

Pulmonary Hypertension

Mechanisms • vascular morphology • clinicopathologic integration

Elevated resistance

Arterial remodeling

Right-heart stress

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Learning objectives

A pathology-centered approach to pulmonary hypertension (PH)

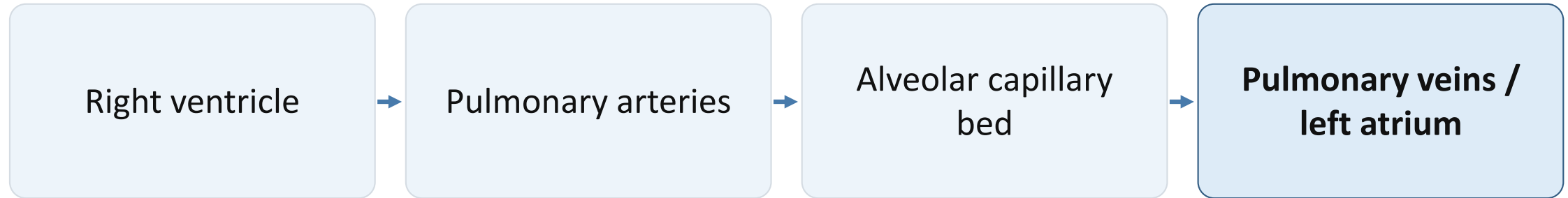
- Differentiate raised pulmonary arterial pressure from pulmonary arterial hypertension (PAH).
- Classify major etiologies by the compartment generating pressure.
- Trace vascular remodeling to right ventricular dysfunction.
- Recognize medial hypertrophy, intimal fibrosis, and plexiform lesions.

Interpretation

Clinical correlations and contemporary hemodynamic definitions are identified separately.

Pulmonary circulation: Low-resistance circuit

Mechanistic sequence



WHY IT MATTERS

Pressure rises when flow increases, distal resistance increases, or downstream venous pressure is elevated.

Definition: Distinguish textbook and current practice

Definition	Mean pulmonary artery pressure	Interpretation
Robbins source (11th ed.)	≥25 mm Hg at rest	Textbook passage retains the historical cutoff.
2022 ESC/ERS	>20 mm Hg at rest	Current guideline threshold; Measured invasively.

Do not present the historical 25-mm Hg cutoff as the current standard.

Pulmonary hypertension $\neq$ pulmonary arterial hypertension

PH describes a hemodynamic state; PAH is a specific disease group.

- PH may arise from left heart disease, hypoxic lung disease, obstruction, or vascular arteriopathy.
- PAH (group 1) primarily involves remodeling of the small pulmonary muscular arteries.
- Etiologic classification is necessary before interpreting vascular findings or planning treatment.

Interpretation Use “PH” for the umbrella diagnosis and “PAH” only when group 1 disease is established.

Three independent drivers of elevated pressure

Driver	Immediate effect	Typical clinical context
Excess pulmonary flow	Greater flow through pulmonary bed	Certain congenital shunts
Elevated arterial resistance	Larger pressure drop across vascular bed	PAH; Obstructive lung disease; CTEPH
High downstream venous pressure	Back-transmission from left-sided circulation	Mitral disease; Left ventricular dysfunction

These are parallel mechanisms—not sequential stages.

Hemodynamics localize the pressure source

Pattern	PAWP	Pulmonary vascular resistance
Pre-capillary PH	≤ 15 mm Hg	> 2 Wood units
Isolated post-capillary PH	> 15 mm Hg	≤ 2 Wood units
Combined post-/pre-capillary PH	> 15 mm Hg	> 2 Wood units

All patterns require mPAP > 20 mm Hg at rest (2022 ESC/ERS).

Five clinical groups: Mechanism and exemplar

Group	Primary mechanism	Representative condition
1 — PAH	Small pulmonary arterial disease	Idiopathic / heritable PAH; Systemic sclerosis
2 — Left heart	Back-transmitted venous pressure	Mitral stenosis; Heart failure
3 — Lung / hypoxia	Loss of vessels; Hypoxic injury	COPD; Interstitial lung disease
4 — Obstruction	Persistent thromboembolic obstruction	CTEPH
5 — Mixed / unclear	Multifactorial pathobiology	Heterogeneous disorders

CTEPH = chronic thromboembolic pulmonary hypertension.

Group 1: Primary small-artery disease

PAH centers on pulmonary vascular cell dysfunction and remodeling.

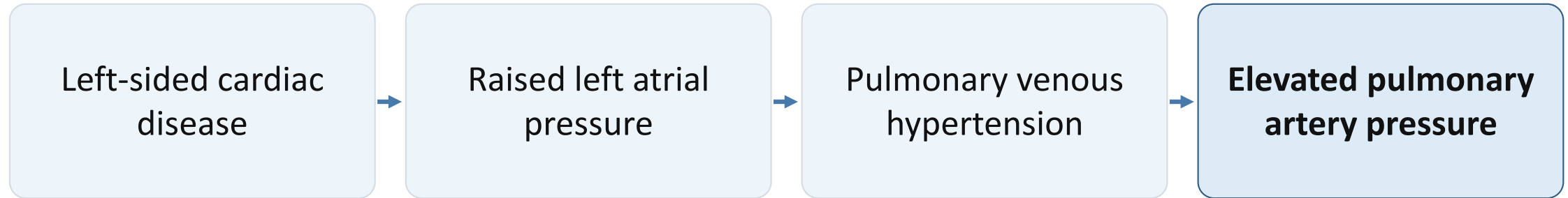
- Muscular arteries and arterioles develop medial hypertrophy and intimal fibrosis.
- Progressive luminal compromise increases pulmonary vascular resistance.
- Plexiform lesions may appear in advanced severe vascular disease.

Interpretation

Heritable and idiopathic PAH are prototypes, but group 1 includes several associated conditions.

Group 2: Left-sided pressure transmits backward

Mechanistic sequence

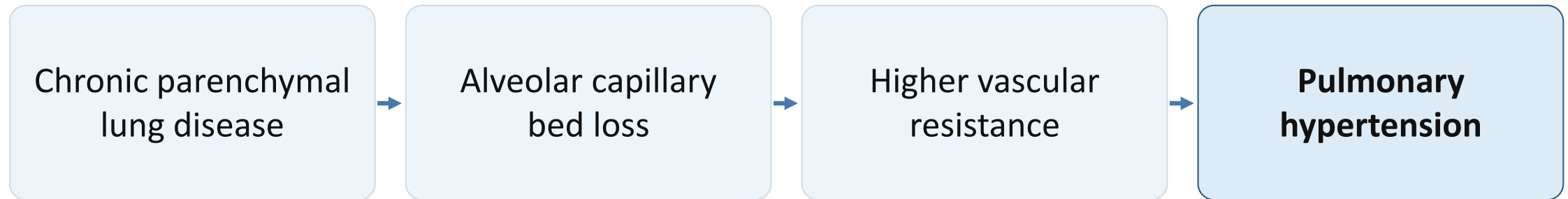


WHY IT MATTERS

A higher pulmonary artery pressure need not mean that primary arterial remodeling initiated the disease.

Group 3: Lung injury increases resistance

Mechanistic sequence

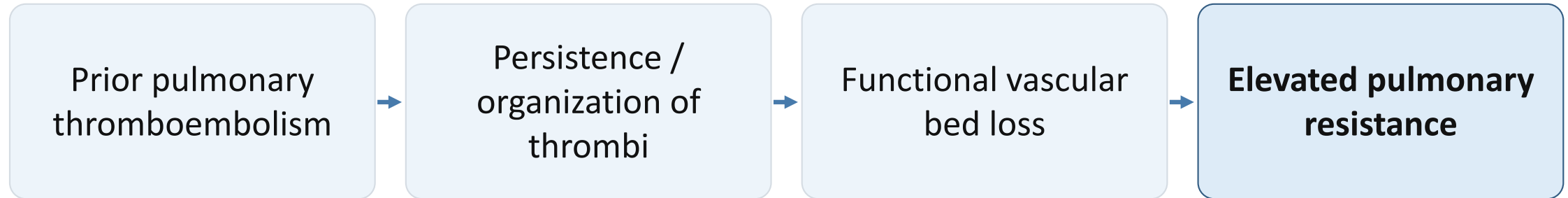


WHY IT MATTERS

Hypoxia and associated vascular responses can add to the loss of functional cross-sectional area.

Group 4: Unresolved thromboembolic obstruction

Mechanistic sequence



WHY IT MATTERS

Multiple small and recurrent emboli may gradually produce chronic PH.

Group 5: Multifactorial mechanisms

Group 5 acknowledges PH that is not explained by one predominant pathway.

- Obstruction, vascular remodeling, and altered flow can contribute simultaneously.
- The category prevents incorrectly assigning every patient to a classic arteriopathy.
- Interpretation requires integration of the underlying systemic disorder and hemodynamics.

Interpretation Robbins groups such cases as unclear and/or multifactorial.

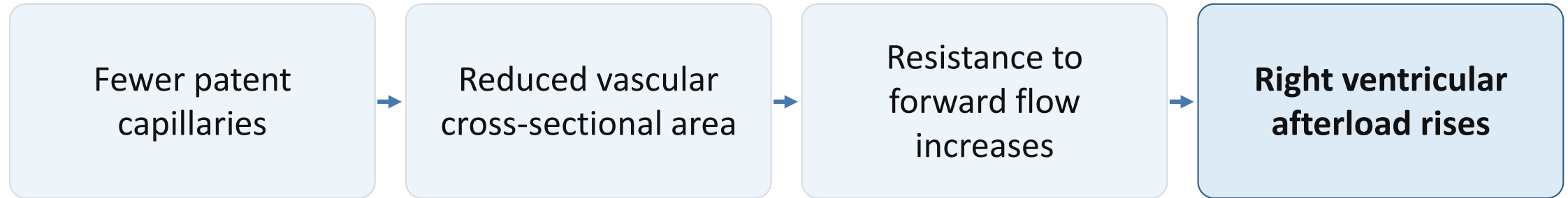
Mechanism determines what pathology to seek

Suspected driver	Helpful clinical context	Expected tissue clue
Recurrent thromboemboli	Prior embolism or risk factors	Organizing / recanalized thrombi
Chronic hypoxic lung disease	Fibrosis, emphysema, chronic bronchitis	Capillary bed loss plus remodeled arteries
Left heart disease	Mitral or ventricular dysfunction	Changes secondary to venous pressure
Group 1 arteriopathy	Idiopathic / familial / associated PAH	Marked small-vessel remodeling

Patterns support an etiologic hypothesis; None is a standalone substitute for clinical correlation.

Capillary loss converts structure into resistance

Mechanistic sequence



WHY IT MATTERS

Explains why diffuse lung disease can cause pulmonary vascular disease without a primary pulmonary arterial mutation.

COPD: Vessel loss accompanies parenchymal injury

Severe emphysema and chronic bronchitis suggest group 3 PH.

- Emphysematous destruction reduces available alveolar-capillary surface.
- Chronic hypoxemia may accentuate pulmonary vascular resistance.
- Morphology requires simultaneous assessment of airspaces, bronchi, and arteries.

Interpretation Pulmonary vessel findings alone should not obscure the responsible lung phenotype.

Interstitial fibrosis: Fewer functional capillaries

Interstitial disease creates a vascular as well as a gas-exchange lesion.

- Architectural distortion and fibrosis destroy or compress capillary networks.
- Less perfused vascular area elevates resistance as disease becomes extensive.
- Pulmonary arterial remodeling may be secondary to chronic parenchymal disease.

Interpretation Extensive fibrosis beside vascular remodeling shifts the etiologic inference toward group 3.

Sleep apnea requires etiologic nuance

Obstructive sleep apnea, obesity, hypoxemia, and PH.

- Sleep-related oxygenation abnormalities can coexist with clinically significant PH.
- Current ESC/ERS guidance cautions that isolated nocturnal OSA alone generally does not cause PH.
- Evaluate daytime hypoventilation, lung disease, obesity-related hypoventilation, and cardiac disease.

Interpretation

A source-era assertion is explicitly qualified by contemporary guidance.

Mitral stenosis: Prototype post-capillary PH

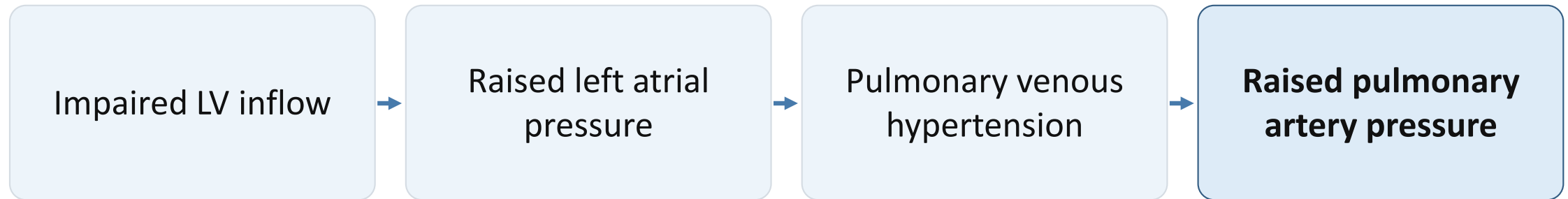
A left atrial pressure barrier can elevate pulmonary arterial pressure.

- The stenotic mitral valve increases left atrial pressure.
- Increased pulmonary venous pressure is transmitted through the lung circulation.
- Prolonged hemodynamic stress can produce secondary vascular changes.

Interpretation Backward transmission of pressure is mechanistically distinct from primary PAH.

Mitral stenosis: Causal chain

Mechanistic sequence



WHY IT MATTERS

This is a post-capillary mechanism and must not be mistaken for clinical group 1 PAH.

Recurrent emboli: Chronic vascular obstruction

Repeated small emboli can be more important than one dramatic event.

- Nonresolving thromboemboli reduce the perfused vascular bed.
- Organization and recanalization may be observed histologically.
- Persistent pulmonary resistance drives chronic right-heart load.

Interpretation

The burden of organized obstruction and the global hemodynamic context must be considered.

Organized thrombus is an etiologic clue

A remodeled artery plus an organizing clot favors chronic thromboembolic disease.

- Organizing thrombi become incorporated into vessel walls.
- Recanalization creates channels through residual thrombotic material.
- Numerous lesions across vessels strengthen suspicion for recurrent thromboemboli.

Interpretation Do not mistake an organized thrombus for a plexiform endothelial proliferation.

Systemic sclerosis: Connective-tissue association

Autoimmune disease can produce pulmonary vascular arteriopathy.

- Systemic sclerosis as a notable associated cause of PH.
- Pulmonary vascular involvement can increase resistance through arterial remodeling.
- Coexisting interstitial disease is a competing or additional mechanism.

Interpretation Distinguish vasculopathy-predominant PAH from PH dominated by fibrotic lung disease.

Associated PAH: Context is essential

Certain infections and drug exposures can accompany group 1 PAH.

- HIV infection and drug-associated cases among settings with severe arteriopathy.
- Plexiform remodeling may occur in these clinical contexts.
- A plexiform lesion signals a morphologic pattern, not a single unique cause.

Interpretation

Diagnostic attribution still depends on history and exclusion of competing hemodynamic mechanisms.

Idiopathic PAH: Diagnosis after evaluation

“Idiopathic” is an etiologic designation rather than a morphologic subtype.

- No recognized secondary cause is established after appropriate evaluation.
- Some apparently sporadic cases have inherited molecular predisposition.
- The term does not mean that molecular pathogenesis is absent.

Interpretation

Why investigation of familial disease informed pathogenesis.

Familial PAH: Variable expression

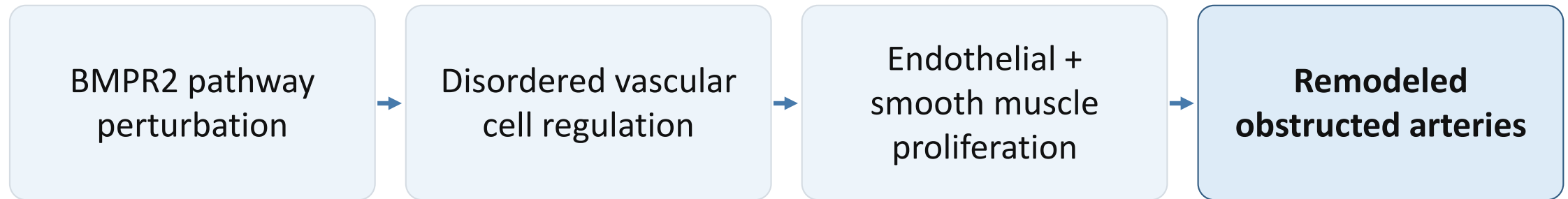
Autosomal dominant inheritance can show strikingly incomplete penetrance.

- A predisposition may segregate through relatives without clinical disease in every carrier.
- Overt disease in only a minority of genetically susceptible relatives.
- Genotype informs susceptibility; It does not guarantee clinical expression.

Interpretation Avoid interpreting a normal phenotype in a relative as proof of absent genetic risk.

BMPR2: Loss of a growth-regulatory brake

Mechanistic sequence



WHY IT MATTERS

The simplified causal model reflects the Robbins text; It does not specify unverified intracellular intermediates.

BMPR2 receptor: Biologic identity

BMPR2 is a receptor within the TGF- β superfamily.

- Bone morphogenetic protein signaling helps regulate cell proliferation and differentiation.
- The pathway also has roles in development and apoptosis.
- Reduced BMPR2 signaling is linked to proliferative pulmonary vasculopathy.

Interpretation

Preserve distinction between a germline variant and reduced receptor expression in affected tissue.

BMPR2 mutation frequencies: Source-era figures

Molecular findings in familial and apparently sporadic PAH.

- Inactivating germline BMPR2 variants: About 75% of familial cases in its account.
- About 25% of sporadic cases are described as harboring BMPR2 variants.
- These are textbook-reported estimates, not universal current genetic-test yields.

Interpretation

Numerical estimates are presented with their original source context; Original chapter PDF unavailable.

BMPR2 pathway may be altered without a variant

Normal DNA sequence does not imply normal pathway signaling.

- Receptor downregulation in some idiopathic cases without BMPR2 mutations.
- Other molecular defects can converge on the same pathway.
- The common theme is dysregulated vascular cell behavior.

Interpretation A negative BMPR2 test cannot exclude PAH.

Two-hit model: Interacting susceptibility and triggers

Component	Proposed contribution	Clinical implication
BMPR2 predisposition	Loss of regulatory signaling	Carrier may remain unaffected
Modifier or environmental insult	Additional effect on vascular cell behavior	Risk and timing vary
Combined susceptibility	Permits proliferative vascular response	Remodeling may emerge

Conceptual model used to explain incomplete penetrance, not a proven universal sequence.

Incomplete penetrance: Mechanistic implications

One genotype can produce several phenotypic outcomes.

- Additional genetic modifiers or exposures may influence the transition to overt disease.
- Timing and magnitude of vascular injury may vary among carriers.
- A mechanistic model is not proof that every trigger has been identified.

Interpretation Avoid deterministic counseling based solely on the presence of a BMPR2 variant.

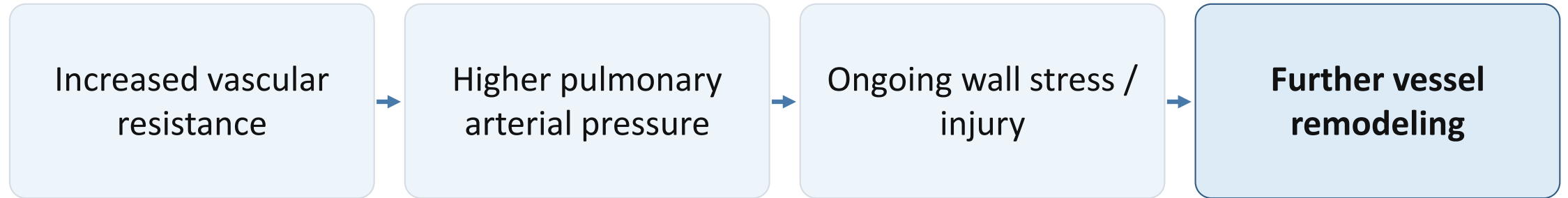
Cellular contributors to vascular remodeling

Compartment	Change emphasized by Robbins	Effect on vessel
Endothelium	Dysfunction and abnormal proliferation	Altered luminal architecture
Vascular smooth muscle	Hypertrophy / proliferation	Increased medial thickness
Intimal connective tissue	Fibrous expansion	Progressive luminal narrowing

Combined remodeling raises resistance throughout affected small vessels.

Positive feedback: Pressure and remodeling

Mechanistic sequence



WHY IT MATTERS

Conceptual clinicopathologic model: Structural narrowing can sustain hemodynamic disease.

Morphology spans the pulmonary arterial tree

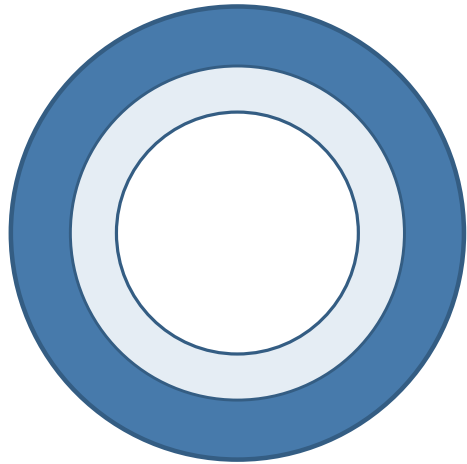
Pulmonary hypertension is not restricted to the smallest vessels.

- Large main or branch pulmonary arteries may show marked wall changes.
- The most diagnostic microscopic remodeling lies in small arteries and arterioles.
- Both lesion distribution and background lung pathology contribute to etiology.

Interpretation Changes extending from main pulmonary arteries to small arterioles.

Small-vessel morphology: Reference architecture

Schematic cross-section (not a clinical specimen)



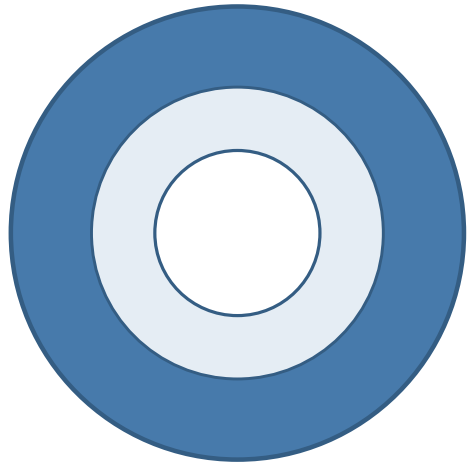
Pulmonary muscular artery

Baseline wall-to-lumen relationship

- Muscular artery with distinct lumen and muscular wall.
- Illustration intentionally simplifies vessel layers.
- Compare subsequent slides at the same illustrative scale.

Medial hypertrophy narrows the effective lumen

Schematic cross-section (not a clinical specimen)



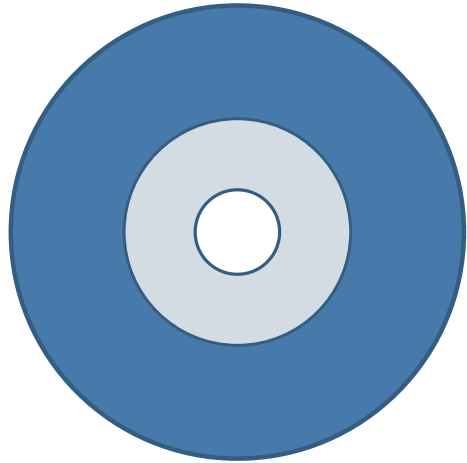
Pulmonary muscular artery

Expanded media

- Pulmonary muscular arteries develop conspicuously thickened media.
- Smooth-muscle expansion occupies more of the wall profile.
- The net consequence is a higher-resistance vascular segment.

Intimal fibrosis may leave a pinpoint lumen

Schematic cross-section (not a clinical specimen)



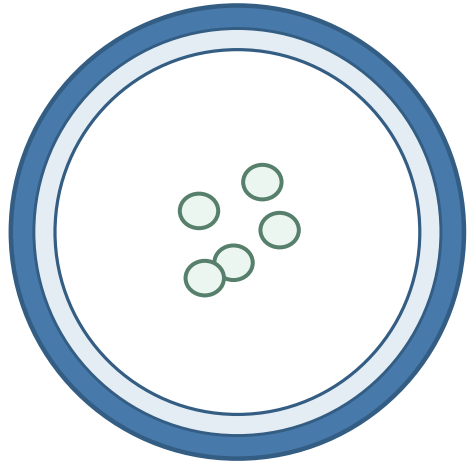
Pulmonary muscular artery

Severe fibrous luminal compromise

- Intimal collagenous expansion compounds medial thickening.
- The 40–300 μm small-artery/arteriole range.
- Severe narrowing can reduce the lumen to a narrow residual channel.

Plexiform lesion: Complex endothelial channels

Schematic cross-section (not a clinical specimen)



Affected small artery

Tuft of capillary-like channels

- A tuft of capillary-like channels forms within / near a remodeled small artery.
- The network may bridge a dilated thin-walled arterial lumen.
- This is a schematic of advanced arteriopathy—not a photomicrograph.

Patterns: Medial, intimal, plexiform

Lesion	Dominant tissue alteration	Practical recognition
Medial hypertrophy	Expanded smooth-muscle layer	Thick arterial media
Intimal fibrosis	Concentric fibrous luminal encroachment	Small residual opening
Plexiform lesion	Disordered capillary-like channels	Tuft / web-like vascular complex

These are associated changes, not three obligatory chronological stages.

Plexiform lesions: Settings and specificity

Plexiform remodeling is characteristic of severe vascular disease but is not pathognomonic.

- It is conspicuous in idiopathic and heritable group 1 PAH.
- Robbins also associates it with unrepaired shunt-related disease, HIV, and drugs.
- Clinical classification of congenital-heart-associated PH requires modern hemodynamic context.

Interpretation The source's group attribution for congenital shunts is not reproduced uncritically.

Large artery change differs from systemic atherosclerosis

Atheroma-like lesions may occur in the pulmonary artery and major branches.

- Large arteries can show wall thickening and plaque-like remodeling.
- The source cautions that classic systemic atherosclerosis is not the universal pattern.
- Small muscular arterial hypertrophy and intimal fibrosis are more prominent lesions.

Interpretation Avoid equating pulmonary vascular remodeling with ordinary coronary atherosclerosis.

Morphology provides clues about cause

Observed accompanying finding	Etiologic inference	Interpretative caution
Recanalized / organized thrombi	Prior recurrent embolic disease	Requires correlation with vascular imaging
Extensive lung fibrosis	Chronic interstitial lung disease	More than one PH mechanism may coexist
Severe emphysema + bronchitis	Chronic hypoxic parenchymal disease	Pressure severity not inferred from image alone
Plexiform arteriopathy	Severe pulmonary arteriopathy	Not etiology-specific by itself

Look beyond the artery: Assess the accompanying lung and heart findings.

Right ventricular hypertrophy is the cardiac counterpart

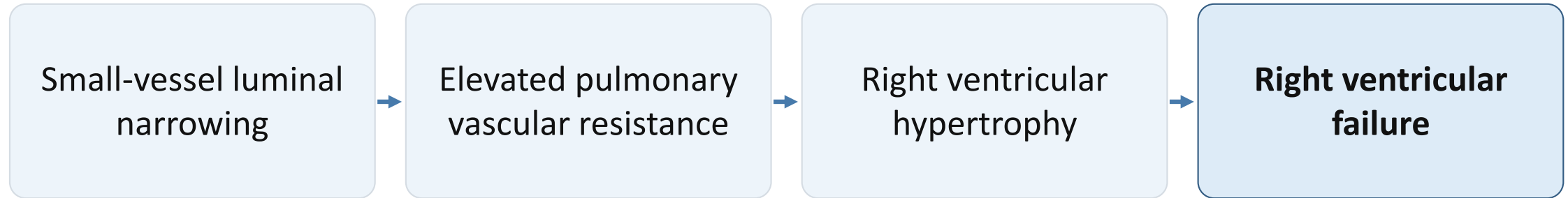
The right ventricle adapts to chronically increased afterload.

- Increased pulmonary vascular resistance raises right ventricular pressure work.
- The right ventricular myocardium hypertrophies during compensation.
- Progressive decompensation produces chronic cor pulmonale.

Interpretation The heart and pulmonary vessels reflect the same sustained hemodynamic load.

From arteriopathy to cor pulmonale

Mechanistic sequence



WHY IT MATTERS

Morphology creates hemodynamic afterload; Prolonged afterload ultimately threatens pump function.

Clinical silence may conceal advanced pathology

Symptoms often appear only after significant vascular and right-heart impairment.

- The pulmonary vascular bed can lose functional reserve progressively.
- Non-specific exertional symptoms may delay recognition.
- Severity cannot be reliably inferred from symptom description alone.

Interpretation

Clinical and hemodynamic evaluations are complementary; Do not diagnose PH from symptoms alone.

Dyspnea: Impaired cardiopulmonary reserve

Breathlessness is a major presenting symptom in idiopathic PAH.

- A high-resistance circuit limits the ability to augment pulmonary blood flow.
- Exercise accentuates the mismatch between demand and cardiac output reserve.
- Dyspnea is non-specific; Competing pulmonary and cardiac causes remain possible.

Interpretation

Relate the symptom to circulatory reserve rather than assuming a primary airway obstruction.

Fatigue: Inadequate forward-flow reserve

Reduced functional cardiac output helps explain early exertional fatigue.

- The right ventricle may fail to maintain flow at increased exertion.
- Patients can report functional limitation before overt resting failure.
- The symptom has low specificity and requires a hemodynamic differential.

Interpretation Exertional intolerance is compatible with PH but is not diagnostic.

Chest discomfort and angina-like pain

Pulmonary hypertension may produce chest pain without coronary plaque rupture.

- An anginal character of pain in some idiopathic cases.
- Increased ventricular pressure work is a plausible contributor.
- Cardiac ischemia and other urgent causes still require evaluation.

Interpretation Do not label a real chest pain episode as benign on the basis of presumed PH.

Cyanosis and right-heart failure are late warnings

Severe disease progresses beyond exertional symptoms.

- Respiratory distress and cyanosis in advanced disease.
- Right ventricular hypertrophy eventually may progress to pump failure.
- Clinical decompensation reflects severe integrated cardiopulmonary dysfunction.

Interpretation Advanced signs should prompt timely specialist assessment.

Cor pulmonale: Structural and functional endpoint

Chronic lung or pulmonary vascular disease can lead to right-heart remodeling.

- Sustained pulmonary afterload produces right ventricular hypertrophy.
- Reduced cardiac output and systemic venous congestion may follow decompensation.
- Identify whether the driving process is vascular, parenchymal, or thromboembolic.

Interpretation

Cor pulmonale is a consequence, not a separate etiologic PH group.

Intercurrent disease can precipitate deterioration

Severely compromised right-heart reserve is sensitive to added insults.

- Robbins mentions superimposed thromboembolism and pneumonia during deterioration.
- Further vascular obstruction can abruptly increase afterload.
- Acute infection may impair oxygenation and destabilize hemodynamics.

Interpretation

Historical source descriptions of untreated survival are not treated as present-day prognosis.

Histologic appearance versus mechanism

Appearance	Mechanistic interpretation	What to verify clinically
Thick media	Chronic arterial muscular response	Right-heart load / PH context
Concentric intimal fibrosis	Reduced vessel lumen	Distribution and background disease
Plexiform complex	Severe endothelial remodeling	PAH-associated conditions
Recanalized thrombus	Chronic organized obstruction	Thromboembolic workup

Histology alone does not classify pulmonary hypertension.

Case: Systemic sclerosis with dyspnea

A patient with systemic sclerosis develops progressive exertional dyspnea. Pulmonary vascular remodeling is suspected.

Discussion prompts

- Could pulmonary vasculopathy be sufficient to explain PH?
- What competing structural lung mechanism must be sought?
- Which tests would identify the dominant hemodynamic driver?

Facilitator note: Answer and rationale are in PowerPoint speaker notes.

Case: Recurrent small pulmonary emboli

A patient has repeated pulmonary embolic episodes and progressive exertional limitation. Histology shows organized material with channels.

Discussion prompts

- What does recanalization indicate?
- How can recurrent small thrombi elevate resistance?
- Which clinical group should be investigated?

Facilitator note: Answer and rationale are in PowerPoint speaker notes.

Case: Asymptomatic BMPR2 variant carrier

A relative of a patient with heritable PAH carries a pathogenic BMPR2 variant but has no symptoms.

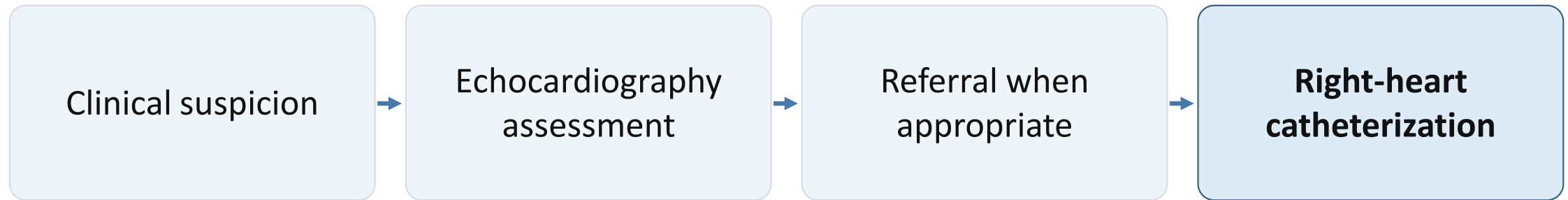
Discussion prompts

- Does a variant guarantee clinically manifest disease?
- Which pathogenetic concept explains discordant relatives?
- Why is “idiopathic” not synonymous with “non-genetic”?

Facilitator note: Answer and rationale are in PowerPoint speaker notes.

Diagnostic pathway: Screen, detect, confirm

Mechanistic sequence



WHY IT MATTERS

The 2022 ESC/ERS diagnostic pathway requires invasive hemodynamic confirmation; Echo alone cannot definitively classify PH.

Right-heart catheterization defines the phenotype

Catheterization integrates pressure measurements and flow.

- Mean pulmonary arterial pressure establishes whether hemodynamic PH is present.
- Wedge pressure distinguishes elevated left-sided filling pressure from pre-capillary patterns.
- Cardiac output is used to calculate pulmonary vascular resistance.

Interpretation

Current 2022 ESC/ERS threshold: MPAP >20 mm Hg; Do not diagnose PAH on mPAP alone.

Pressure phenotype directs the differential

Hemodynamics	Initial etiologic direction	Why
Elevated PAWP	Left-heart disease evaluation	Venous pressure can transmit upstream
High PVR + normal PAWP	PAH, lung disease, CTEPH evaluation	Resistance lies predominantly before capillaries
Combined elevation	Mixed post- and pre-capillary process	Chronic venous pressure may coexist with vascular remodeling

These are organizing heuristics; The complete diagnosis requires clinical context.

Management is cause-directed

A pathology diagnosis is useful when it identifies the driving pathway.

- Secondary PH requires treatment of the precipitating disorder where possible.
- Hypoxemia and chronic thromboembolic disease require different assessments.
- PAH-directed vasodilator therapy is selected within the appropriate clinical group.

Interpretation Treating the underlying trigger and specialized care for group 1.

Why PAH drugs do not fit every PH group

An elevated pressure is not, by itself, a mechanism-specific treatment indication.

- Group 2 is dominated by elevated left-sided filling pressure.
- Group 3 is often dominated by chronic lung injury and hypoxic physiology.
- Group 4 includes potentially addressable organized arterial obstruction.

Interpretation

Avoid indiscriminate treatment transfer between PH groups; Therapeutic details require specialist guideline review.

Lung transplantation in selected advanced disease

Robbins recognizes transplantation as definitive treatment for selected patients.

- Advanced progressive pulmonary vascular injury may be difficult to reverse.
- Clinical candidacy depends on disease phenotype, comorbidities, and severity.
- Transplantation is a highly selected option rather than routine management.

Interpretation

The textbook gives no universal eligibility criteria; None are invented here.

High-yield differential: Vessel versus parenchyma

Primary clue	Prefer this mechanism	Do not overlook
Small arteries remodeled; Background lung relatively preserved	Pulmonary arteriopathy	Cardiac, thromboembolic, or inherited causes
Severe emphysema or fibrosis	Capillary bed loss / hypoxia	Coexisting pulmonary arteriopathy
Organized recanalized thrombi	Chronic thromboembolic obstruction	Secondary adaptive hypertrophy
Raised left atrial pressure	Post-capillary PH	Combined pre-capillary component

The final classification is etiologic and hemodynamic, not microscopic alone.

Diagnostic pitfalls that change classification

Four errors can produce a convincing but wrong pulmonary hypertension diagnosis.

- Equating elevated pulmonary artery pressure with group 1 PAH.
- Using ≥ 25 mm Hg as though it remains the current guideline threshold.
- Treating plexiform remodeling as pathognomonic for idiopathic disease.
- Ignoring left-heart disease, chronic lung disease, or thromboemboli.

Interpretation

Correct classification integrates pressures, structural disease, clinical risk factors, and pathology.

Synthesis: Five causal anchors

Anchor	Core mechanism	Signature correlation
Pressure	Resistance, flow, or venous back pressure	Measure and phenotype hemodynamics
Etiology	Five broad clinical groups	Identify the predominant driver
Molecular	BMPR2 pathway / modifiers	Variable inherited susceptibility
Morphology	Medial + intimal + plexiform changes	Reduced small-vessel caliber
Clinical	Chronic right ventricular afterload	Cor pulmonale / right-heart failure

Use these anchors to explain why the findings occur—not only to memorize them.

Application question: Interpret a narrowed arteriole

A small pulmonary artery has severe medial hypertrophy and intimal fibrosis. The patient also has extensive pulmonary fibrosis.

Discussion prompts

- Does the artery alone establish idiopathic PAH?
- Which group becomes plausible from the lung background?
- What confirms hemodynamic pulmonary hypertension?

Facilitator note: Answer and rationale are in PowerPoint speaker notes.

Selected references

Core textbook and separately verified guideline

Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed. Elsevier; 2026. Chapter 15, “The Lung,” pp. 640–642.

Humbert M, et al. 2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension. Eur Heart J. 2022;43:3618–3731. doi:10.1093/eurheartj/ehac237.