

Cor pulmonale

Pulmonary (right-sided) hypertensive heart disease

Right ventricular adaptation and failure caused by pulmonary vascular pressure overload

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Learning outcomes

- Define cor pulmonale and distinguish it from secondary right-sided failure.
- Classify pulmonary and vascular causes of right ventricular pressure overload.
- Recognize the acute–chronic gross morphological differences.
- Explain systemic venous congestion and its organ effects.

Cor pulmonale is a ventricular response, not a lung diagnosis

Pulmonary vascular pressure overload drives changes in the right ventricular myocardium.

Implication 1

- Identify the upstream pulmonary disorder.

Implication 2

- Then evaluate right ventricular remodeling and function.

The normal right ventricle works at lower pressure

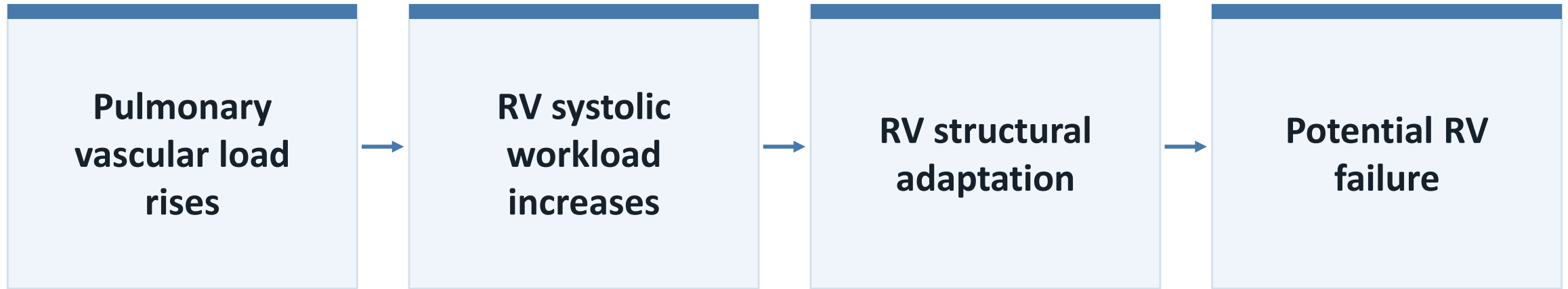
Right ventricle

- Thin, compliant free wall
- Designed for low-pressure pulmonary circulation

Left ventricle

- Thicker muscular wall
- Supports higher-pressure systemic circulation

Pressure overload links pulmonary disease to heart disease



The cardiac consequence is downstream of the pulmonary hemodynamic problem.

Isolated cor pulmonale and other right-sided failure differ

Isolated cor pulmonale

- Primary lung or pulmonary vascular disorder
- RV overload arises from pulmonary hypertension

Secondary right-sided failure

- Left-sided heart disease often comes first
- Pulmonary venous pressure transmits RV load

Acute and chronic cor pulmonale express different remodeling

Acute onset

- Abrupt RV dilation
- No time for compensatory hypertrophy

Chronic exposure

- RV wall hypertrophy
- Dilation may develop and failure may follow

The time course determines the phenotype

Early / acute course

- Abrupt rise in RV afterload
- Rapid chamber expansion
- Marked dysfunction possible

Sustained / chronic course

- Sustained pressure burden
- Myocyte hypertrophy
- Later dilation and failure

Four source-defined families predispose to cor pulmonale

Pulmonary parenchymal disease

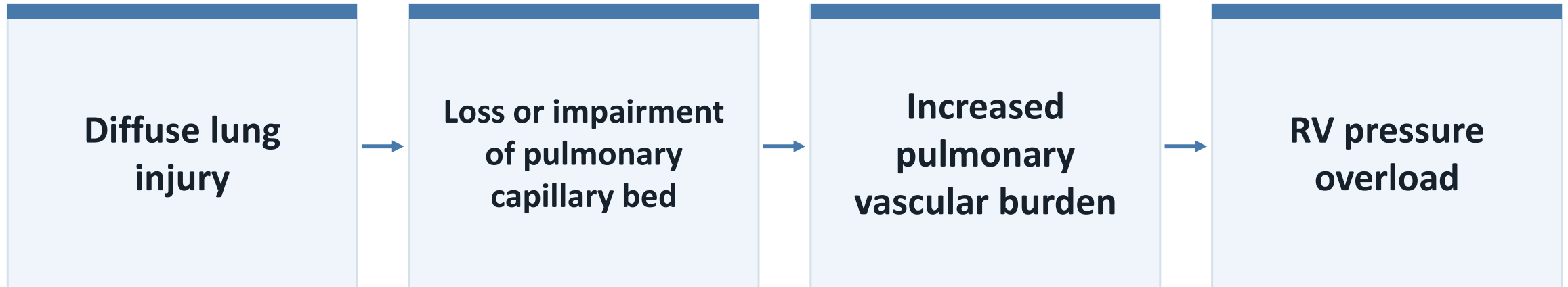
Pulmonary vascular disease

Impaired chest movement

Pulmonary arterial constriction

Robbins Table 12.7 organizes etiologies by the affected component.

Parenchymal disease reduces effective vascular capacity

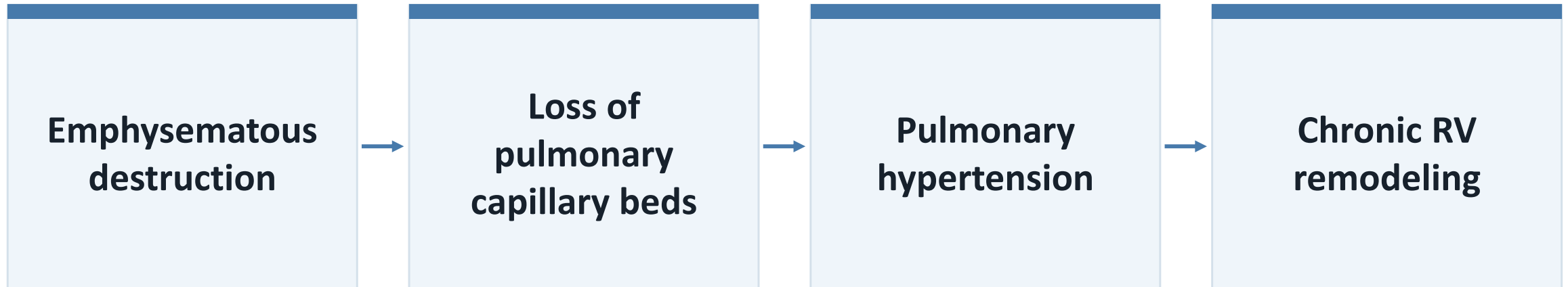


The relation between parenchymal disease and RV disease is a lung–vessel–heart continuum.

Chronic obstructive pulmonary disease is a major substrate

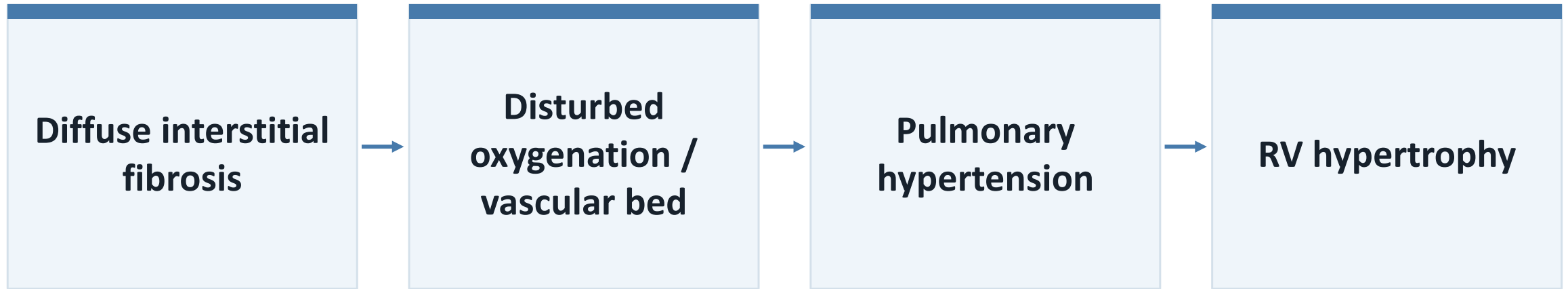
- Chronic obstructive pulmonary disease appears Table 12.7.
- Emphysema is emphasized as a frequent chronic parenchymal cause.
- Advanced lung disease can combine hypoxemia with loss of capillary beds.

Emphysema links parenchymal destruction to vascular load



Vascular bed loss is an initiating event described in the pulmonary hypertension chapter.

Diffuse interstitial fibrosis adds a second parenchymal route



Fibrotic lung disease and pulmonary vascular disease can coexist.

Additional parenchymal causes belong in the differential

Pneumoconioses

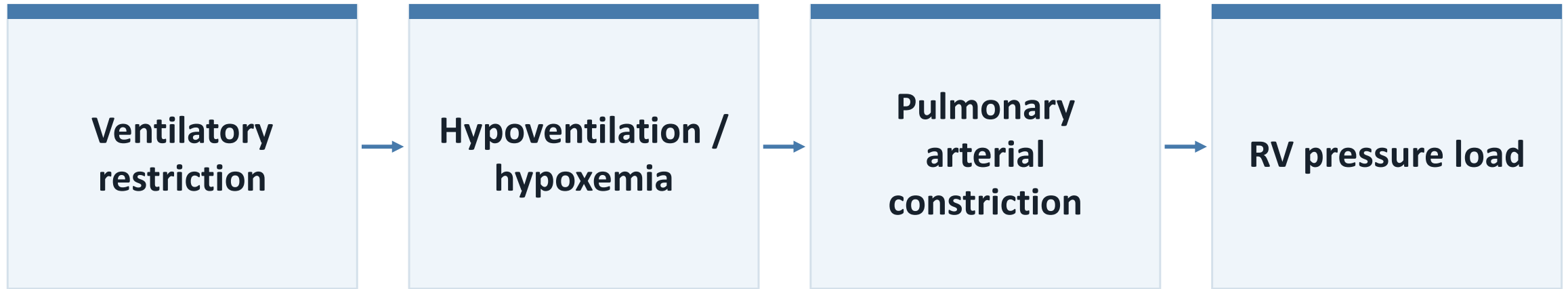
Cystic fibrosis

Bronchiectasis

**Chronic obstructive pulmonary
disease**

These are distinct entries Table 12.7, not interchangeable diagnoses.

Restricted chest movement can initiate pulmonary hypertension



Use as an explanatory schematic; Mechanisms vary by underlying disorder.

Chest movement disorders are etiologically diverse

Kyphoscoliosis

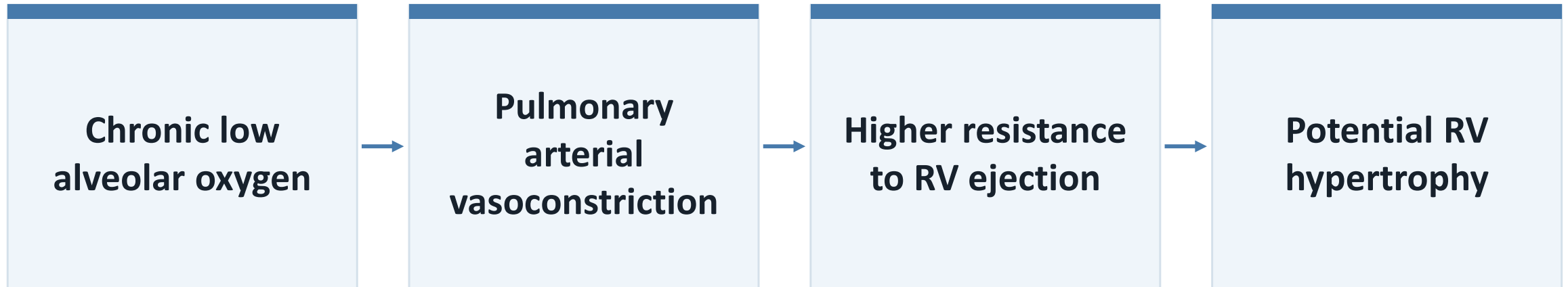
Marked obesity / sleep apnea

Neuromuscular disease

Chest-wall restriction

First three are explicit causes; Fourth labels their shared functional category.

Sustained hypoxemia is a pulmonary vasoconstrictor



Hypoxemia among disorders inducing pulmonary arterial constriction.

Altitude and airway obstruction can trigger the same response

- Chronic altitude sickness is listed among constrictor-associated causes.
- Obstruction of major airways may provoke sustained respiratory compromise.
- Idiopathic alveolar hypoventilation is a separate listed cause.
- Metabolic acidosis also appears in the table.

Pulmonary vessel diseases impose primary vascular load

Recurrent thromboembolism

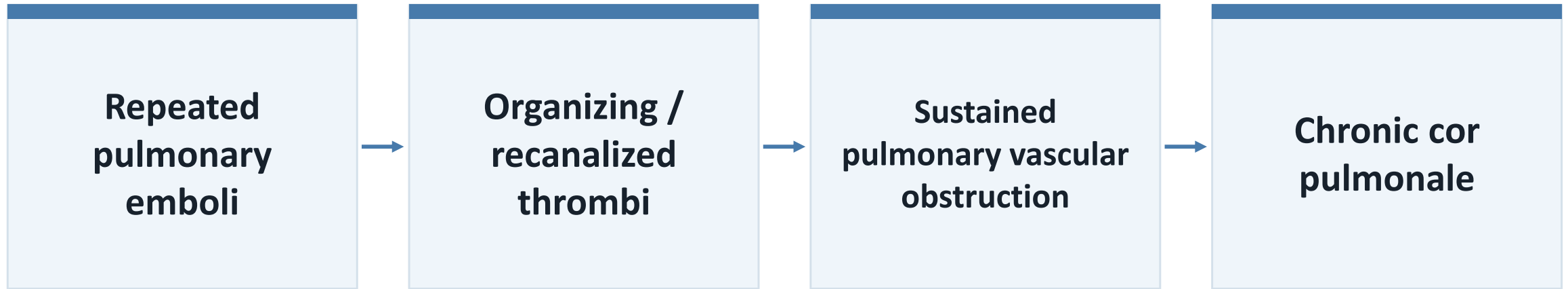
Primary pulmonary hypertension

Extensive pulmonary arteritis

**Vascular obstruction from toxins or
tumor**

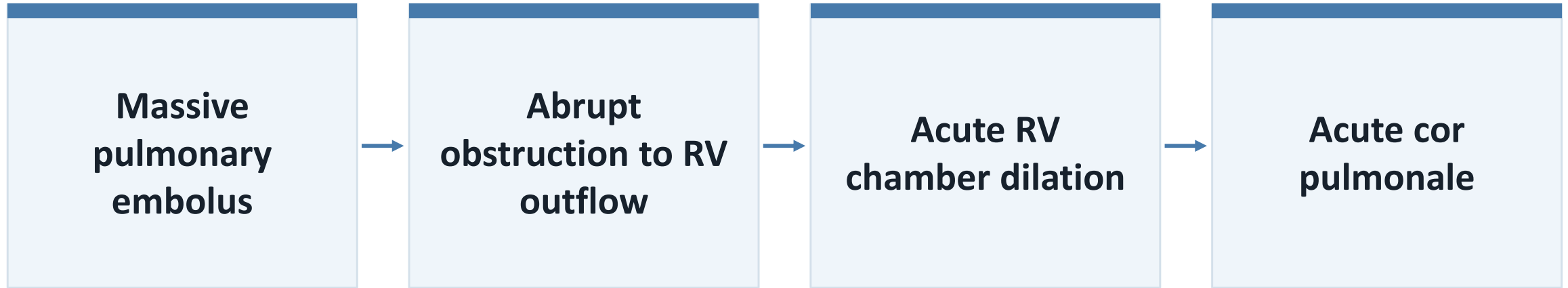
All four routes act through disease in the pulmonary vascular compartment.

Recurrent embolization leads to cumulative vascular disease



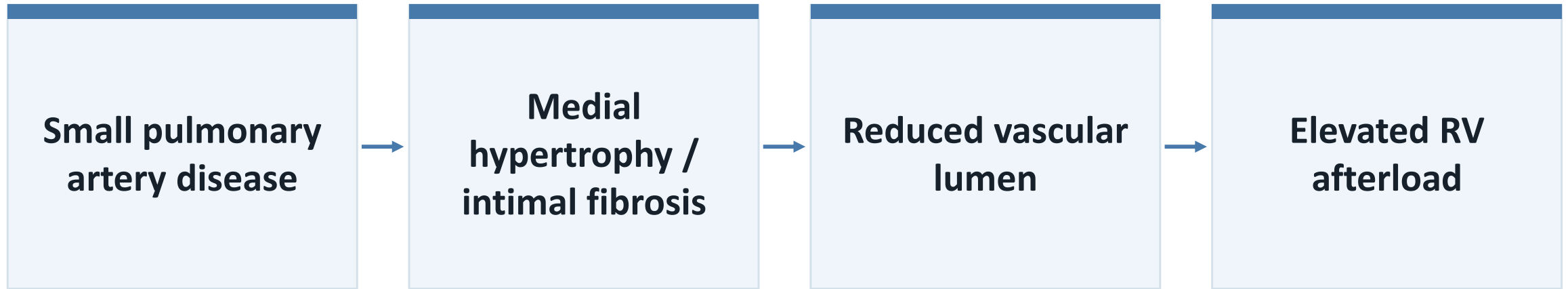
Organized thrombi are a pathological clue to thromboembolic causation.

Acute massive pulmonary embolism is a different temporal injury



The acutely overloaded RV has not undergone pressure-induced hypertrophy.

Pulmonary arterial hypertension targets small muscular arteries



Pulmonary arterial hypertension is a pulmonary vascular—not a myocardial—starting lesion.

Vasculitis and vascular obstruction are separate causes

- Extensive pulmonary arteritis, including granulomatosis with polyangiitis, is listed.
- Drug-, toxin-, or radiation-induced vascular obstruction may predispose.
- Extensive pulmonary tumor microembolism is another vascular cause.
- The same cardiac endpoint does not establish the same pulmonary pathology.

Pulmonary hypertension often originates in the left heart

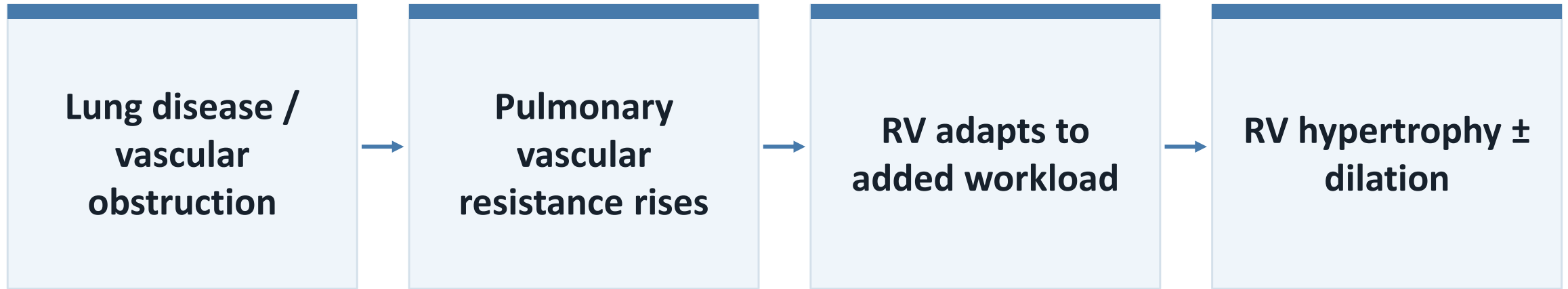
Lung / vascular origin

- Pulmonary disorder precedes RV pressure overload
- Cor pulmonale in the isolated-right-sided sense

Left-heart origin

- Left-sided cardiac disease elevates pulmonary pressure
- Secondary RV strain is not the same etiologic category

An etiologic synthesis: Different insults, common endpoint

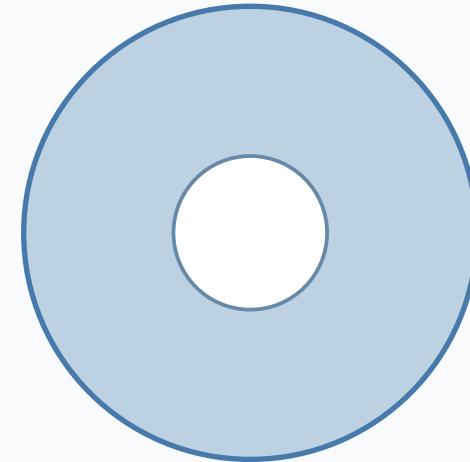


The common final pathway is increased RV pressure workload.

Progressive pulmonary vascular narrowing amplifies afterload

- Vascular remodeling thickens the arterial wall.
- Intimal fibrosis narrows the lumen in small arteries.
- Persistent narrowing increases resistance to forward flow.

Simplified pulmonary artery cross-section



Media / arterial wall

Lumen

Schematic—not microscopic evidence

Pulmonary hypoxia and vascular destruction may coexist

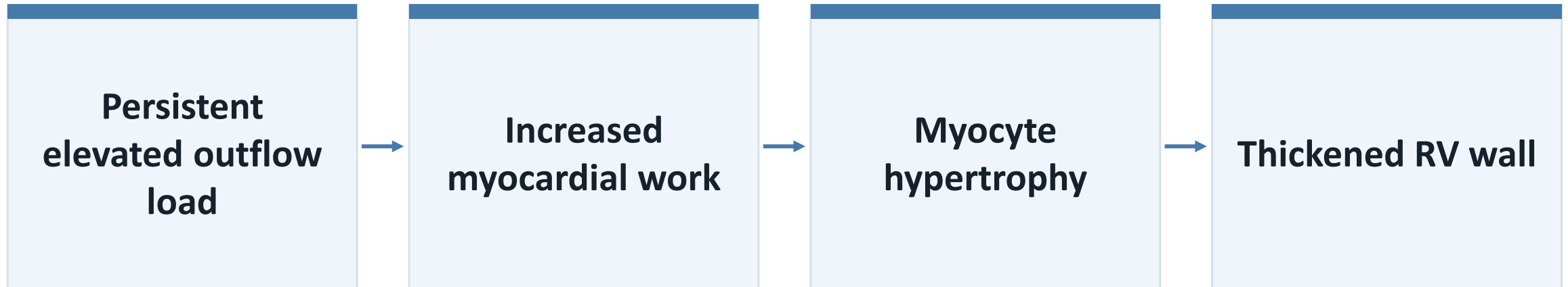
Hypoxemia pathway

- Pulmonary vasoconstriction
- Pressure load can rise without a primary heart lesion

Capillary-loss pathway

- Fewer available capillary channels
- Vascular surface area declines in emphysema/fibrosis

RV wall stress initiates myocardial adaptation



Pressure overload is the initiating cardiac stress; The response is initially compensatory.

Chronic adaptation does not guarantee normal function

Early / acute course

- Compensated wall thickening
- Sustained elevated load
- Chamber dilation
- Right-sided failure may develop

Sustained / chronic course

- Morphologic progression is possible, not inevitable in every patient.

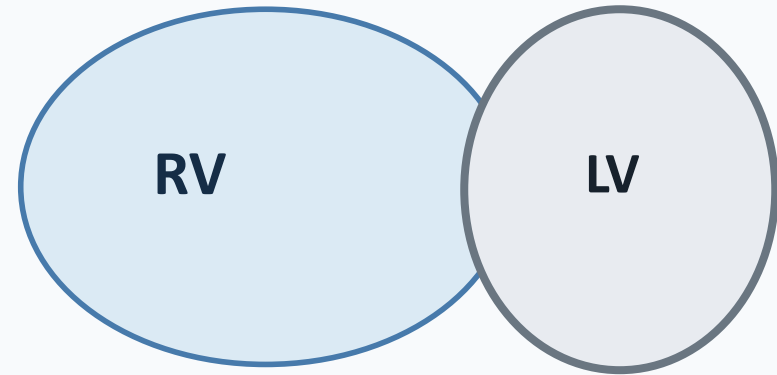
Acute cor pulmonale is primarily chamber dilation

- No established RV hypertrophy

- Marked RV dilation

- Cross-section becomes ovoid

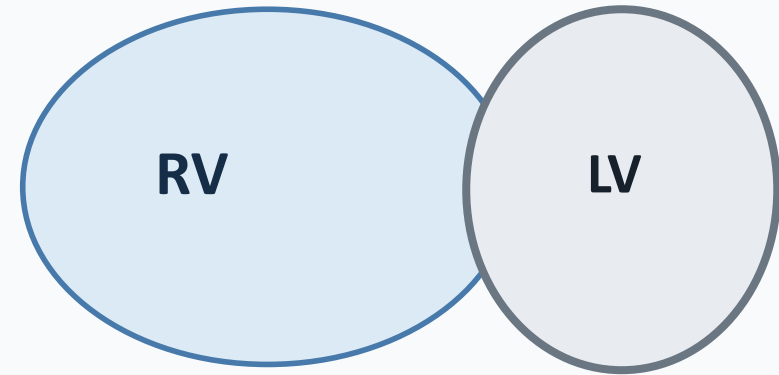
Schematic, not a specimen



The normally crescentic RV cavity becomes ovoid

- Normal short-axis RV cavity: Crescent-shaped
- Acute severe pressure load: Expanded, ovoid RV cavity
- This change can occur without a thickened RV wall

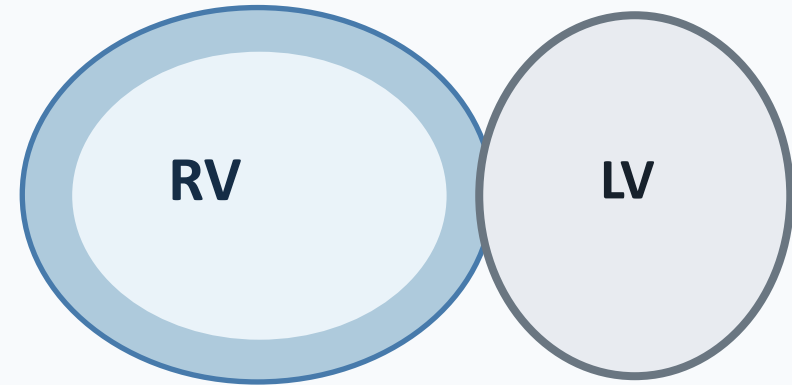
Schematic, not a specimen



Chronic cor pulmonale produces right ventricular wall thickening

- Chronic RV pressure overload
- Wall thickness may reach 1.0 cm or greater
- Often accompanied by ventricular dilation

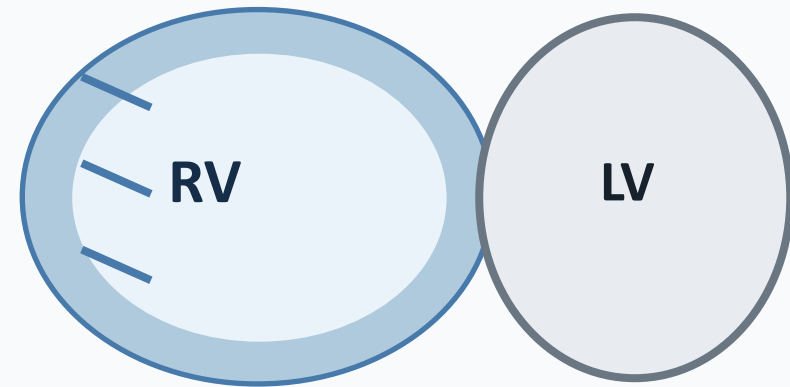
Schematic, not a specimen



Hypertrophied RV trabeculae are a gross clue

- Prominent hypertrophied trabeculae
- RV enlargement Fig. 12.19B
- Gross changes may distort the opposing LV cavity

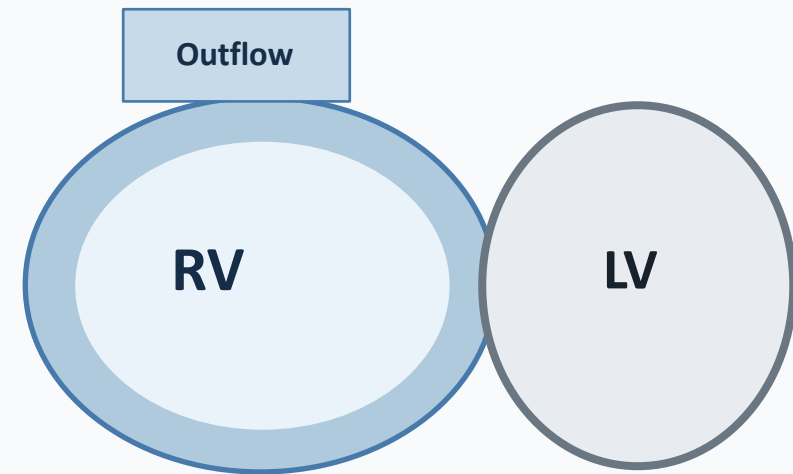
Schematic, not a specimen



Outflow tract thickening may reveal subtle RV hypertrophy

- Hypertrophy can localize to muscular bundles below the pulmonary valve
- It may be apparent despite relatively subtle free-wall enlargement
- Inspect the subpulmonic outflow region

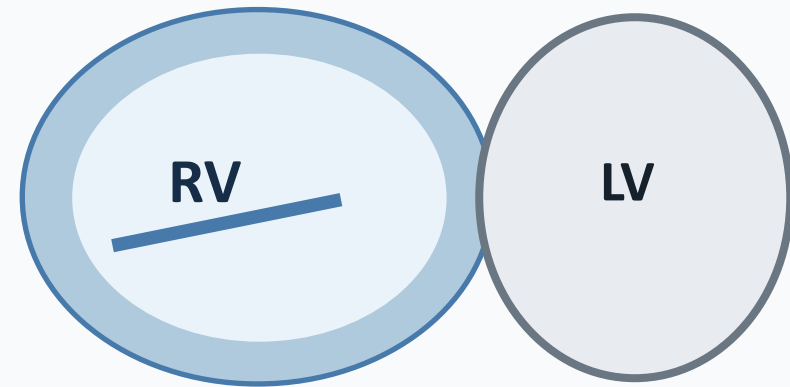
Schematic, not a specimen



The moderator band may be thickened

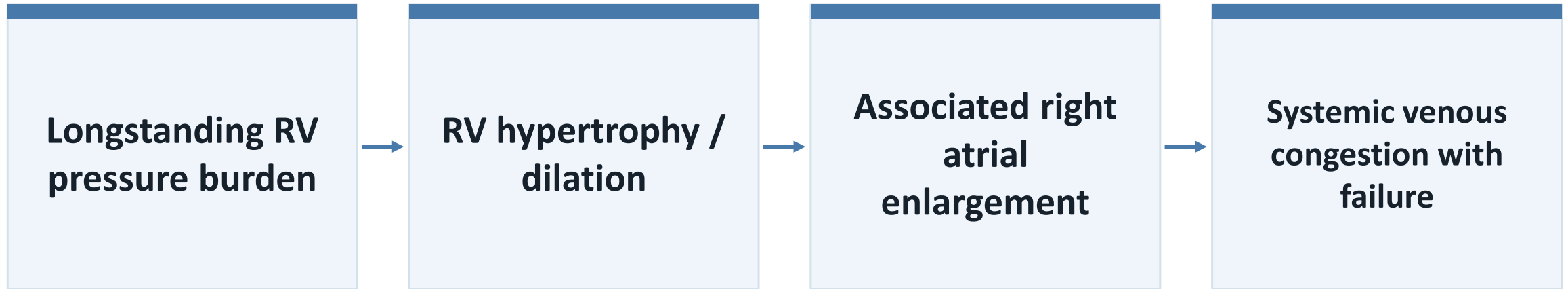
- Moderator band: Septum to anterior RV papillary muscle
- Thickening is a more subtle manifestation of RV hypertrophy
- This is an anatomical feature—not an isolated disease marker

Schematic, not a specimen



Moderator band

Right atrial remodeling often accompanies chronic overload

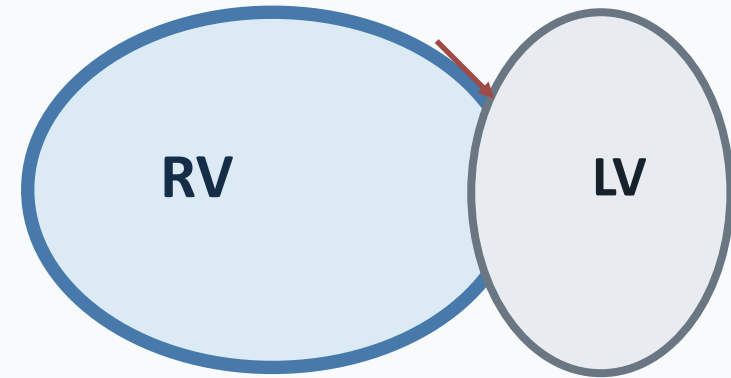


Robbins key concepts include right atrial hypertrophy and dilation.

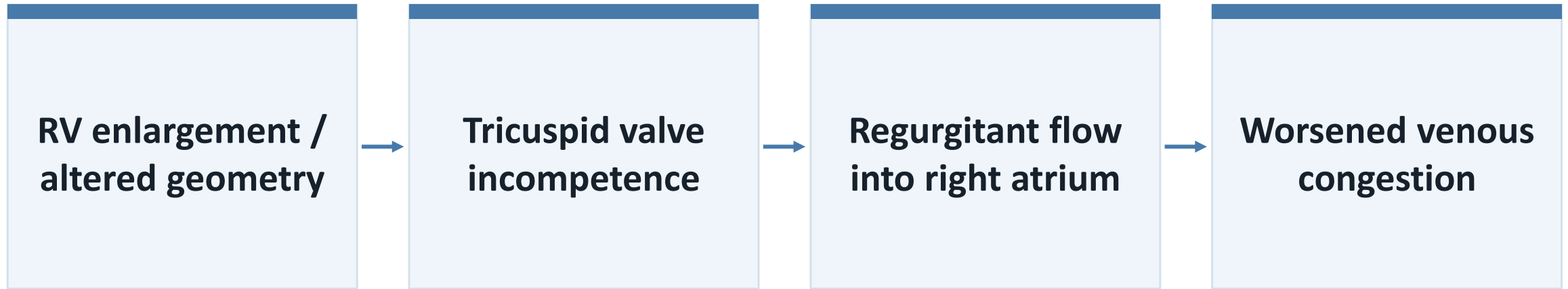
An enlarged RV can distort the left ventricle

- RV chamber expansion alters septal geometry
- The LV can be compressed or distorted
- Interventricular dependence can impair LV filling/function

Schematic, not a specimen



Functional tricuspid regurgitation may complicate remodeling



Robbins also describes fibrous thickening of the tricuspid valve.

Acute versus chronic morphology: Three discriminators

Acute cor pulmonale

- Marked RV dilation
- No established hypertrophy
- Crescent-to-ovoid cavity change

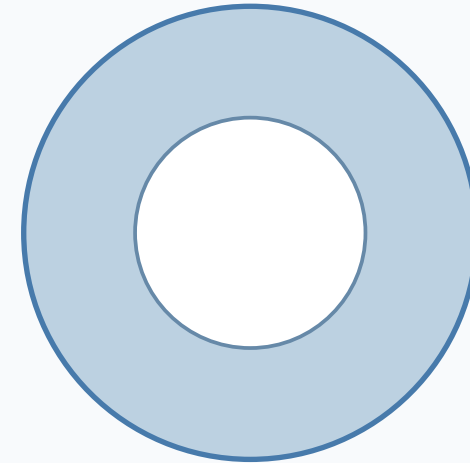
Chronic cor pulmonale

- RV free-wall thickening
- Hypertrophied muscle bundles
- Dilation may coexist

Pulmonary vessel morphology provides causal evidence

- Medial hypertrophy involves muscular and elastic arteries.
- Changes can extend into small arteries and arterioles.
- The lesion distribution helps explain increased afterload.

Simplified pulmonary artery cross-section



Media / arterial wall

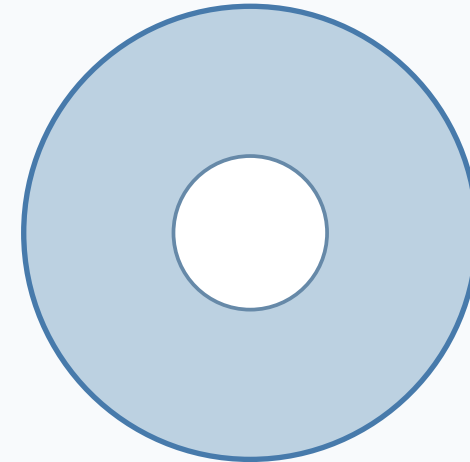
Lumen

Schematic—not microscopic evidence

Intimal fibrosis progressively compromises arterial lumens

- Small arteries show medial thickening with intimal fibrosis.
- In severe disease the residual lumen may be very narrow.
- The lesion is vascular; The right ventricle bears its hemodynamic consequence.

Simplified pulmonary artery cross-section



Media / arterial wall

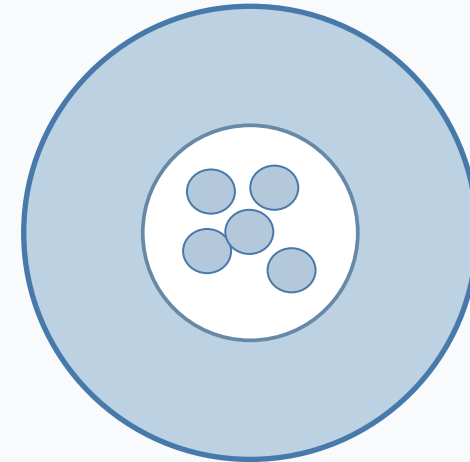
Lumen

Schematic—not microscopic evidence

Plexiform change represents advanced arteriopathy

- A plexiform lesion is a tuft-like complex of capillary channels.
- It is associated with severe pulmonary vascular disease.
- It is not necessary for the diagnosis of every cause of cor pulmonale.

Simplified pulmonary artery cross-section



Media / arterial wall

Lumen

Schematic—not microscopic evidence

Organizing pulmonary thrombi indicate prior embolization

Vascular observation

- Multiple organizing or recanalized thrombi
- Supports recurrent pulmonary embolization

Interpretive boundary

- Findings support an upstream etiology
- RV hypertrophy alone cannot identify the pulmonary cause

Pulmonary fibrosis and emphysema are etiologic histologic clues

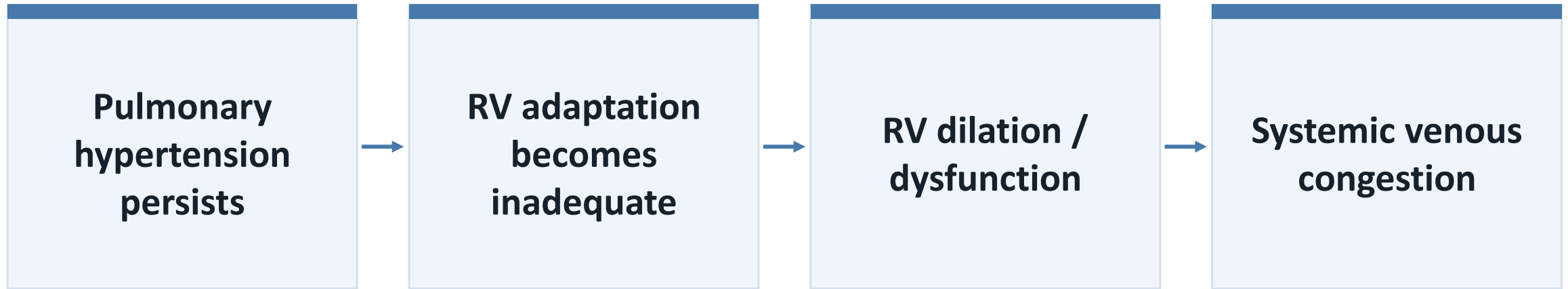
Fibrotic lung disease

- Diffuse interstitial pulmonary fibrosis
- Associated with pulmonary vascular burden

Emphysematous lung disease

- Parenchymal destruction and capillary-bed loss
- Often accompanies chronic hypoxia

Cor pulmonale can eventually become right-sided failure



Remodeling and clinical failure are related but distinct states.

Isolated RV failure preferentially congests systemic veins

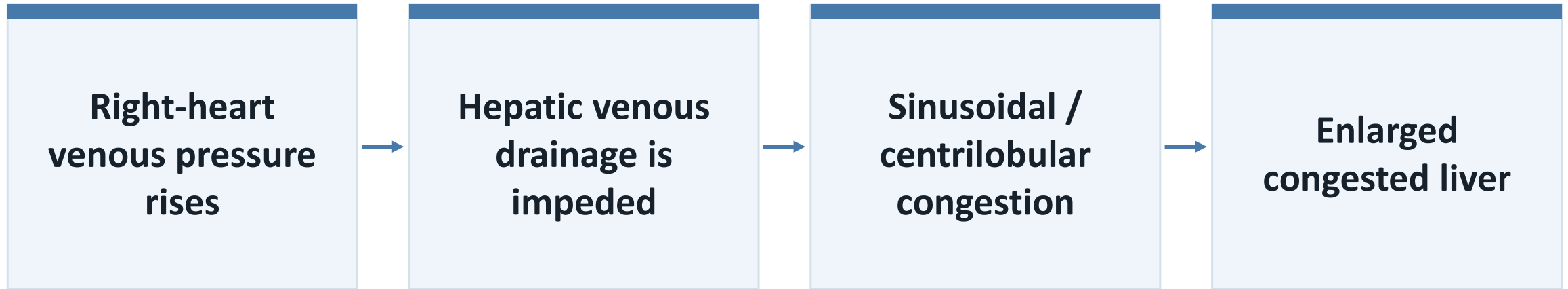
Systemic / portal beds

- Engorgement of peripheral and visceral veins
- Edema, organ congestion, effusions

Pulmonary circuit

- Congestion may be relatively limited in primary RV failure
- Lung disease itself can still cause dyspnea

Venous hypertension produces congestive hepatomegaly

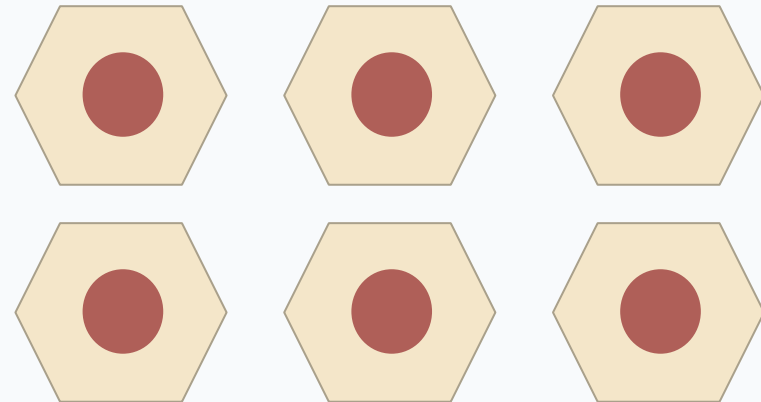


The venous back-pressure originates at the failing right heart.

A nutmeg liver reflects zonal passive congestion

- Red-brown congested centrilobular zones
- Relatively tan periportal regions
- Mottling reflects uneven vascular congestion

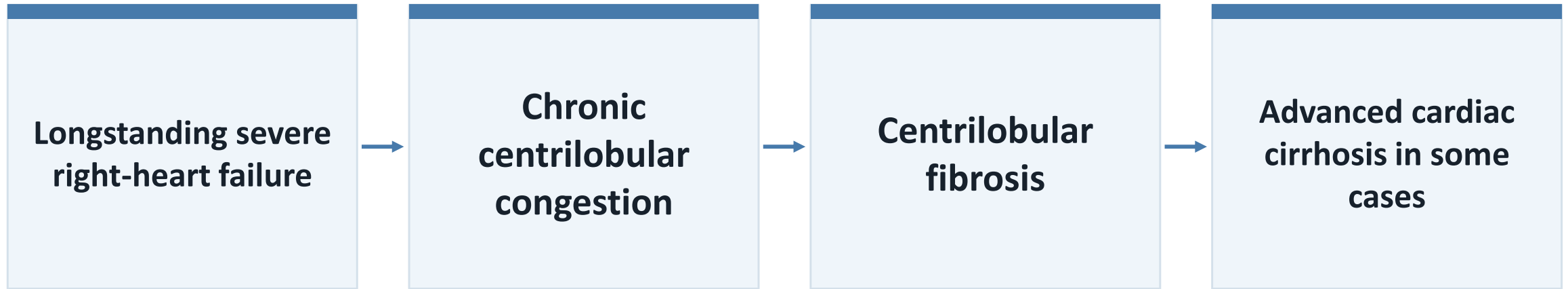
Simplified lobular congestion pattern



Red-brown: Centrilobular congestion

Conceptual map, not histology

Prolonged severe congestion can progress to cardiac fibrosis



Centrilobular necrosis may be more likely with additional low perfusion.

Ascites and serous effusions follow venous congestion

Peritoneal fluid → ascites

Pleural effusions

Pericardial effusions

Reduced ventilation if effusions are large

Body cavity fluid is a consequence of systemic venous pressure and altered fluid handling.

Dependent peripheral edema is a major clinical clue

- Pedal and pretibial edema are characteristic of right-sided failure.
- Sacral edema may be predominant in bedridden patients.
- Severe generalized edema can progress to anasarca.
- Edema reflects congestion, enhanced by renal fluid retention.

Portal congestion also affects spleen and gut

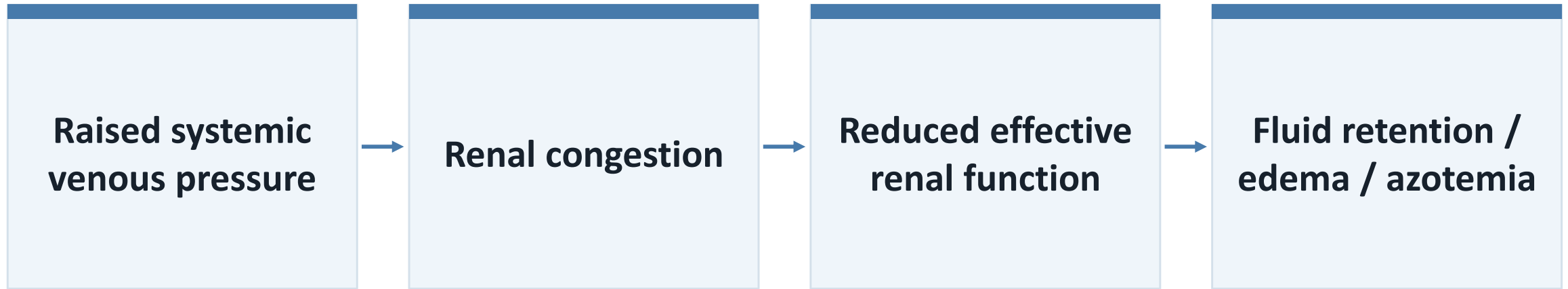
Spleen

- Congestive enlargement
- Associated platelet sequestration

Bowel

- Wall edema and chronic congestion
- Possible impaired nutrient / drug absorption

Renal congestion reinforces fluid retention

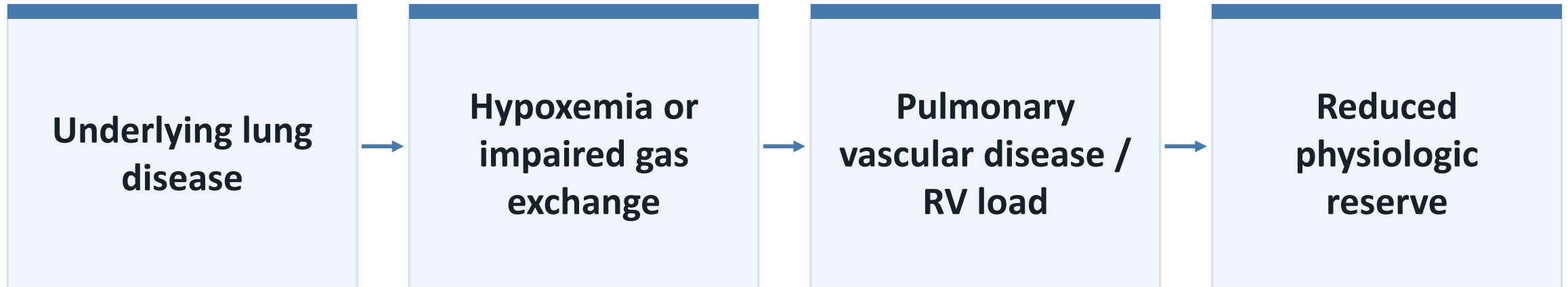


The renal contribution can amplify volume-related manifestations.

Pulmonary arterial hypertension may present late

- Dyspnea and fatigue are described as common initial manifestations.
- Anginal-type chest pain can occur in idiopathic disease.
- Advanced disease may produce cyanosis and respiratory distress.
- Decompensated right-heart failure is a major late complication.

Breathlessness may have multiple proximate mechanisms



Do not infer a single cause of dyspnea solely from the presence of RV remodeling.

Anatomic assessment requires more than wall thickness

RV cavity size

RV free-wall thickness

Septal / LV geometry

Right atrium and tricuspid valve

Each anatomic feature carries different information about chronicity and consequence.

Echocardiography links structure to clinical physiology

- Echocardiography evaluates chamber size, motion and valvular function.
- RV enlargement and dysfunction must be interpreted in clinical context.
- Assess whether left-sided dysfunction may account for pulmonary pressure elevation.
- A cardiac study does not replace identifying the pulmonary cause.

A cause-directed assessment begins in the lung and vessels

**Recognize RV
hypertrophy /
dilation**



**Establish the
hemodynamic
context**



**Investigate lung,
vascular and chest-
wall causes**



**Exclude
predominant left-
heart origin**

Educational reasoning framework, not a formal validated diagnostic algorithm.

Right- versus left-dominant failure has different congestion

Predominant right-sided failure

- Peripheral / portal venous congestion
- Edema, hepatomegaly, ascites

Predominant left-sided failure

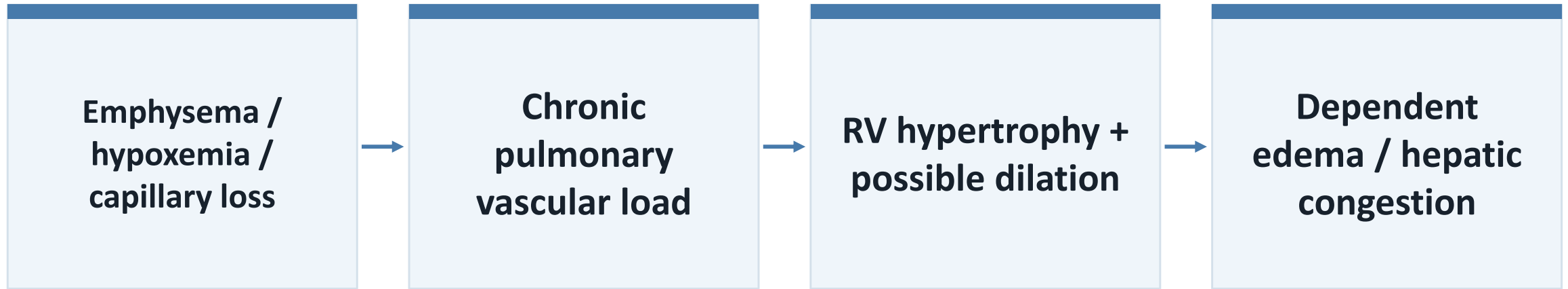
- Pulmonary venous congestion
- Pulmonary edema and related respiratory symptoms

Case: Chronic emphysema with progressive edema

A patient with advanced emphysema develops exertional breathlessness, dependent edema and RV wall thickening with dilation.

- Which pulmonary mechanism is likely?
- What cardiac morphology indicates chronic exposure?
- Which extra-cardiac findings support failure?

Case analysis: Chronic pulmonary pressure loading



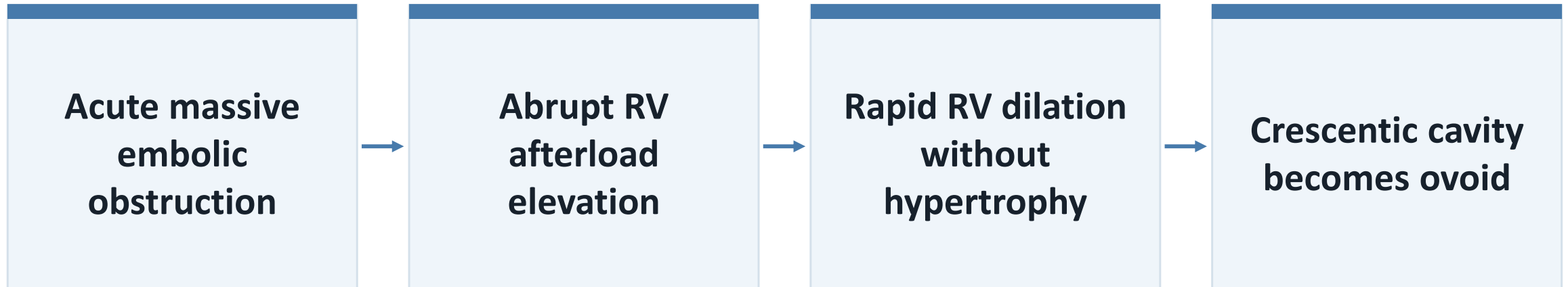
The chain links source pathology, heart morphology and systemic consequences.

Case: Sudden circulatory deterioration

A patient develops abrupt hemodynamic deterioration after a large pulmonary embolus; Gross examination finds a dilated RV without wall thickening.

- What is the most likely cardiac syndrome?
- Why is the RV wall not hypertrophied?
- What chamber-shape change is expected?

Case analysis: Acute cor pulmonale



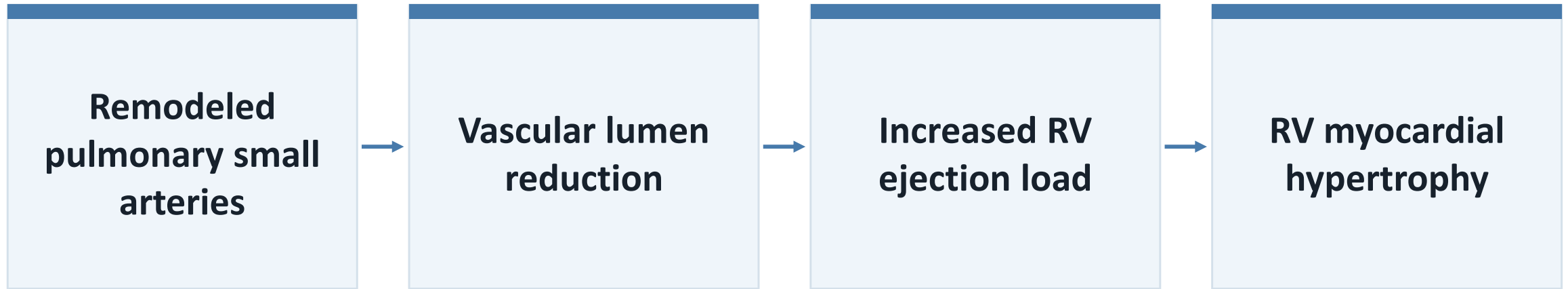
Temporal mismatch: Chamber dilation develops faster than myocyte hypertrophy.

Case: A histologic cause of pressure overload

Lung vascular sections show markedly thickened small-artery walls with intimal fibrosis; The heart has RV hypertrophy.

- Which compartment contains the primary lesion?
- How is RV morphology explained?
- Why does RV hypertrophy not specify the cause?

Case analysis: Primary pulmonary vascular disease



A vascular lesion establishes the upstream compartment; Evaluation determines its etiologic subclass.

Diagnostic pitfall: RV hypertrophy is not etiologically specific

What morphology establishes

- Evidence of chronic RV pressure adaptation
- Possible dilation and right-sided decompensation

What morphology cannot establish alone

- The exact pulmonary initiating disease
- Whether left-heart disease is responsible

Pattern synthesis: Chamber, vessel and organ

Heart: Hypertrophy / dilation

**Pulmonary vessels: Constriction /
obstruction**

**Lungs: Underlying parenchymal
disease**

Organs: Systemic venous congestion

Integrate compartments rather than relying on one isolated finding.

Core summary: Three decisions clarify cor pulmonale

- 1 Identify pulmonary pressure overload and its cause**
- 2 Determine acute dilation versus chronic hypertrophy**
- 3 Assess RV failure and systemic venous complications**

Cor pulmonale is a clinicopathologic chain from lung or pulmonary vessel to right heart.

Primary sources and scope

- Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th ed. (2026).
- Chapter 12, The Heart: hypertensive heart disease; right-sided heart failure; Table 12.7.
- Chapter 15, The Lung: pulmonary hypertension; pulmonary thromboembolism.
- Original textbook figures not reproduced; diagrams are original editable schematics.