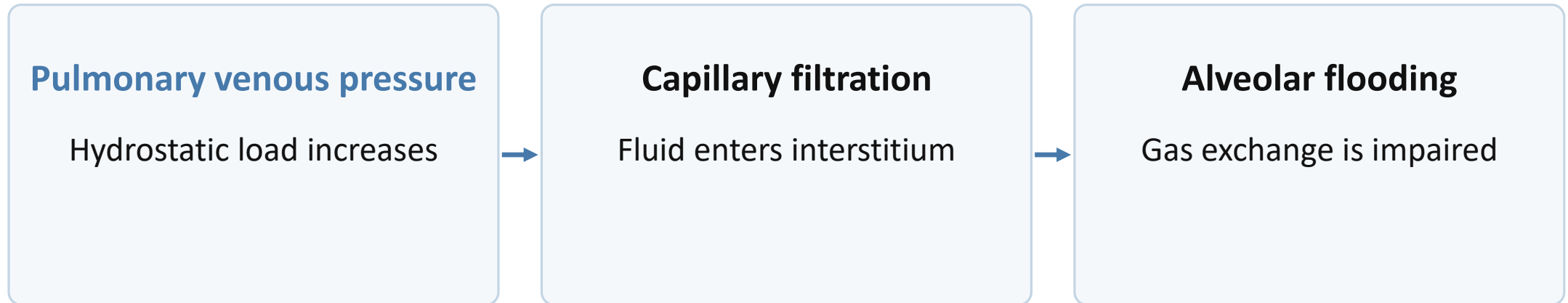


Hemodynamic Pulmonary Edema

Mechanism, morphology, and clinicopathologic interpretation



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Learning objectives

- Distinguish hemodynamic pulmonary edema from increased-permeability edema.
- Trace elevated pulmonary venous pressure to interstitial and alveolar fluid accumulation.
- Recognize acute and chronic pulmonary congestion on gross and microscopic grounds.
- Explain why edema causes hypoxemia, dyspnea, basal crackles, and susceptibility to infection.

Pulmonary edema: The defining compartment

Excess extravascular lung fluid

Pulmonary edema compromises normal air-filled alveoli by accumulating fluid first in the interstitial compartment and, when more severe, in alveolar spaces.

Pulmonary congestion often accompanies edema, but congestion denotes venous blood pooling—not fluid itself.

Two distinct pathogenetic routes

Hemodynamic / hydrostatic

- Increased vascular hydrostatic pressure or reduced oncotic pressure.
- Fluid is typically protein poor (transudate).
- Most often associated with left-sided heart failure.

Microvascular / alveolar injury

- Endothelial or epithelial injury increases permeability.
- Inflammatory protein-rich exudate leaks out.
- Diffuse alveolar damage belongs to a separate topic.

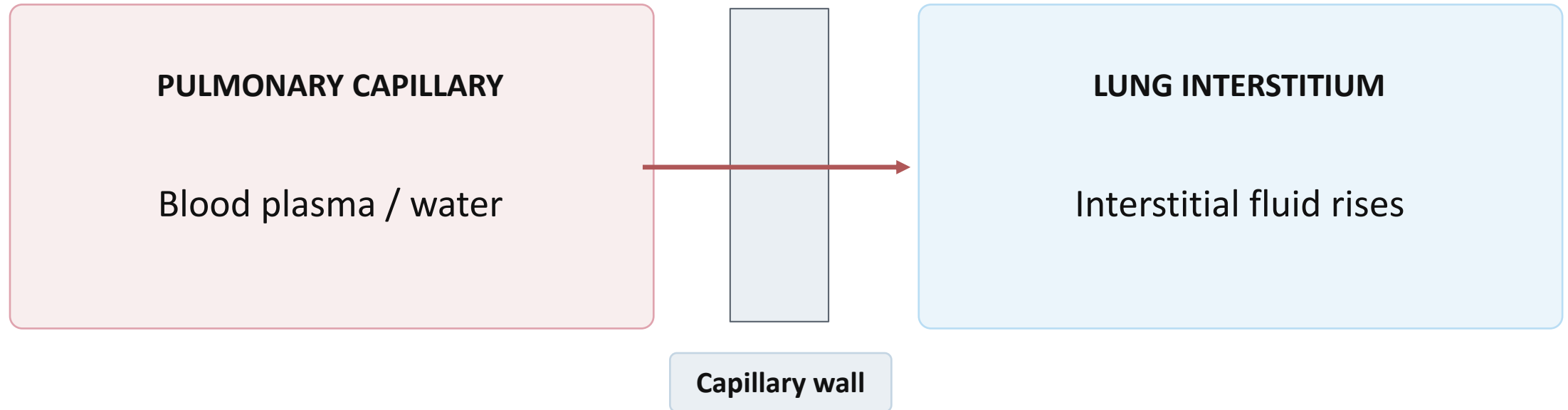
Normal lung: Fluid is continually cleared



Edema develops when net filtration exceeds the capacity for fluid clearance.

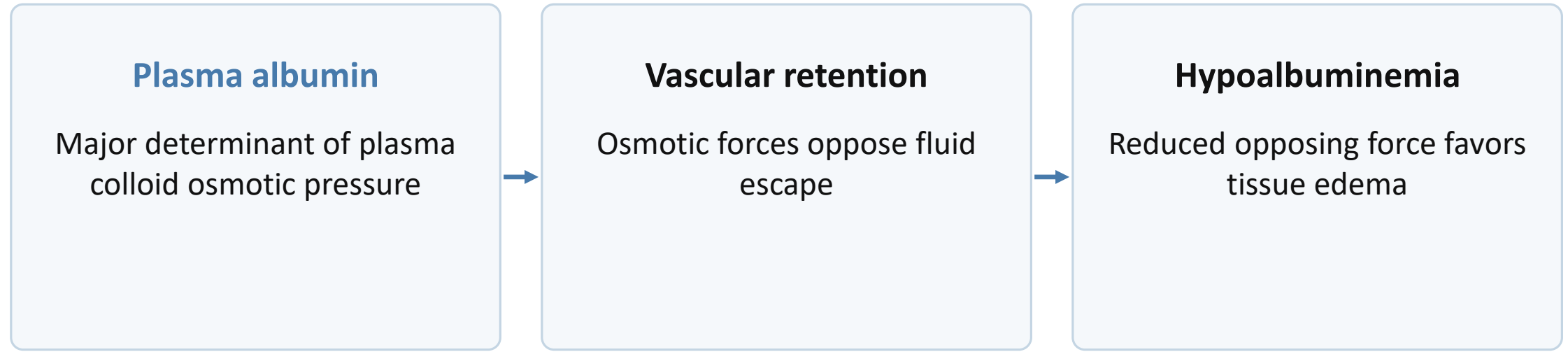
Hydrostatic pressure promotes filtration

Higher pulmonary capillary hydrostatic pressure



Net filtration is from capillary lumen across the vessel wall into lung interstitium.

Albumin opposes outward filtration



Loss of albumin can cause edema even without primary left-ventricular failure.

Lymphatic drainage provides a reserve

Interstitial filtration

Low-volume fluid entry is physiologic



Lymphatic removal

Returns excess interstitial fluid



Overwhelmed clearance

Visible edema develops when influx exceeds removal

Edema requires a net fluid imbalance

Filtration into lung interstitium

>

Lymphatic removal



Interstitial accumulation → alveolar edema

A rise in hydrostatic pressure is the characteristic primary disturbance.

Causes: Three hemodynamic categories

Raised hydrostatic pressure

Left-sided heart failure (common), volume overload, pulmonary vein obstruction.

Reduced oncotic pressure

Hypoalbuminemia from renal, hepatic, or gastrointestinal disease (less common).

Lymphatic obstruction

Impaired lymphatic fluid clearance is rare as a cause of pulmonary edema.

Left-sided heart failure is the leading cause

Impaired left-heart function

Elevated left-sided filling pressure



Pulmonary venous congestion

Blood backs into lung circulation



Raised capillary pressure

Edema fluid leaves the microvasculature

Heart failure causes pulmonary edema by its backward-failure component.

Cardiac diseases upstream of congestion

Ischemic heart disease

Myocardial ischemia or infarction reduces effective ventricular function.

Pressure or valve disease

Hypertension and aortic or mitral valve disease raise left-sided workload or pressure.

Myocardial disease

Intrinsic myocardial disease also compromises effective left-heart pump function.

Systolic dysfunction: Impaired ejection

Reduced LV ejection

Systolic pump function
deteriorates



Raised residual filling load

Left-sided pressures increase

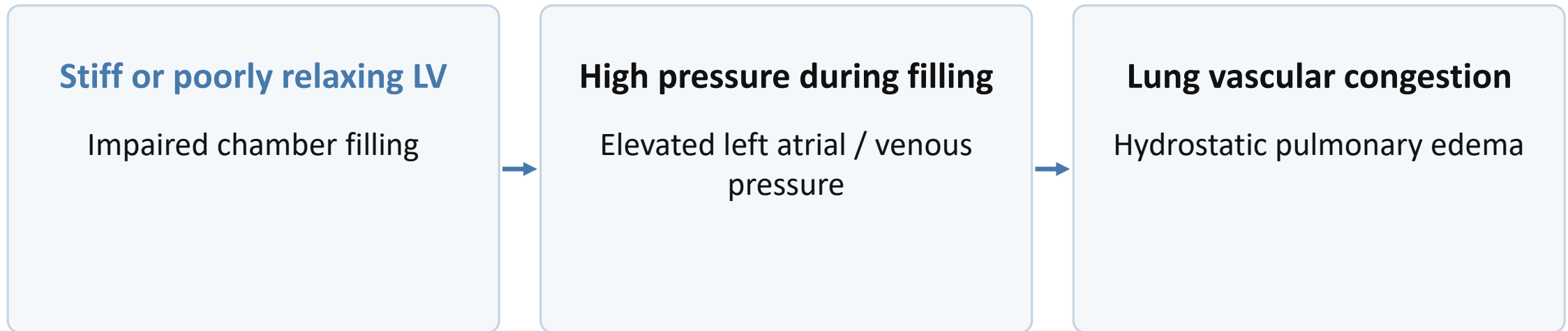


Pulmonary venous backup

Capillary hydrostatic load
increases

Pulmonary edema is a pressure consequence; It is not confined to a single ejection-fraction category.

Diastolic dysfunction can also cause edema



Normal or preserved ejection fraction does not exclude a hydrostatic mechanism.

Mitral valve disease: A pulmonary pressure load

Mitral stenosis

Resistance to left atrial emptying



Left atrial pressure rises

Pressure transmitted to pulmonary veins



Chronic venous congestion

Hemosiderin and septal fibrosis can accumulate

Pressure transmits backward to alveoli

- 01 Left ventricular dysfunction or obstruction to left atrial outflow
- 02 Left atrial and pulmonary venous pressures become elevated
- 03 Alveolar capillary beds become congested and hydrostatic pressure rises
- 04 Interstitial edema develops; Severe edema extends into airspaces

Why left- and right-sided failure differ

Predominant left-sided failure

- Pulmonary venous congestion is prominent.
- Interstitial and intraalveolar edema may result.
- Cough and dyspnea are early effects.

Predominant right-sided failure

- Systemic and portal venous engorgement dominate.
- Peripheral edema and visceral congestion may occur.
- Primary pulmonary congestion is generally minimal.

Volume overload amplifies pulmonary edema

Volume overload

Expanded intravascular fluid
volume



Increased vascular pressure

Pulmonary circulation becomes
congested



Transudation

Interstitial and alveolar fluid
increase

Renal compensation becomes maladaptive

Reduced cardiac output

Renal blood flow may fall



Neurohumoral activation

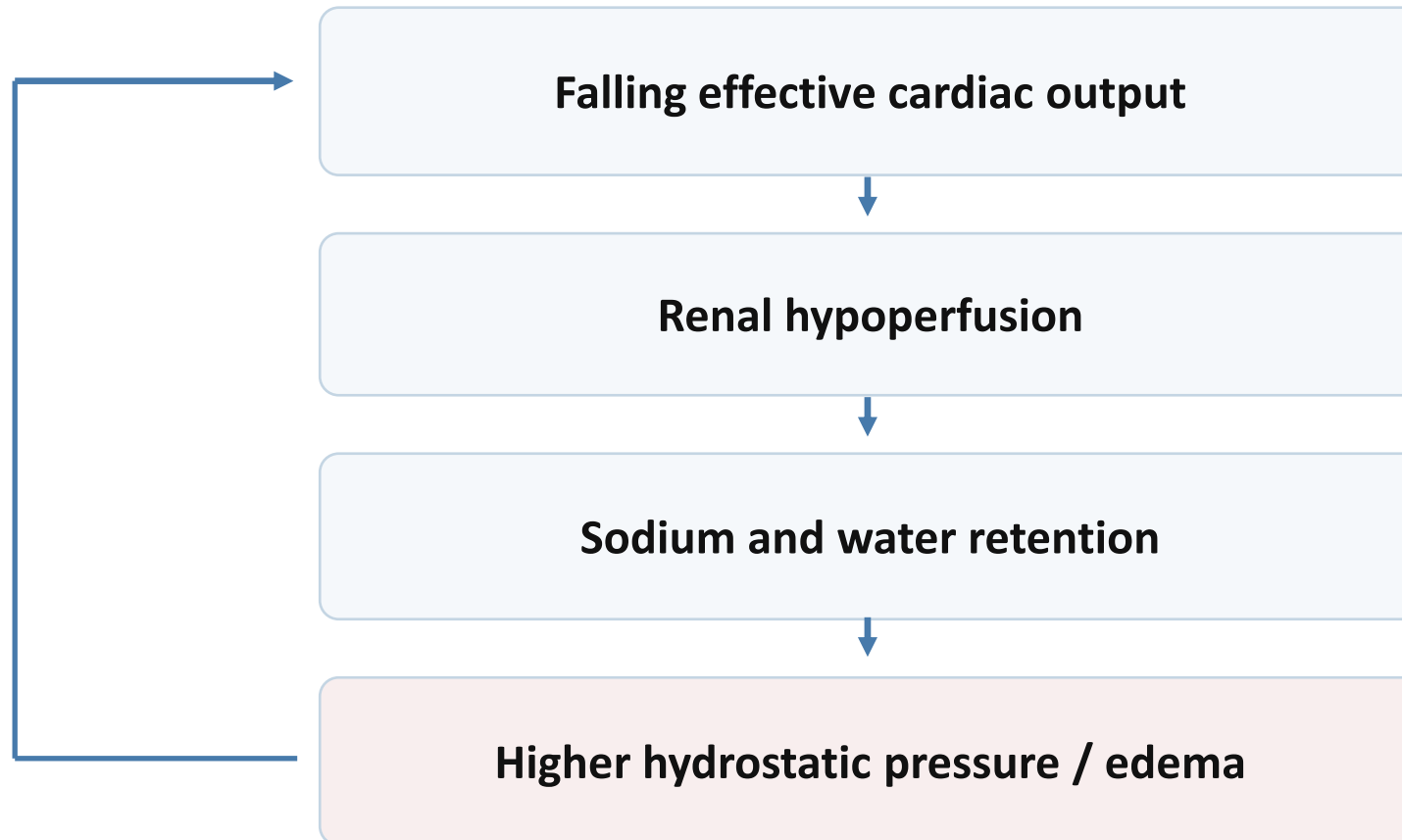
Renin–angiotensin–aldosterone
pathway



Sodium + water retention

Volume expansion can
exacerbate edema

The congestion–retention feedback loop



Progressive cardiac dysfunction sustains the cycle

Reduced oncotic pressure: A second pathway

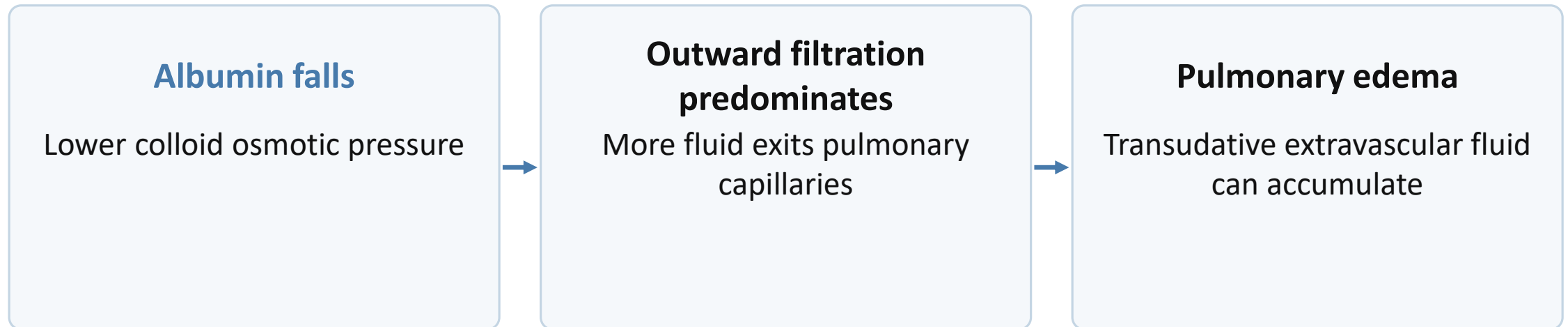


Table 15.1 classifies this route as less common than elevated hydrostatic pressure.

Nephrotic syndrome lowers plasma albumin

Glomerular protein loss

Albumin escapes into urine



Plasma albumin decreases

Lower colloid osmotic pressure



Edema

Enhanced movement of fluid
into tissues

Liver disease can reduce albumin synthesis

Severe liver disease

Impaired albumin synthesis



Low plasma albumin

Reduced intravascular oncotic pressure



Fluid accumulation

Systemic or pulmonary edema may develop

Protein-losing enteropathy: Intestinal loss

Gastrointestinal protein loss

Enteric depletion of plasma proteins



Hypoalbuminemia

Lower vascular oncotic pressure



Edema tendency

Fluid leaves the circulation more readily

Lymphatic obstruction is an uncommon cause

Normal lymphatic function

- Excess interstitial fluid is continuously removed.
- Drainage helps prevent edema despite physiologic filtration.

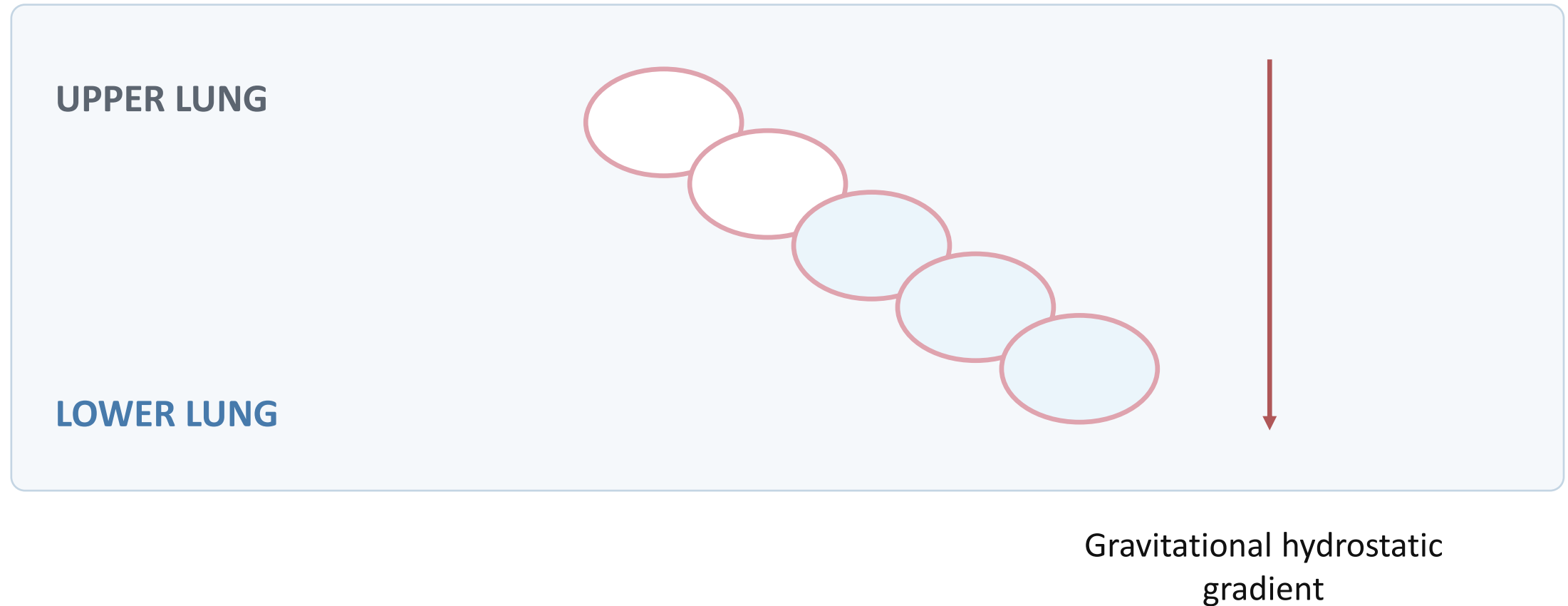
Lymphatic obstruction

- Impaired clearance permits tissue fluid accumulation.
- Pulmonary lymphatic obstruction as rare.

Etiologic hierarchy Table 15.1

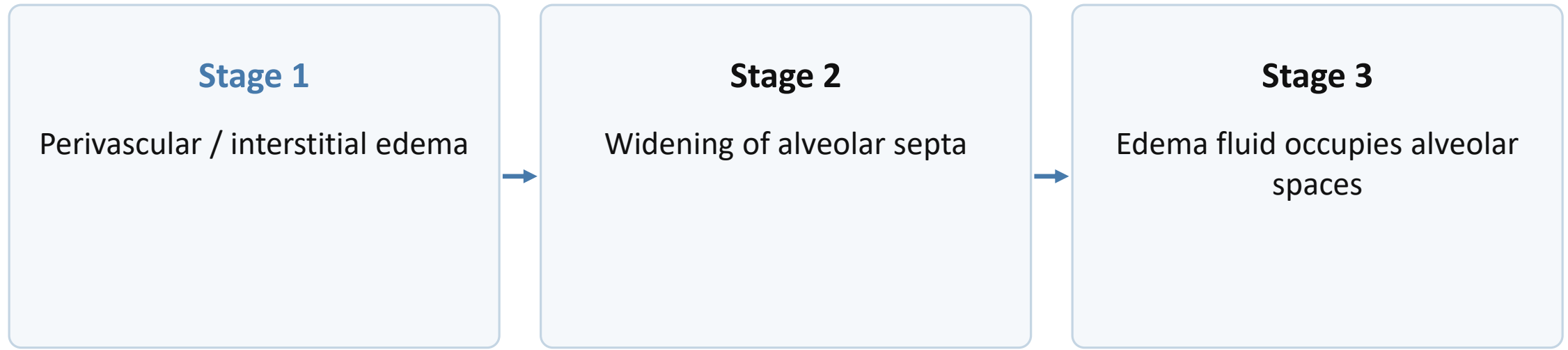
Mechanism	Examples	Relative emphasis
Increased hydrostatic pressure	Left-sided failure; Volume overload; Pulmonary vein obstruction	Most typical
Reduced oncotic pressure	Nephrotic syndrome; Liver disease; Protein-losing enteropathy	Less common
Lymphatic obstruction	Impaired lymphatic fluid removal	Rare

Dependent edema starts at the lung bases



Edema appears first in basal, dependent regions because pressure is highest there.

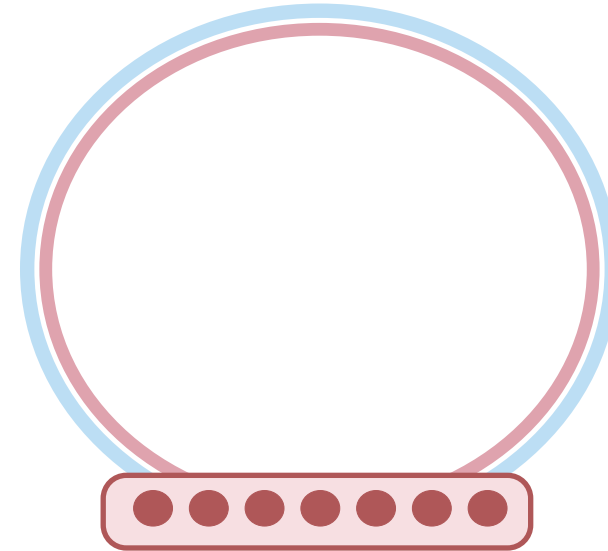
Fluid follows a recognizable sequence



The stages describe increasing fluid accumulation, not separate diseases.

Early edema involves the interstitium

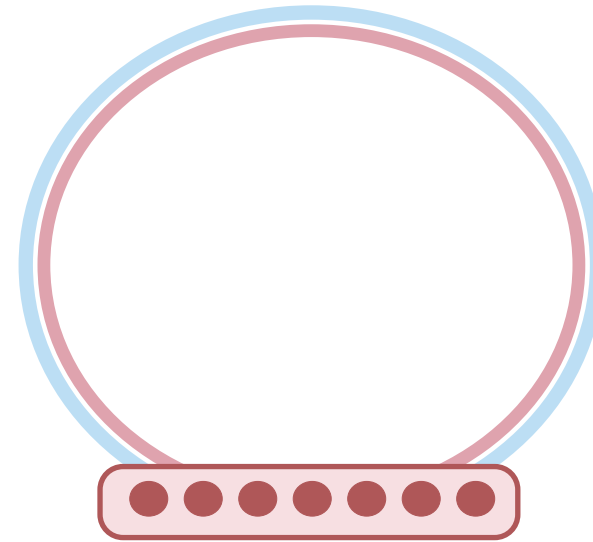
- Earliest changes include perivascular and interstitial accumulation.
- Fluid can be conspicuous in the interlobular septa.
- Alveolar spaces may still be predominantly aerated.



Schematic alveolus

Edematous septa become widened

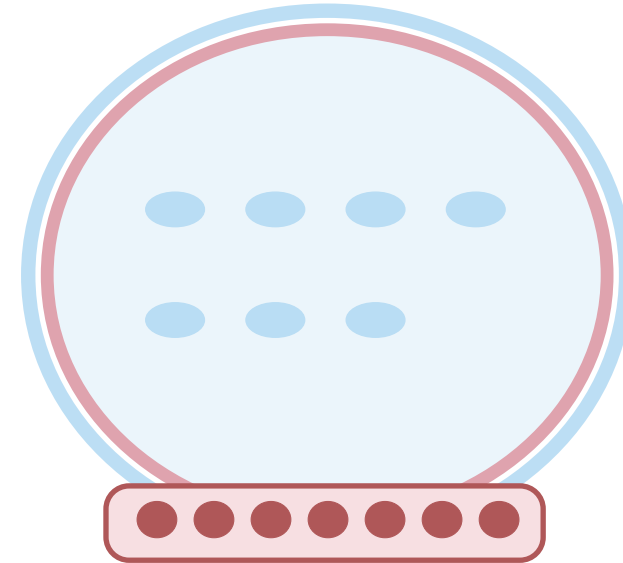
- Widening of alveolar septa reflects interstitial fluid accumulation.
- Edema increases the separation between capillary blood and alveolar gas.
- Septal expansion develops before substantial alveolar flooding.



Schematic alveolus

Alveolar flooding impairs aeration

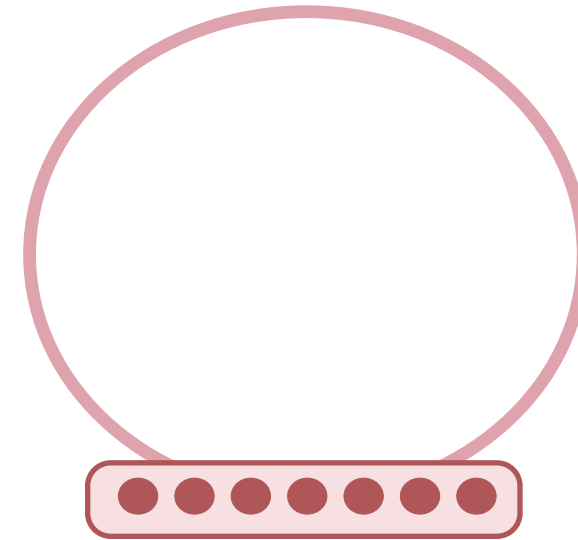
- Increasing extravascular fluid reaches the alveolar lumen.
- Formerly air-containing spaces accumulate edema fluid.
- Gas exchange becomes compromised by loss of functional airspace.



Schematic alveolus

Engorged alveolar capillaries

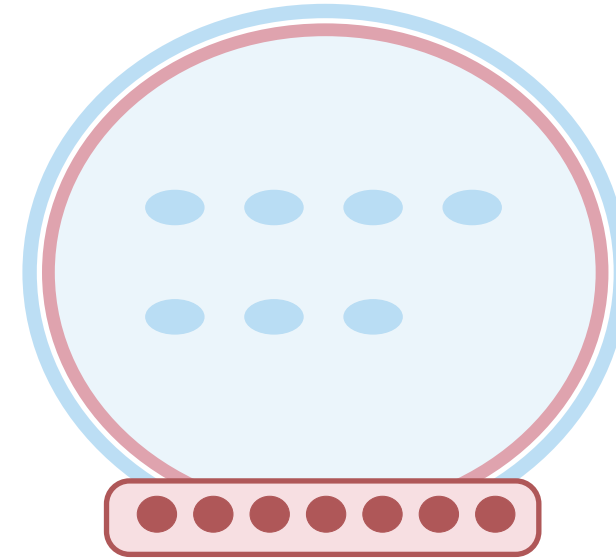
- Passive pulmonary venous congestion distends the capillary network.
- Engorged capillaries are a prominent early microscopic finding.
- Capillary congestion accompanies—but is not synonymous with—edema.



Schematic alveolus

What does transudate look like on H&E?

- Fluid within alveolar lumens appears finely granular and pale pink.
- This is a protein-poor transudate resulting from altered fluid forces.
- Morphologic interpretation requires vascular and clinical context.

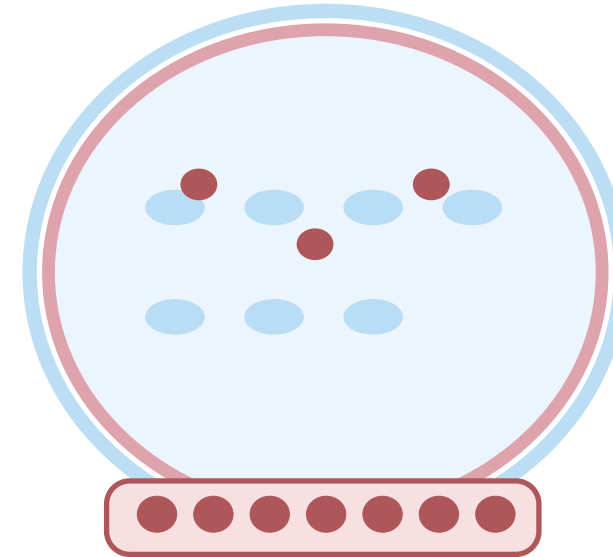


Schematic alveolus

Diagram only — not an H&E micrograph

Capillary microhemorrhage can coexist

- Congested capillaries may leak or rupture focally.
- Erythrocytes then accumulate in some alveolar spaces.
- Microhemorrhages provide the substrate for later hemosiderin formation.



Schematic alveolus

Heart failure cells indicate prior bleeding

Extravasated erythrocytes

Red cells enter alveolar space



Macrophage phagocytosis

Erythrocyte breakdown



Hemosiderin pigment

Iron accumulates in
macrophages

“Heart failure cells” are hemosiderin-laden macrophages (siderophages).

From red cells to hemosiderin

- 01** Microhemorrhage places erythrocytes outside pulmonary capillaries.
- 02** Resident alveolar macrophages phagocytose extravasated cells.
- 03** Heme degradation releases iron, stored as hemosiderin.
- 04** Repeated bleeding produces abundant brown-pigmented macrophages.

Siderophages: What they signify

Positive inference

- Previous alveolar hemorrhage or congestion has occurred.
- Chronic left-heart disease is a classic setting.
- More numerous cells support repeated episodes.

Interpretive limit

- Hemosiderin-laden macrophages are not unique to heart failure.
- Correlate with septal fibrosis, venous congestion and clinical history.
- A single pigmented cell does not establish etiology.

Acute and chronic congestion are different

Feature	Acute congestion / edema	Chronic congestion
Capillaries	Engorged	May remain engorged
Airspaces	Edema fluid $\pm$ focal red cells	Siderophages often numerous
Alveolar septa	Edema-related widening	Fibrotic thickening
Gross impression	Wet, heavy lung	Brown, firm indurated lung

Gross edema: Heavy, wet lungs

Increased lung weight

Edematous lungs may become markedly heavier than normal.

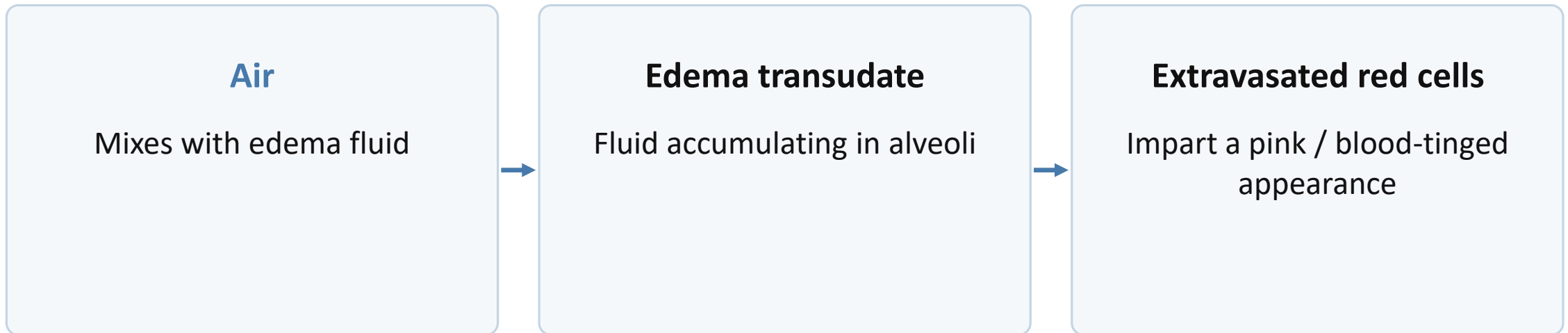
Wet cut surface

Cutting can release frothy fluid containing air and edema fluid.

Pulmonary congestion

Venous blood pooling contributes a dark congested appearance.

Frothy, blood-tinged fluid: Its components



Foam reflects physical admixture: Air + edema liquid ± red blood cells.

Chronic passive congestion remodels septa

Repeated congestion

Ongoing vascular stasis and episodes of hemorrhage



Persistent tissue injury

Chronic hypoxia and remodeling



Septal fibrosis

Thickened alveolar walls and reduced flexibility

Brown induration: Two processes combined

**Brown pigment from hemosiderin-laden
macrophages**

Induration from fibrotic alveolar septa

Firm, brown lungs

Classic in long-standing pulmonary venous congestion, including mitral stenosis.



Brown induration versus acute edema

Acute / active edema

- High water content produces heavy, soggy lungs.
- Congested capillaries and intraalveolar fluid dominate.
- Chronic scarring is not required.

Long-standing congestion

- Hemosiderin imparts brown pigment.
- Fibrotic septal thickening increases firmness.
- The combination is described as brown induration.

Pleural effusions may worsen impairment

Raised pleural capillary pressure

Fluid filters into pleural space



Serous pleural effusion

Additional extravascular
thoracic fluid



Lung compression

Less ventilated parenchyma may
worsen gas exchange

How edema impairs oxygenation

Interstitial edema

Diffusion path becomes less efficient



Alveolar edema

Functional air-filled area is reduced



Gas-exchange impairment

Hypoxemia and dyspnea can result

Edematous lungs are vulnerable to infection

- Fluid in interstitial and alveolar spaces impairs normal respiratory function.
- An edematous environment can favor secondary pulmonary infection.
- Superimposed infection may intensify hypoxemia and respiratory symptoms.

Early clinical pattern in left-heart failure

- Exertional cough and dyspnea may be the earliest respiratory manifestations.
- As edema progresses, breathlessness occurs at lower activity levels.
- Symptoms reflect impaired gas exchange and elevated pulmonary vascular load.

Orthopnea: Positional worsening

Supine posture

Redistribution of circulating volume



Higher lung vascular load

Congestion may worsen in susceptible patients



Orthopnea

Dyspnea is relieved by sitting or standing

Paroxysmal nocturnal dyspnea

- Abrupt nocturnal episodes of severe dyspnea may occur as left-heart failure progresses.
- Patients may awaken with a sensation of suffocation.
- The symptom is a clinical clue to pulmonary congestion, not a histologic diagnosis.

Basal fine rales: Fluid-filled airspaces

Edematous basal alveoli

Alveoli are fluid affected



Inspiration

Small airspaces abruptly reopen



Fine basal crackles

Characteristic auscultatory finding

Cardiac clues support the pulmonary pattern

Cardiac / clinical clue	Pathologic link
Cardiomegaly	May reflect underlying heart disease or ventricular remodeling
Tachycardia	Compensatory / associated finding in heart failure
S3 or S4 gallop	Volume loading or increased ventricular stiffness
Mitral valve abnormality	May elevate pulmonary venous pressure

Integrating symptoms with morphology

Clinical finding	Morphologic / physiologic correlate
Dyspnea / hypoxemia	Interstitial or alveolar fluid limits gas exchange
Fine basal crackles	Reopening of fluid-affected small airspaces
Blood-tinged edema fluid	Microhemorrhage and fluid mixed with air
Chronic impairment	Septal fibrosis and repeated congestion

Hydrostatic versus permeability edema

Feature	Hemodynamic edema	Alveolar wall injury
Initial disturbance	Elevated hydrostatic / reduced oncotic force	Increased capillary / epithelial permeability
Characteristic fluid	Protein-poor transudate	Inflammatory protein-rich exudate
Common setting	Left-sided heart failure	Infection, inhalation injury, sepsis, etc.
Interpretation	Venous pressure and filtration problem	Alveolar barrier injury problem

Transudate and exudate are not synonyms

Transudate

- Relatively protein poor.
- Associated with changes in pressure or plasma protein balance.
- Typical of hemodynamic pulmonary edema.

Exudate

- Relatively protein rich.
- Associated with inflammation and increased vascular permeability.
- Characteristic of alveolar microvascular injury.

Pulmonary edema is not atelectasis

Edema

- Increased lung water and fluid in alveoli.
- Grossly heavy, wet lungs; Microscopy may show transudate.
- Often follows pulmonary venous congestion.

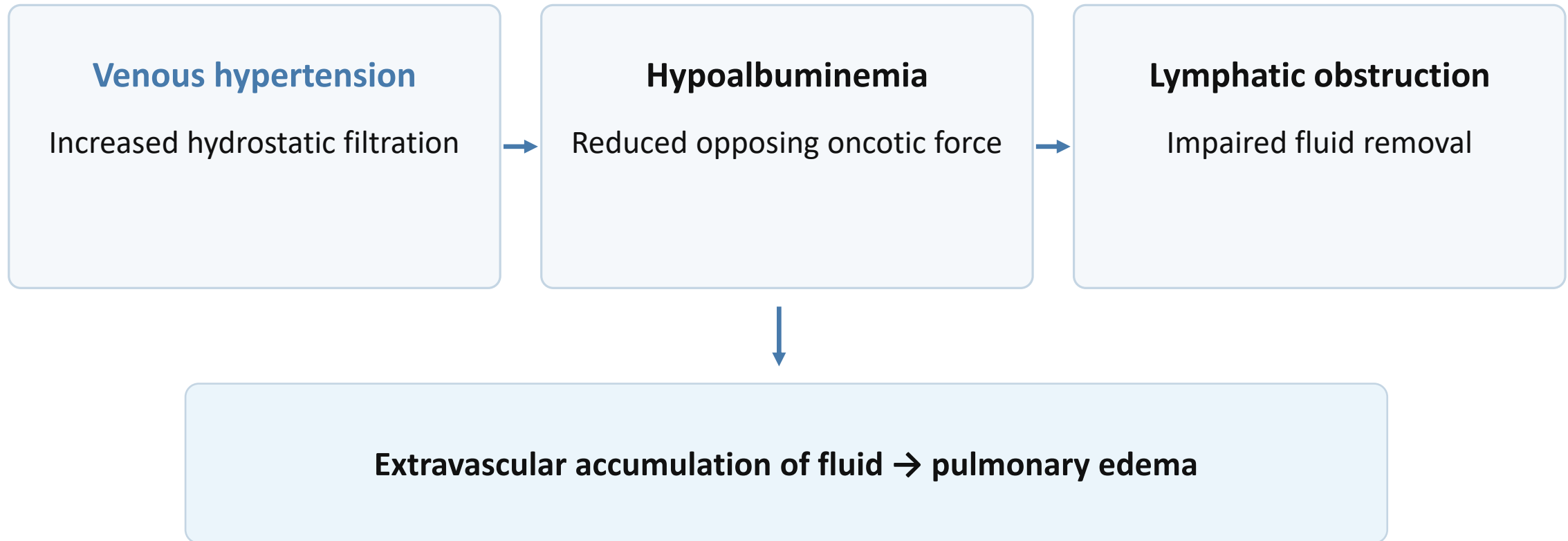
Atelectasis

- Reduced lung expansion or collapse of previously inflated lung.
- Mechanisms include airway obstruction, compression and contraction.
- Can coexist with edema or pleural effusions.

Microscopic pitfalls and safeguards

- 01** Pale pink alveolar material should be interpreted alongside vascular congestion.
- 02** Siderophages support prior hemorrhage but do not exclusively prove cardiac failure.
- 03** Thickened septa may reflect acute edema or established chronic fibrosis.
- 04** Clinical history and cardiac context help identify the hemodynamic cause.

Three hemodynamic pathways, one outcome



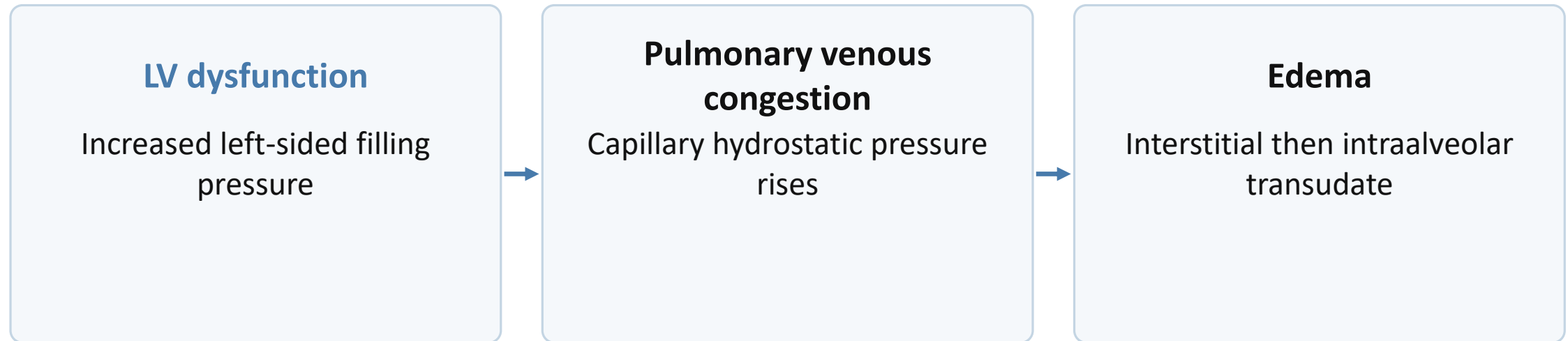
Case 1 — worsening dyspnea and crackles

Case vignette

A patient with established left-ventricular dysfunction develops increasing exertional dyspnea, orthopnea, and fine basal crackles. At autopsy the lungs are heavy and wet.

Which mechanism best explains the pulmonary fluid?

Case 1 — connect the findings



The constellation is typical of hemodynamic edema accompanying left-heart failure.

Case 2 — mitral stenosis and brown lungs

Case vignette

A patient with long-standing mitral stenosis has firm, brown lungs. Histology shows fibrotic alveolar septa and numerous hemosiderin-laden macrophages.

Which chronic process produces the brown color and firmness?

Case 2 — explain brown induration

Chronic venous pressure

Repeated capillary engorgement
/ bleeding



Pigmented macrophages

Hemosiderin from
phagocytosed red cells



Septal fibrosis

Firm brown induration of the
lung

Case 3 — nephrotic syndrome with edema

Case vignette

A patient with substantial urinary protein loss develops hypoalbuminemia and generalized edema. There is no primary evidence of an injured alveolar barrier.

Which Starling force is most directly altered?

Case 3 — oncotic loss, not barrier injury

Urinary albumin loss

Nephrotic glomerular permeability



Lower plasma oncotic pressure

Opposition to filtration falls



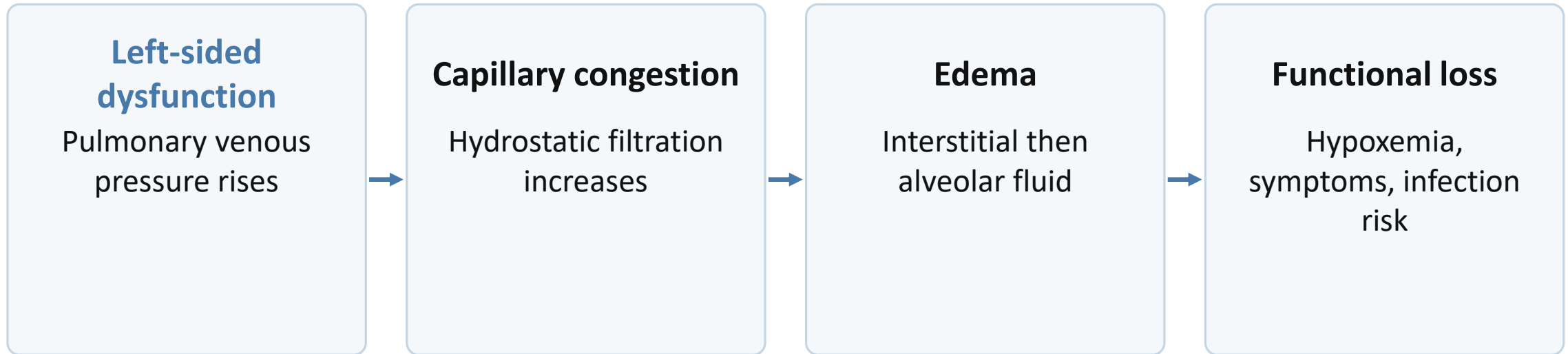
Transudative edema

Fluid can accumulate in lungs and other tissues

Clinicopathologic interpretation algorithm

- 01** Establish that excess interstitial or alveolar lung fluid is present.
- 02** Assess for left-sided failure, volume load, and pulmonary venous obstruction.
- 03** Consider hypoalbuminemia or rare lymphatic impairment when relevant.
- 04** Match acute congestion/transudate or chronic siderophages/fibrosis to duration.
- 05** If barrier injury predominates, evaluate noncardiogenic edema separately.

Pathogenesis in a single causal sequence



Morphology: Findings by compartment

Compartment	Principal changes
Pulmonary capillary	Engorgement and venous congestion
Interstitial / alveolar septum	Edema-related widening; Fibrosis in chronic disease
Alveolar airspace	Pale pink transudate; Occasional red cells
Alveolar macrophage	Hemosiderin accumulation after hemorrhage

High-yield distinctions to retain

- Hydrostatic pulmonary edema is most often associated with left-sided heart failure.
- Dependent lower-lobe regions are involved early.
- Acute histology: Engorged capillaries, pale transudate, $\pm$ microhemorrhage.
- Chronic changes: Siderophages and fibrotic septa $\rightarrow$ brown induration.
- Edema impairs gas exchange and increases vulnerability to infection.

Knowledge check: Choose the best inference

Case vignette

A lung sample shows congested alveolar capillaries, finely granular pale pink intraalveolar fluid, focal extravasated erythrocytes, and no established septal fibrosis.

What is the most defensible morphologic interpretation?

Knowledge check: Interpretation

- The pattern supports acute pulmonary congestion with intraalveolar edema.
- The pale fluid is compatible with a hydrostatic transudate.
- Focal erythrocytes indicate microhemorrhage; Absence of fibrosis argues against established chronic remodeling.
- Clinical evidence of elevated pulmonary venous pressure remains necessary to assign the etiology.

References and source boundaries

Robbins, Cotran & Kumar: Pathologic Basis of Disease. 11th ed. (2026).

- Chapter 15, “Pulmonary Edema,” “Hemodynamic Pulmonary Edema,” and Table 15.1 (pp. 614–615).
- Chapter 4, “Edema and Effusions” and “Hyperemia and Congestion” (pp. 100–102).
- Chapter 12, “Heart Failure” and “Left-Sided Heart Failure” (pp. 481–484).
- Original explanatory diagrams are native PowerPoint schematics, not histologic images.