

Hypertensive Heart Disease

Pressure overload • Ventricular remodeling • Clinical consequences

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Learning objectives

- Define hypertensive heart disease and distinguish systemic from pulmonary forms.
- Explain how pressure overload produces myocyte hypertrophy and interstitial fibrosis.
- Recognize gross and microscopic changes in systemic and pulmonary HHD.
- Connect remodeling to ischemia, arrhythmia, heart failure, and reversibility.
- Apply clinicopathologic criteria and avoid common diagnostic misattribution.

One disease concept, two ventricular targets

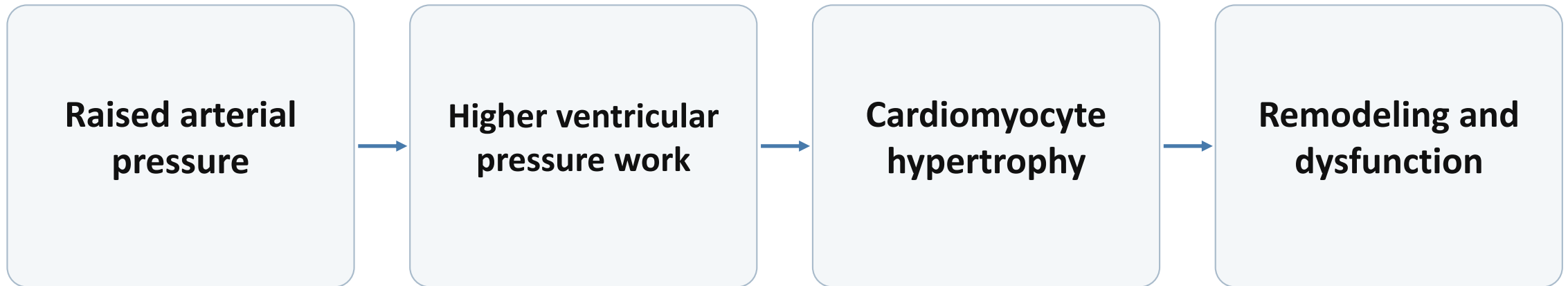
Systemic arterial hypertension

- Sustained pressure work falls primarily on the left ventricle.
- Adaptive response: Concentric LV hypertrophy.
- Late effects: Stiffness, ischemia and dysfunction.

Pulmonary arterial hypertension

- Pulmonary vascular load primarily affects the right ventricle.
- Chronic response: RV hypertrophy, often with dilation.
- Acute pressure crisis: RV dilation can precede hypertrophy.

Pressure overload is the initiating lesion



Hypertension increases afterload; The ventricle must generate greater pressure to eject blood. The gross phenotype depends on which vascular circuit is hypertensive.

Hypertrophy initially compensates—then can harm

Early compensation

- Greater muscle mass helps meet the increased mechanical demand.
- The chamber may initially retain systolic performance.

Later maladaptation

- Increased stiffness impairs filling.
- Increased oxygen demand and fibrosis erode functional reserve.
- Dilation, congestive failure or sudden death may follow.

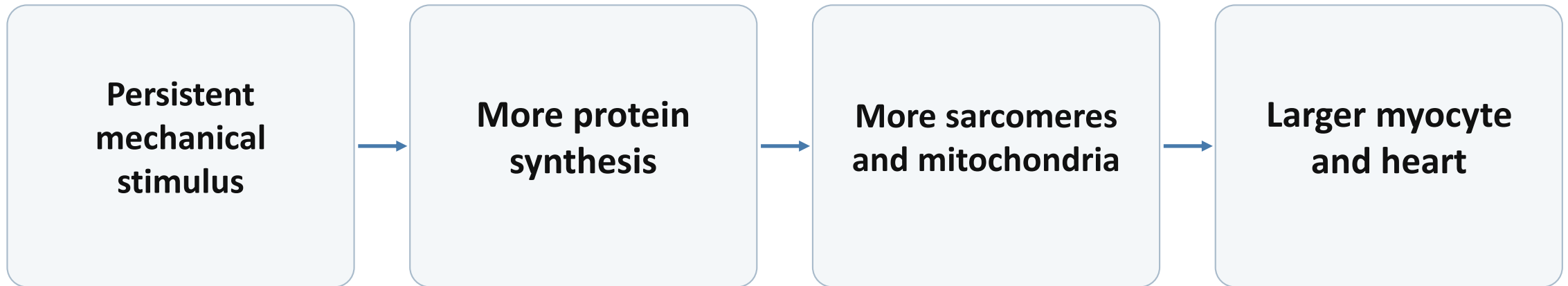
Systemic and pulmonary disease are not interchangeable

Question	Systemic HHD	Pulmonary HHD
Major load	Systemic pressure	Pulmonary vascular pressure
Primary target	Left ventricle	Right ventricle
Characteristic pattern	Concentric LV hypertrophy	RV hypertrophy ± dilation
Alternative term	Left-sided HHD	Cor pulmonale when isolated

Pressure versus volume overload

Property	Pressure overload	Volume overload
Examples	Hypertension; Aortic stenosis	Valvular regurgitation
Sarcomere addition	Predominantly parallel	Predominantly in series
Early geometry	Thickened wall	Expanded chamber
Principal remodeling	Concentric hypertrophy	Dilation with hypertrophy

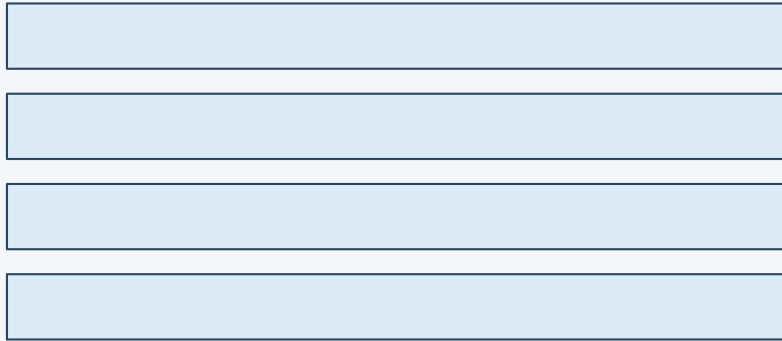
Cardiomyocytes enlarge rather than divide



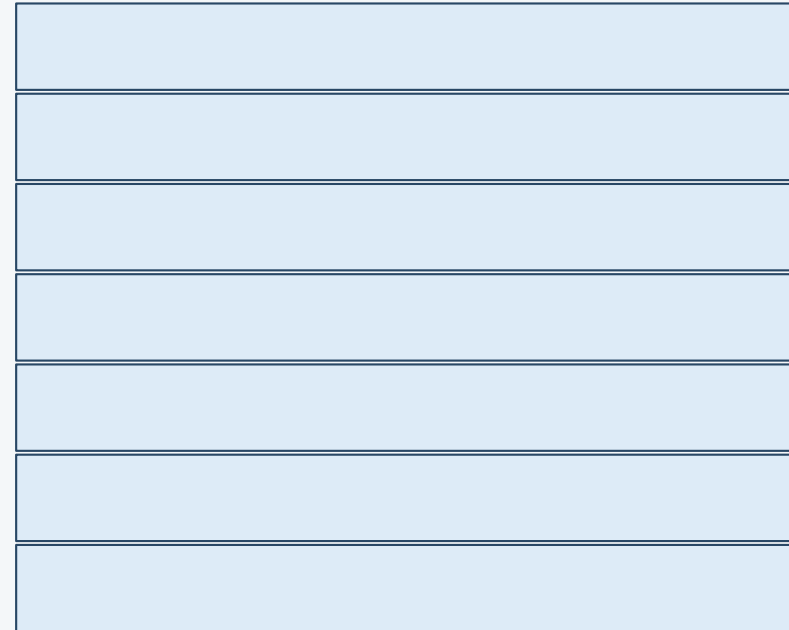
Myocardial hypertrophy reflects cellular enlargement rather than replacement by newly divided cardiomyocytes. The cumulative effect is increased cardiac mass.

Parallel sarcomeres enlarge the myocyte cross-section

Before pressure overload



With pressure overload

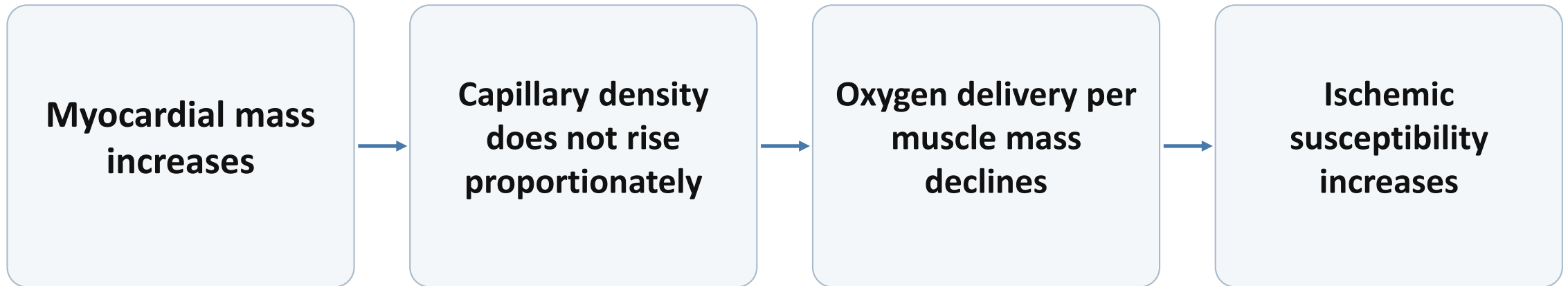


Schematic of sarcomeres: Parallel addition expands cell width rather than simply elongating the cell.

Nuclear enlargement accompanies myocyte hypertrophy

- Hypertrophic myocytes often show enlarged or multiple nuclei.
- Increased nuclear DNA content follows replication without cell division.
- A large, enlarged nucleus supports cellular hypertrophy but is not disease-specific.
- Size differences may require comparison with unaffected myocardium.

Why capillary supply becomes relatively inadequate



This supply–demand vulnerability is especially important in pressure-overload hypertrophy. Do not confuse relative capillary insufficiency with proof of epicardial coronary stenosis.

Why oxygen demand rises in hypertrophied hearts

More muscle mass

Increased workload

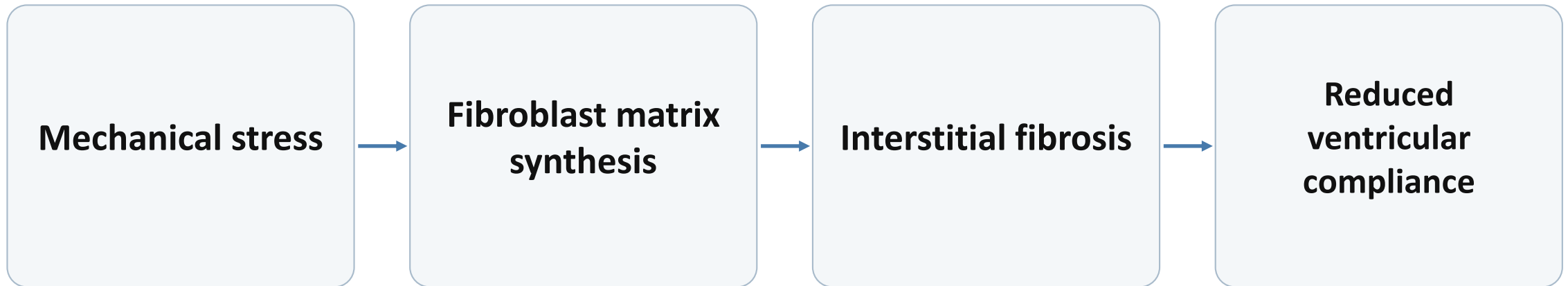
Greater myocardial O₂ demand

Heart rate / contractility

Reduced supply reserve

Mass, rate and contractile state contribute to myocardial oxygen consumption. • Ischemia can arise from supply–demand imbalance even without a newly occluded coronary artery.

Fibrosis is a second major remodeling pathway



Mechanical forces activate myocardial fibroblasts and extracellular matrix deposition. The structural cost is resistance to diastolic filling.

Cellular hypertrophy and fibrosis are distinct findings

Myocyte response

- Increased transverse cell diameter.
- Nuclear enlargement and altered gene expression.
- More contractile and mitochondrial material.

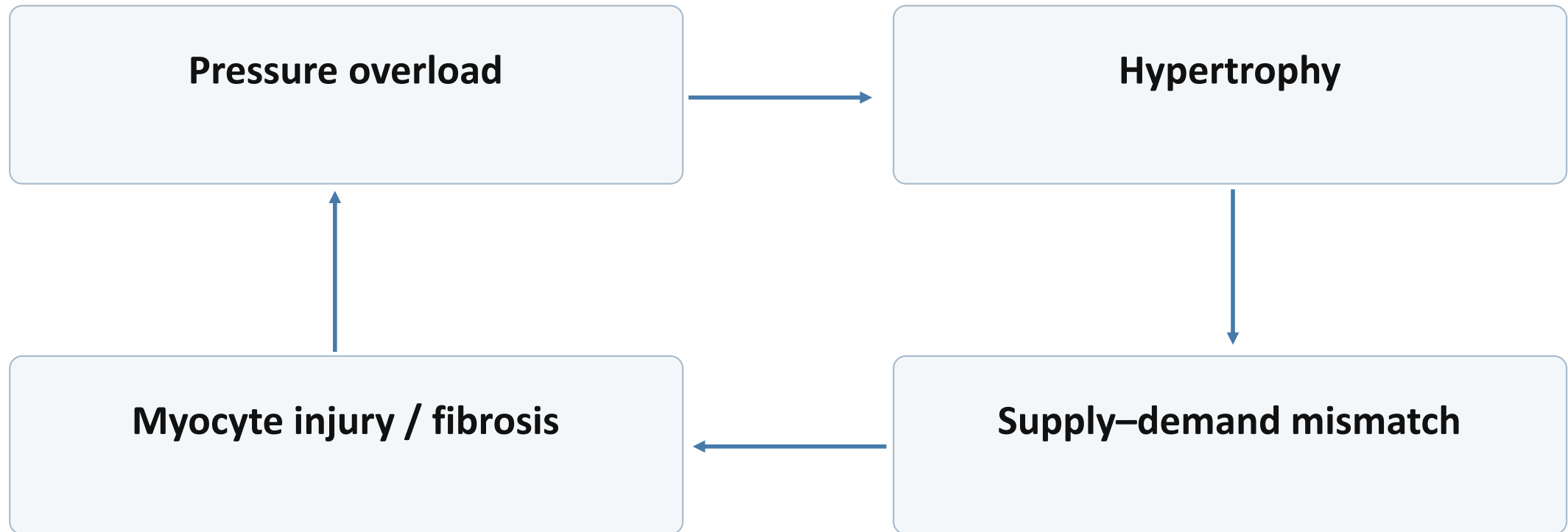
Stromal response

- Perivascular collagen deposition.
- Interstitial collagen between myocytes.
- Stiffer ventricular wall and impaired filling.

Hypertrophic signaling includes gene reprogramming

- Immediate-early growth-associated genes include FOS, JUN, MYC and EGR1.
- Persistent overload may reactivate proteins associated with fetal cardiac development.
- Examples include fetal myosin forms, natriuretic peptides and collagen programs.
- These molecular changes accompany tissue remodeling; They are not standalone HHD diagnostic tests.

A reinforcing cycle reduces functional reserve



- Fibrosis decreases compliance while hypertrophy increases metabolic demand.
- The originally protective response becomes increasingly maladaptive.

Systemic HHD: Core pathologic criteria

Required element	What pathology must establish
1. Left ventricular hypertrophy	Usually concentric; Not otherwise explained by another cardiovascular lesion
2. Evidence of hypertension	Clinical history or pathologic evidence in another organ, such as kidney
Diagnostic interpretation	Both elements must be considered together

LV hypertrophy alone does not establish systemic HHD

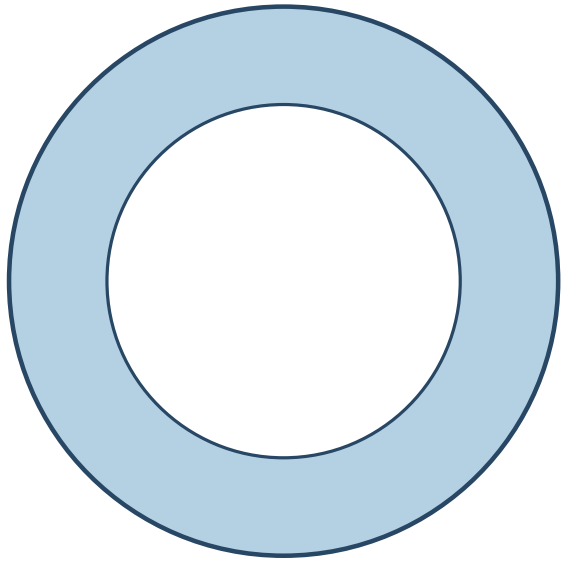
Supports hypertensive cause

- Documented longstanding systemic hypertension.
- Concentric left ventricular enlargement.
- Concordant renal or other vascular hypertensive pathology.

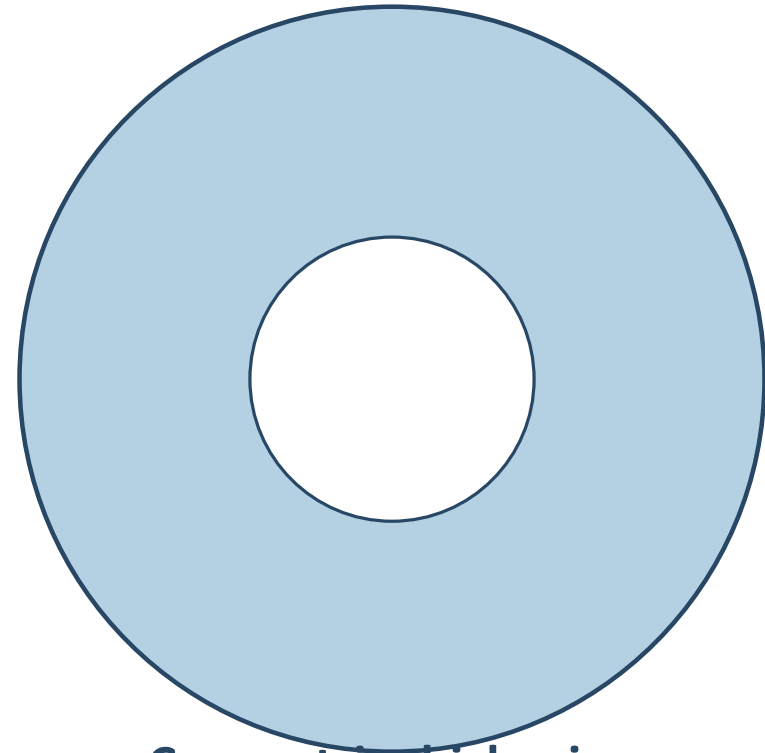
Requires alternative consideration

- Pressure overload from aortic stenosis.
- Primary myocardial disease with hypertrophy.
- Other cardiovascular causes of increased LV mass.

The typical LV phenotype is concentric



Reference geometry

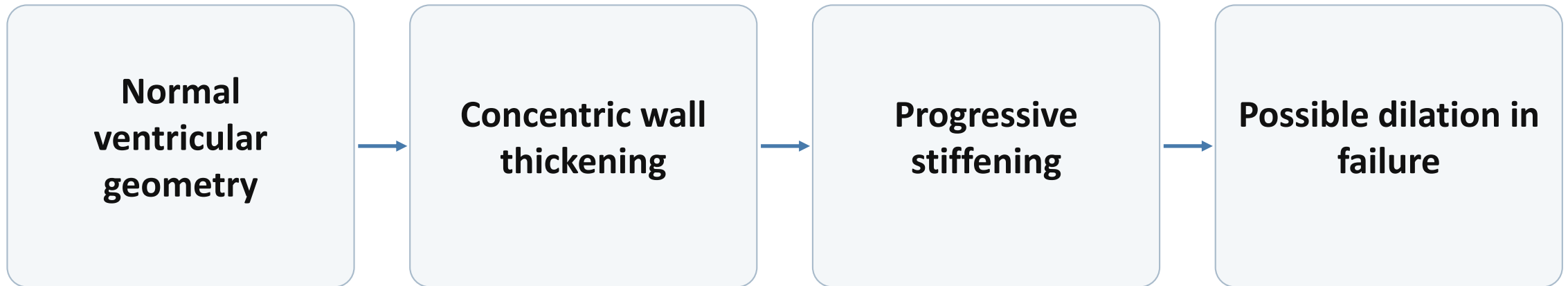


Concentric thickening

Conceptual cross-section — schematic, not a specimen

Ventricular wall becomes thicker with initially little or no chamber dilation. • Heart weight rises more than overall heart dimensions.

LV wall thickness increases before dilation



The first three stages describe the common pressure-overload trajectory. Late dilation is possible, not inevitable.

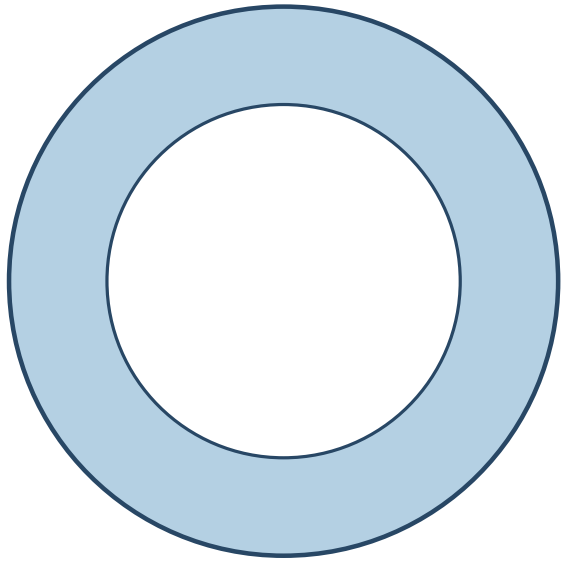
Gross pathology: Increased mass without equal enlargement

- Pressure overload causes disproportionate thickening of the left ventricular wall.
- Overall cardiac dimensions may be less impressive than the increase in heart weight.
- Heart weight may exceed 500 g in advanced examples.
- This example is not a threshold required for diagnosis.

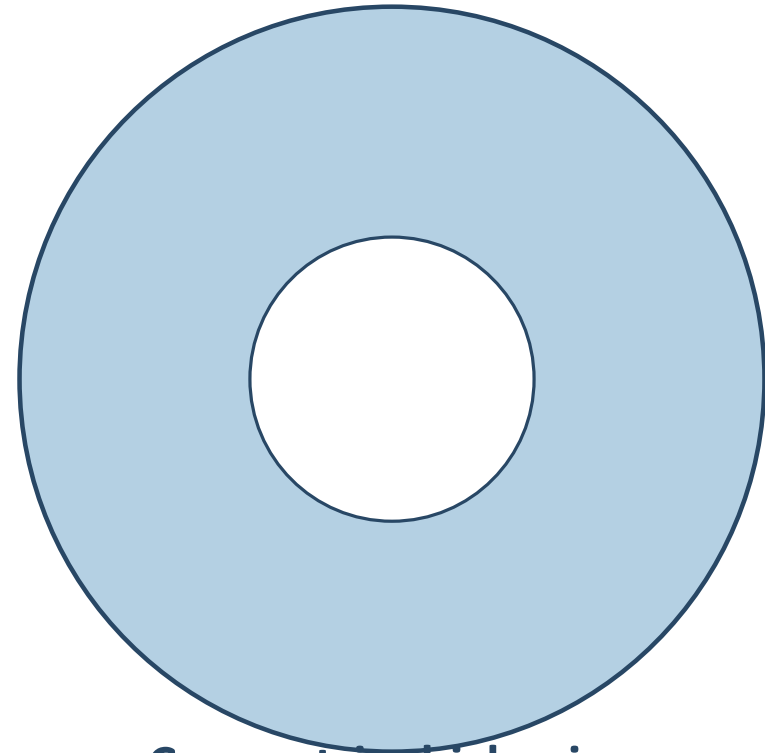
Marked LVH may exceed 2 cm in wall thickness

- The hypertrophied left ventricular wall may exceed 2.0 cm in severe systemic HHD.
- Such dimensions illustrate severity; They are not the minimal diagnostic criterion.
- Correlate measurements with specimen orientation, patient context and competing causes.
- Pressure-overloaded hearts may later dilate and alter apparent wall thickness.

How concentric LVH alters chamber geometry



Reference geometry



Concentric thickening

Conceptual cross-section — schematic, not a specimen

Compared with a normal LV, concentric hypertrophy increases wall-to-cavity proportion. • A small cavity need not signify severe systolic impairment.

Heart weight can reveal hypertrophy when the wall looks normal

- A dilated, hypertrophied ventricle may show normal or reduced apparent wall thickness.
- Therefore wall thickness alone underestimates hypertrophy in some failing hearts.
- Heart weight is a better measure of hypertrophy in dilated hearts.
- Interpret mass and chamber geometry together.

Early microscopic change: Greater myocyte diameter

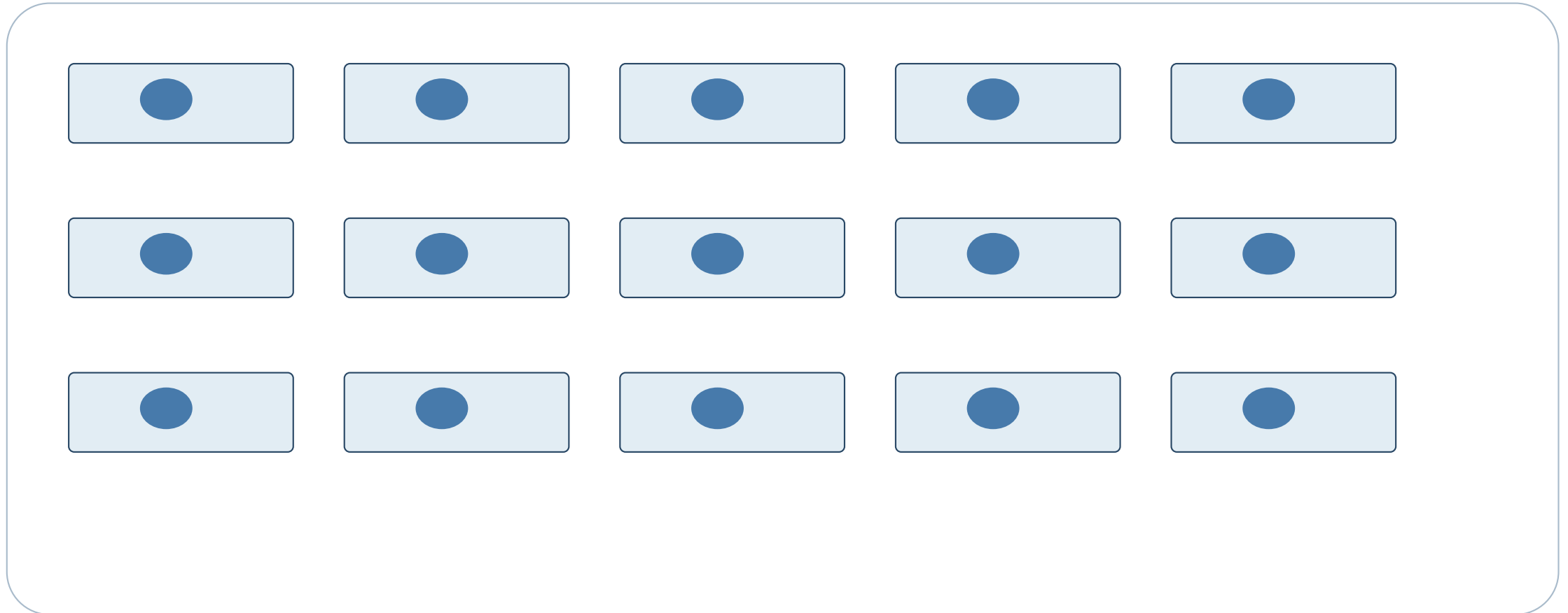
Very early change

- Increased myocyte transverse dimension is the first microscopic abnormality.
- The change can precede striking nuclear morphology.

Why it may be missed

- Routine histology may not make small size increases obvious.
- Section orientation and sampling can obscure comparisons.
- Do not overdiagnose isolated myocyte enlargement.

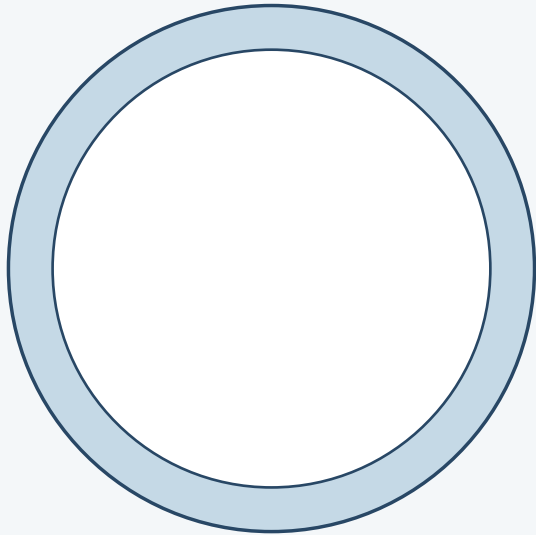
Advanced myocardial hypertrophy: Cellular and nuclear changes



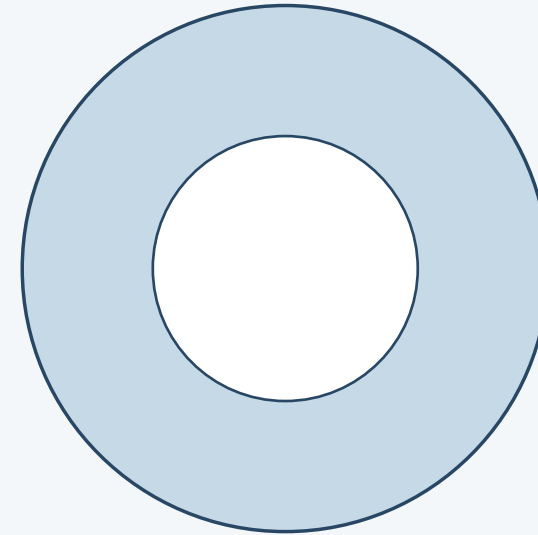
Original schematic: Cardiomyocyte profiles and enlarged myocyte nuclei

Perivascular fibrosis: Collagen around small vessels

Vessel without prominent fibrosis

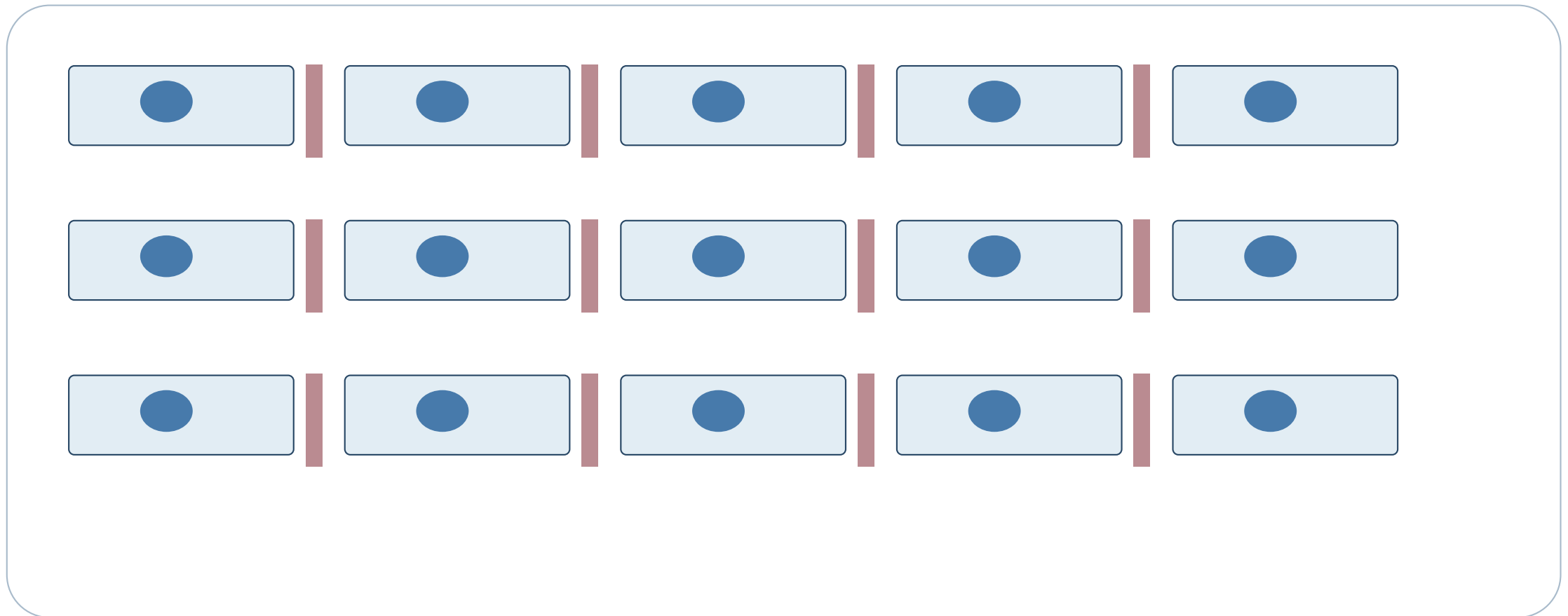


Vessel with perivascular fibrosis



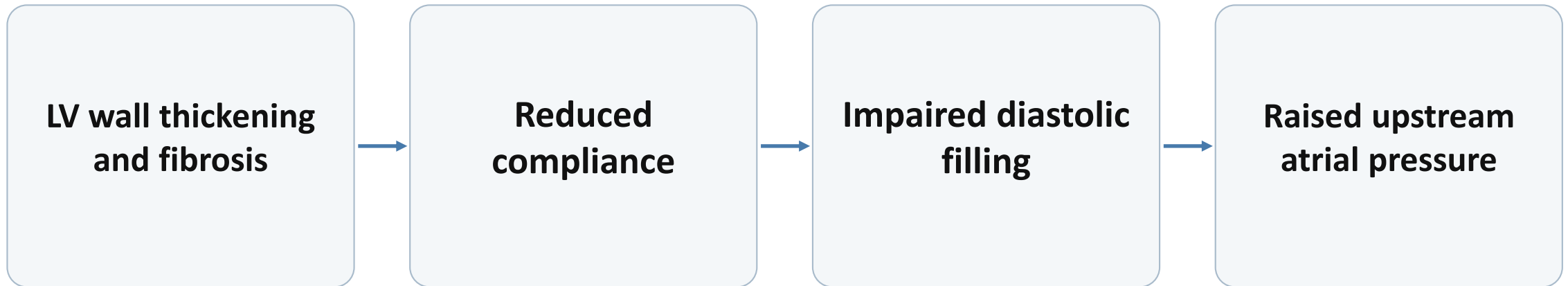
Original conceptual schematic—not a histologic specimen.

Interstitial fibrosis: Stiffness between myocytes



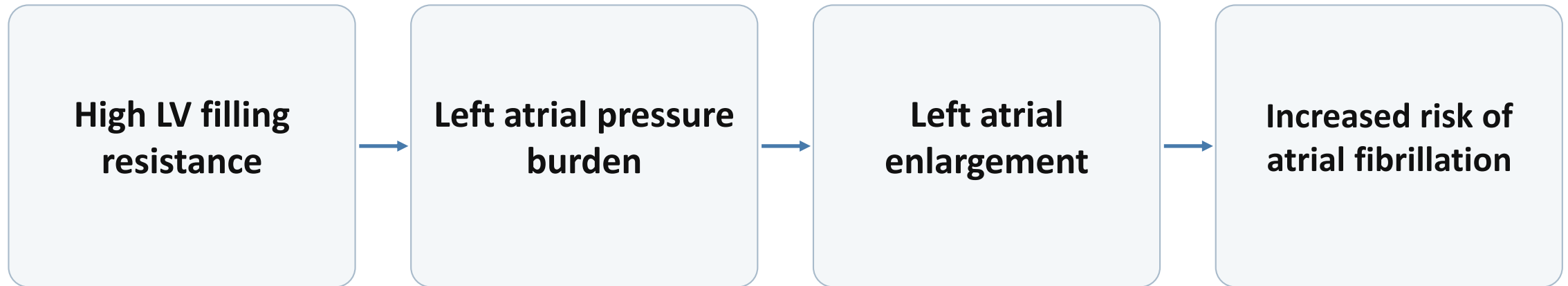
Original schematic: Cardiomyocyte profiles and interstitial collagen bands

The stiff LV fills at higher pressure



Mechanical stiffness is a direct mechanistic bridge from gross LVH to atrial consequences.

Why the left atrium enlarges



Left atrial enlargement may be an important secondary structural sign of diastolic dysfunction.

Atrial fibrillation may be the first presentation

- Compensated hypertensive LVH may be clinically silent.
- Enlargement of the left atrium creates an arrhythmogenic substrate.
- Some patients first come to attention because of newly recognized atrial fibrillation.
- The rhythm abnormality does not by itself prove hypertensive etiology.

Compensated disease can be detected before symptoms

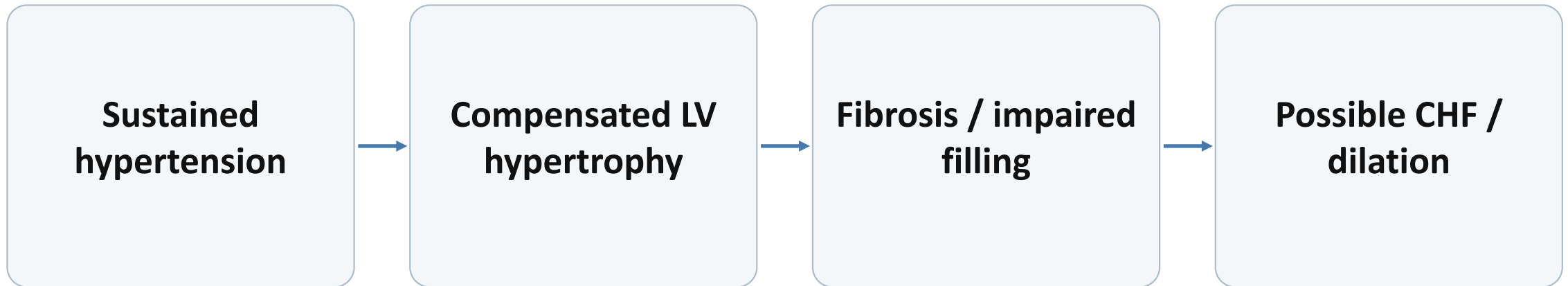
Subclinical course

- Myocardium maintains adequate output initially.
- No symptoms are required for substantial LV hypertrophy.

Available clinical clues

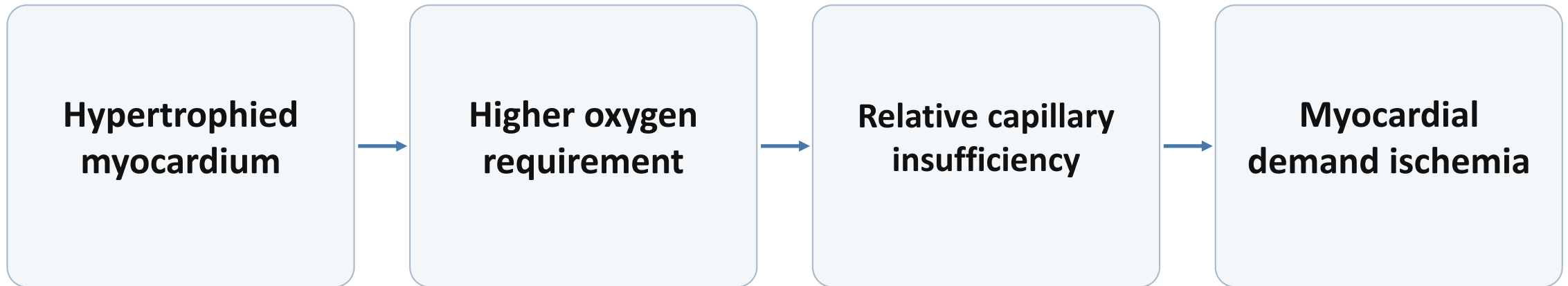
- Electrocardiographic evidence of LV enlargement.
- Echocardiographic evidence of ventricular hypertrophy.
- Longstanding hypertension and associated organ changes.

From silent hypertrophy to overt failure



Progression depends on burden, duration and adequacy of blood-pressure control. Not every patient traverses every stage.

Demand ischemia: Increased need without enough reserve



Increased oxygen requirement is a direct mechanism of demand ischemia in systemic HDD.

Systemic hypertension also promotes coronary disease

Epicardial coronary pathway

- Hypertension potentiates coronary atherosclerosis.
- Coronary disease can therefore coexist with LV hypertrophy.

Intramyocardial demand pathway

- More muscle increases myocardial oxygen consumption.
- Reduced supply reserve makes ischemia possible without plaque rupture.

Cardiac dilation signals loss of compensation

- Initially concentric LV hypertrophy is commonly non-dilated.
- With worsening myocardial dysfunction, chamber dilation may supervene.
- A dilated heart may have greater mass without dramatically thick walls.
- Do not equate late dilation solely with a primary dilated cardiomyopathy.

CHF links myocardial remodeling to systemic illness

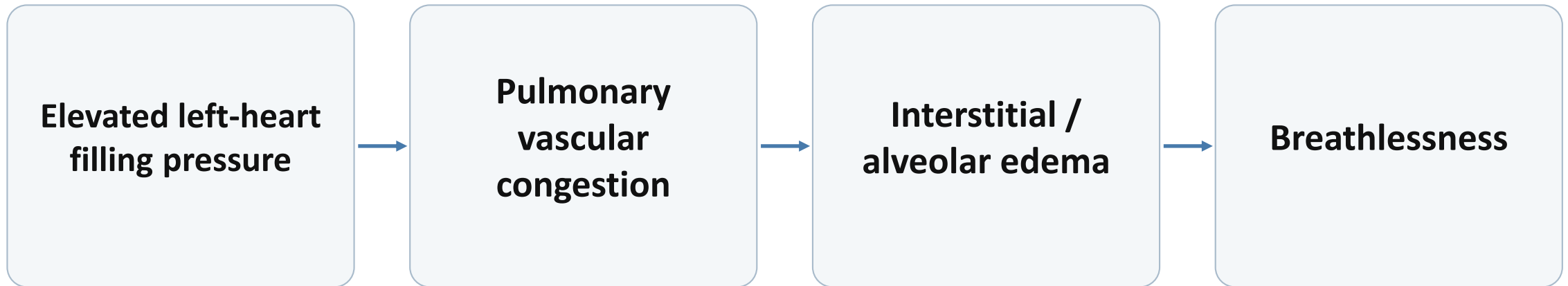
Cardiac basis

- LV dysfunction or elevated filling pressure.
- Left atrial and pulmonary venous pressure may rise.

Downstream effects

- Pulmonary congestion and edema.
- Reduced forward perfusion of organs.
- Symptoms evolve with severity of decompensation.

Left-sided failure produces pulmonary congestion



This section is heart-failure context, not required to diagnose compensated HHD.

Diastolic and systolic problems must be distinguished

Question	Predominant diastolic problem	Advanced contractile impairment
Structural contributor	Thick, fibrotic, noncompliant LV	Decompensated remodeled ventricle
Main physiologic issue	Resistance to ventricular filling	Reduced pumping ability
Potential consequence	Elevated filling pressures	Dilation and CHF
Caution	May precede overt systolic dysfunction	Not universal in HHD

Clinical outcomes are heterogeneous

Stable compensation

IHD / ischemia

Systemic HHD trajectory

Progressive CHF

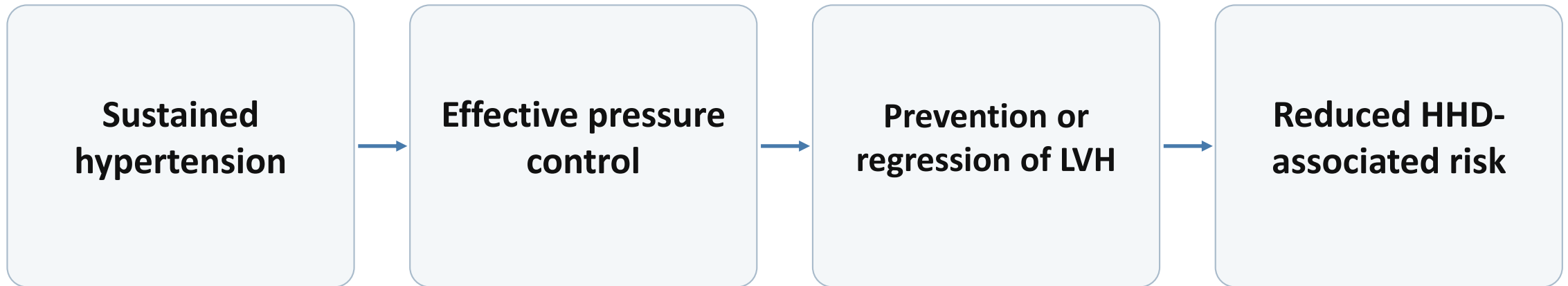
Sudden cardiac death

Some patients remain compensated; Others develop ischemia, CHF, or sudden cardiac death. • Blood-pressure severity, duration, cause and control influence outcome.

Sudden death is possible—but not inevitable

- Marked HHD increases vulnerability to clinically serious outcomes.
- Sudden cardiac death is among the possible clinical outcomes.
- Fibrosis, ischemia and electrical instability are relevant pathophysiologic context.
- Avoid inferring an individual patient's mode of death from LVH alone.

Blood-pressure control can reverse remodeling



Controlling pressure load may prevent hypertrophy or permit its regression. Regression is possible but depends on clinical circumstances.

The RV normally operates in a low-pressure circuit

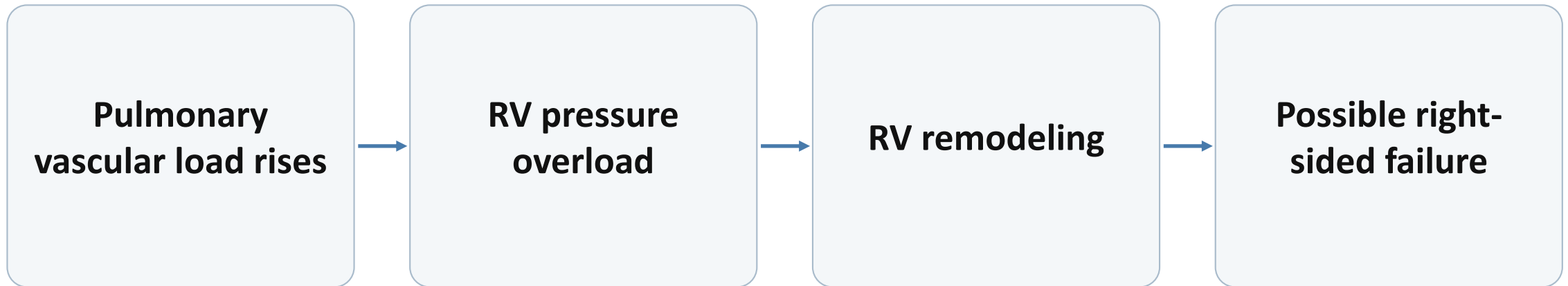
Physiologic RV design

- Thinner wall and greater compliance than the LV.
- Adapted to the lower resistance of the pulmonary circulation.

Effect of pulmonary hypertension

- Higher pressure work stresses the RV.
- Acute load may dilate the chamber.
- Persistent load favors hypertrophy.

Cor pulmonale is an RV pressure-overload phenotype

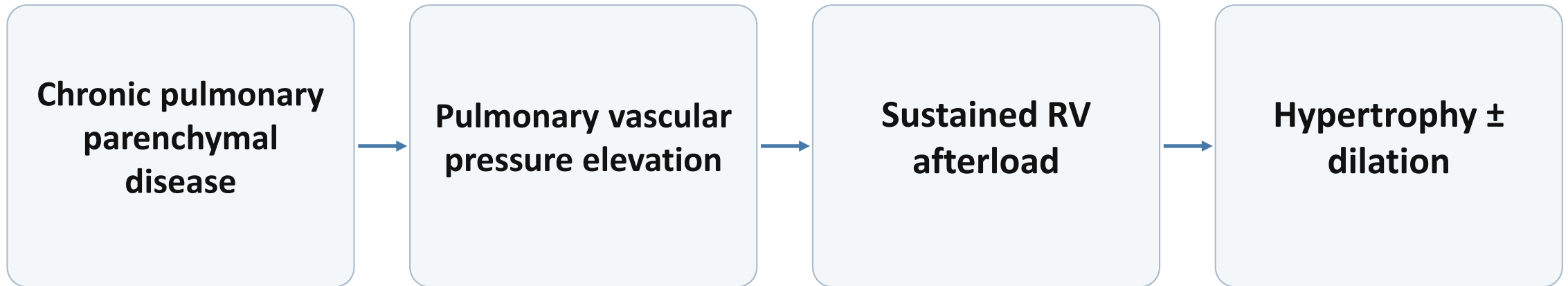


Isolated pulmonary hypertensive heart disease is termed cor pulmonale. Pulmonary hypertension from left heart disease is common, but should be etiologically distinguished from isolated cor pulmonale.

Pulmonary hypertension can originate in different places

Pathway	Illustrative cause	RV implication
Pulmonary parenchymal disease	Emphysema / chronic lung disease	Chronic RV loading
Primary pulmonary vascular disease	Primary pulmonary hypertension	Chronic RV loading
Pulmonary vascular obstruction	Massive embolism	Acute RV pressure crisis
Left-heart pathology	Secondary pulmonary hypertension	RV involvement secondary to left disease

Chronic lung disease is a typical cor pulmonale driver

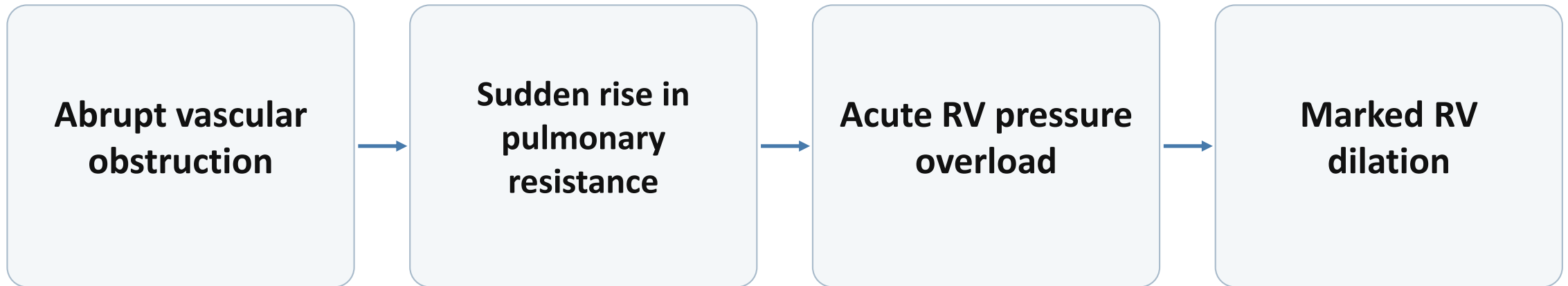


Emphysema is explicitly given as an example. The source does not require all chronic lung disease to cause cor pulmonale.

Primary pulmonary hypertension can remodel the RV

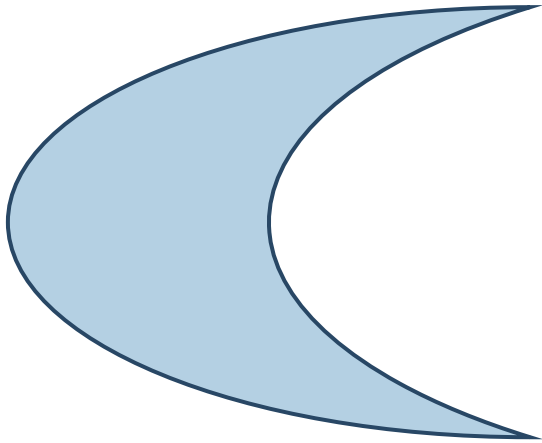
- A pulmonary vascular disease can impose sustained pressure work without primary LV failure.
- The ventricle adapts by hypertrophy but may subsequently dilate.
- Progressive pressure loading may culminate in isolated right-sided failure.
- Persistent pulmonary arterial pressure overload determines the RV remodeling response.

Massive pulmonary embolism causes acute cor pulmonale

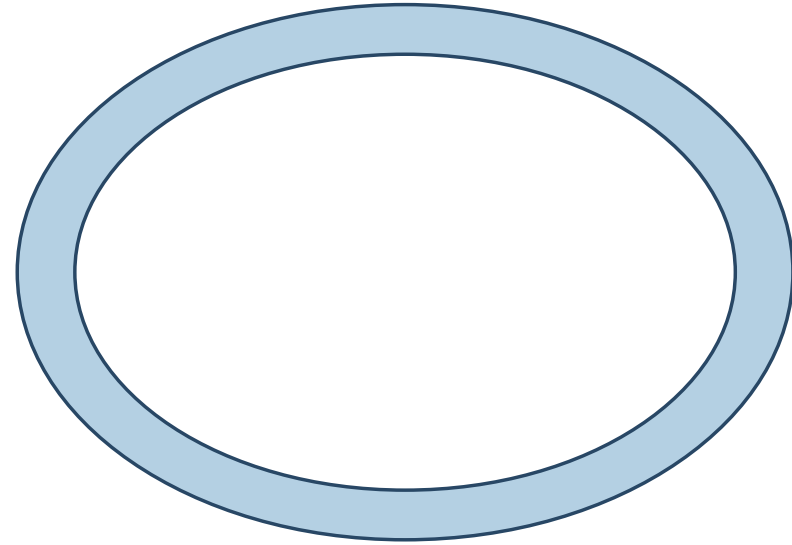


A rapid obstruction does not allow time for established hypertrophy.

Acute cor pulmonale: Dilated RV without hypertrophy



Crescent-shaped RV

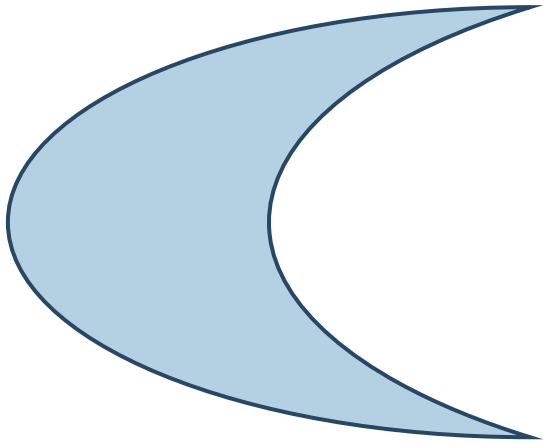


Acute dilation

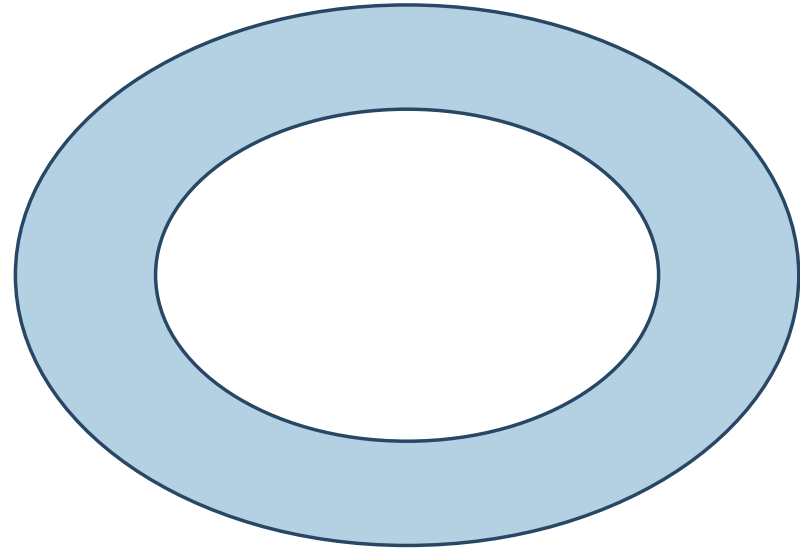
Conceptual cross-section — schematic, not a specimen

Right ventricular dilation dominates the gross picture. • In cross-section, the normally crescent-shaped RV may become ovoid.

Chronic cor pulmonale: RV hypertrophy and dilation



Crescent-shaped RV



Chronic hypertrophy + dilation

Conceptual cross-section — schematic, not a specimen

Right ventricular free wall thickens under sustained load. • Dilation often coexists with hypertrophy.

RV wall thickness can reach 1 cm or more

- Right ventricular wall thickness may reach 1.0 cm or more in advanced chronic cases.
- This describes advanced morphology, not an obligatory cutoff for cor pulmonale.
- Relative chamber enlargement and associated tissue changes still matter.
- Interpret measurements in anatomical and clinical context.

Subtle RV hypertrophy may involve the outflow tract

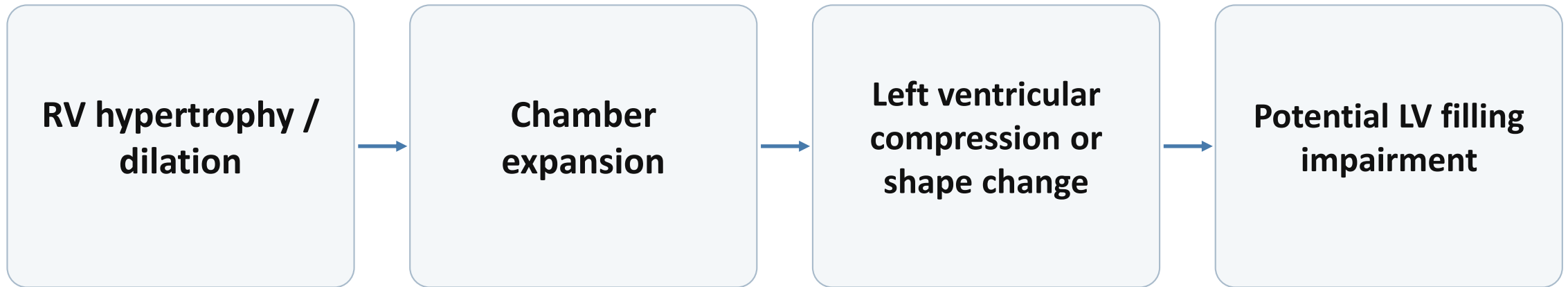
RV outflow tract

- Muscle bundles just below the pulmonary valve may thicken.
- Changes may reveal early or limited RV hypertrophy.

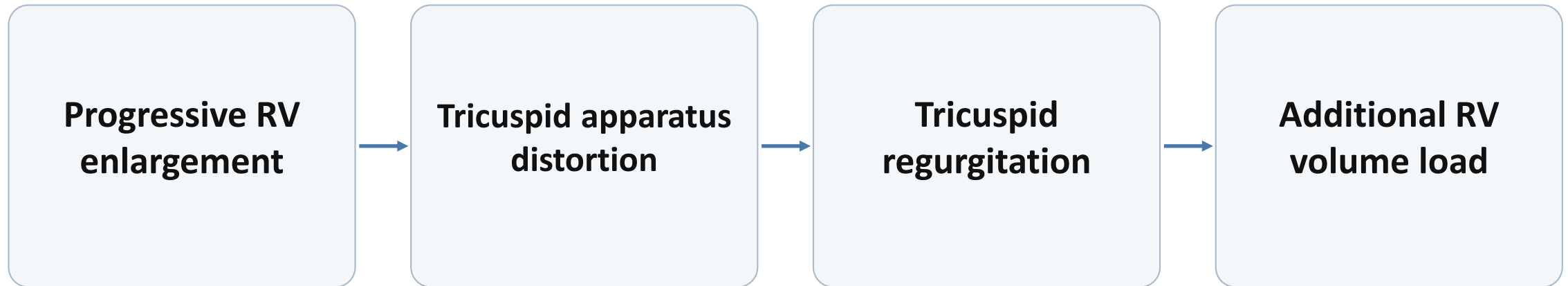
Moderator band

- The moderator band may become prominent.
- It spans septum to the anterior RV papillary muscle.
- This is a normal muscular structure that can hypertrophy.

Severe RV remodeling can distort the left ventricle



Tricuspid dysfunction can complicate RV enlargement



Some affected hearts also show fibrous thickening of the tricuspid valve.

Chronic and acute cor pulmonale differ in tempo

Feature	Acute cor pulmonale	Chronic cor pulmonale
Time course	Abrupt pressure loading	Persistent pressure loading
Common context	Massive pulmonary embolism	Lung or pulmonary vascular disease
Dominant gross change	Marked RV dilation	RV hypertrophy ± dilation
Wall hypertrophy	Not established	May be pronounced

RV failure produces a different congestion pattern

Left-sided failure

- Pulmonary venous congestion and edema.
- Dyspnea reflects pulmonary fluid accumulation.

Predominantly right-sided failure

- Systemic and portal venous engorgement.
- Organ congestion predominates.
- Pulmonary congestion can be relatively limited if isolated.

Most right-sided failure follows left-sided disease

- An elevated pulmonary pressure can transmit downstream from failing left chambers.
- Therefore right-sided failure is most commonly secondary to left-sided failure.
- This differs from isolated cor pulmonale caused by pulmonary disease.
- Clinical attribution requires knowing where the pressure elevation originated.

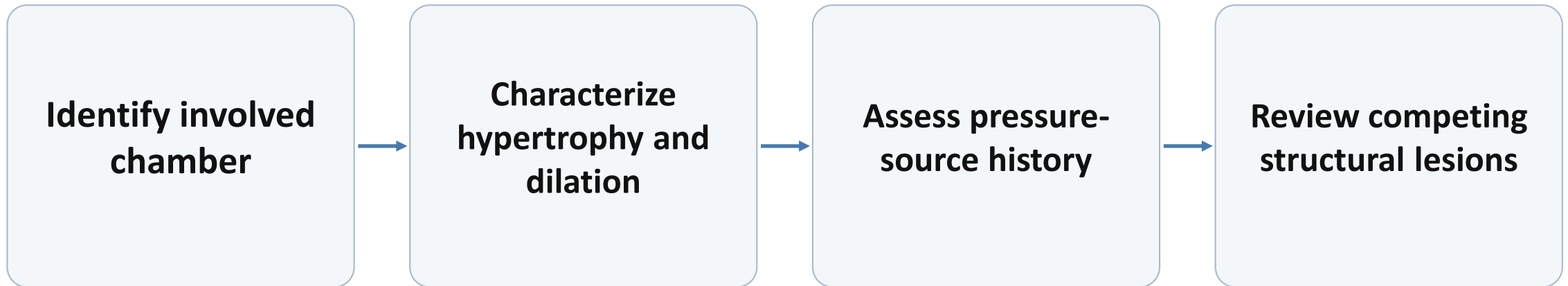
Systemic versus pulmonary HHD: Gross patterns

Feature	Systemic hypertension	Pulmonary hypertension
Chamber most affected	Left ventricle	Right ventricle
Classic pattern	Concentric LV wall thickening	RV enlargement ± hypertrophy
Associated chamber change	Left atrial enlargement	Possible LV shape distortion
Advanced consequence	LV dysfunction / CHF	Right-sided failure

Systemic versus pulmonary HHD: Clinical reasoning

Clinical clue	Favor systemic HHD	Favor cor pulmonale
Pressure history	Systemic hypertension	Pulmonary hypertension / lung disease
Structure	LVH and possible LA enlargement	RV hypertrophy or dilation
Possible vascular pathology	Renal hypertension-related changes	Pulmonary parenchymal / vascular abnormalities
Important caution	Rule out alternative LVH causes	Rule out secondary PH from left-heart disease

A morphologic diagnosis needs an etiologic correlation



Systemic HHD diagnosis requires two complementary pathologic criteria. For right-sided disease, establish why RV pressure overload occurred.

Diagnostic pitfalls in hypertensive remodeling

- Concentric LVH is suggestive but not specific for systemic hypertension.
- Late dilatation may mask the original concentric pattern.
- Perivascular/interstitial fibrosis occurs in several cardiac disorders.
- An enlarged RV may reflect left-sided disease rather than isolated cor pulmonale.
- The listed wall/weight measurements are examples, not universal thresholds.

Case: Concentric LVH with atrial fibrillation

Clinical vignette

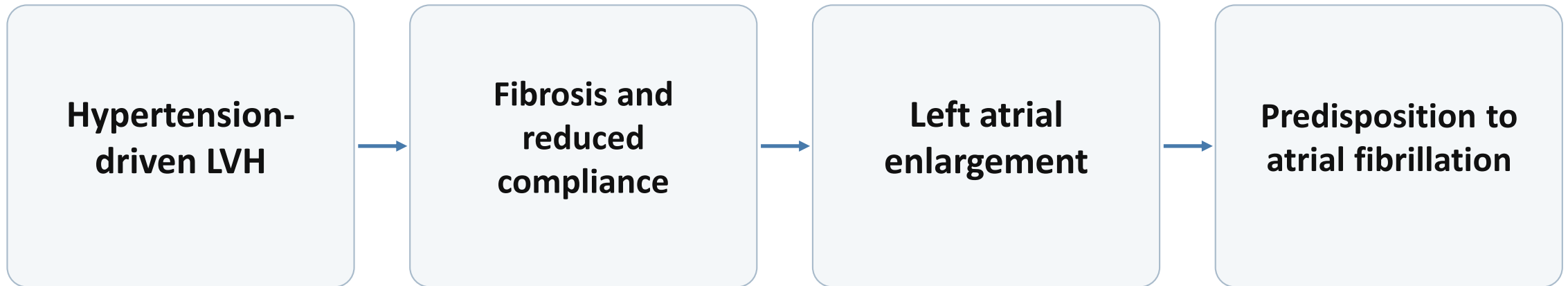
A patient with years of systemic hypertension has echocardiographic concentric LV thickening and a new diagnosis of atrial fibrillation. No primary valve lesion is evident.

Reasoning task

What structural link best explains the arrhythmia?

- Identify the pressure-loaded chamber.
- Relate compliance to left atrial pressure.
- Predict the key secondary chamber change.

Case interpretation: LV stiffness $\rightarrow$ LA enlargement



Atrial arrhythmia is clinically compatible with HHD but is not specific for it.

Case: Collapse with sudden RV dilation

Clinical vignette

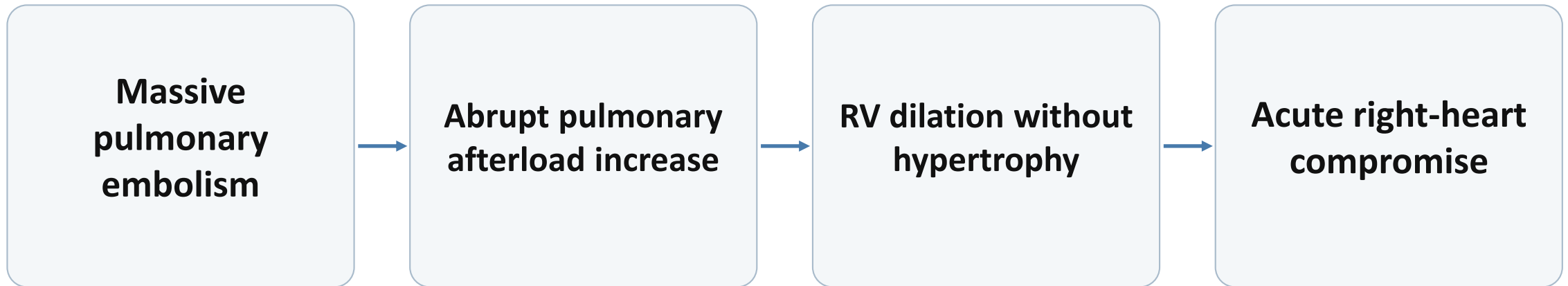
A patient deteriorates abruptly after a large pulmonary embolus. Cardiac examination demonstrates a prominently dilated RV without convincing wall hypertrophy.

Reasoning task

Does the morphology favor acute or chronic pressure overload?

- Judge the time available for structural remodeling.
- Contrast dilation with muscular hypertrophy.
- Connect the finding to pulmonary vascular obstruction.

Case interpretation: Acute cor pulmonale



Conclude an acute pressure-overload phenotype. A chronic substrate cannot be excluded solely from the schematic case.

Case: A remodeled RV in chronic lung disease

Clinical vignette

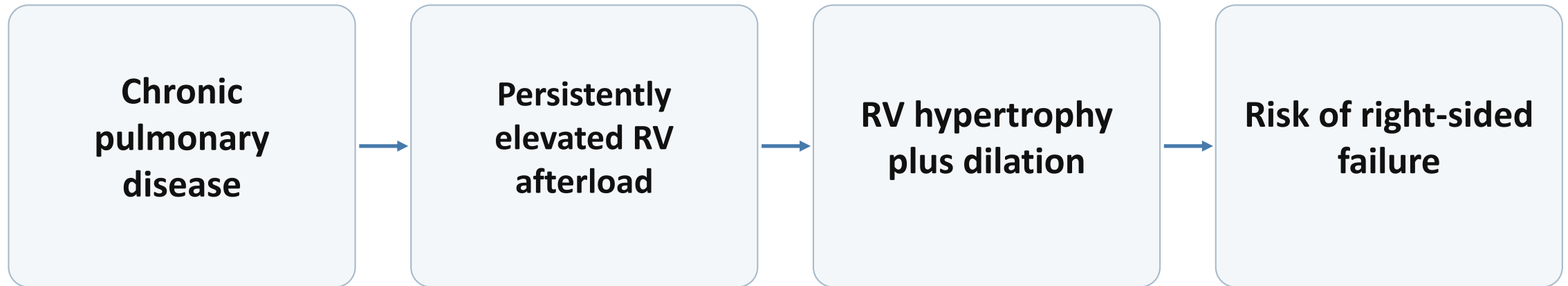
A patient with longstanding emphysema has RV free-wall thickening, a prominent moderator band and chamber dilation.

Reasoning task

What lesion and mechanism connect these findings?

- Explain the role of sustained pulmonary vascular load.
- Recognize chronic RV hypertrophy with dilation.
- Consider whether left-sided disease also contributes.

Case interpretation: Chronic cor pulmonale



Moderator band thickening may provide an additional morphologic clue.

Integrated synthesis: Load → structure → consequences

Pressure circuit	Primary remodeling	Major potential consequences
Systemic	LV concentric hypertrophy; Fibrosis	LA enlargement, ischemia, CHF, SCD
Pulmonary—acute	RV dilation	Acute RV dysfunction
Pulmonary—chronic	RV hypertrophy ± dilation	RV failure, tricuspid regurgitation, LV distortion

Four high-yield principles

- Pressure overload is the defining initiating mechanism of HHD.
- Hypertrophy compensates initially; Fibrosis and oxygen mismatch reduce reserve.
- Systemic HHD classically produces concentric LVH; Chronic cor pulmonale targets the RV.
- Etiologic correlation is essential: Morphology alone rarely identifies the pressure source.

Final application: assign the pressure source

- Concentric LVH with corroborating systemic hypertension and no alternative load → systemic HHD.
- Abrupt RV dilation following massive embolism without developed wall hypertrophy → acute cor pulmonale.
- RV wall and moderator-band hypertrophy in chronic pulmonary disease → chronic cor pulmonale.
- Coexisting LV and RV disease requires investigation of pulmonary pressure secondary to left-sided dysfunction.