

Pulmonary Embolism

Thromboembolism, vascular obstruction, and pulmonary infarction

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Learning objectives

Trace venous thrombi to the pulmonary arterial circulation.

Relate embolic burden and cardiac reserve to respiratory and hemodynamic consequences.

Distinguish pulmonary hemorrhage from hemorrhagic infarction.

Interpret the main morphologic, clinical, diagnostic, and preventive principles.

Pulmonary embolism: Core definition

Embolism = material traveling to and lodging in a vessel

A circulating embolus lodges in the pulmonary arterial tree and obstructs downstream perfusion.

Most clinically important pulmonary emboli are thromboemboli originating in the systemic venous circulation.

The consequences range from clinically silent obstruction to sudden cardiovascular collapse.

Embolic obstruction is not the same as local thrombosis

Pulmonary thromboembolism	In situ pulmonary thrombosis
Clot formed elsewhere and migrated	Clot formed within pulmonary vasculature
Typical mechanism of major pulmonary artery occlusion	Rare in large pulmonary vessels
Commonly linked to venous DVT	Described with pulmonary hypertension and heart failure

Why pulmonary embolism matters

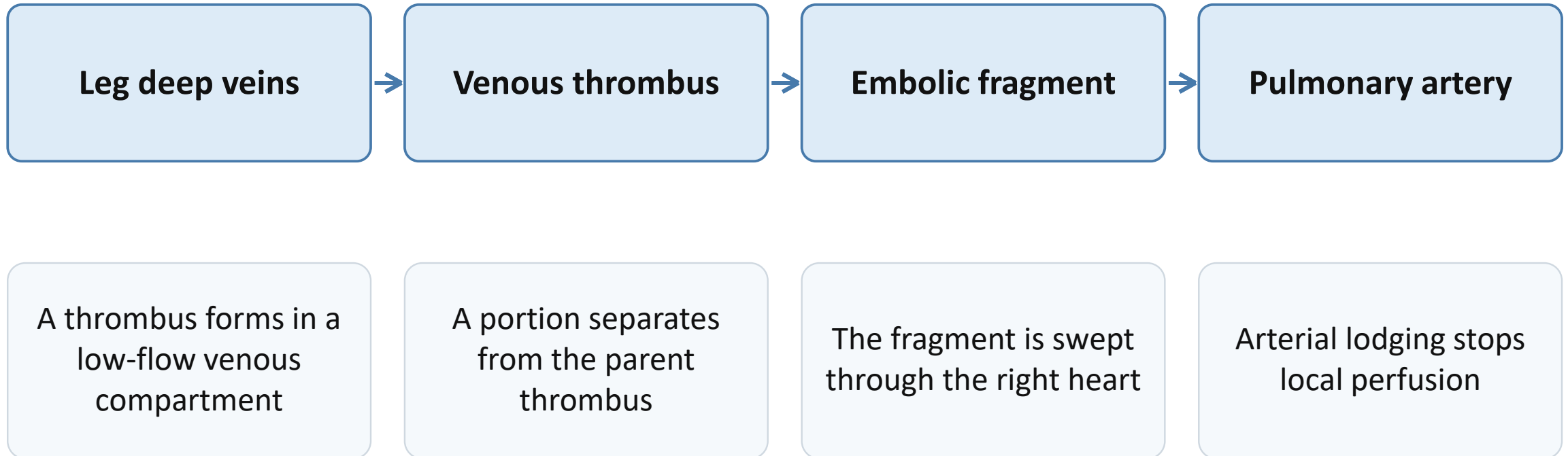
It is a major cause of morbidity and death in immobilized or thrombosis-prone patients.

A single event may produce abrupt pulmonary perfusion failure.

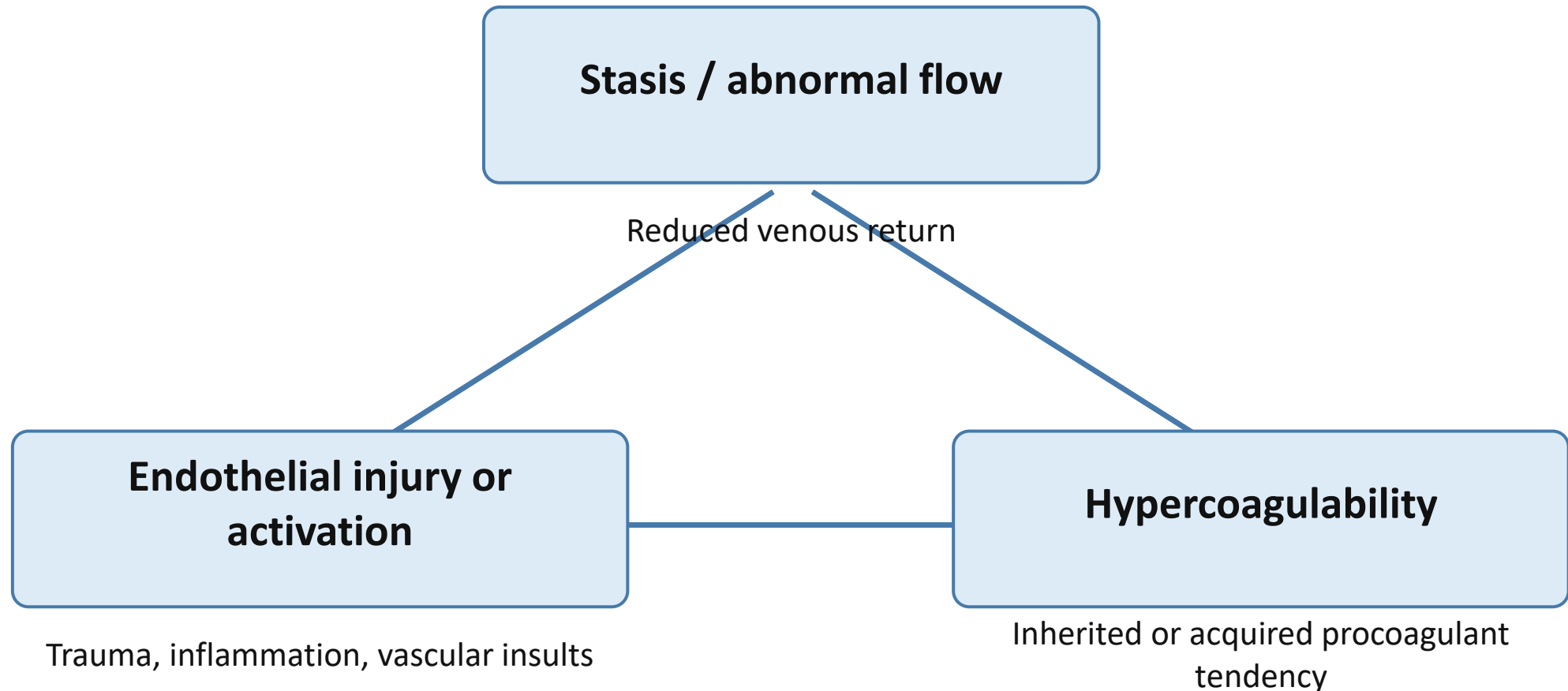
A small embolus can be an early warning of subsequent larger emboli.

Prevention requires recognition of the underlying thrombotic predisposition.

The usual source: Deep venous thrombosis

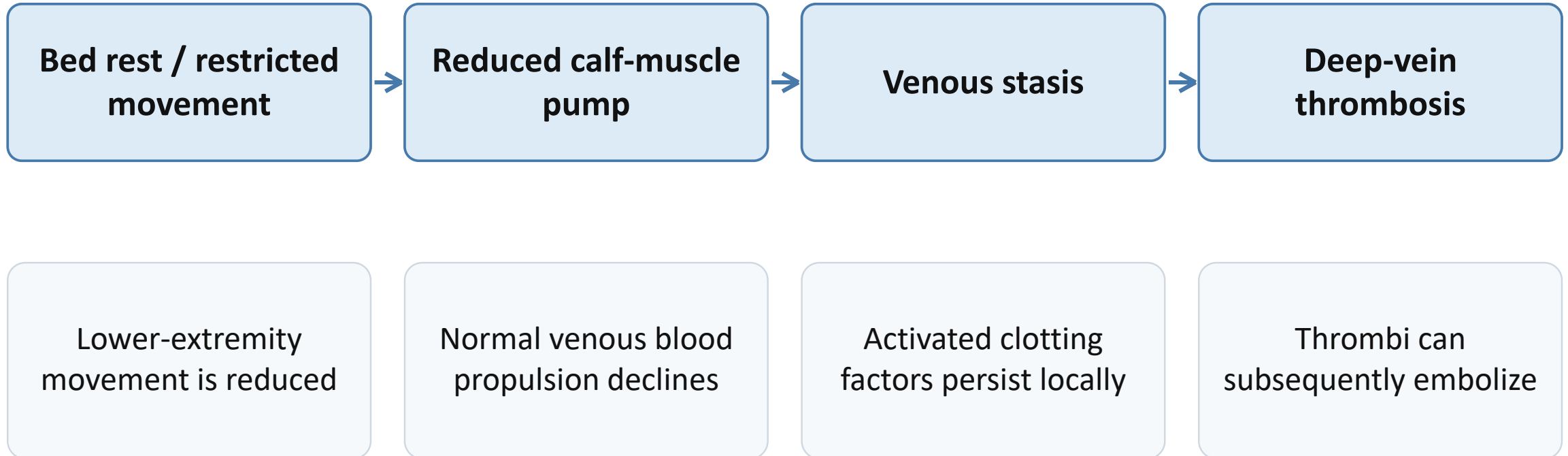


Virchow triad explains thrombus formation



Predisposition to venous thrombosis

Stasis: Why immobility increases risk



Heart failure also promotes venous stasis

Hemodynamic setting

Impaired venous return favors thrombus formation.

Stasis is an important contributor to venous thrombosis.

Pathologic consequence

An embolic fragment enters the venous return.

Right-heart transit delivers it to pulmonary arteries.

Surgery, trauma, and burns are multifactorial triggers

Altered flow and injury

Immobilization promotes venous stasis.

Injured tissue and vessels provide prothrombotic stimuli.

Coagulation balance

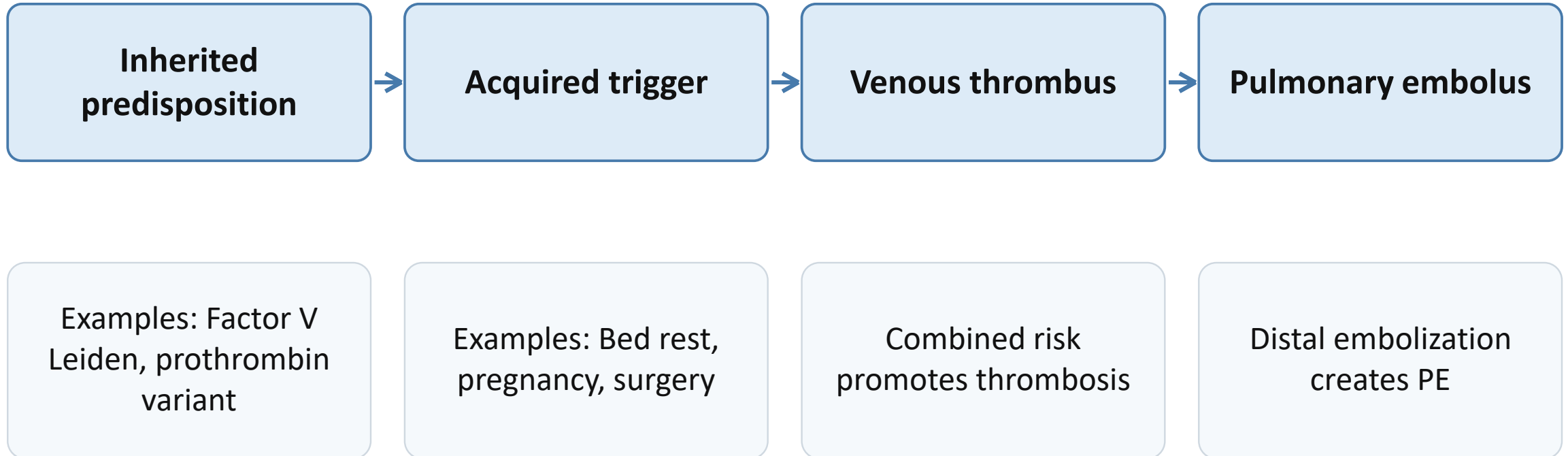
Procoagulants are released from injured tissue.

Reduced fibrinolytic activity can sustain thrombi.

Inherited thrombophilia: Mechanisms

Inherited state	Biologic effect
Factor V Leiden	Reduced susceptibility of factor V to protein C inactivation
Prothrombin G20210A variant	Higher prothrombin synthesis / levels
Antithrombin deficiency	Loss of physiologic anticoagulant effect
Protein C or protein S deficiency	Impaired anticoagulant regulatory pathway

Inherited and acquired risks can combine



Acquired prothrombotic states

Clinical setting	Prominent contributing process
Malignancy	Tumor-associated procoagulants and inflammation
Pregnancy / hormonal exposure	Systemic hypercoagulability; Altered venous return
Obesity	Acquired hypercoagulable predisposition
Major surgery / trauma	Stasis, tissue injury, and procoagulant activation

Cancer-associated thromboembolism

Malignancy



Procoagulant release



Venous thrombosis



**Pulmonary
embolization**

Tumor-associated
inflammation
contributes

Tumor-derived tissue
factor or mucin may
act as procoagulants

Thrombi may develop
or recur

Fragments may travel
to the lungs

Pregnancy and postpartum thrombotic risk

Venous component

Reduced venous return from leg veins may contribute.

Peripartum reduced mobility may add stasis.

Systemic component

Hormonal changes are associated with hypercoagulability.

Pregnancy and the postpartum period increase venous thrombotic risk.

Orthopedic injury: A high-risk clinical context

Patient scenario: An adult remains largely immobile after a hip fracture.

Which mechanisms in Virchow triad increase the risk of subsequent PE?

Venous stasis from immobility

Tissue / vascular injury after trauma

Acquired procoagulant changes

Central lines and right atrial thrombi

**Indwelling central
venous line**



Right atrial thrombus



Embolization



Pulmonary circulation

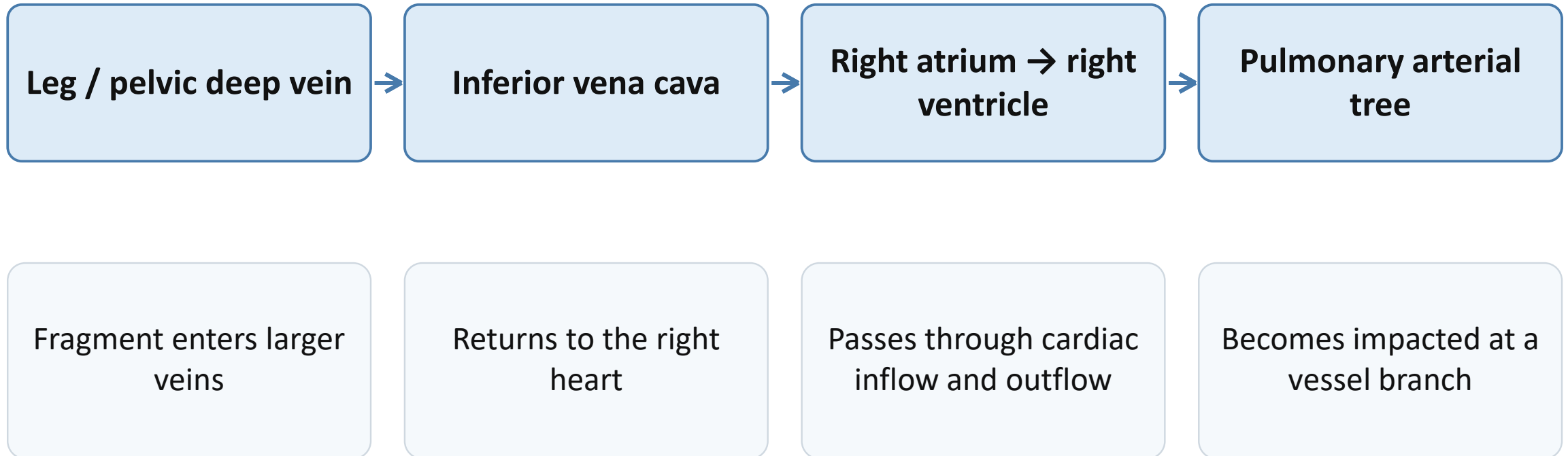
A potential local nidus

Thrombus forms near
an intravascular device

An attached mass can
detach

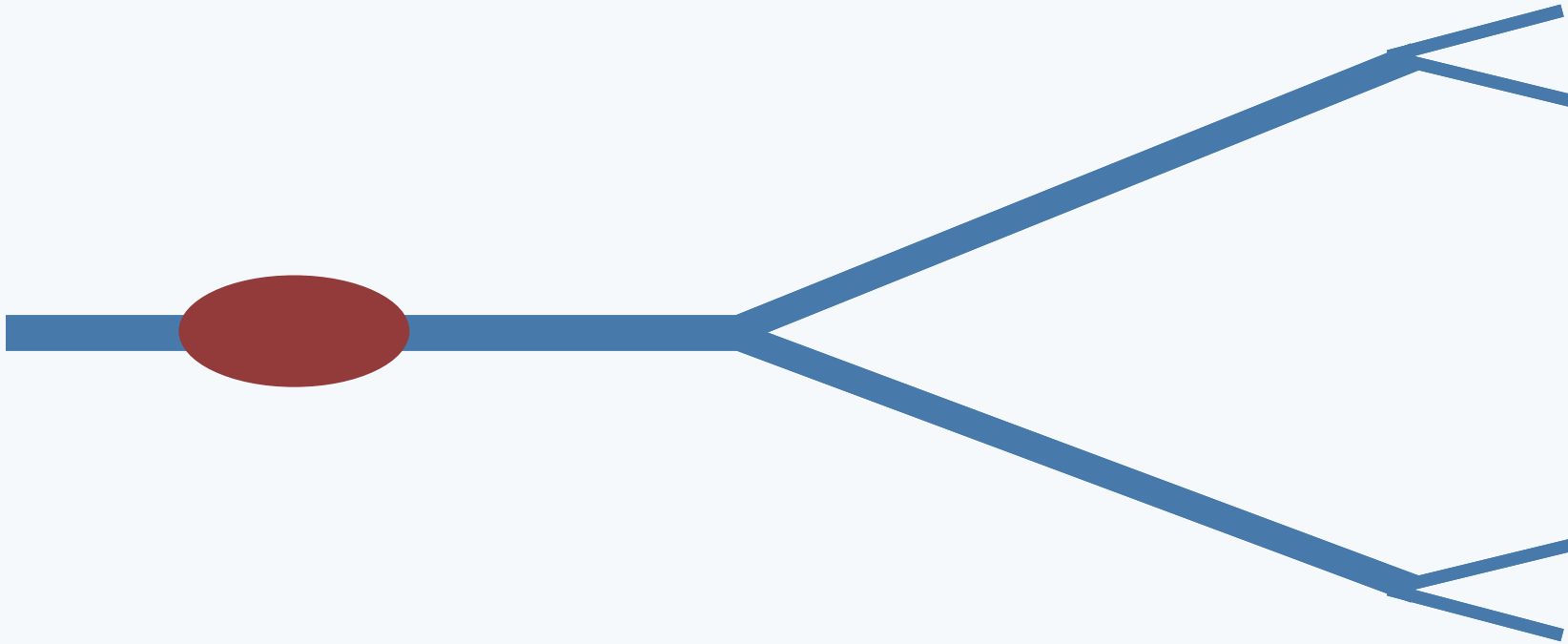
Emboli enter the
pulmonary arterial tree

The embolus follows the right-heart circulation



Anatomical lodging determines embolic effects

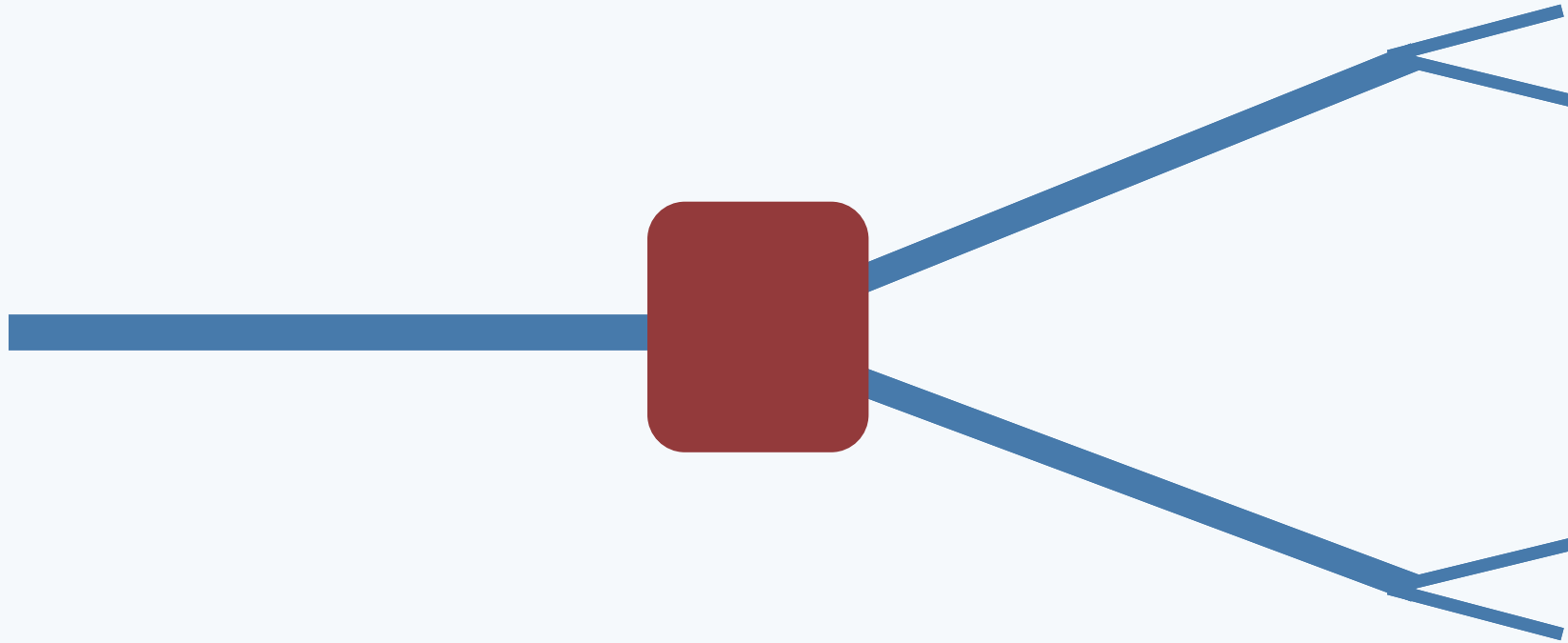
Pulmonary arterial bifurcation



Proximal pulmonary arterial occlusion

Saddle embolus: Obstruction at the bifurcation

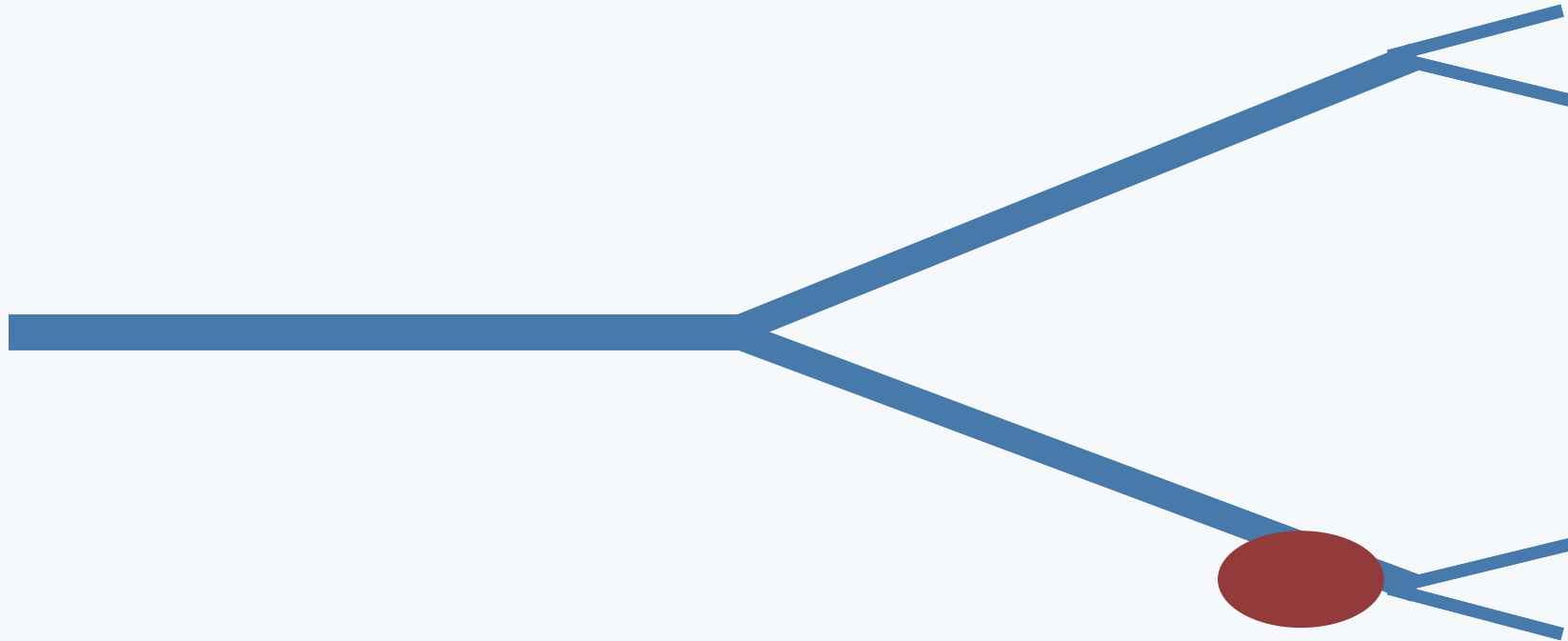
Pulmonary arterial bifurcation



Saddle embolus lies across the bifurcation

Small emboli lodge in peripheral branches

Pulmonary arterial bifurcation



Distal embolus occludes a peripheral branch

Multiple emboli may be simultaneous or recurrent

Shower of emboli

Several fragments may detach from one large thrombus.

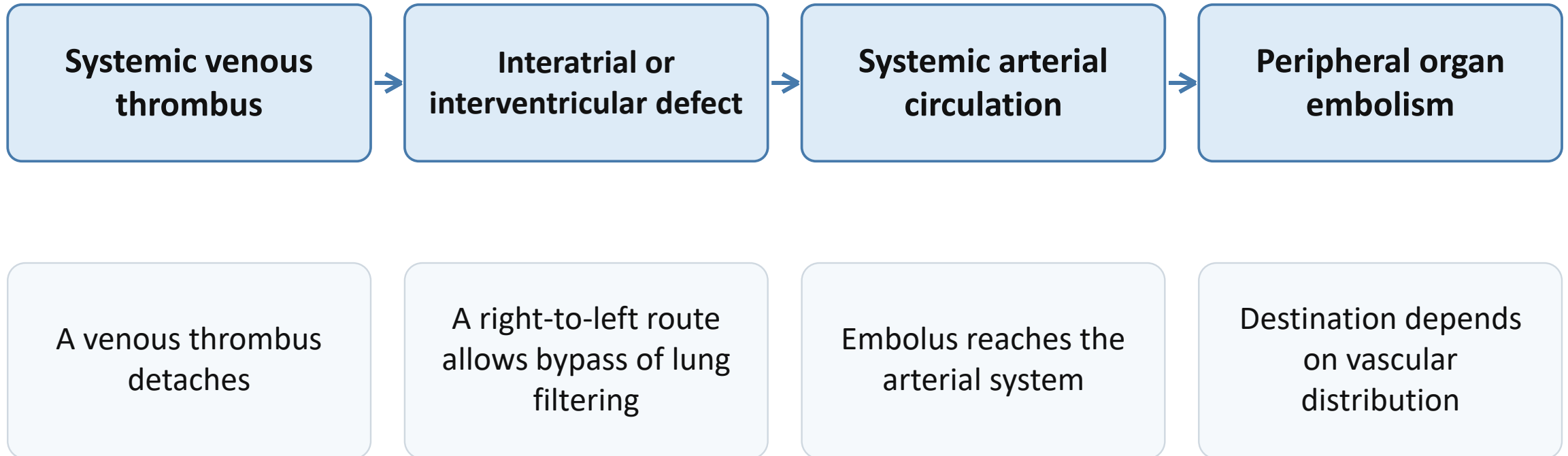
Obstruction occurs at multiple sites at once.

Serial embolization

Repeated episodes can occur over time.

Each event may reduce the effective vascular bed.

Paradoxical embolism: Venous-to-systemic passage



Not every pulmonary embolus is a blood clot

Embolic material	Context in the supplied source
Thrombus	Usual substrate of pulmonary embolism
Fat	Rare nonthrombotic pulmonary embolus
Air	Rare nonthrombotic pulmonary embolus
Tumor	Rare nonthrombotic pulmonary embolus

Two parallel pathophysiologic consequences

Respiratory compromise

Ventilation continues in poorly perfused lung units.

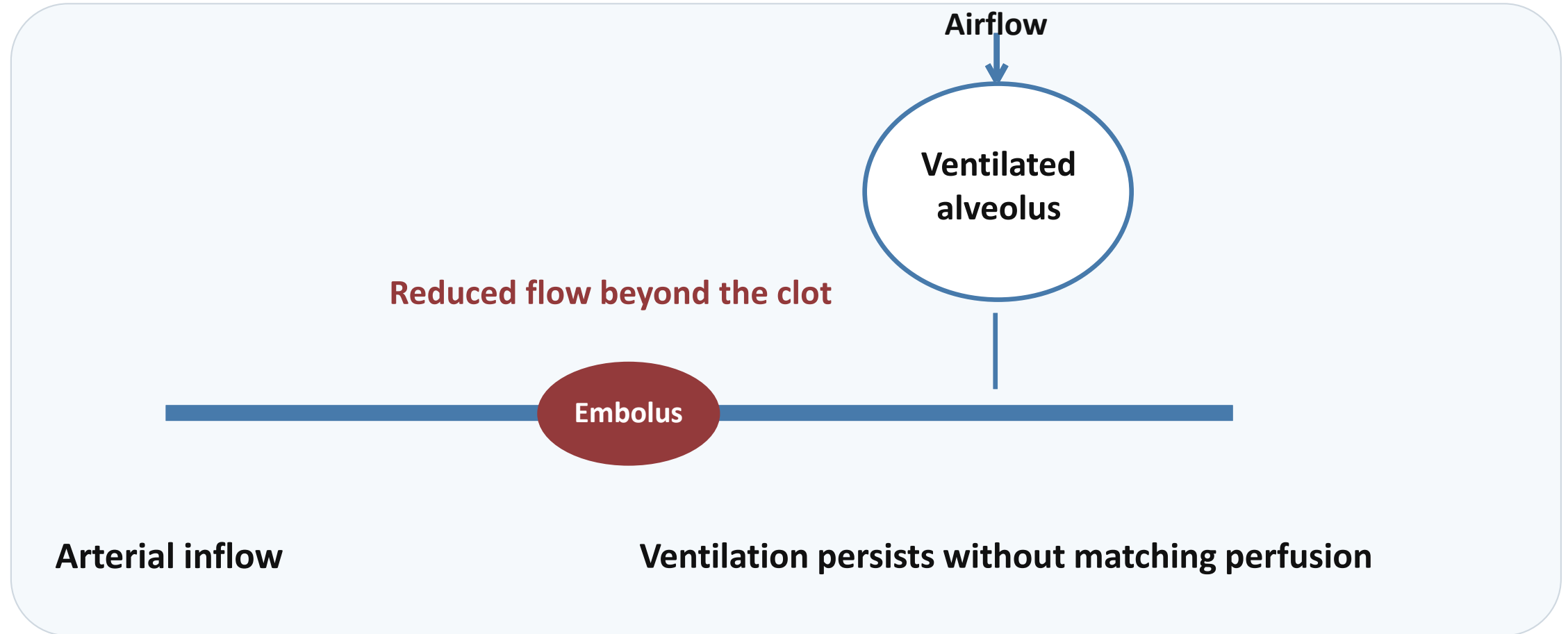
Gas-exchange efficiency is reduced.

Hemodynamic compromise

Obstruction increases resistance to pulmonary blood flow.

Right ventricular function may fail acutely.

Ventilated but nonperfused lung: The mismatch



Physiologic schematic — not a clinical imaging study

Vascular obstruction raises right-heart afterload

**Pulmonary arterial
occlusion**



**Increased vascular
resistance**



**Right-ventricular
pressure load**



**Acute right-heart
failure**

Available perfused
vascular bed falls

Blood must traverse
fewer channels

The RV faces an acute
outflow burden

Severe cases progress
to shock

What determines clinical severity?

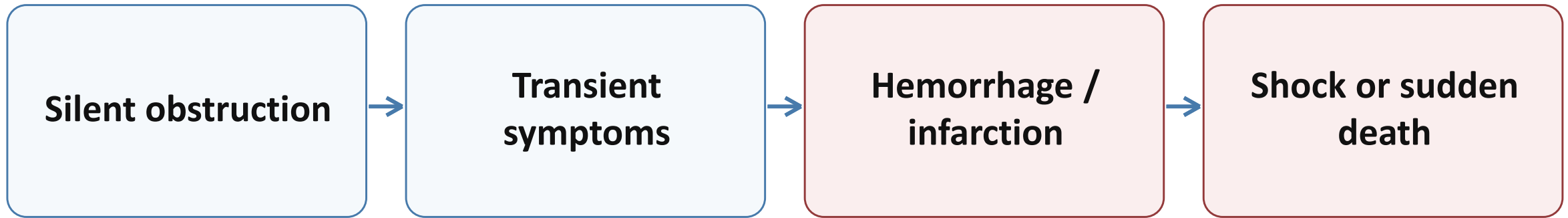
Degree of arterial obstruction

Caliber of occluded vessels

Number of emboli

Pre-existing cardiac reserve

Clinical spectrum: Small to catastrophic



Outcome depends on embolic burden, vessel caliber, embolus number, and cardiac reserve

Why many small emboli remain clinically silent

Small emboli may obstruct only a limited fraction of the pulmonary vascular bed.

The remaining perfused lung can maintain adequate function.

Thromboemboli may subsequently organize into the arterial wall.

The absence of early symptoms does not eliminate the risk of another embolus.

Massive obstruction can produce abrupt collapse

Extensive pulmonary obstruction



Marked fall in pulmonary blood flow



Acute right ventricular failure



Circulatory collapse

A substantial part of the vascular bed becomes unavailable

Right-heart output through the lungs declines

Acute cor pulmonale may ensue

Severe obstruction may be rapidly fatal

Pulseless electrical activity in catastrophic PE

Electrical rhythm $\neq$ effective circulation

Electrical activity may remain visible on the ECG despite absence of a palpable pulse.

Severe vascular blockage prevents effective forward flow through the lungs.

This mechanism explains electromechanical dissociation during resuscitation in some fatal PE cases.

Cardiovascular reserve modifies the outcome

Preserved reserve

Bronchial arterial flow may maintain lung tissue viability.

Hemorrhage may occur without infarction.

Compromised reserve

Pre-existing heart or lung disease reduces tolerance.

Pulmonary infarction is more likely.

The lung has a dual arterial supply

Pulmonary arterial inflow

May be blocked by PE

Bronchial arterial inflow

May preserve perfusion

Alveolar tissue viability

Dual inflow helps explain why embolic hemorrhage may occur without infarction.

Schematic; Flow contributions not drawn to scale

Hemorrhage does not necessarily mean infarction

Pulmonary hemorrhage

Blood may extravasate after embolic vascular injury.

Viable tissue can remain because bronchial perfusion persists.

Pulmonary infarction

Ischemic necrosis develops when compensatory perfusion fails.

The resulting infarct is characteristically hemorrhagic.

Small distal branches can cause infarction

**Distal arterial
embolus**



**Reduced local
perfusion**



**Insufficient
alternative flow**



**Hemorrhagic
infarction**

Occlusion occurs near
the microvascular bed

The affected territory
is perfusion-limited

Reserve and bronchial
supply determine
survival

Alveolar wall necrosis
accompanies
hemorrhage

Infarction is not the usual outcome of PE

Pulmonary infarction in approximately one tenth of embolic episodes.

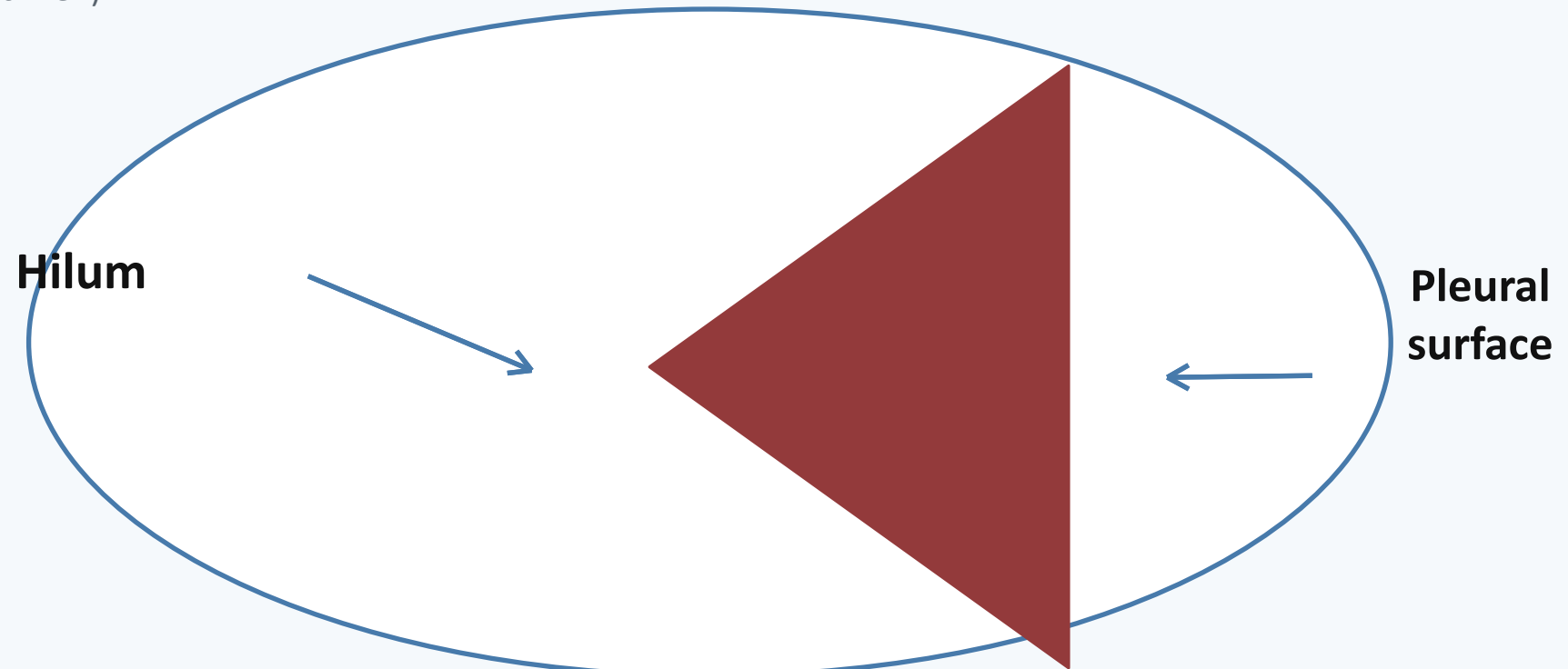
Most emboli do not infarct lung tissue because of the dual arterial supply.

Pre-existing cardiopulmonary impairment makes infarction more likely.

Infarction therefore reflects both vascular obstruction and reduced collateral reserve.

Gross distribution of pulmonary infarcts

Schematic (not a specimen)



Typical pleural-based infarct; Most occur in lower lobes

The classic infarct is wedge-shaped

Hilar apex ← wedge → pleural base

The broad end reaches the pleural surface; The apex points toward the hilum.

An occluded vessel may be found near the apex.

The wedge shape reflects the downstream territory of an obstructed arterial branch.

Early pulmonary infarction: Gross morphology

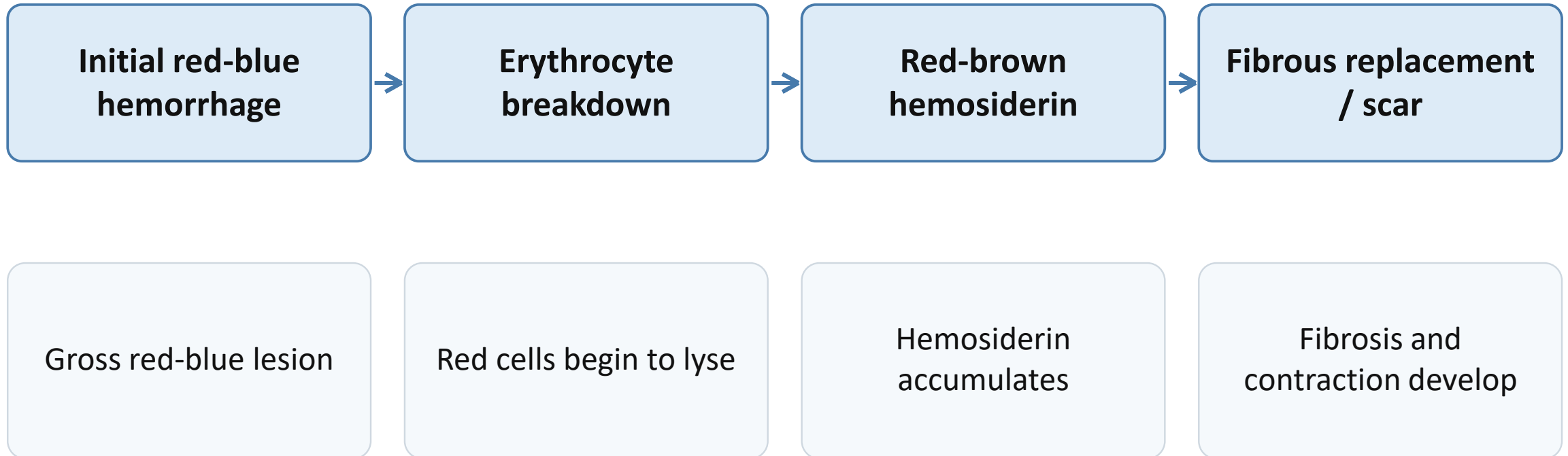
Infarcted lung

Raised, red-blue hemorrhagic area.
Often extends to the pleural surface.

Pleural reaction

Fibrinous exudate may cover the adjacent pleura.
The pleuritis helps explain pleuritic chest pain.

The infarct changes appearance with time



Temporal sequence — schematic; Not a photographed specimen

Infarct healing ends in contraction and scarring

Necrotic lung region



**Marginal fibrous
replacement**



**Gray-white peripheral
zone**



Contracted scar

Tissue is not
recoverable in the
infarcted territory

Repair advances
inward from the
margins

Fibrosis progressively
replaces necrotic tissue

The mature lesion
contracts

Microscopic hallmark: Ischemic parenchymal necrosis

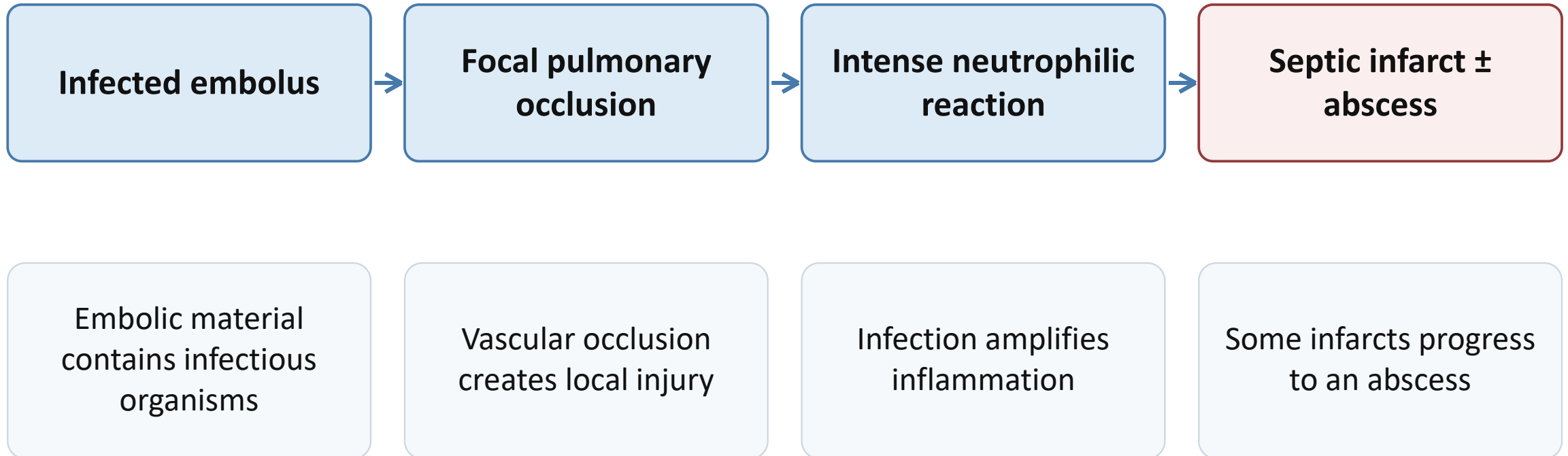
Injured compartments

Alveolar walls undergo ischemic necrosis.
Bronchiolar and vascular walls may also be involved.

Associated changes

Hemorrhage fills or surrounds injured parenchyma.
The morphology reflects both ischemia and blood extravasation.

Septic emboli produce a different inflammatory pattern



An antemortem thrombus differs from a postmortem clot

Antemortem thrombus	Postmortem clot
Formed in flowing blood before death	Develops after circulation stops
May show lines of Zahn	Does not display genuine flow-related lines of Zahn
Can embolize and occlude arteries	Does not explain antemortem thromboembolism

Most common symptomatic presentations

Dyspnea is a prominent symptom.

Pleuritic chest pain is common in symptomatic cases.

Cough may accompany pulmonary embolization.

Symptom intensity varies with the amount and location of obstruction.

Look for clues to the deep-vein source

Leg symptoms

Calf or thigh swelling may be present.

Pain in the affected limb may accompany DVT.

Clinical interpretation

Leg findings support a thromboembolic source.

Absence of obvious limb symptoms does not rule out PE.

Infarction adds fever, hemoptysis and pleuritis

Parenchymal injury

Hemorrhagic infarction can cause hemoptysis.

Fever may accompany tissue injury.

Pleural involvement

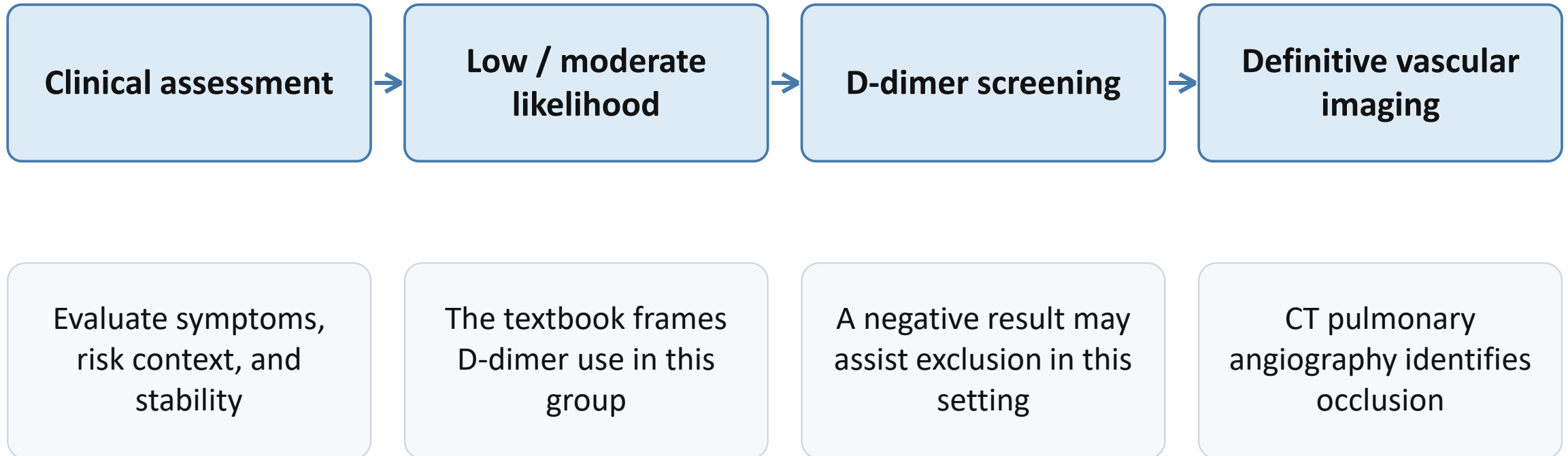
Pleuritic pain may reflect fibrinous pleuritis.

An audible pleural friction rub can occur.

Large emboli may mimic myocardial infarction

Shared clinical features	PE-specific context
Severe chest pain	Recent immobility or thrombotic predisposition
Dyspnea	Possible accompanying leg DVT
Shock	Acute pulmonary vascular obstruction

Diagnostic testing must reflect clinical likelihood



D-dimer is a rule-out test in selected patients

Interpret a test in its pretest-probability context

D-dimer in hemodynamically stable patients at low-to-moderate clinical risk.

A normal result can help exclude PE when appropriately interpreted in that group.

D-dimer alone is not presented as an imaging substitute in patients with high clinical suspicion.

CT pulmonary angiography identifies the obstruction

Anatomic question

Is contrast flow interrupted in a pulmonary artery?

Which central or branch vessels are affected?

Role

Usually provides definitive diagnosis.

Displays obstructed pulmonary arterial branches.

Ventilation–perfusion imaging: An alternative

Ventilation study

Maps inspired-air distribution within the lungs.

Air entry may be relatively preserved in an embolized region.

Perfusion study

Assesses regional pulmonary blood flow.

A corresponding perfusion deficit may reveal mismatch.

Duplex ultrasonography evaluates DVT

Suspected PE



**Evaluate thrombotic
source**



**Venous duplex
ultrasound**



**Identify deep-vein
thrombosis**

Clinical syndrome
prompts vascular
assessment

DVT can coexist with
pulmonary embolism

Ultrasonography
examines deep venous
flow and
compressibility

Support for a venous
source

Chest radiography may be initially unrevealing

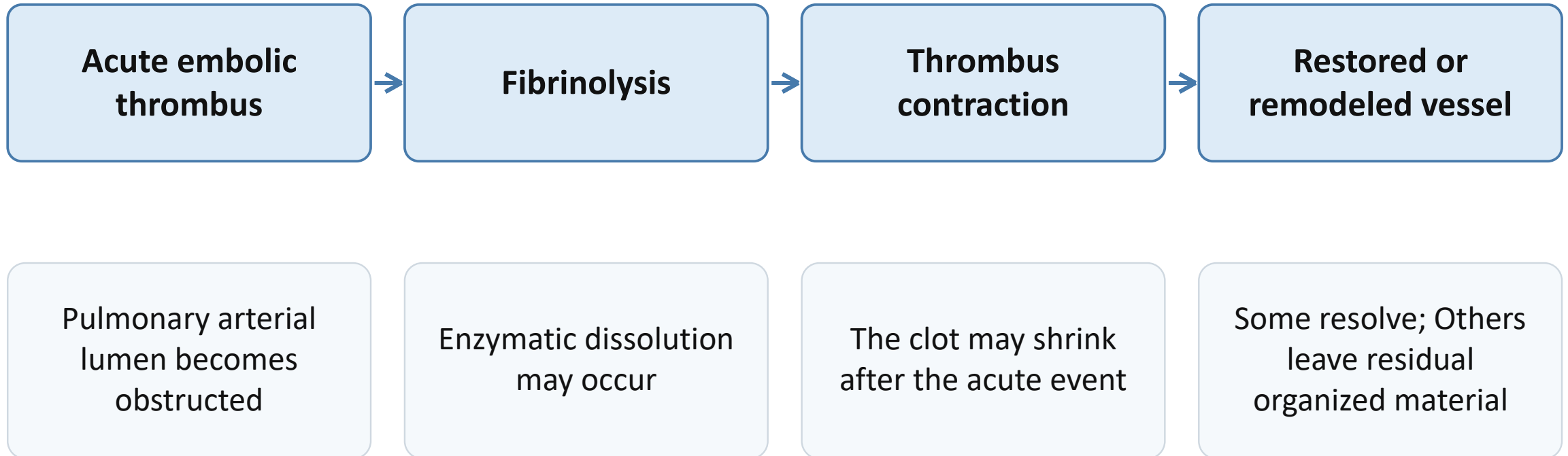
Uncomplicated PE

Chest radiograph may appear normal.
A normal radiograph does not exclude PE.

Pulmonary infarction

A peripheral wedge-shaped infiltrate may appear.
Visibility about 12–36 h after infarction.

Clot resolution follows fibrinolysis and contraction



Organized emboli can leave fibrous webs

Persistent embolus



**Thrombus
organization**



**Incorporation into
arterial wall**



Bridging fibrous web

Not all thrombus
dissolves completely

Fibrous tissue replaces
thrombotic material

Chronic remodeling
changes the lumen

Thin residual webs may
persist

Recurrent emboli: Cumulative vascular injury

**Repeated embolic
episodes**



**Loss of perfused
vascular bed**



**Higher pulmonary
resistance**



**Right-ventricular
strain**

Small events occur
over time

Functional cross-
sectional area
diminishes

Hemodynamic load
increases

Chronic cor pulmonale
can develop

Chronic thromboembolic pulmonary hypertension

Recurrent obstruction → pulmonary hypertension → cor pulmonale

Persistent or recurrent thromboemboli narrow the effective pulmonary vascular bed.

Increased pulmonary vascular resistance may result in pulmonary hypertension.

Long-standing pressure overload can progress to right-ventricular failure.

Preventing venous thrombosis prevents PE

Preventive strategy	Mechanistic purpose
Early postoperative / postpartum ambulation	Limit venous stasis
Mechanical compression in appropriate patients	Improve venous return
Anticoagulation when clinically indicated	Reduce thrombus formation and extension

Anticoagulation and supportive care

Anticoagulation

Central element of PE treatment.
Targets the underlying thromboembolic process.

Supportive measures

Address the consequences of respiratory compromise.
Support hemodynamics when cardiac output is impaired.

Thrombolysis: Potential benefit and bleeding risk

Potential role	Important limitation
Considered in severe complications such as shock	Can cause serious bleeding
Targets obstructing thrombus more rapidly	Decision requires careful clinical risk assessment

Inferior vena cava filter: Selected use

High recurrence risk



**Anticoagulation
contraindicated**



IVC filter placed



Venous clot capture

Further embolic events
remain a concern

Anticoagulant therapy
cannot be used

A mechanical barrier is
deployed in the vena
cava

May intercept clots
before reaching the
lungs

Case: Dyspnea after prolonged immobilization

A patient is bedridden after surgery and suddenly develops dyspnea with a painful swollen calf.

What source and pathway best explain the pulmonary findings?

Deep-vein thrombosis in an immobilized leg

Fragment transit via vena cava and right heart

Obstruction of pulmonary arterial branches

Case: Hemorrhage versus true infarction

Two patients have peripheral pulmonary arterial emboli. One has preserved cardiac function; The other has severe pre-existing cardiac disease.

Why may only the second patient develop infarction?

Bronchial circulation can preserve tissue perfusion

Cardiopulmonary compromise reduces compensatory reserve

Vascular hemorrhage does not itself prove necrosis

Case: Abrupt shock and absent pulses

A high-risk patient develops sudden severe dyspnea, hypotension, and pulseless electrical activity.

What pathophysiology links a large embolus to collapse?

Major pulmonary outflow obstruction

Abruptly increased right-ventricular afterload

Failure of effective forward pulmonary blood flow

Compare three major outcomes of pulmonary emboli

Outcome	Typical mechanism	Major implication
Clinically silent	Limited perfusion loss / preserved reserve	Risk of later embolization may persist
Hemorrhagic infarction	Distal occlusion plus insufficient collateral perfusion	Wedge-shaped pleural lesion
Acute cor pulmonale / collapse	Extensive pulmonary vascular obstruction	Acute right-heart failure

Integrating source, mechanism and morphology

Predisposing state



**Deep-vein
thrombosis**



**Pulmonary arterial
obstruction**



**Anatomic / clinical
consequence**

Stasis, endothelial
injury, or
hypercoagulability

Embolization is the key
intermediate

Location and burden
determine perfusion
loss

Silent PE, infarction, or
acute RV failure

Core principles to retain

Most pulmonary thromboemboli begin as thrombi in systemic deep veins.

Perfusion loss and increased pulmonary vascular resistance account for major consequences.

Dual circulation limits infarction; Compromised reserve increases its likelihood.

Recurrent or unresolved emboli may culminate in pulmonary hypertension and right-heart failure.

Primary references

Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th edition (2026).

Pulmonary Embolism — Hemodynamic Disorders, Thromboembolic Disease, and Shock.

Pulmonary Embolism and Infarction — The Lung.

The clinical and morphologic content of this lecture is grounded in these textbook sections.