

Right-Sided Heart Failure

Mechanisms, morphology, and systemic consequences

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Learning objectives

- Distinguish secondary right-sided failure from isolated pulmonary causes.
- Explain right-ventricular pressure overload, remodeling, and septal effects.
- Trace systemic venous congestion to organ-specific pathology.
- Integrate clinical manifestations with textbook diagnostic principles.

Heart failure: A pump–perfusion disorder

- The heart cannot adequately meet tissue metabolic demands—or does so only at elevated filling pressures.
- Congestive heart failure combines variable forward hypoperfusion with backward venous pooling.
- Right-sided failure particularly exposes the systemic venous circulation to congestion.

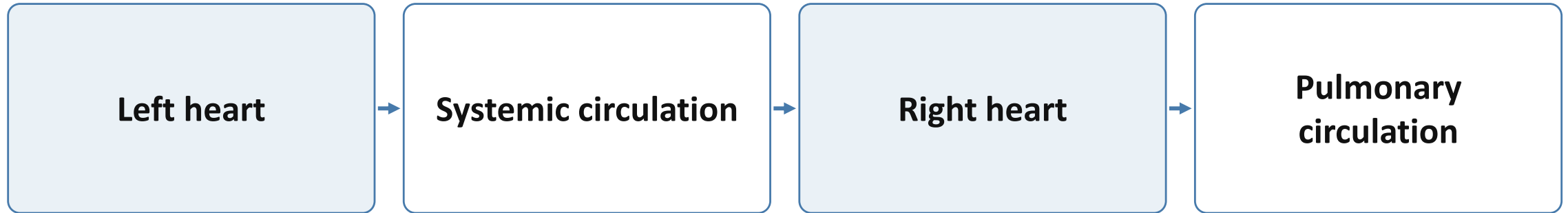
Forward failure

Reduced delivery to tissues

Backward failure

Blood pools upstream of an ineffective ventricle

A closed circulation links both ventricles



- The pulmonary circuit returns blood to the left heart.
- One ventricular problem can strain the other.

Why left-sided failure causes right-sided failure

- Left-heart dysfunction raises pressure backward into the pulmonary circuit.
- The resulting pulmonary pressure burden increases right-ventricular work.
- Chronic strain may progress from compensatory remodeling to right-sided failure.

Initial defect

Left-heart failure

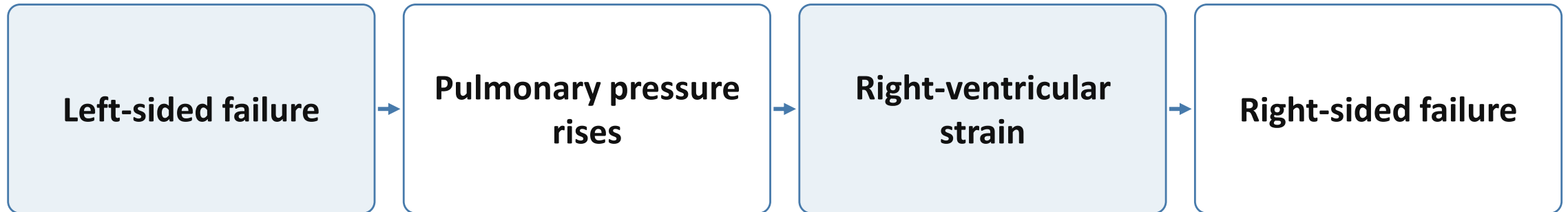
Transmitted burden

Pulmonary pressure elevation

Consequence

Right-heart overload

The dominant causal sequence



- This is the most common pathway to right-sided heart failure.

Left-sided etiologies also matter on the right

- Ischemic heart disease and systemic hypertension are common antecedents.
- Aortic or mitral valve disease can initiate the same sequence.
- Primary myocardial disorders can ultimately impair both sides.

Ischemic disease

Myocardial injury

Pressure load

Systemic hypertension

Structural load

Aortic / mitral disease

Muscle disease

Primary myocardial pathology

Two routes to right-sided heart failure

Feature	Secondary to left-heart failure	Isolated pulmonary pathway
Frequency	Most common	Less common
Initial burden	Left-heart dysfunction	Lung / pulmonary vascular disorder
Hemodynamic link	Pulmonary pressure burden	Pulmonary hypertension
Dominant outcome	Often biventricular	Systemic venous congestion

Isolated right-sided failure is uncommon

- When relatively isolated, the origin is usually pulmonary.
- Lung parenchymal disease and pulmonary vascular disorders raise right-ventricular afterload.
- The subsequent heart phenotype can be described as cor pulmonale.

Lung disease

Parenchymal impairment

Pulmonary vessels

Obstructive / hypertensive disease

Right heart

Pressure overload

Cor pulmonale: An etiologic distinction

- Cor pulmonale denotes pulmonary disease–related right-heart pressure overload.
- Chronic disease produces right-ventricular hypertrophy and often dilation.
- Right-heart failure is a possible downstream outcome, not a necessary starting point.

Initiating process

Pulmonary pathology

Cardiac adaptation

Right-ventricular remodeling

Clinical endpoint

Possible right-heart failure

Pulmonary parenchymal disease

- Chronic parenchymal lung disorders are important causes of cor pulmonale.
- The relevant cardiac consequence is increased load in the pulmonary circuit.
- Emphysema as a representative chronic pulmonary cause.

Parenchymal disorder

Altered pulmonary circulation

Pulmonary hypertension

Increased right-heart pressure work

Pulmonary vascular disease

- Primary pulmonary hypertension directly loads the right ventricle.
- Recurrent pulmonary thromboembolism can produce chronic vascular strain.
- Both routes share the final pathway of increased pulmonary vascular pressure.

Pulmonary hypertension

Vascular pressure elevation

Recurrent emboli

Vascular obstruction / pressure burden

Pulmonary vasoconstrictive settings

- Obstructive sleep apnea is listed as a setting associated with pulmonary vasoconstriction.
- Altitude-related illness can also raise pulmonary vascular pressure.
- These examples illustrate causes beyond structural lung parenchymal disease.

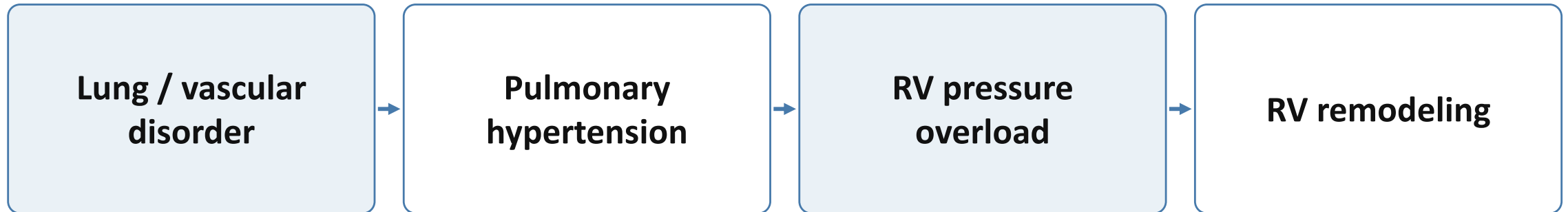
Obstructive sleep apnea

Vasoconstrictive context

Altitude sickness

Vasoconstrictive context

Different pulmonary causes, one pathway



- Hypertrophy and dilation are characteristic structural responses.

Do not equate the cause with the consequence

Term	Describes	Main teaching distinction
Pulmonary hypertension	Elevated pressure in pulmonary circulation	Hemodynamic burden
Cor pulmonale	Right-heart disease attributable to pulmonary disorder	Etiologic and structural concept
Right-sided heart failure	Failure with systemic venous congestion	Functional / clinical syndrome

Why the right ventricle is pressure-sensitive

- The pulmonary circulation is normally the lower-pressure circuit.
- The right ventricle consequently has a thinner, more compliant wall than the left.
- Sustained pulmonary pressure elevation imposes an abnormal mechanical load.

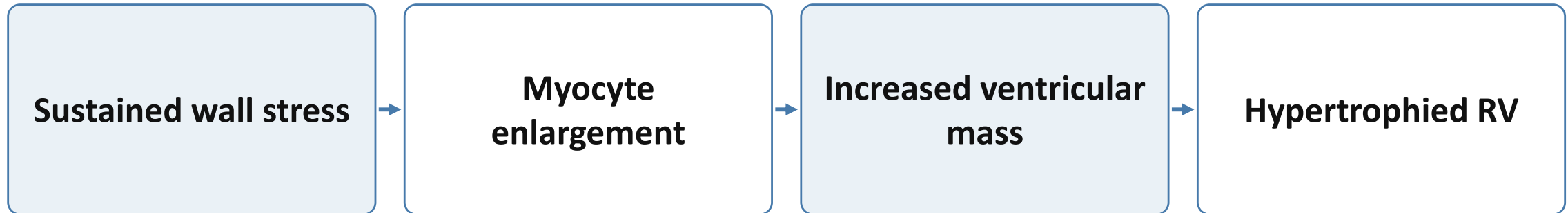
Normal context

Low-pressure pulmonary circuit

Disease context

Sustained RV pressure overload

Pressure overload initiates hypertrophy



- Hypertrophy is initially an adaptation to increased mechanical work.

How cardiomyocytes enlarge

- Increased protein synthesis supplies new contractile elements.
- Additional sarcomeres accumulate within individual cells.
- Cardiomyocytes may show enlarged or multiple nuclei.

Cell-level change

Larger myocytes

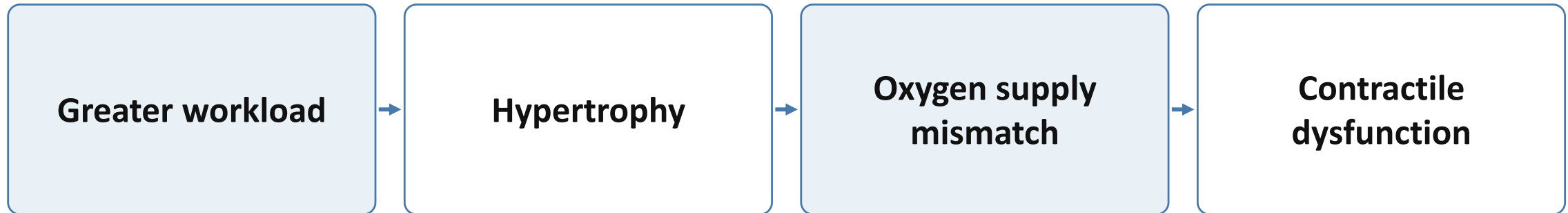
Tissue-level consequence

Greater myocardial mass

Pressure versus volume loading

Feature	Pressure overload	Volume overload
Dominant stimulus	Resistance to ejection	Increased chamber volume
Sarcomeres	Predominantly parallel	Predominantly serial
Chamber response	Concentric wall thickening	Dilation predominates
Right-heart application	Pulmonary hypertension	Additional chamber-loading states

Compensation can become maladaptive



- Mechanical adaptation does not ensure indefinitely preserved function.

Capillary supply fails to scale with hypertrophy

- Capillary density does not increase proportionally with myocyte enlargement.
- Greater myocardial mass increases oxygen and nutrient requirements.
- The hypertrophied ventricle becomes vulnerable to ischemia-related decompensation.

Demand

Rises with tissue mass / work

Supply reserve

Relatively constrained

Interstitial fibrosis changes filling properties

- Mechanical overload can stimulate myocardial fibroblasts.
- Increased extracellular matrix produces interstitial fibrosis.
- Fibrosis increases resistance to ventricular filling and contributes to dysfunction.

Cardiomyocytes

Hypertrophy

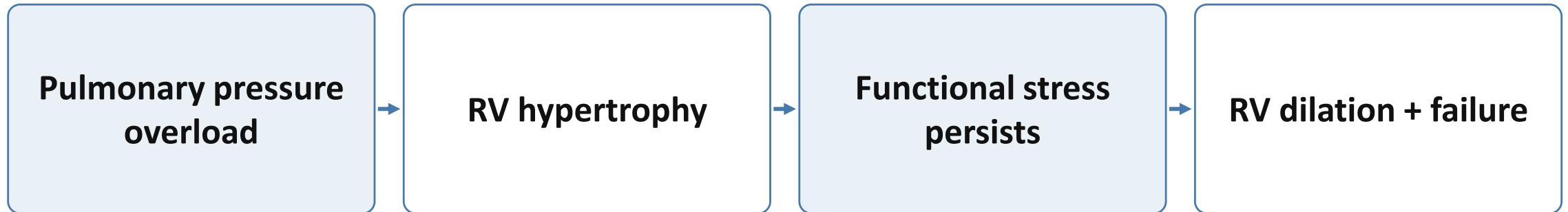
Stroma

Fibrosis

Function

Greater stiffness

Hypertrophy and dilation can coexist



- The actual wall thickness depends on the loading history and degree of dilation.

Right atrium and ventricle: Gross phenotype

- The common morphologic pattern is right-atrial and right-ventricular enlargement.
- The extent of hypertrophy and dilation varies with etiology and duration.
- In isolated pulmonary causes, primary valve lesions need not be present.

Right ventricle

Hypertrophy ± dilation

Right atrium

Dilation / enlargement

Unusual structural cardiac causes

- Tricuspid or pulmonary valvular defects may be the dominant lesion.
- Endocardial fibrosis is another uncommon substrate.
- Carcinoid heart disease is specifically cited as an example of endocardial pathology.

Valve abnormality

Tricuspid / pulmonary

Endocardial disease

Fibrosis; Carcinoid example

Microscopic response is not pathognomonic

- Hypertrophied myocytes are a general response to sustained workload.
- Interstitial fibrosis can accompany maladaptive ventricular remodeling.
- Morphology must be interpreted alongside the primary disease and hemodynamic context.

Possible findings

Myocyte enlargement

Also possible

Interstitial fibrosis

Diagnostic caution

Etiology is contextual

Extreme pressure can displace the septum

Marked RV pressure elevation



Septum bows leftward



LV filling / function impaired

- This is a mechanical interaction between the two ventricles.

Septal shift links right-sided load to left-sided dysfunction

- Right-ventricular pressure is transmitted mechanically across the interventricular septum.
- Severe right-sided pressure can cause leftward septal bulging.
- Left-ventricular dysfunction may occur despite a pulmonary-origin trigger.

Primary load

Right ventricular

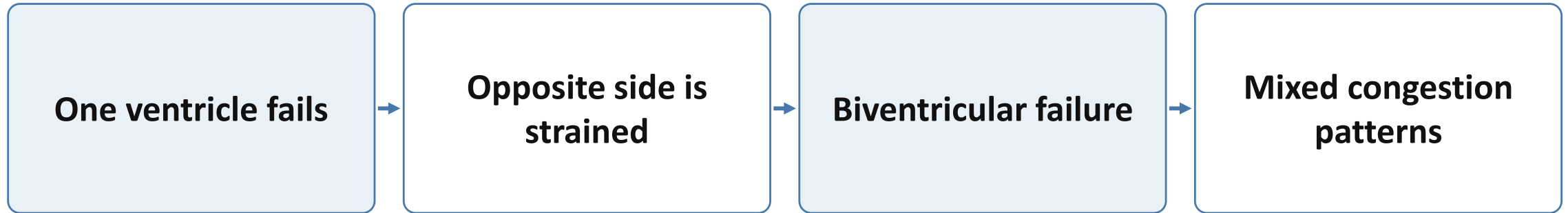
Mechanical effect

Leftward septal displacement

Secondary effect

Left ventricular dysfunction

An interdependent circulation can fail globally

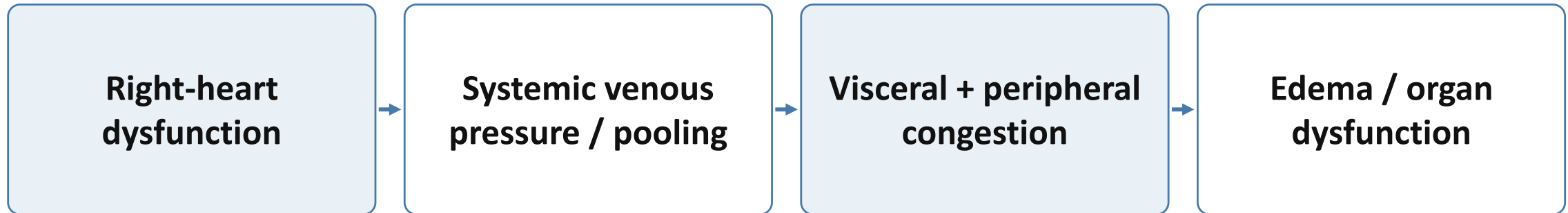


- Left-to-right progression is especially common.

Primary right versus left failure: Dominant pathology

Domain	Predominantly right-sided	Predominantly left-sided
Back-pressure	Systemic / portal venous systems	Pulmonary circulation
Edema / effusions	Peripheral edema; ascites	Pulmonary edema prominent
Organ impact	Liver, spleen, bowel, kidneys	Lungs; downstream organs
Overlap	Frequent in biventricular CHF	Frequent in biventricular CHF

Venous congestion explains multisystem injury



- The organ distribution of venous backup explains much of the clinical picture.

The systemic and portal veins are central

- The signature of primary right-sided failure is venous engorgement outside the lungs.
- Blood pools in systemic and portal venous beds.
- Consequences differ by organ anatomy, pressure, and chronicity.

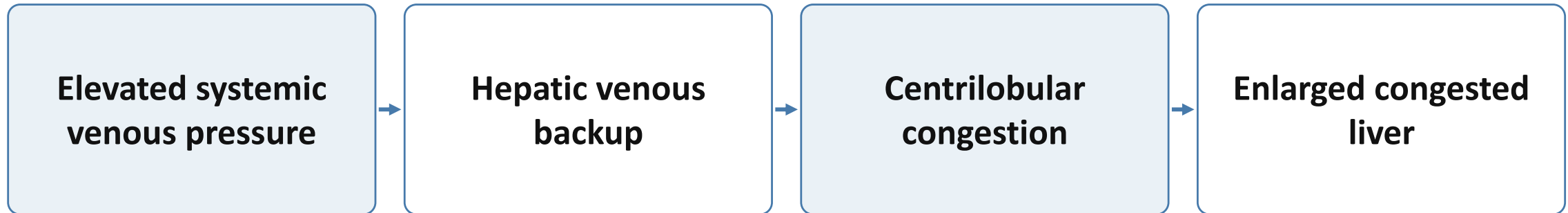
Systemic compartment

Peripheral tissue + serous spaces

Portal compartment

Liver, spleen + bowel

Hepatic congestion begins around central veins



- Passive congestion is greatest near the central veins.

Congestive hepatomegaly

- Passive venous engorgement increases liver size and weight.
- Congestion is especially pronounced in centrilobular regions.
- The gross appearance reflects uneven distribution of congested blood.

Macroscopic finding

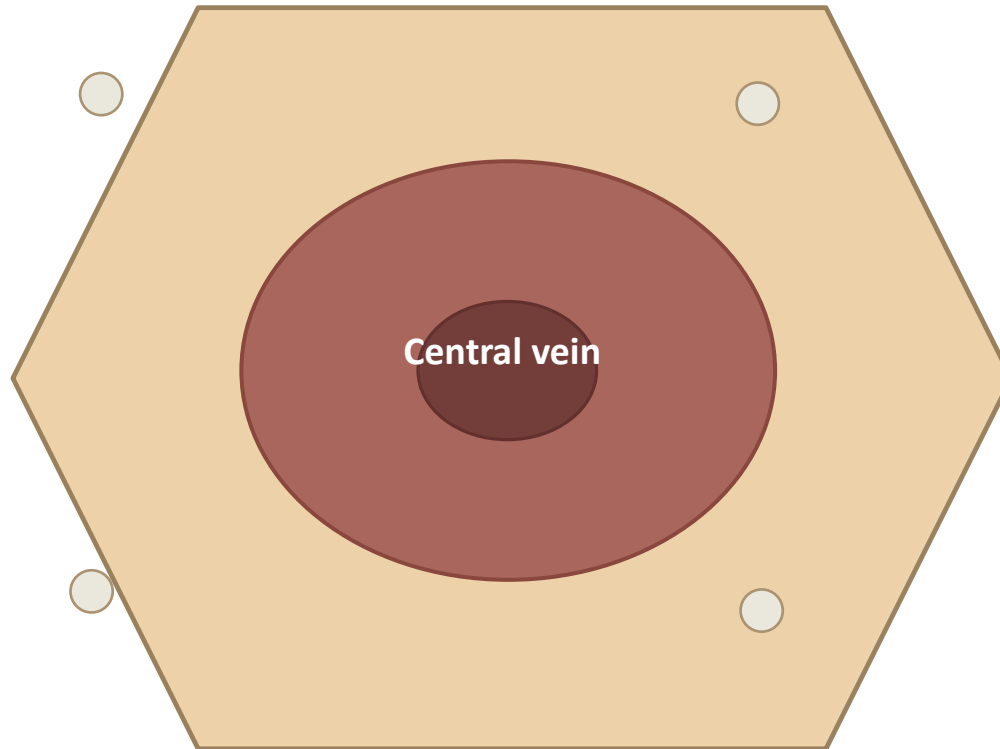
Hepatomegaly

Spatial pattern

Centrilobular prominence

Why the liver develops a “nutmeg” pattern

Simplified zonal schematic — not a gross specimen or histologic image



Tan periportal regions

Less visibly congested

Red-brown central zones

Blood pooling near central veins

Gross “nutmeg” mottling reflects preferential centrilobular congestion.

Centrilobular zones are vulnerable

- The central-vein regions bear the greatest venous congestion.
- When combined with impaired perfusion, hypoxia may become severe.
- This distribution can culminate in centrilobular hepatocyte necrosis.

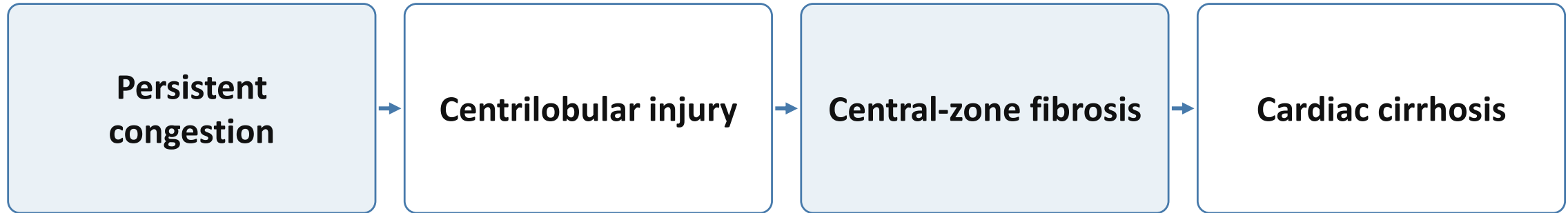
Passive congestion

Pericentral blood accumulation

Severe hypoxia

Potential central-zone necrosis

Congestion can progress to hepatic fibrosis

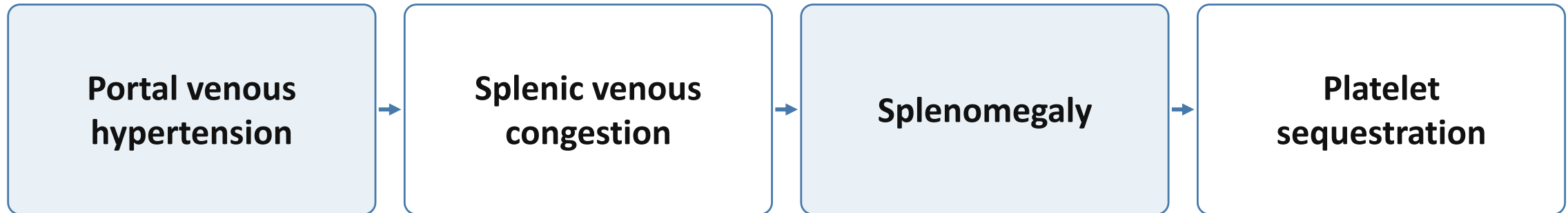


- This is an outcome of longstanding severe right-sided failure.

Hepatic changes: Stage and substrate

Morphology	Dominant pathology	Clinical interpretation
Congestive hepatomegaly	Venous pooling	Early / established passive congestion
Nutmeg appearance	Red-brown centers and tan periphery	Spatial heterogeneity of congestion
Centrilobular necrosis	Severe hypoxic injury	Especially if hypoperfusion coexists
Cardiac cirrhosis	Chronic central-zone fibrosis	Longstanding, severe disease

Portal congestion affects the spleen



- Congestive splenomegaly is a secondary visceral consequence.

Congestive splenomegaly

- Elevated portal pressure produces splenic enlargement.
- An enlarged spleen can sequester platelets.
- This association is a direct consequence of the portal venous component.

Primary process

Portal venous congestion

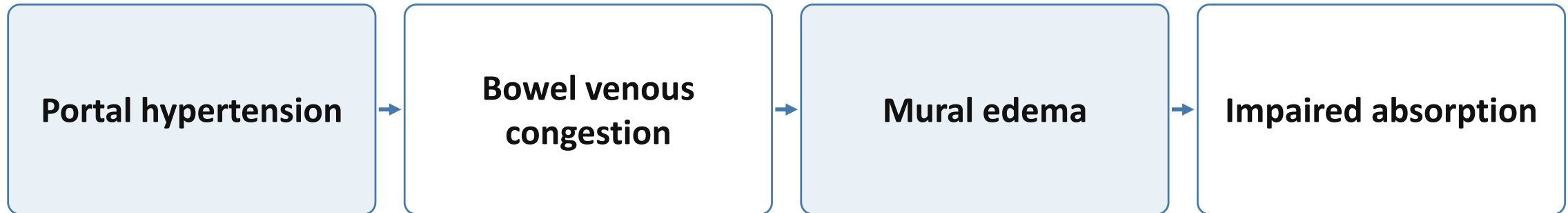
Organ change

Splenic enlargement

Hematologic effect

Platelet sequestration

Bowel-wall congestion can alter absorption



- Nutrient and drug absorption may be affected when edema is severe.

Bowel edema: A less visible manifestation

- Chronic venous engorgement causes edema of the gastrointestinal wall.
- The degree of edema can become functionally important.
- Possible impairment of nutrient and medication absorption.

Pathology

Congested, edematous bowel

Functional result

Absorption may decline

Serous cavity effusions

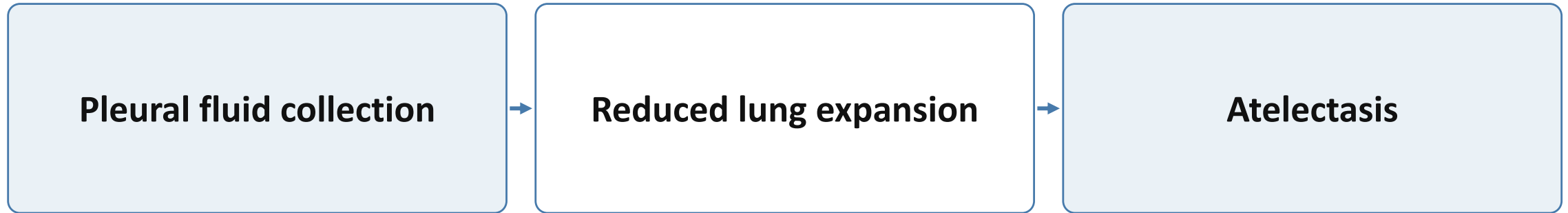
- Venous congestion may cause fluid collections in pleural spaces.
- The pericardial cavity may also accumulate fluid.
- Peritoneal fluid accumulation is termed ascites.

Pleural
Effusion

Pericardial
Effusion

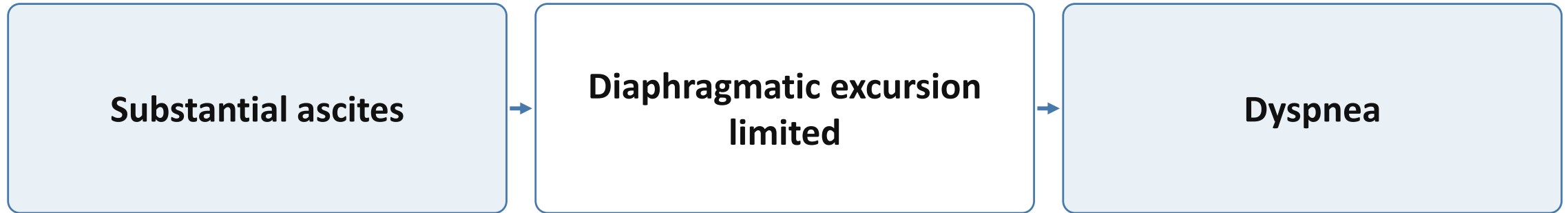
Peritoneal
Ascites

Pleural effusion can impair lung expansion



- Mechanical restriction is distinct from primary alveolar pulmonary edema.

Ascites can produce mechanical dyspnea



- A respiratory symptom may originate from abdominal fluid burden.

Peripheral edema is a hallmark

- Venous back-pressure favors edema of subcutaneous tissues.
- Gravity and body position influence where edema becomes apparent.
- Pedal and pretibial regions are classic dependent sites.

Mechanism

Dependent venous congestion

Clinical manifestation

Foot, ankle or shin edema

The distribution of edema depends on posture

Clinical setting	Characteristic edema	Reason for teaching
Ambulatory patient	Feet / ankles; pretibial tissue	Gravity-dependent localization
Chronically bedridden patient	Sacral region may predominate	Dependent region shifts with posture
Advanced generalized edema	Anasarca	Widespread fluid accumulation

Anasarca is advanced generalized edema

- Massive generalized edema can occur in marked right-sided failure.
- This extends beyond localized pedal or pretibial swelling.
- It reflects severe systemic fluid accumulation rather than a single-organ lesion.

Localized

Pedal / sacral edema

Generalized

Anasarca

The kidney can amplify volume overload

Renal venous congestion



Greater fluid retention



Peripheral edema worsens

- Renal congestion is especially prominent with right-sided failure.

Right-heart failure and azotemia

- Renal congestion impairs renal handling of retained fluid.
- Right-sided failure can produce more pronounced azotemia than isolated left-sided failure.
- Renal dysfunction and venous congestion can reinforce the systemic edema burden.

Kidney

Venous congestion

Functional effect

Fluid retention

Laboratory effect

Azotemia

Cerebral dysfunction is also possible

- Cerebral venous congestion and hypoxia can impair mental function.
- A similar phenotype may result from systemic hypoperfusion in left-sided failure.
- Neurologic findings therefore require integration with the whole circulation.

Venous mechanism

Cerebral congestion

Oxygen mechanism

Hypoxia

Clinical consequence

Mental-function deficits

A multisystem morphology-to-symptom map

Organ / space	Morphologic process	Clinical effect
Liver	Hepatic venous congestion	Hepatomegaly; possible injury
Spleen	Congestive enlargement	Platelet sequestration
Bowel	Congestion / edema	Absorption may be impaired
Body wall	Dependent tissue edema	Pedal / sacral swelling
Renal system	Congestion	Retention and azotemia

Biventricular failure combines both phenotypes

- Failure of one side can overburden the other in a closed circuit.
- Chronic decompensation frequently becomes biventricular.
- Pulmonary findings may coexist with peripheral edema and visceral congestion.

Left-sided pattern

Pulmonary congestion

Right-sided pattern

Systemic venous congestion

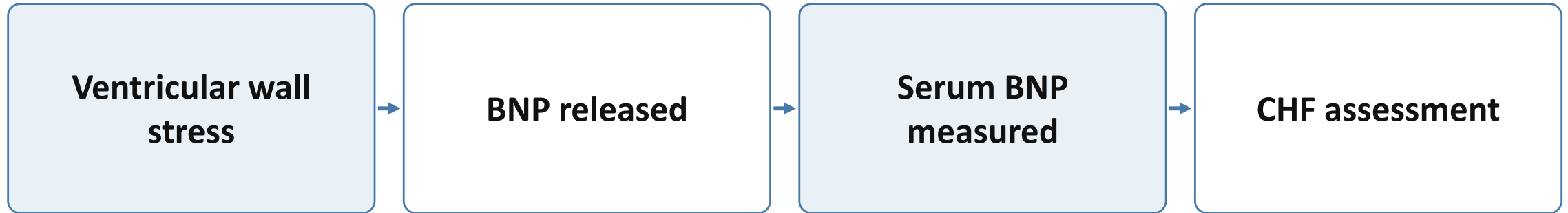
Combined syndrome

Both patterns coexist

Distinguishing pulmonary edema from pleural fluid

Finding	Principal mechanism in source	Interpretation
Pulmonary edema	Backward pressure from left-sided failure	Alveolar / interstitial fluid
Pleural effusion	Elevated venous / pleural fluid pressure	Fluid around lung; can restrict inflation
Ascites-related dyspnea	Peritoneal fluid burden	Diaphragm mechanically restricted

BNP links myocardial wall stress to assessment



- A low BNP has useful negative predictive value for CHF; No cutoff is supplied.

Echocardiography evaluates cardiac consequences

- Echocardiography informs ventricular ejection fraction.
- It assesses wall motion and valve function.
- It can also identify possible mural thrombosis during CHF assessment.

Pump function

Ejection fraction

Wall mechanics

Motion

Structural assessment

Valves / thrombus

Clinical assessment must integrate organs

- Clinical history and physical examination establish the pattern of congestion.
- BNP provides one laboratory measure in the evaluation of CHF.
- Echocardiography adds functional and structural information; No single result substitutes for overall assessment.

History + examination

Congestion pattern

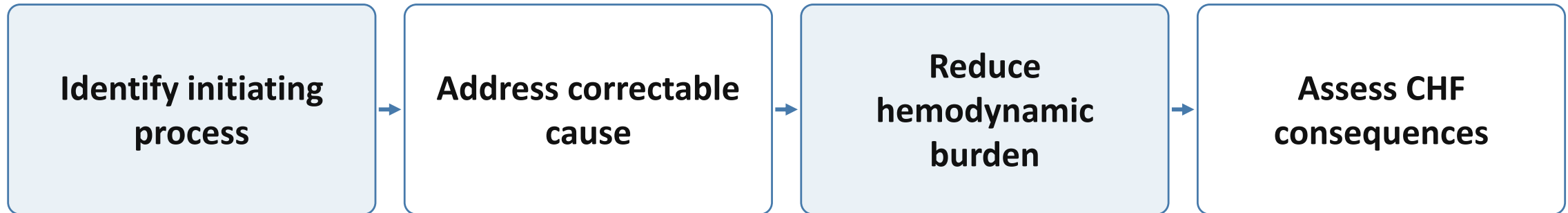
BNP

Wall-stress signal

Echocardiography

Anatomic / functional assessment

Management begins with the cause



- Valvular defects and inadequate cardiac perfusion as examples.

Decongestion in the CHF framework

- Salt restriction addresses fluid burden.
- Diuretics reduce volume overload.
- These measures target congestion but do not by themselves identify its initiating pathology.

Dietary measure

Salt restriction

Pharmacologic measure

Diuretic decongestion

Other textbook CHF treatment principles

- Positive inotropes increase myocardial contractility when used in selected circumstances.
- Afterload reduction and neurohumoral modulation are additional therapeutic concepts.
- The source discusses adrenergic blockade and ACE inhibition, but does not supply a right-heart-specific treatment algorithm.

Contractility

Positive inotropes

Afterload / signaling

Adrenergic blockade; ACE inhibitors

Case 1: Identify the pathway

A patient with established left-sided heart failure develops progressive ankle edema and congestive hepatomegaly. What mechanism best explains the new findings?

A. Pulmonary pressure burden extends strain to the right ventricle

B. Primary alveolar edema directly causes hepatic injury

C. The liver is the initial source of the cardiac pressure load

Case 1: Secondary right-heart involvement

A. Pulmonary pressure burden extends strain to the right ventricle

- Left-heart failure is the commonest antecedent of right-sided failure.
- Right-sided decompensation creates systemic venous backup.
- Pedal edema and congestive hepatomegaly fit this distribution.

Case 2: Connect the organ lesions

A patient with chronic pulmonary hypertension has right-ventricular enlargement, a mottled congested liver, splenomegaly, and bowel edema. Which shared process explains the extracardiac findings?

A. Portal and systemic venous congestion

B. Primary left atrial thromboembolism

C. Isolated pulmonary alveolar edema

Case 2: A systemic venous syndrome

A. Portal and systemic venous congestion

- Hepatic congestion emphasizes central-vein regions.
- Portal venous hypertension can enlarge the spleen and congest the bowel.
- These findings are downstream consequences of right-sided dysfunction.

Case 3: Explain dyspnea without alveolar edema

A patient with right-sided failure has marked ascites and a large pleural effusion but little primary pulmonary congestion. Which mechanism can still cause breathlessness?

A. Limited diaphragmatic excursion and lung inflation

B. Pulmonary edema is obligatory in isolated right-heart failure

C. Splenic enlargement directly causes alveolar flooding

Case 3: Mechanical respiratory restriction

A. Limited diaphragmatic excursion and lung inflation

- Ascites can mechanically restrict movement of the diaphragm.
- Large pleural effusions can interfere with lung expansion and cause atelectasis.
- This is distinct from the dominant left-failure mechanism of pulmonary edema.

Five pathology anchors

Pathway	Tissue / organ consequence
Left heart → lungs → right heart	Common route to secondary right-sided failure
Pulmonary disease → pulmonary hypertension	Cor pulmonale pathway
RV pressure overload	Hypertrophy, dilation, possible septal shift
Systemic / portal venous engorgement	Edema, effusions and visceral congestion
Chronic severe congestion	Centrilobular fibrosis; possible cardiac cirrhosis

Primary source and scope

- Robbins, Cotran & Kumar: Pathologic Basis of Disease. 11th ed. (2026). Chapter 12, The Heart.
- Core section: “Right-Sided Heart Failure,” followed by the systemic-organ morphology discussion.
- Parent context: Heart Failure and Pathophysiology and Progression to Heart Failure; related cor pulmonale discussion.