

Chronic Ischemic Heart Disease

Clinicopathologic mechanisms · Morphology · Heart failure

From ischemic injury to progressive ventricular dysfunction

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Learning outcomes

- Define chronic ischemic heart disease (IHD) and its clinical context
- Trace the transition from infarction or severe coronary disease to heart failure
- Recognize the gross and microscopic morphologic constellation
- Integrate structural lesions with ventricular dysfunction

Where chronic IHD fits within IHD

Coronary ischemia



Chronic myocardial injury



Progressive heart failure

- IHD encompasses several presentations of myocardial ischemia
- Chronic IHD is the presentation associated with progressive heart failure
- Angina, myocardial infarction, and sudden cardiac death are other IHD presentations

Core pathologic definition

Progressive congestive heart failure follows accumulated ischemic myocardial damage

- Compensatory responses may become inadequate to preserve function
- The entity is defined by its ischemic substrate and functional course

“Ischemic cardiomyopathy”: Clinical usage

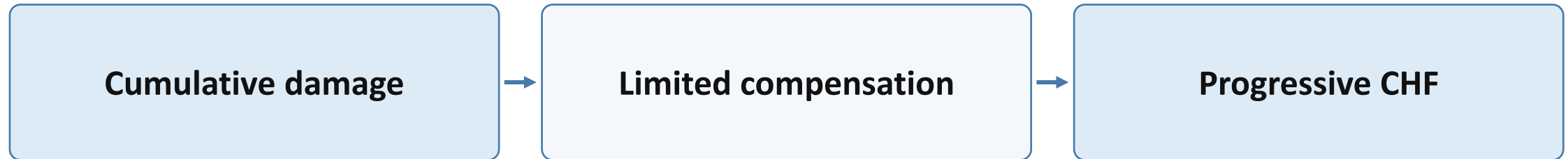
Pathology term

- Chronic ischemic heart disease
- Progressive heart failure

Clinical usage

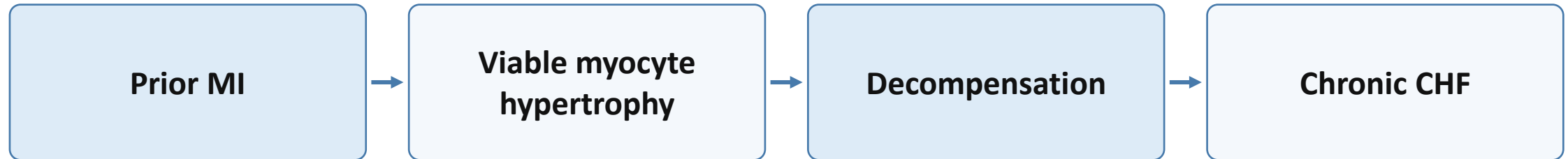
- Ischemic cardiomyopathy
- Ischemic ventricular dysfunction

Two linked contributors to progression



- Cumulative ischemic damage reduces functioning myocardial tissue
- Compensatory responses cannot indefinitely sustain cardiac performance
- Either injury burden or inadequate adaptation can dominate an individual course

The usual postinfarction pathway



- Most chronic IHD cases have a preceding myocardial infarction
- Noninfarcted tissue hypertrophies to compensate for loss of myocardium
- Later functional decompensation of that hypertrophied tissue promotes CHF

A prior infarction is common—not essential

Common pathway

- Healed infarct
- Compensatory hypertrophy
- Progressive failure

Alternative pathway

- Severe coronary obstruction
- No prior infarction required
- Chronic failure

Coronary intervention in the history

- Patients may have undergone coronary arterial interventions
- Some have had coronary bypass surgery
- Such a history can coexist with chronic ischemic dysfunction rather than excluding it

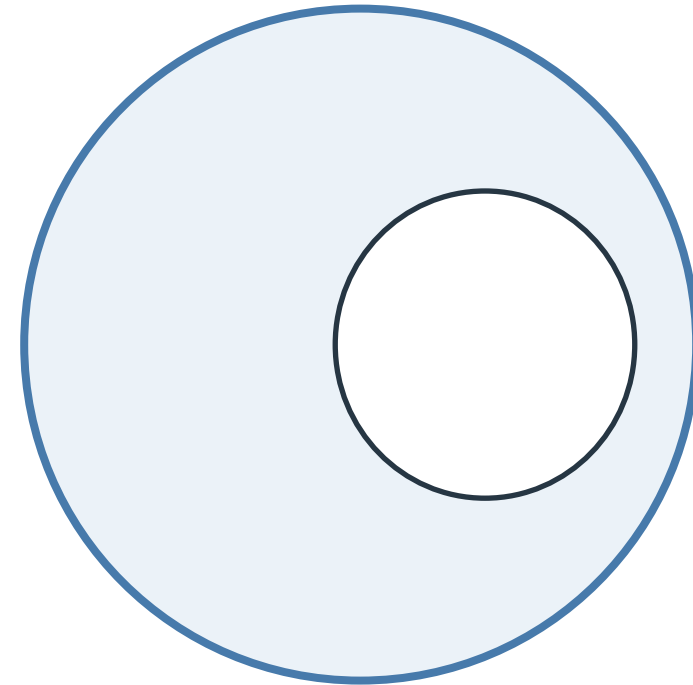
Advanced clinical burden

~40%

- The Robbins text associates chronic IHD with roughly 40% of cardiac transplant recipients
- This statistic describes the transplant-recipient population, not the prevalence of IHD
- Progression to advanced cardiac failure is a clinically important outcome

The indispensable coronary substrate

- Some degree of stenotic coronary atherosclerosis is invariably present in the described morphology
- Epicardial coronary atherosclerosis dominates the wider ischemic heart disease spectrum
- The myocardium must therefore be interpreted together with its coronary arteries



Schematic — stenotic vessel

Less common routes to myocardial ischemia

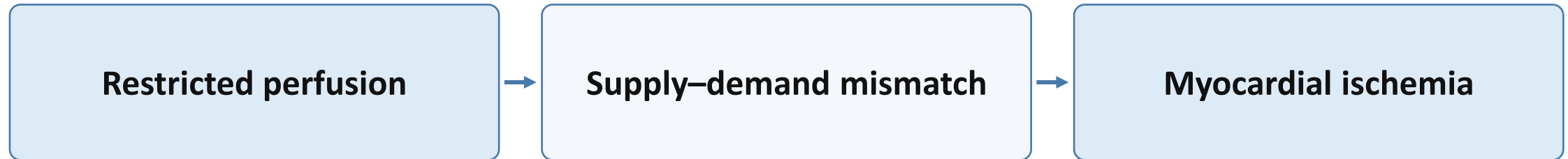
Dominant cause

- Coronary atherosclerosis

Less common causes

- Vasospasm
- Coronary embolism
- Vasculitis

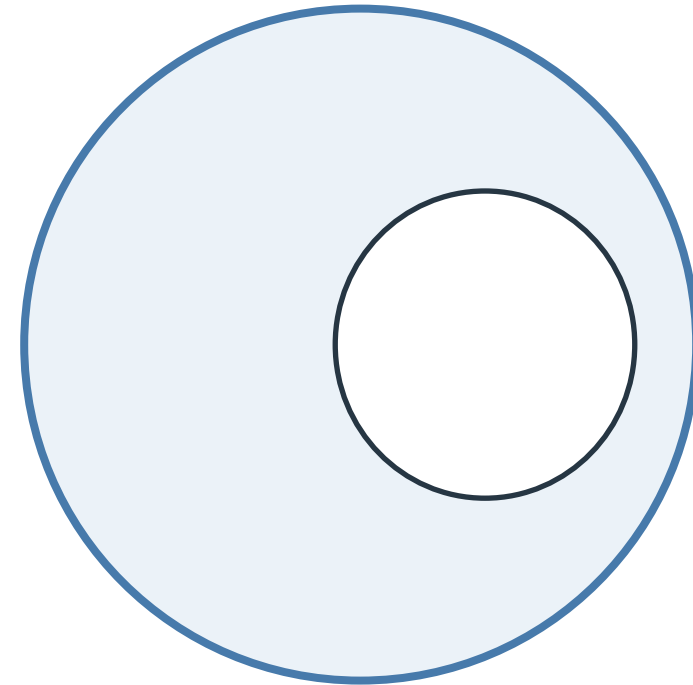
Supply–demand imbalance: The central mechanism



- Myocardial ischemia develops when coronary perfusion is insufficient for oxygen demand
- Reduced delivery of oxygen and nutrients impairs ATP production
- Underlying ischemia may intensify as myocardial workload increases

Chronic coronary narrowing

- Chronic progressive atherosclerotic narrowing limits coronary blood flow
- With advanced stenosis, compensatory dilation may not meet increased cardiac demand
- Reduced flow reserve is more informative than stenosis severity alone in multifocal disease

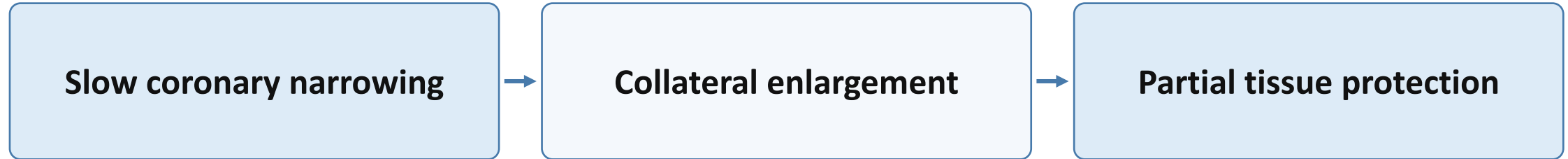


Schematic — stenotic vessel

More than one coronary bed may be involved

- Atherosclerosis can involve one, two, or all three major epicardial coronary vessels
- Different territories may contribute to the viable but ischemia-prone ventricle
- Widespread obstructive disease helps explain functional vulnerability beyond an old scar

Collateral circulation: Partial protection



- Gradually narrowing coronary lesions can promote collateral channels
- These channels may reduce ischemia in jeopardized myocardial regions
- Protection is variable and does not erase the underlying obstructive coronary disease

Why hypertrophy raises oxygen requirements

Loss of contractile mass



Viable myocyte hypertrophy



Higher oxygen demand

- Surviving myocardium develops increased muscle mass after infarction
- Greater tissue mass and heightened contractile work increase oxygen consumption
- Compensatory enlargement therefore adds metabolic demand to a coronary-limited system

Hypertrophy is not matched by capillary growth

Growing demand

- Larger myocytes
- Higher myocardial workload

Limited supply

- No proportional capillary increase
- Stenotic coronaries

Tachycardia is a double stressor

Increased demand

- More contractions per minute
- Greater oxygen consumption

Reduced supply time

- Shorter relative diastole
- Less time for coronary perfusion

Systemic hypotension can worsen ischemia

- Even moderate coronary disease may become consequential when systemic perfusion falls
- Reduced coronary inflow adds to the pre-existing oxygen supply limitation
- Perfusion pressure and coronary anatomy must be considered together

Postinfarct tissue redistribution

Infarcted myocardium



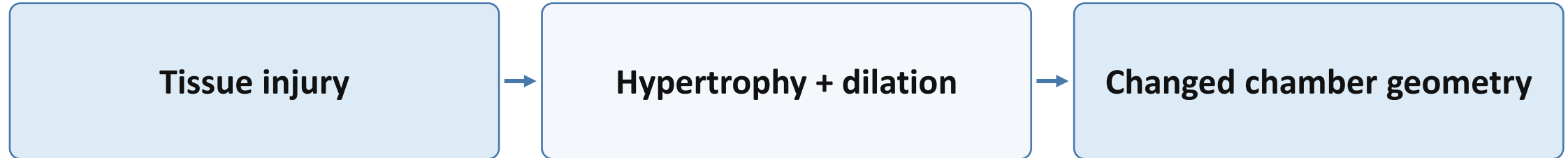
Viable myocardium adapts



Compensatory hypertrophy

- Infarction reduces functioning myocardial mass
- Viable segments bear a greater compensatory workload
- Work-related hypertrophy occurs in noninfarcted myocardium

Ventricular remodeling: What the term covers



- After infarction, noninfarcted myocardium can hypertrophy and dilate
- Changes in chamber shape and wall properties accumulate
- Remodeling includes molecular, cellular, and structural adaptation to injury

Compensation initially supports perfusion

- Frank–Starling recruitment increases force as filling stretches myocytes
- Neurohumoral responses can maintain pressure and perfusion initially
- Hypertrophy increases contractile capacity in the early adaptive phase

The Frank–Starling trade-off

Increased filling



Temporary output support



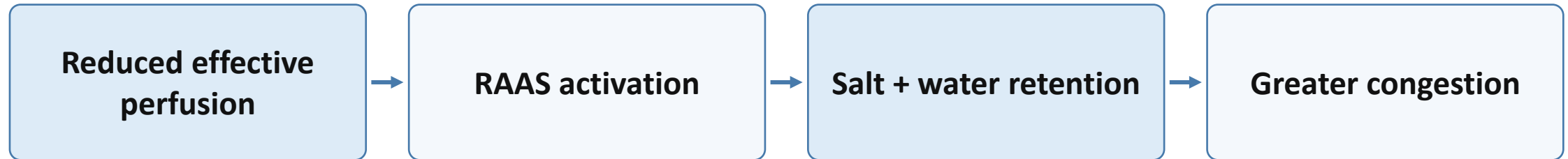
**Progressive dilation may
dominate**

- Greater filling can temporarily augment contraction
- Persistent chamber dilation is not equivalent to preserved pump function
- The compensatory effect may be overwhelmed as the ventricle remodels

Adrenergic compensation

- Norepinephrine increases heart rate and myocardial contractility
- Vascular resistance also increases under adrenergic activation
- These compensations alter the same demand–supply balance faced by ischemic myocardium

RAAS: A volume-preserving response



- Renin–angiotensin–aldosterone activation retains salt and water
- It also increases vascular tone
- Persistent fluid retention can exacerbate the congestion of failing hearts

Natriuretic counter-regulation

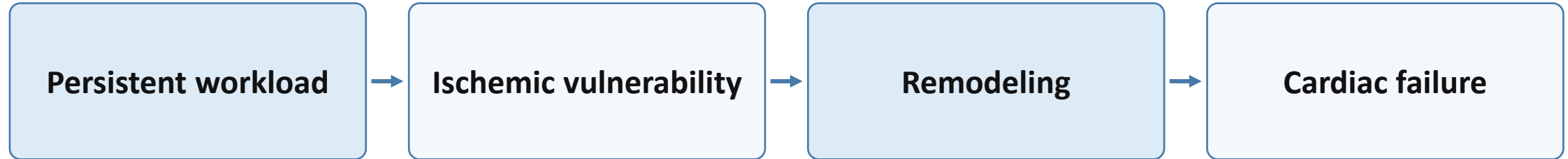
Volume-retaining response

- RAAS
- Salt and water retention
- Increased vascular tone

Counter-response

- Atrial natriuretic peptide
- Diuresis
- Vasorelaxation

Why adaptation eventually fails

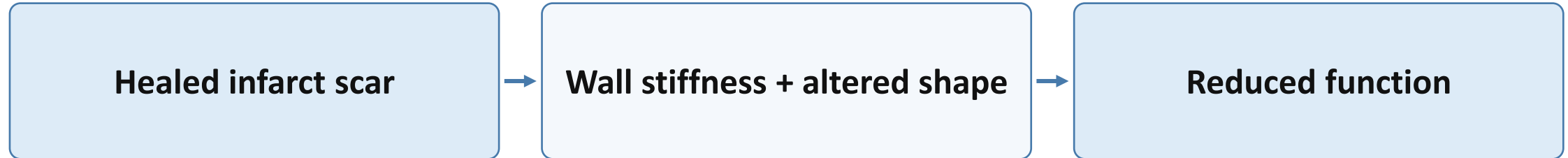


- Surviving myocytes must sustain persistent mechanical work
- Hypertrophy increases oxygen demand while coronary supply remains impaired
- Dilation and fibrosis further compromise mechanical efficiency

Dilation as a late dominant change

- Increasing chamber size may overwhelm the initial benefit of hypertrophy
- Altered ventricular shape contributes to depressed output
- Dilation can occur with or without a discrete ventricular aneurysm

Scar formation changes mechanics



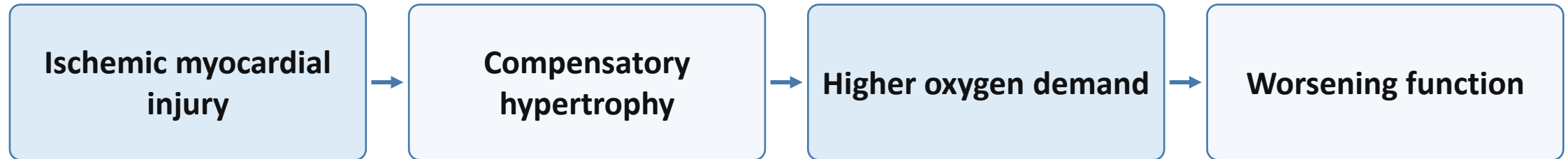
- Healed infarcts replace lost myocardium with scar
- Scar formation can stiffen the ventricular wall
- Stiffening and altered geometry further reduce effective cardiac output

Interstitial fibrosis adds filling resistance



- Mechanical forces stimulating myocyte growth also activate fibroblast matrix synthesis
- Interstitial collagen accumulation increases resistance to diastolic filling
- This is a structural route from hypertrophy to functional impairment

A self-reinforcing ischemic cycle



- Coronary obstruction restricts myocardial perfusion
- Compensatory hypertrophy elevates oxygen consumption
- Progressive dysfunction increases the burden on remaining viable muscle

Systolic dysfunction in the remodeled heart

- Loss of viable muscle and adverse dilation can depress left ventricular contraction
- Reduced ejection fraction reflects impaired systolic pump performance
- Forward cardiac output can become inadequate for tissue needs

Diastolic dysfunction from stiffness

Hypertrophy + fibrosis



Impaired relaxation



Elevated filling pressures

- Hypertrophy and interstitial fibrosis limit ventricular relaxation and filling
- Preserved contraction alone cannot guarantee normal filling
- Higher filling pressures can contribute to pulmonary congestion

Systolic and diastolic dysfunction

Systolic impairment

- Diminished contractile output
- Reduced ejection fraction
- Loss of viable myocardium / dilation

Diastolic impairment

- Stiff, poorly relaxing ventricle
- Impaired diastolic filling
- Hypertrophy / fibrosis

Forward failure: Tissue hypoperfusion

Reduced cardiac output



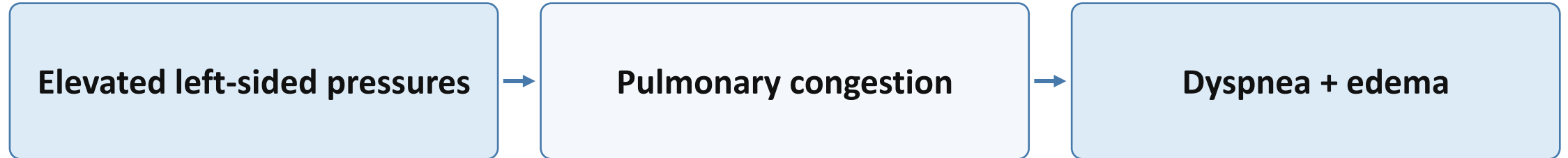
Peripheral hypoperfusion



Organ consequences

- Heart failure may prevent adequate tissue perfusion
- Renal hypoperfusion stimulates fluid-retaining responses
- Advanced hypoperfusion can compromise kidney and brain function

Backward failure: Venous congestion



- Raised filling pressures allow blood to pool upstream
- Left-sided failure particularly causes pulmonary venous congestion
- Pulmonary edema contributes substantially to the clinical syndrome

Pulmonary consequences of left-sided failure

- Pulmonary congestion progresses from interstitial edema to alveolar fluid
- Chronic episodes can yield hemosiderin-laden alveolar macrophages
- This lung morphology reflects failure of the left heart, not a new coronary lesion

Dyspnea links myocardium to the lung

- Exertional breathlessness can precede symptoms at rest
- Orthopnea and paroxysmal nocturnal dyspnea emerge with worsening congestion
- Fine basal rales correspond to reopening of fluid-affected alveoli

Clinical signs related to remodeling

- Cardiomegaly reflects cardiac enlargement
- Tachycardia may accompany heart failure
- S3 can reflect volume overload; S4 can reflect increased stiffness

Dilation can contribute to mitral regurgitation



- Progressive left ventricular enlargement can displace papillary muscles
- This may produce functional mitral regurgitation
- The resultant volume burden can aggravate chamber remodeling

Left-sided failure can burden the right heart

Left ventricular failure



Pulmonary vascular burden



Right ventricular failure

- Elevated pulmonary circulatory pressure increases right ventricular workload
- Persistent strain may contribute to right-sided failure
- The clinical course can therefore progress to combined ventricular failure

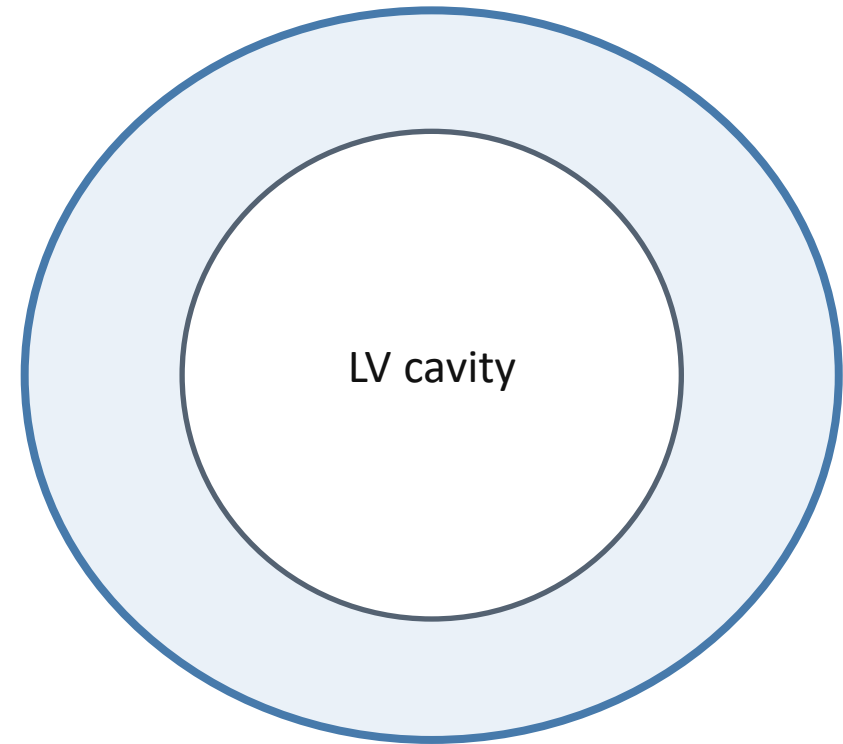
Gross morphology: Cardiomegaly

Chronic IHD hearts are enlarged

- Myocardial hypertrophy and chamber dilation coexist
- Gross enlargement is a structural signature but does not alone quantify dysfunction

Left ventricular hypertrophy plus dilation

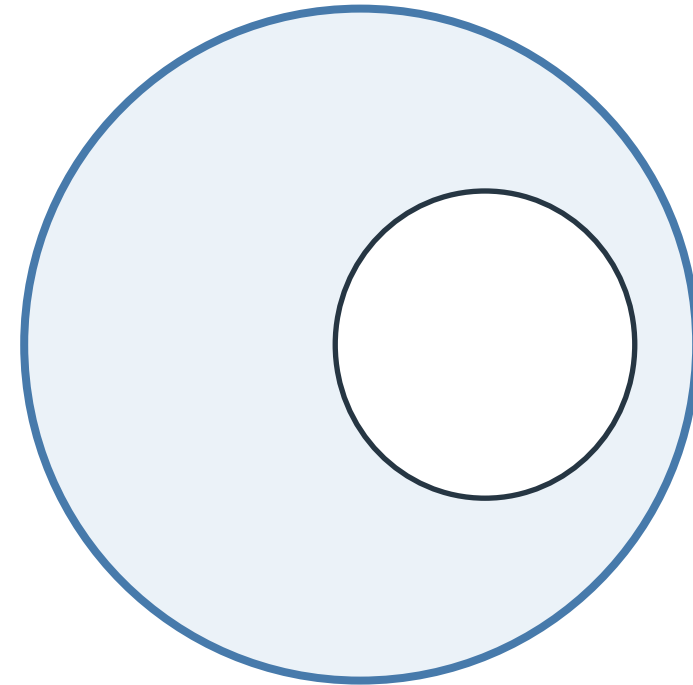
- Both thickened myocardium and an enlarged cavity may be present
- In a dilated heart, wall thickness can be misleading as a measure of hypertrophy
- Heart weight better reflects increased mass in markedly dilated ventricles



Schematic — not a specimen

The coronary arteries must be examined

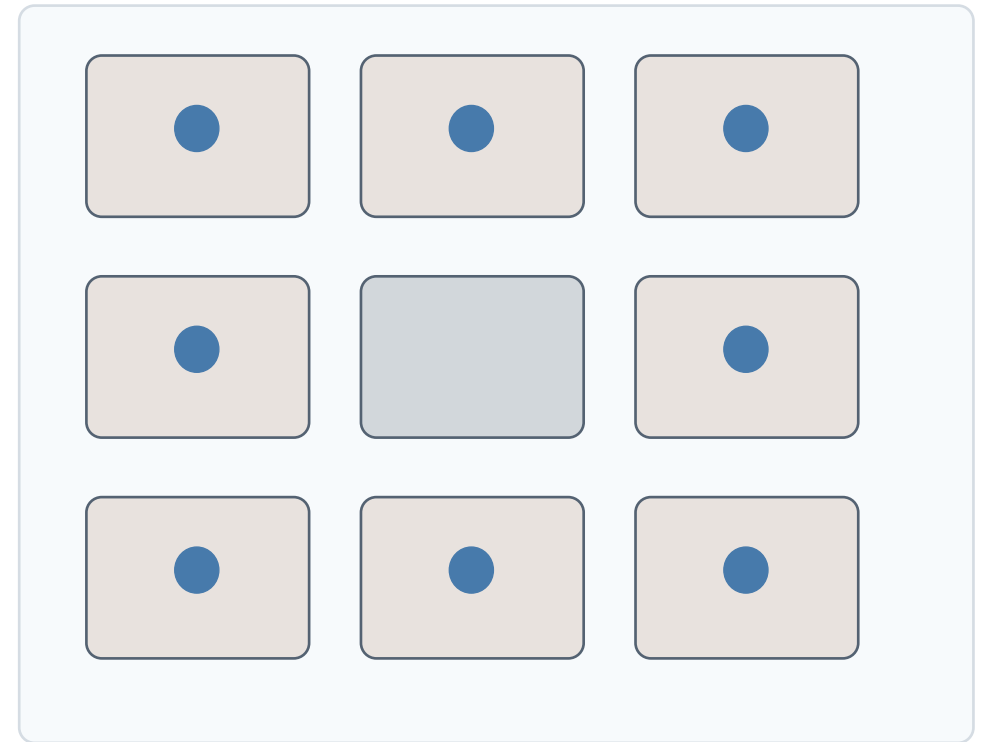
- Stenosing coronary atherosclerosis is a constant feature in the chronic IHD morphology described
- Severity and distribution of obstruction provide essential etiologic evidence
- Coronary disease is interpreted alongside myocardial scars and remodeling



Schematic — stenotic vessel

Healed infarct scars: Discrete lesions

- Discrete myocardial scars usually represent previous infarctions
- They record earlier irreversible myocardial damage
- The presence of a scar is compatible with later decompensation of surviving myocardium



Conceptual tissue map — not histology

Absence of a visible scar does not exclude IHD

Evidence often present

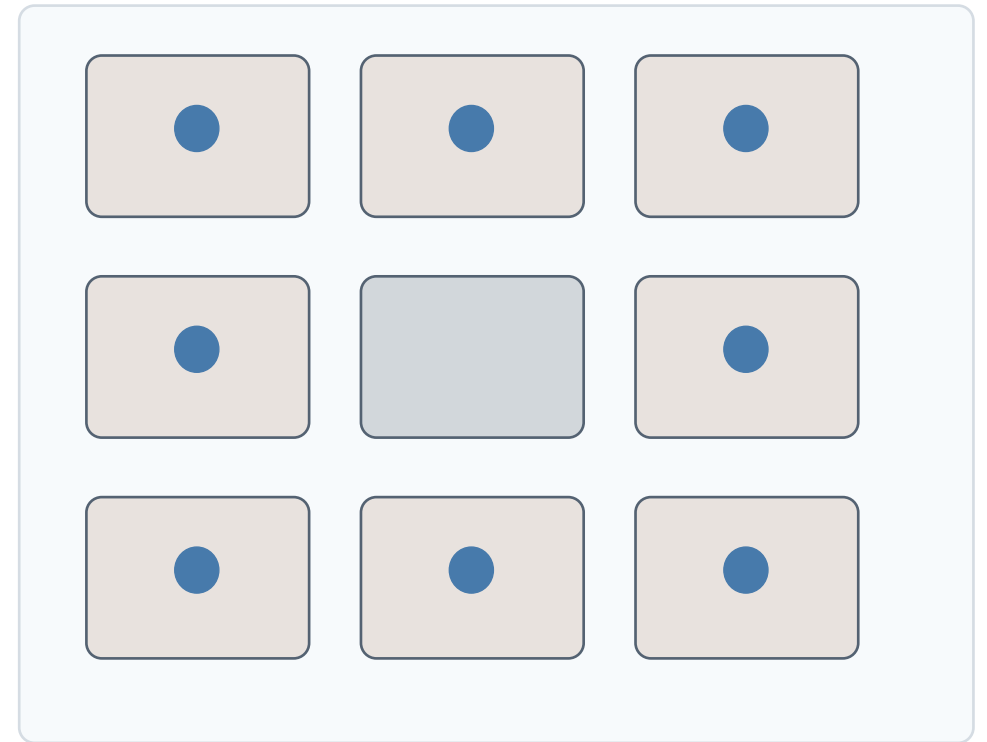
- Healed infarct scars
- LV hypertrophy and dilation

Still compatible with chronic IHD

- No documented earlier MI
- Severe stenotic coronary disease

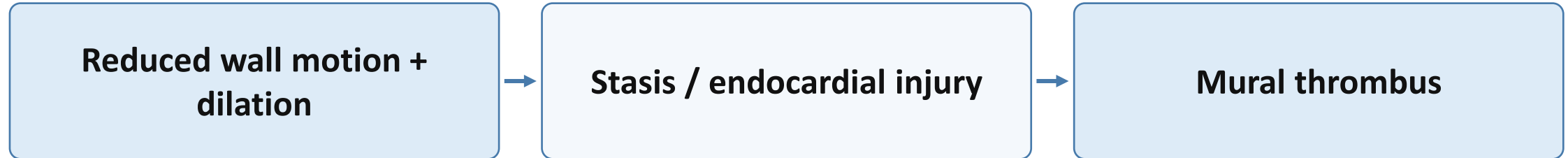
Mural endocardial fibrous thickening

- Patchy fibrosis of the mural endocardium is a recognized morphologic feature
- This change to abnormal wall shear forces
- The lesion reflects altered intracavitary mechanics accompanying remodeling



Conceptual tissue map — not histology

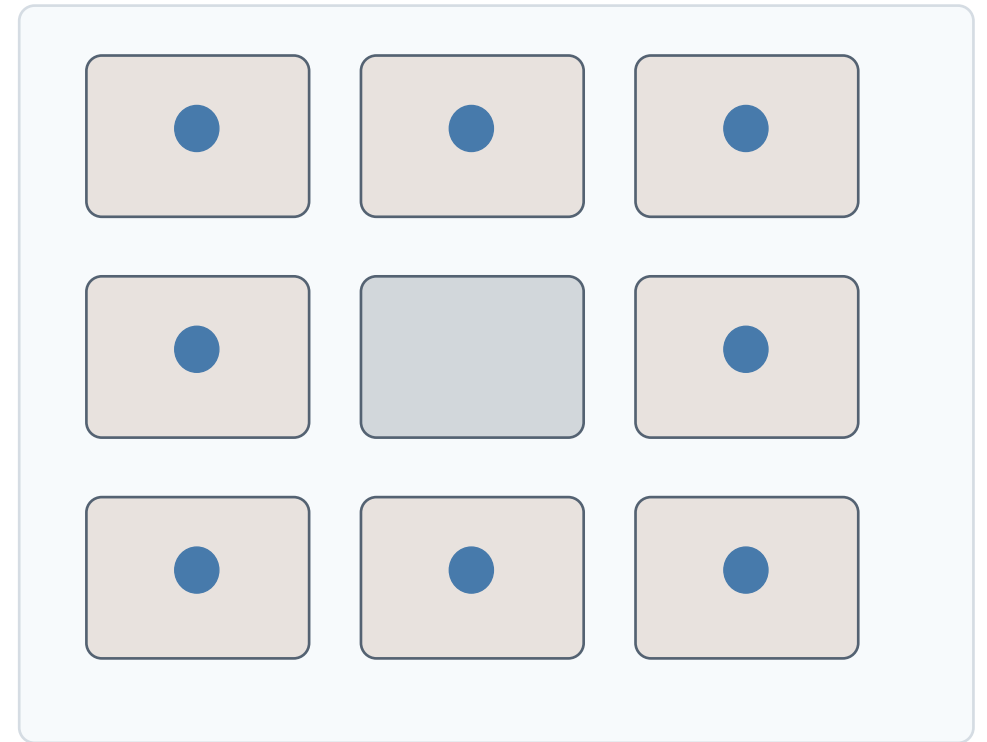
Mural thrombi: A possible complication



- Mural thrombi may be found in chronic IHD
- Postinfarct stasis, chamber dilation, and endocardial injury can favor thrombosis
- Intracardiac thrombus creates a potential embolic source

Microscopy: Hypertrophied myocytes

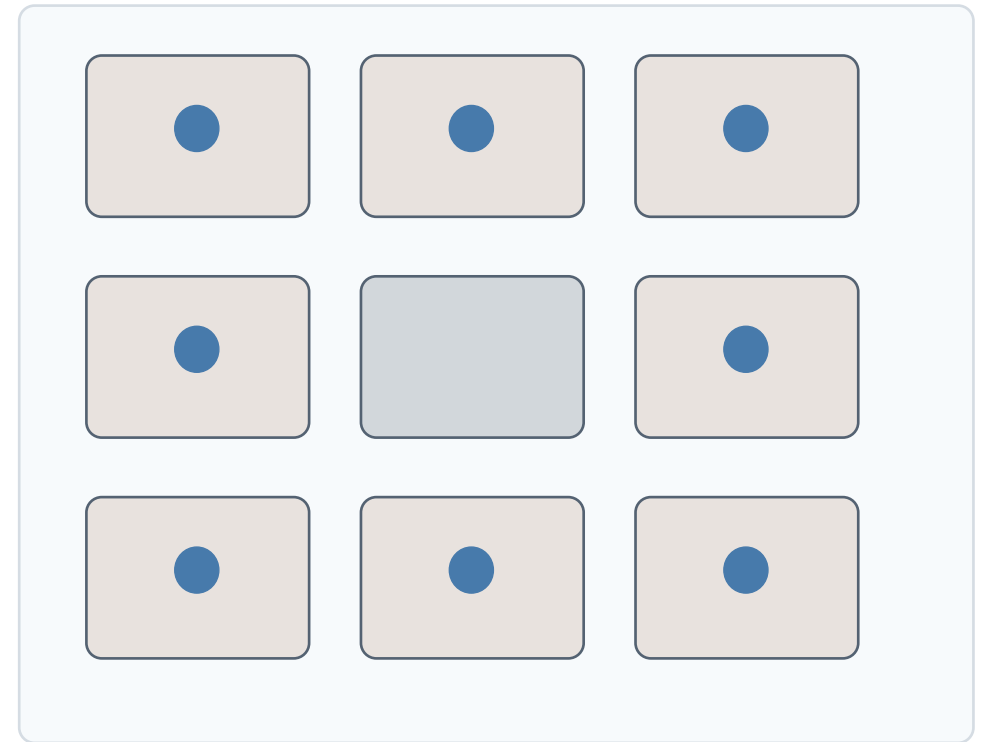
- Myocyte hypertrophy is a characteristic microscopic finding
- It represents adaptation of viable tissue to persistent workload
- It commonly coexists with interstitial fibrosis in failing hearts



Conceptual tissue map — not histology

Subendocardial myocyte vacuolization

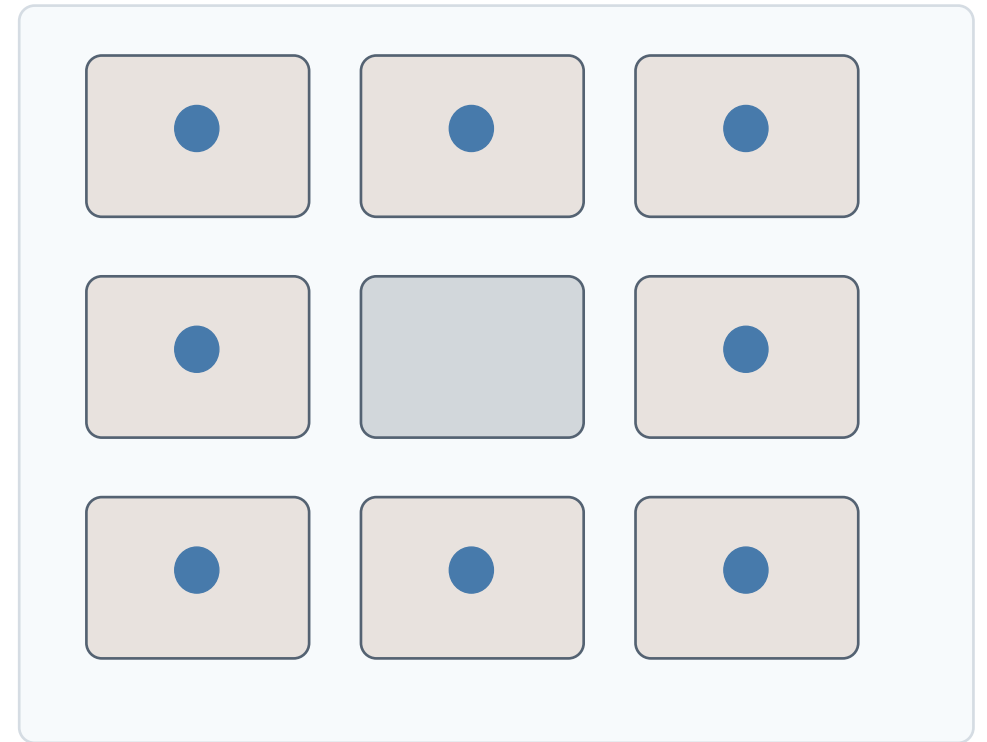
- Diffuse vacuolization of subendocardial myocytes is specifically described
- Its location links the finding to the ischemia-vulnerable subendocardial region
- Do not mistake an abstract schematic for diagnostic histologic evidence



Conceptual tissue map — not histology

Interstitial fibrosis: Between surviving myocytes

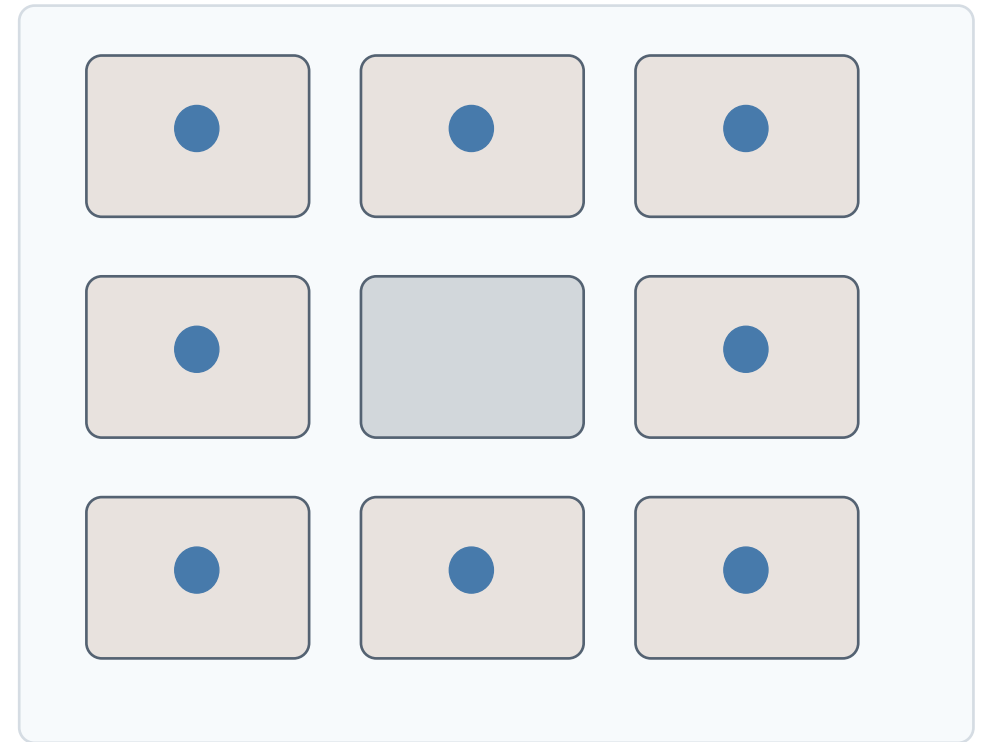
- Fibrous matrix accumulates in myocardial interstitium
- The change accompanies hypertrophy and raises myocardial stiffness
- It is distinct from scars that replace infarcted tissue



Conceptual tissue map — not histology

Replacement fibrosis: Loss followed by scar

- Replacement fibrosis occupies regions of previous myocardial injury
- Healed infarcts commonly leave discrete areas of scar
- This reduces functioning muscle mass available to sustain contraction



Conceptual tissue map — not histology

Fibrosis is not a single lesion

Interstitial fibrosis

- Among surviving myocytes
- Associated with stiffness

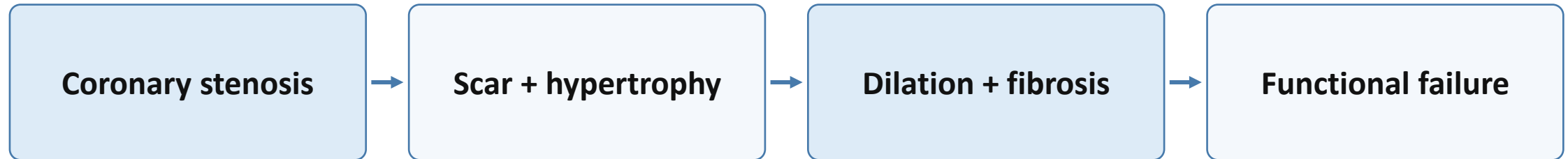
Replacement fibrosis

- Within areas of tissue loss
- Associated with old infarcts

Integrated morphologic checklist

Compartment	Characteristic change
Coronary arteries	Stenotic atherosclerosis
Cardiac size	Cardiomegaly; LV hypertrophy and dilation
Myocardium	Healed scars; hypertrophied myocytes
Endocardium	Patchy fibrosis; possible mural thrombus
Microscopy	Subendocardial vacuoles and fibrosis

Morphology and physiology are linked



- Coronary stenosis corresponds to constrained myocardial perfusion
- Scar and hypertrophy alter ventricular work distribution
- Dilation and fibrosis help explain impaired pumping and filling

A late aneurysm differs from diffuse dilation

General ventricular dilation

- Chamber enlargement
- May occur without aneurysm

Postinfarct aneurysm

- Localized thinned scarred wall
- Late sequel of transmural MI

Infarct characteristics shape later outcomes

- Larger infarcts impair pump function to a greater extent
- Transmural infarcts carry a greater likelihood of late CHF
- Extent, location, and wall thickness of injury influence complications

The surviving ventricle matters for prognosis

Cardiac functional reserve

- Residual LV function
- Extent of loss from infarction

Coronary supply to viable muscle

- Remaining vascular obstructions
- Risk to viable myocardium

Prevention in the chronic ischemic course

Before any MI

- Primary prevention
- Avoid initial infarction

After prior MI

- Secondary prevention
- Avoid reinfarction

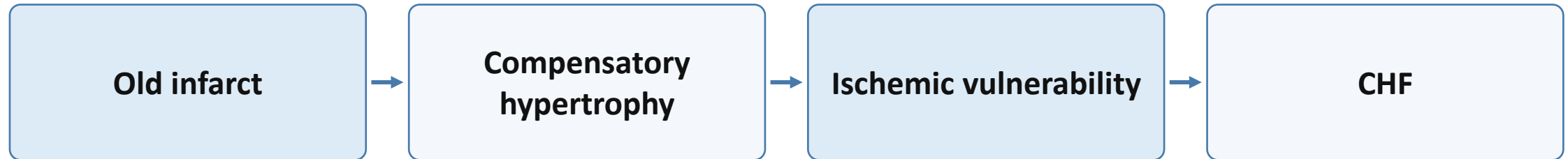
Teaching case: Deterioration after infarction

Constructed teaching vignette

- Hypothetical course: Remote infarction followed by progressive cardiac enlargement and CHF
- Expected pathology: Coronary atherosclerosis with healed scar, hypertrophy, and dilation
- Mechanistic question: Why did initially compensatory viable myocardium decompensate?

Interpretation is developed on the following slide.

Case interpretation: Adaptation has limits



- Infarction permanently reduces myocardial mass
- Noninfarcted myocardium hypertrophies and increases its oxygen requirement
- Further dilation and stiffening can diminish output despite the earlier compensatory response

Teaching case: CHF without a known infarct

Constructed teaching vignette

- Hypothetical course: Progressive CHF with severe obstructive coronary artery disease
- No previous MI needs to be assumed
- Question: Is the absence of an infarct history sufficient to exclude chronic IHD?

Interpretation is developed on the following slide.

Case interpretation: Stenotic CAD can suffice

Robbins explicitly describes severe obstructive CAD presenting as chronic CHF without prior infarction

- The coronary substrate remains essential to the diagnosis
- An infarct is usual but not required by this formulation

Teaching case: Interpret combined pathology

Constructed teaching vignette

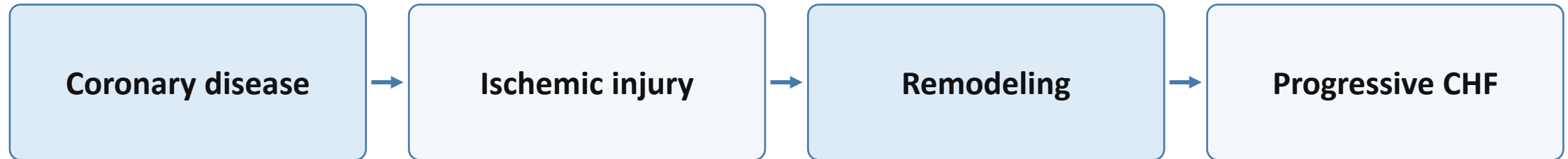
- Hypothetical postmortem constellation: Cardiomegaly, severe coronary stenosis, patchy mural endocardial fibrosis
- Microscopy: Hypertrophied myocytes, subendocardial vacuolization, and interstitial fibrosis
- Question: What additional myocardial lesion is often present?

Interpretation is developed on the following slide.

Case interpretation: Look for healed scars

- Healed infarcts commonly appear as discrete myocardial scars
- Replacement fibrosis can coexist with diffuse interstitial fibrosis
- Mural thrombosis is possible, but is not required in every case

Distinguish cause, response, and consequence



- Cause: Stenotic coronary disease limits myocardial supply
- Response: Surviving myocardium undergoes hypertrophy and remodeling
- Consequence: Accumulated injury and ineffective adaptation produce progressive CHF

High-yield synthesis

- Chronic IHD is an ischemic cause of progressive congestive heart failure
- Usually postinfarction; Severe obstructive CAD may also cause it without prior infarct
- Cardiomegaly, LV hypertrophy/dilation, scars, endocardial changes, and fibrosis form the pathologic pattern
- The transition from adaptive hypertrophy to adverse remodeling explains progressive dysfunction

Reference

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th edition (2026).
- The Heart — “Chronic Ischemic Heart Disease” and the parent ischemic heart disease section.
- Related material: myocardial infarction, ventricular remodeling, and the pathophysiology of heart failure.