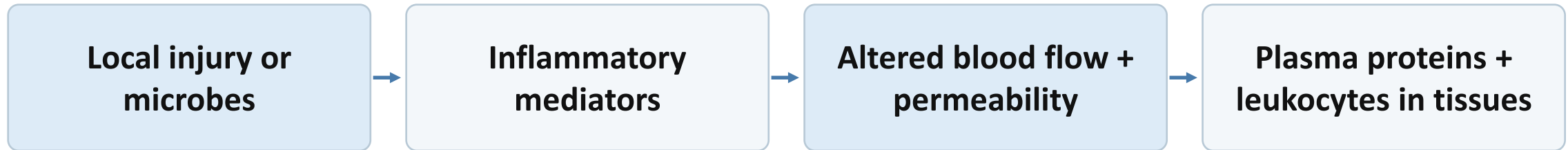
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Learning objectives

- Explain the changes in vascular caliber and blood flow after injury.
- Connect permeability changes with exudation and tissue edema.
- Distinguish exudates, transudates, edema, and purulent exudates.
- Explain how stasis favors leukocyte margination.

**From vascular change to
tissue defense**

Vascular responses deliver inflammatory defense



Local host defense requires regulated transfer across the microvascular wall.

Three major components of acute inflammation

Vascular dilation

Increases local blood flow

Microvascular leakage

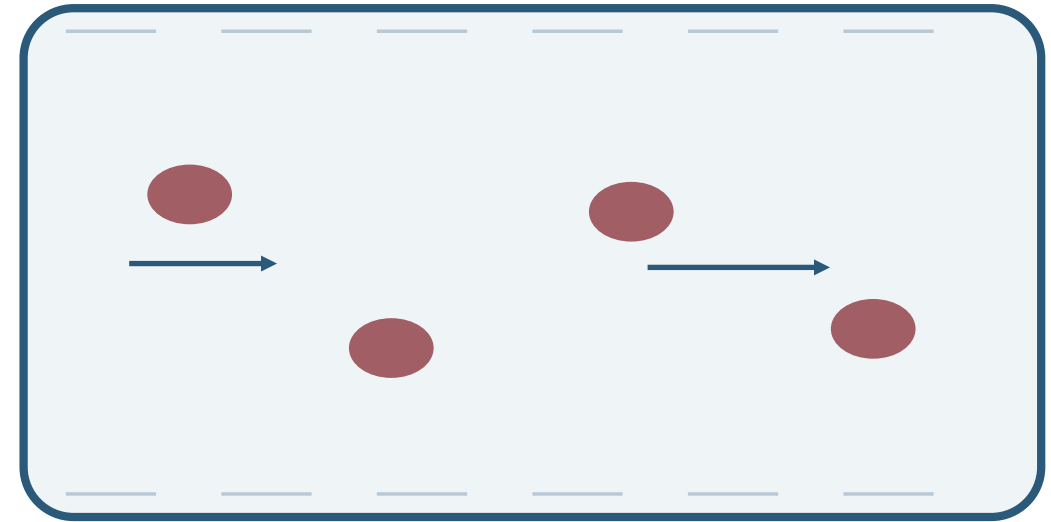
Allows plasma proteins
into tissue

Leukocyte recruitment

Delivers circulating
effector cells

A microvessel separates plasma from injured tissue

- The endothelial lining is the key interface.
- In normal conditions, most plasma proteins remain intravascular.
- Inflammation alters both local flow and endothelial barrier function.



Schematic microvessel (not histology)

Vascular reactions have two distinct axes

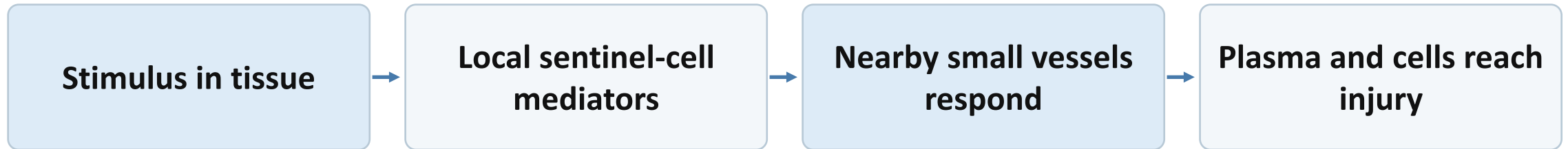
Hemodynamics

- Changes in vessel caliber
- Changes in blood flow
- Stasis and vascular congestion

Barrier function

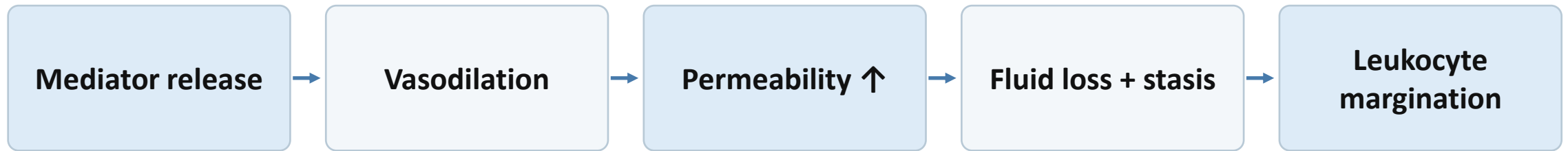
- Increased vascular permeability
- Loss of protein-rich fluid
- Edema and exudate formation

The response is spatially targeted



Circulating leukocytes and plasma proteins are redirected to the focus of injury.

Order of early vascular events



Vasodilation is an early vascular event

- Chemical mediators act on vascular smooth muscle.
- Affected small vessels increase in caliber.
- Local blood flow initially rises.
- This contributes directly to erythema and warmth.

**Early: Greater caliber →
greater local flow**

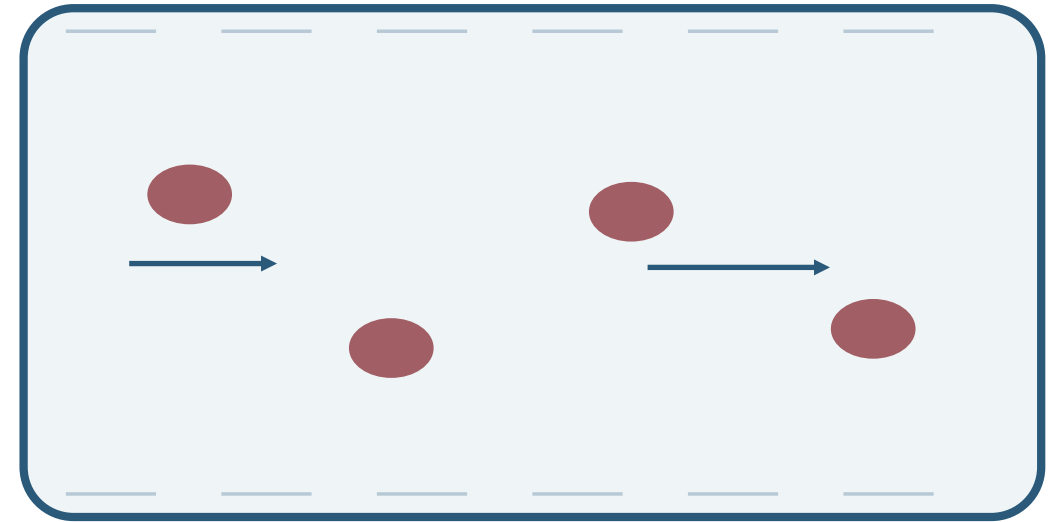
Histamine is a prominent vasodilator

- Histamine as a notable mediator.
- The mediator acts on vascular smooth muscle.
- Vasodilation begins soon after the inflammatory stimulus.
- The same mediator can also participate in vascular leakage.

**One mediator, multiple
vascular effects**

Postcapillary venules are central responders

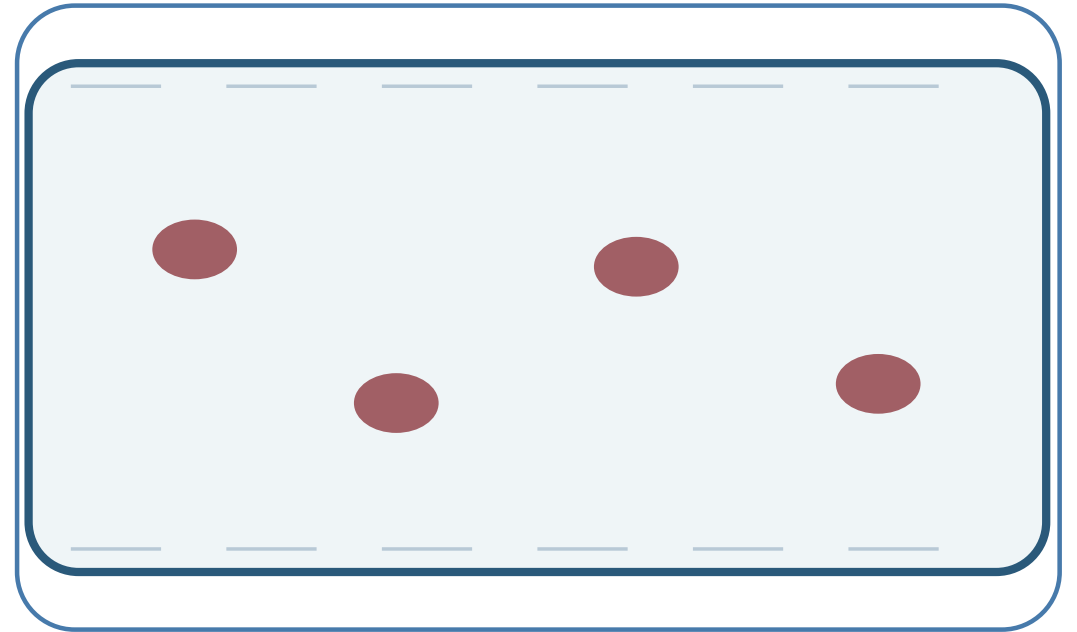
- The source emphasizes postcapillary venules.
- These vessels participate in permeability changes.
- They also provide an interface for leukocyte adhesion and exit.



Schematic microvessel (not histology)

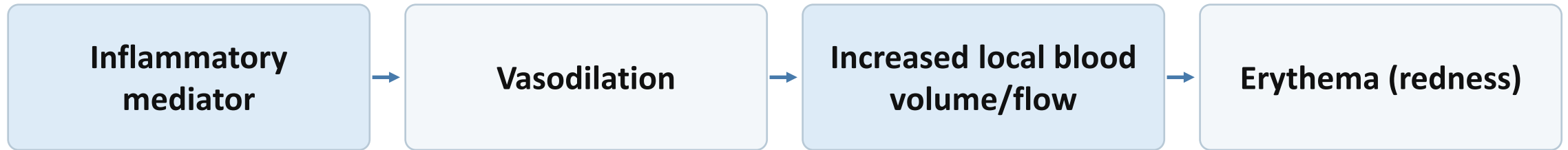
Increased caliber raises blood flow locally

- Vasodilation expands the functional vascular channel.
- More blood reaches the site of injury initially.
- The inflamed area therefore appears red and warm.



Schematic microvessel (not histology)

Why acute inflammation causes redness



Why the inflamed area feels warm

Dilation of small vessels



Increased warm blood delivery



Local heat (calor)

Hyperemia and congestion describe different flow states

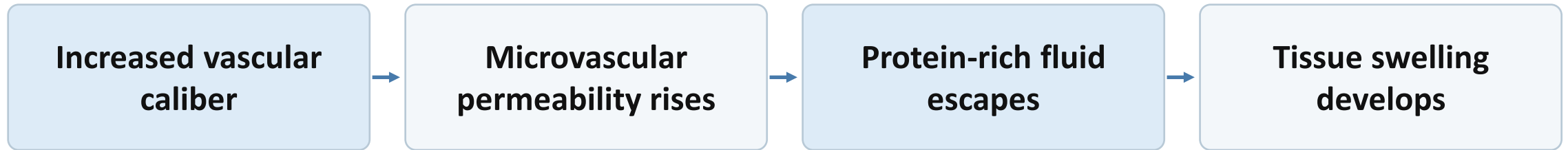
Early hyperemia

- Vessels dilate
- Local blood flow rises
- Supports warmth and redness

Developing congestion

- Microvascular flow slows
- Red cells accumulate in vessels
- Engorgement becomes apparent

Vasodilation precedes the major fluid leak



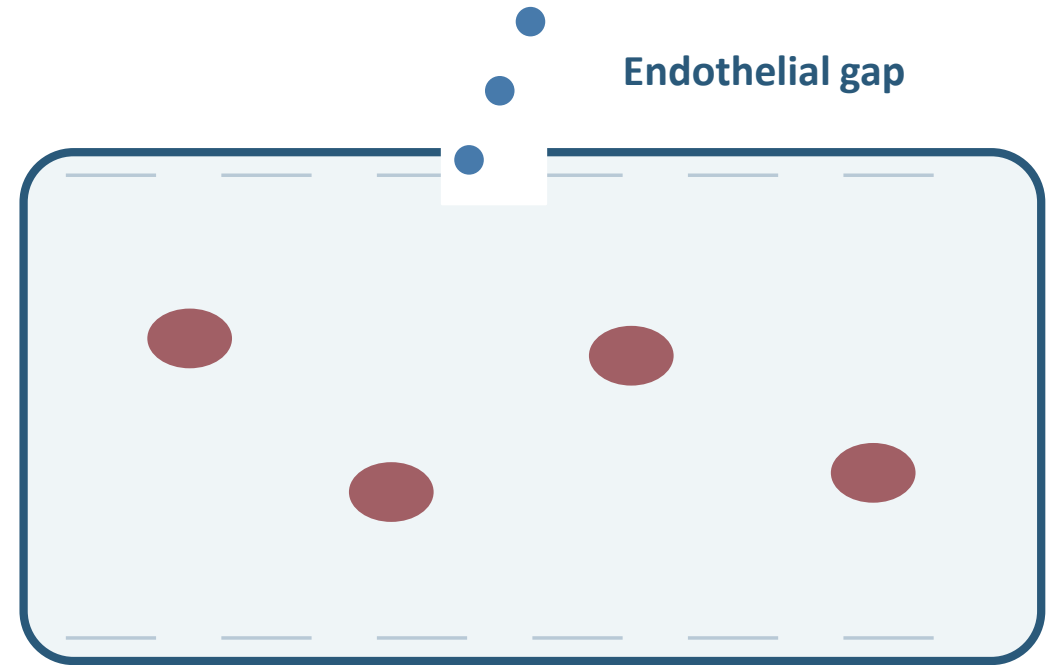
Microvascular permeability controls exudation

- Endothelial barrier integrity regulates protein escape.
- Inflammatory mediators can increase passage between endothelial cells.
- Injury can cause a more sustained barrier defect.
- Increased permeability is the defining vascular change in exudation.

**A protein-permeable
vessel generates an
exudate**

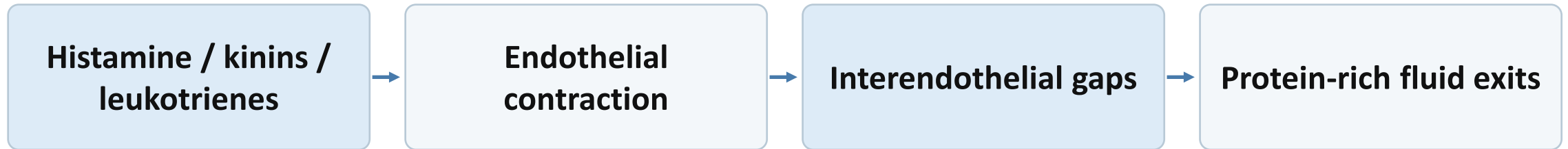
Inflammatory permeability is especially venular

- Postcapillary venules are a key leakage site.
- Junctional changes allow plasma components to exit.
- The response supplies tissue with soluble host-defense proteins.



Schematic microvessel (not histology)

Endothelial contraction creates temporary gaps



The immediate transient leak is short-lived

- Interendothelial gaps appear rapidly after mediator exposure.
- A usual duration of 15–30 minutes.
- The underlying mechanism is endothelial contraction.
- This is the most common described leakage mechanism.

**Immediate transient
response: Usually 15–30
min**

Different mediators can converge on a gap response

Histamine

Induces endothelial
contraction

Bradykinin

Promotes junctional
leakage

Leukotrienes

Can increase permeability

Delayed prolonged leakage has a different tempo

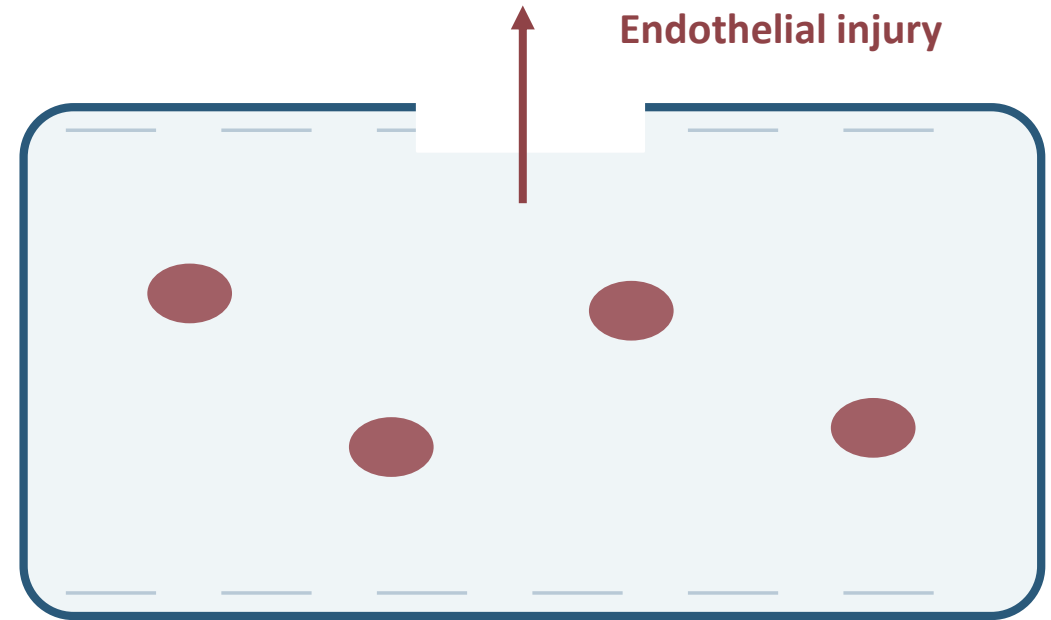
- In some mild injuries, leakage begins after 2–12 hours.
- It may continue for several hours or even days.
- Contraction or mild endothelial damage may contribute.
- Immediate and delayed leakage can lie on a continuum.

**Delayed onset does not
exclude inflammatory
leakage**

Sunburn illustrates delayed vascular leakage

Endothelial necrosis produces sustained leakage

- Severe insults can injure and detach endothelial cells.
- Direct barrier destruction permits vascular contents to escape.
- Leakage can continue until vessels thrombose or repair.



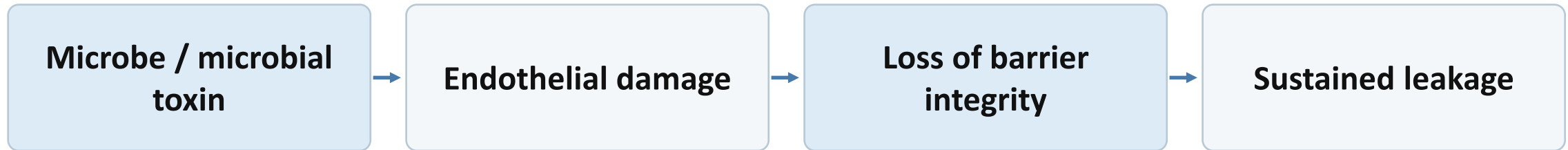
Schematic microvessel (not histology)

Thermal burns combine several leakage mechanisms

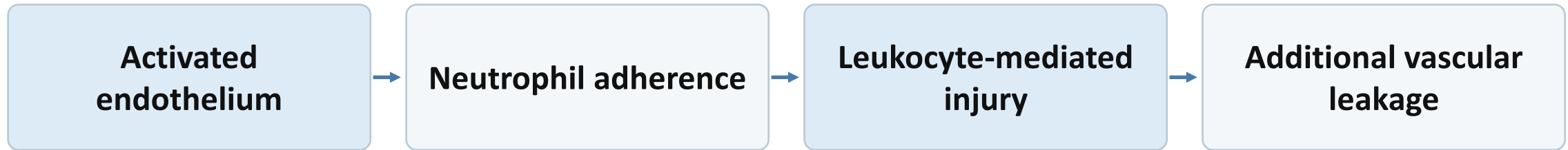
- Chemically mediated endothelial contraction may occur first.
- Direct endothelial injury can extend the permeability defect.
- Adherent leukocytes may add endothelial damage.
- Extensive leakage can cause dangerous fluid loss.

**Severe burns can produce
life-threatening fluid
depletion**

Microbes and toxins may directly injure endothelium



Adherent neutrophils can amplify vascular injury



Vascular permeability mechanisms can overlap

Mediator-driven contraction

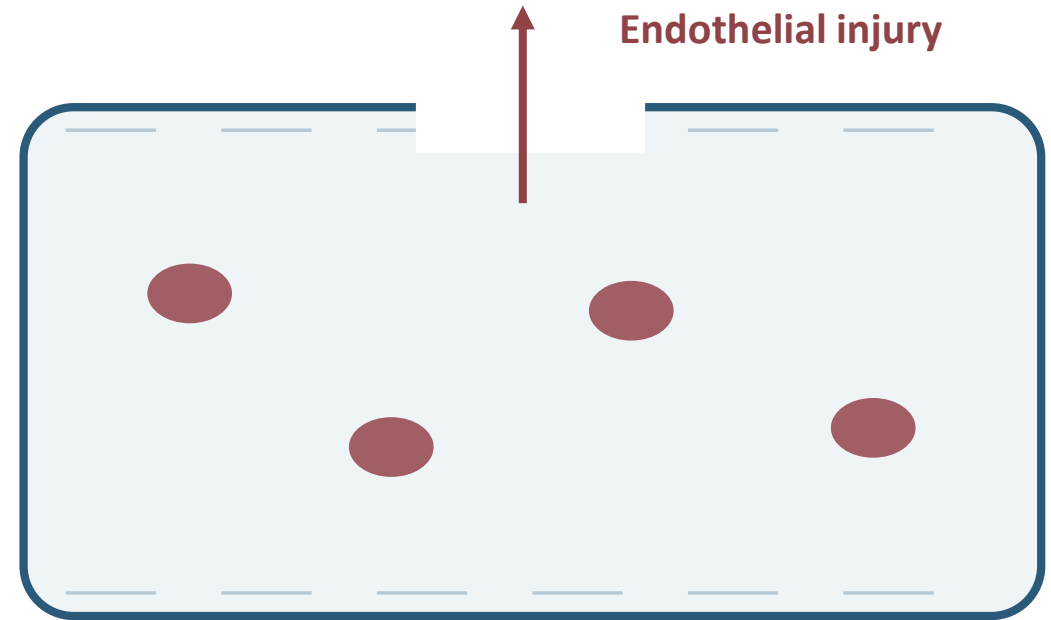
- Endothelium retracts
- Gaps are created
- Often rapid and transient

Endothelial cell injury

- Cells may undergo necrosis
- Cells detach from vessel wall
- Leakage may be sustained

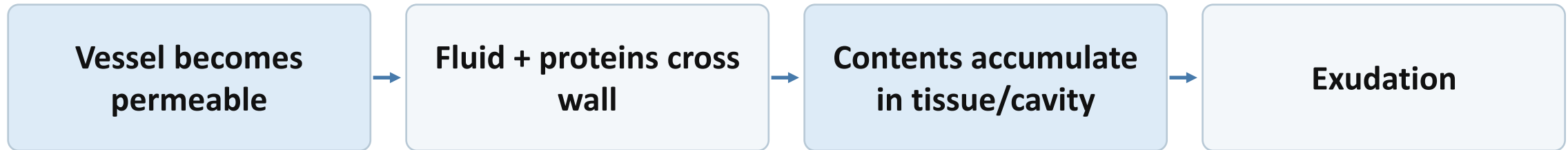
An injured vessel is not equivalent to a retracted vessel

- Contraction preserves living endothelial cells.
- Injury disrupts or removes part of the endothelial lining.
- The mechanism influences the duration of the leak.



Schematic microvessel (not histology)

Exudation is escape from the vascular compartment



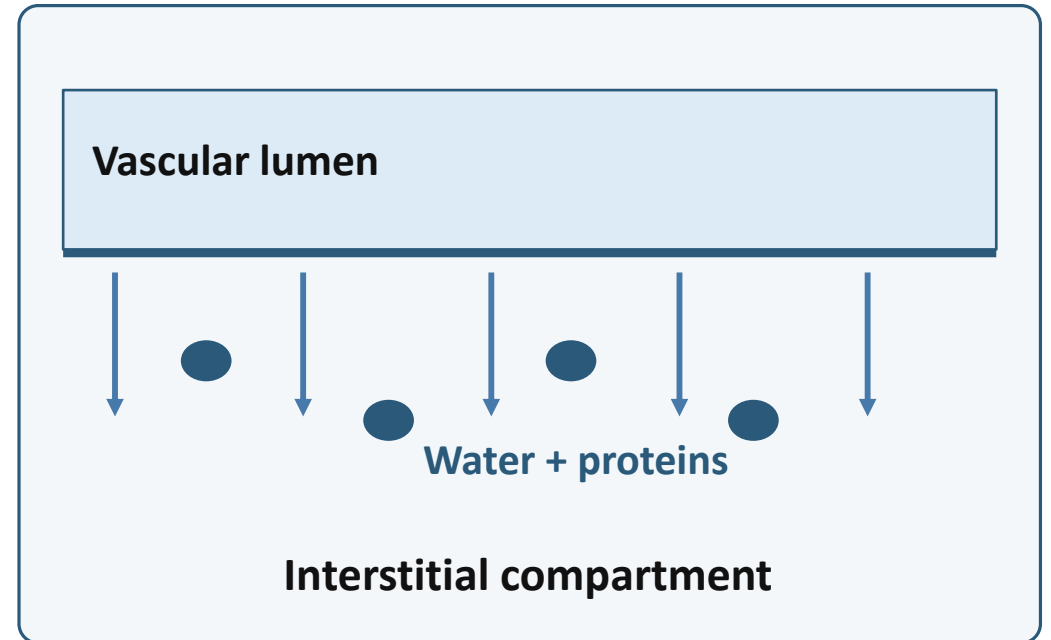
Exudates indicate an inflammatory permeability defect

- An exudate is an extravascular protein-rich fluid.
- It may contain cellular debris and sometimes blood cells.
- Its presence implies increased permeability of small vessels.
- Exudation connects vascular alteration to tissue findings.

**Exudate → inflammatory
vessel leak**

Exudates contain fluid and plasma proteins

- Inflammation allows more than water to leave vessels.
- Plasma proteins move into the interstitial space.
- An inflammatory exudate can also contain cell fragments.



The protein content distinguishes exudates

Exudate

- Relatively protein-rich
- Inflammatory permeability increased
- Cells or debris may be present

Transudate

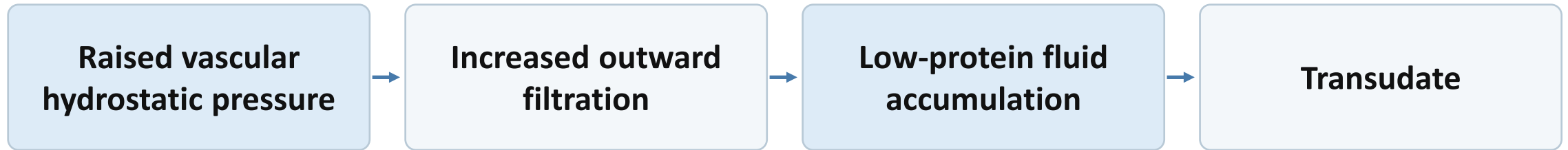
- Relatively protein-poor
- No primary permeability increase
- Few or no cells

A transudate is essentially a plasma ultrafiltrate

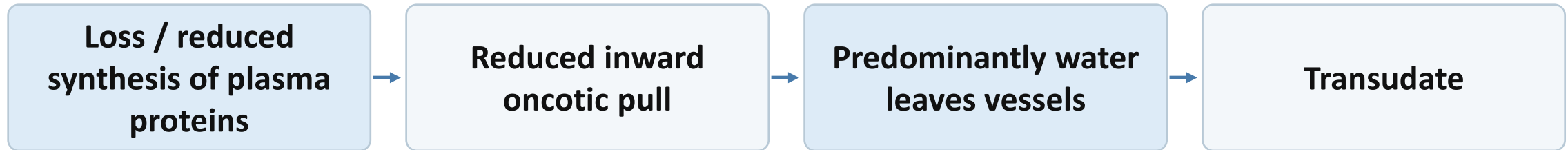
- Fluid escapes without an inflammatory barrier defect.
- Protein content is low, largely albumin.
- Little or no cellular material is present.
- Pressure or osmotic imbalance drives formation.

**Transudate → hydrostatic
/ oncotic imbalance**

Hydrostatic pressure can cause transudation



Reduced colloid osmotic pressure also causes transudation



Typical pressure-related transudate settings

Venous outflow obstruction

Raised hydrostatic
pressure

Liver disease

Reduced protein synthesis

Kidney disease

Increased protein loss

Exudate and transudate: Causal comparison

Feature	Exudate	Transudate
Primary disturbance	Endothelial permeability ↑	Pressure / osmotic imbalance
Protein content	High	Low
Cells / debris	May be present	Little or none
Main implication	Inflammatory process	Noninflammatory fluid shift
Vascular wall	More permeable	No primary increase in permeability

Edema means excess extravascular fluid

- Edema can occur in interstitial tissue.
- It may also collect in serous cavities.
- Edema describes the location and volume of fluid, not its composition.
- Either an exudate or a transudate can produce edema.

**Edema is not synonymous
with exudate**

Two distinct pathways can produce edema

Inflammatory edema

- Vascular permeability increases
- Proteins accompany fluid
- Exudate forms

Noninflammatory edema

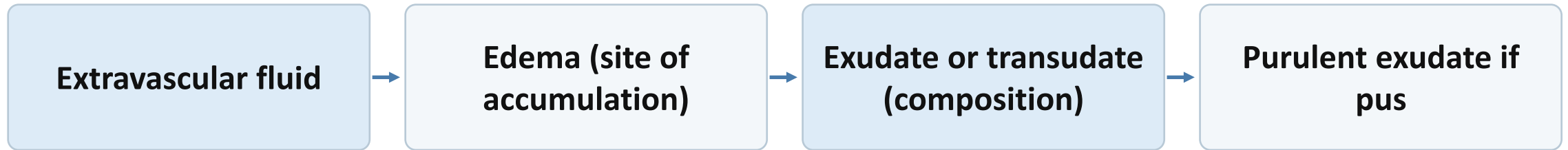
- Hydrostatic / oncotic imbalance
- Mainly fluid leaves vessels
- Transudate forms

A purulent exudate is not merely watery edema

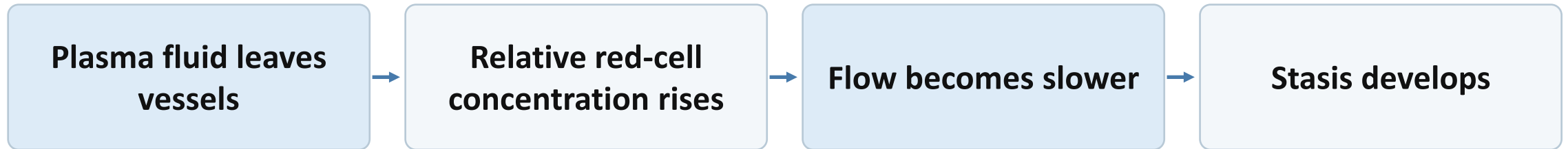
- Pus is an inflammatory exudate.
- It is rich in leukocytes, mainly neutrophils.
- Dead-cell debris is present.
- Microbes are present in many, but not necessarily all, cases.

**Pus = neutrophils +
necrotic debris ± microbes**

Fluid terminology follows a hierarchy



Loss of plasma fluid changes microvascular flow



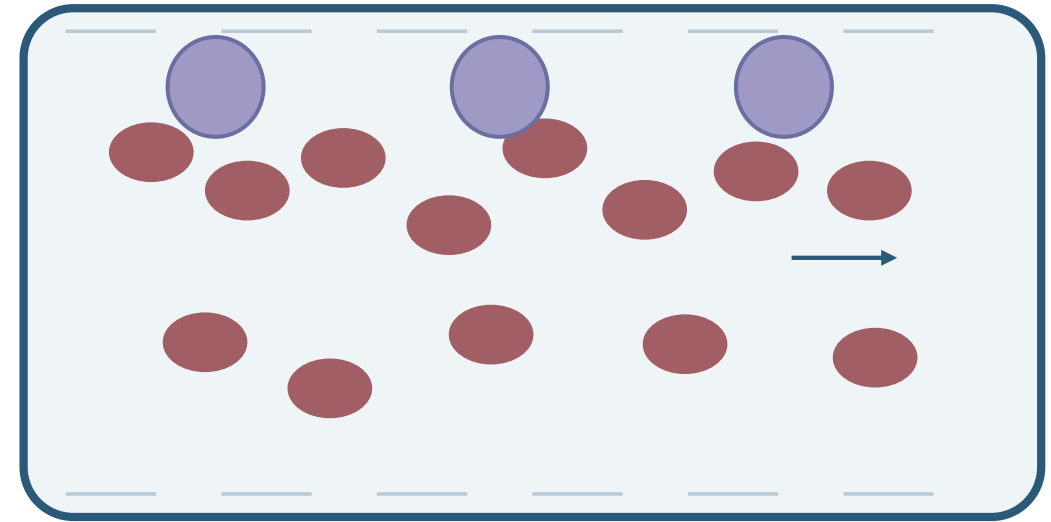
Hemoconcentration accompanies vascular leakage

- Inflammatory fluid loss removes plasma from the vessel.
- Red cells become more concentrated in remaining blood.
- The dilated vessel now contains more slowly moving blood.
- These changes promote microvascular stasis.

**Plasma loss + dilation →
slower flow**

Stasis develops after the early hyperemic phase

- Blood flow progressively slows in small vessels.
- Red cells accumulate within the dilated vascular lumen.
- Vessels appear engorged, a state described as congestion.



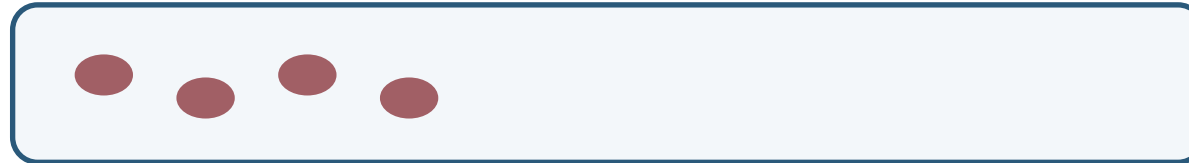
Schematic microvessel (not histology)

The hemodynamic sequence is not static

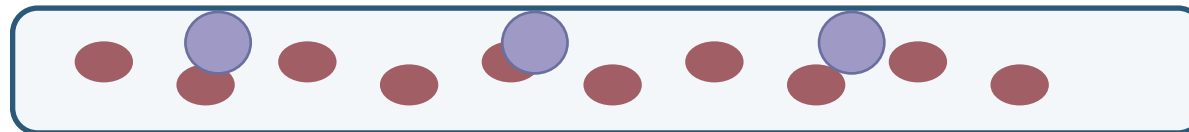
Normal flow



Early vasodilation



Later stasis + margination



Schematic; Vessel diameter and cell density are illustrative, not quantitative

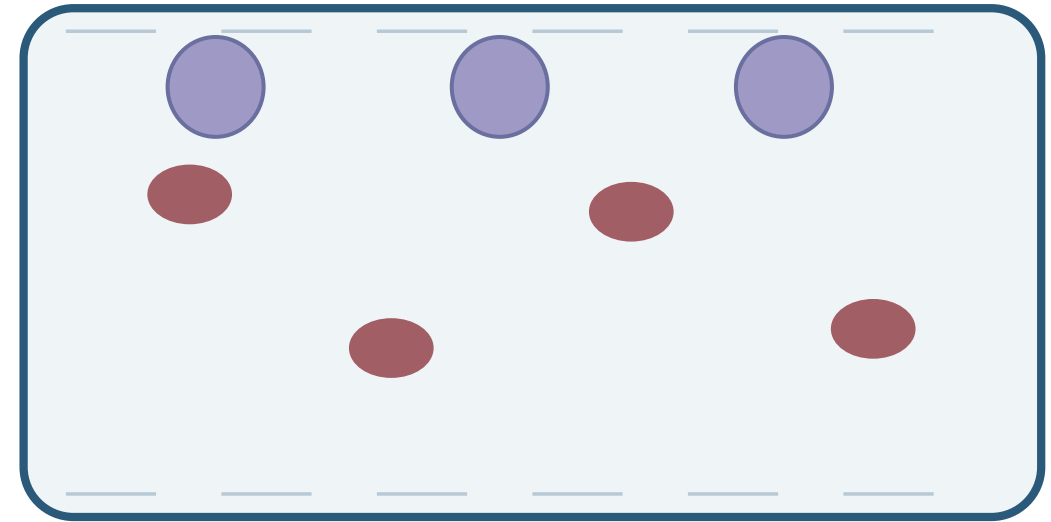
Vascular congestion is a histologic observation

- Congestion means small vessels are engorged with erythrocytes.
- It reflects accumulation of slowly moving red cells.
- The accompanying edema may separate tissue structures.
- The diagram is schematic rather than an actual H&E image.

**Morphologic correlate:
Engorged small vessels**

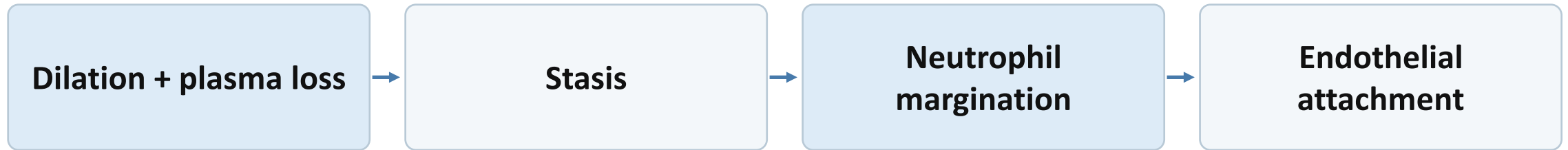
Stasis shifts leukocytes toward endothelium

- Slower flow favors peripheral positioning of leukocytes.
- Neutrophils collect along the endothelial surface.
- This redistribution is termed margination.



Schematic microvessel (not histology)

Margination bridges vascular and cellular responses

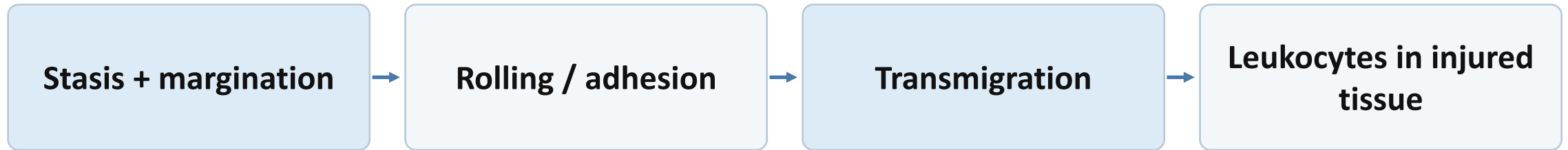


Endothelial activation enables firm cellular contact

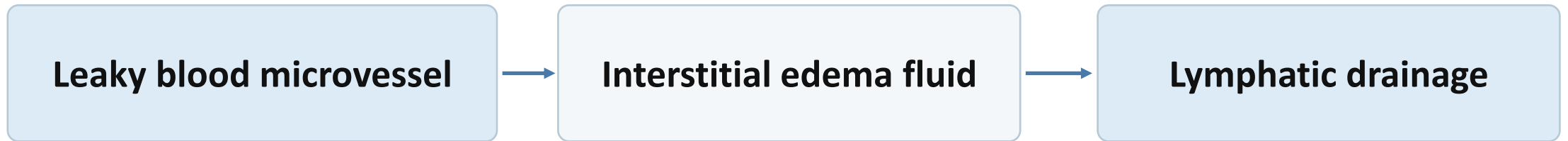
- Injury-associated mediators activate endothelial cells.
- Endothelial adhesion-molecule expression rises.
- Leukocytes attach to the endothelium.
- The next phase is migration across the vessel wall.

Vascular changes set the stage for leukocyte exit

Vascular change precedes leukocyte emigration



The lymphatic system helps clear extravascular fluid



Increased lymph flow can remove some of the accumulating extravascular fluid.

Lymph flow rises during acute inflammation

- Lymphatics normally collect small amounts of extravascular fluid.
- Inflammatory edema increases the drainage load.
- Lymph can carry leukocytes, debris, and microbes.
- This provides context for spread toward draining nodes.

Lymph drainage is a companion, not a substitute, for vascular defense

Inflamed lymphatics and nodes can become clinically visible

- Lymphangitis denotes inflammation of lymphatic vessels.
- Lymphadenitis denotes inflammation of lymph nodes.
- A red streak extending from a wound suggests lymphangitis.
- Enlarged painful draining nodes can accompany it.

Regional lymphatic findings can indicate local infection

Redness and swelling arise by different mechanisms

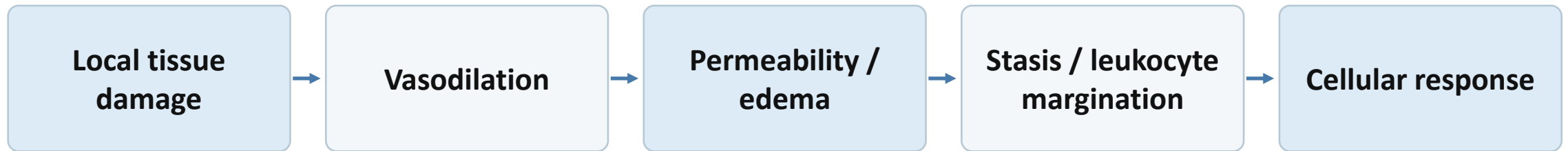
Redness and warmth

- Early vasodilation
- Greater local blood flow
- Engorgement may persist during stasis

Swelling

- Increased vascular permeability
- Protein-rich fluid enters tissue
- Edema becomes visible

Small local inflammation produces a coordinated response



Severe systemic fluid loss is a possible consequence

- Barrier disruption may involve many vessels in severe burns.
- Large quantities of plasma fluid may leave the circulation.
- Vascular reactions that protect locally can be harmful when extensive.
- Potentially life-threatening fluid loss.

**Extent of leakage
determines systemic
significance**

Clinical interpretation: An erythematous warm wound

Clinical setting

Following a superficial injury, the surrounding skin becomes red and warm before pronounced swelling develops.

Interpretation question

Which vascular event is most directly responsible for the initial signs?

Source-based explanation

Early mediator-induced vasodilation with increased local blood flow.

Erythema and warmth precede or accompany the later permeability-related swelling.

Clinical interpretation: Delayed sunburn edema

Clinical setting

After ultraviolet exposure, local tissue swelling develops several hours after the exposure.

Interpretation question

Is a delayed leak necessarily noninflammatory?

Source-based explanation

No. It is compatible with delayed prolonged inflammatory leakage.

Robbins explicitly describes sunburn as a delayed-onset permeability response.

Clinical interpretation: Fluid in heart failure

Clinical setting

A patient with venous congestion develops a relatively protein-poor edema fluid without local vascular injury.

Interpretation question

Is the fluid best interpreted as exudate or transudate?

Source-based explanation

Transudate.

A hydrostatic pressure abnormality can generate low-protein fluid without increased vessel permeability.

Clinical interpretation: A purulent wound

Clinical setting

A wound contains thick fluid rich in neutrophils and necrotic cellular debris.

Interpretation question

Which form of extravascular fluid is present?

Source-based explanation

Purulent inflammatory exudate (pus).

The cellular composition identifies pus rather than simple transudative edema.

Clinical interpretation: Major thermal injury

Clinical setting

A patient with extensive burns develops widespread tissue swelling and substantial intravascular fluid loss.

Interpretation question

Why can local permeability mechanisms become dangerous?

Source-based explanation

Extensive, sustained plasma leakage reduces circulating fluid volume.

Endothelial contraction, direct injury, and leukocyte-mediated injury may all contribute.

A single causal chain explains many findings



Distinguish a permeability defect from a pressure defect

Finding	Interpretation	Reason
Protein-rich swelling	Inflammatory exudation	Barrier permeability increases
Low-protein swelling	Transudation	Hydrostatic / oncotic shift
Slow red-cell-filled venules	Congestion / stasis	Plasma loss and dilation
Neutrophils along endothelial wall	Margination	Slowing changes leukocyte distribution

Assessment: Which finding implies vascular leakage?

An extravascular fluid has high protein content and scattered cell debris. What does this imply?

A. Isolated increase in hydrostatic pressure

B. Increased permeability of small vessels

C. Reduced plasma protein synthesis alone

D. An unchanged endothelial barrier

Rationale: High-protein inflammatory exudates indicate permeability increase.

Assessment: What prepares neutrophils for adhesion?

After vasodilation and plasma-fluid loss, which event positions neutrophils near the vessel wall?

A. Transudation

B. Margination during stasis

C. Immediate endothelial thrombosis

D. Reduced endothelial adhesion

Rationale: Slowed blood flow shifts leukocytes to the endothelial periphery.

Five distinctions to retain

Dilation

Initial $\uparrow$ local flow $\rightarrow$
warmth/redness

Leakage

Plasma proteins escape $\rightarrow$
exudate

Edema

Excess extravascular fluid
of either type

Stasis

Flow slows; Red cells
concentrate

Margination

Leukocytes move toward
endothelium

Primary source and figure provenance

Core reading

- Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th edition (2026).
- Chapter 3: Inflammation and Repair; acute inflammation, pp. 67–69.
- Assigned section: Reactions of Blood Vessels in Acute Inflammation.

All instructional drawings were constructed as native PowerPoint schematics; no synthetic micrographs or unlicensed pathology images are used.