

Pulmonary Embolism and Infarction

Pathogenesis • Morphology • Clinicopathologic correlation

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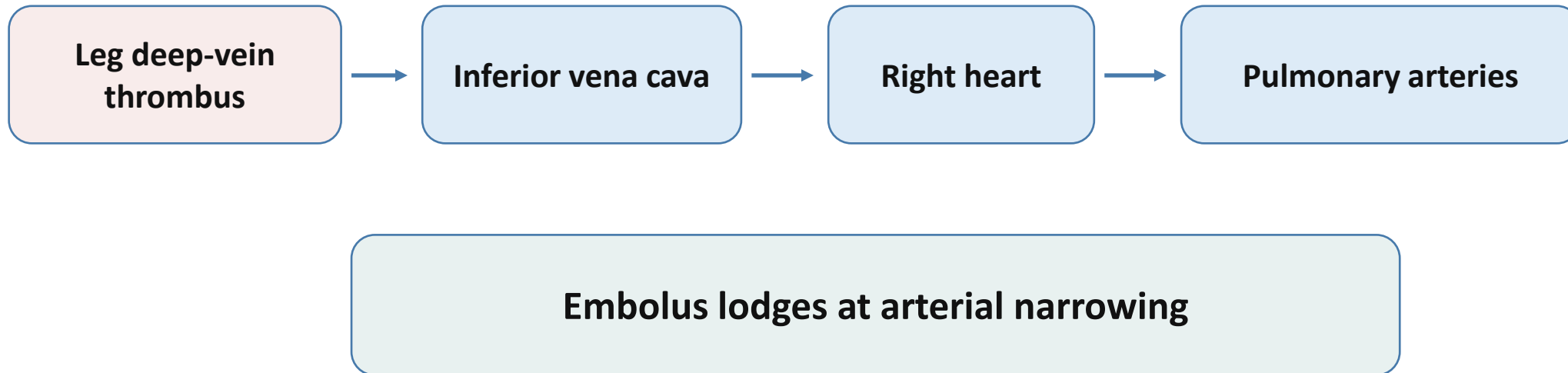
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Learning objectives

- Distinguish pulmonary thromboembolism from primary thrombosis and nonthrombotic emboli.
- Explain why some emboli cause silent obstruction, whereas others cause shock or infarction.
- Identify characteristic gross and microscopic findings of hemorrhagic pulmonary infarction.
- Integrate clinical findings, diagnostic tests, and mechanisms of recurrent embolic disease.

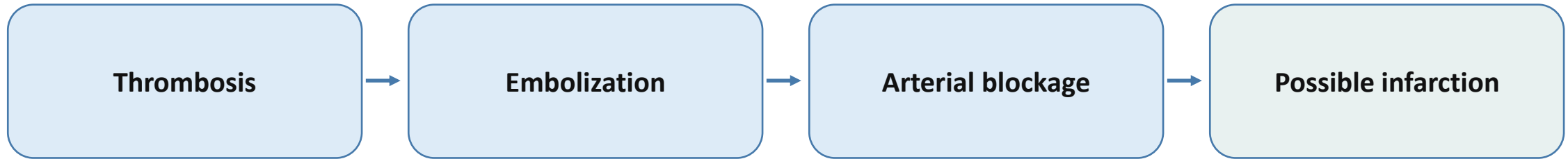
Pulmonary embolism: Central concept



Schematic: Thrombus detachment → transport → pulmonary obstruction

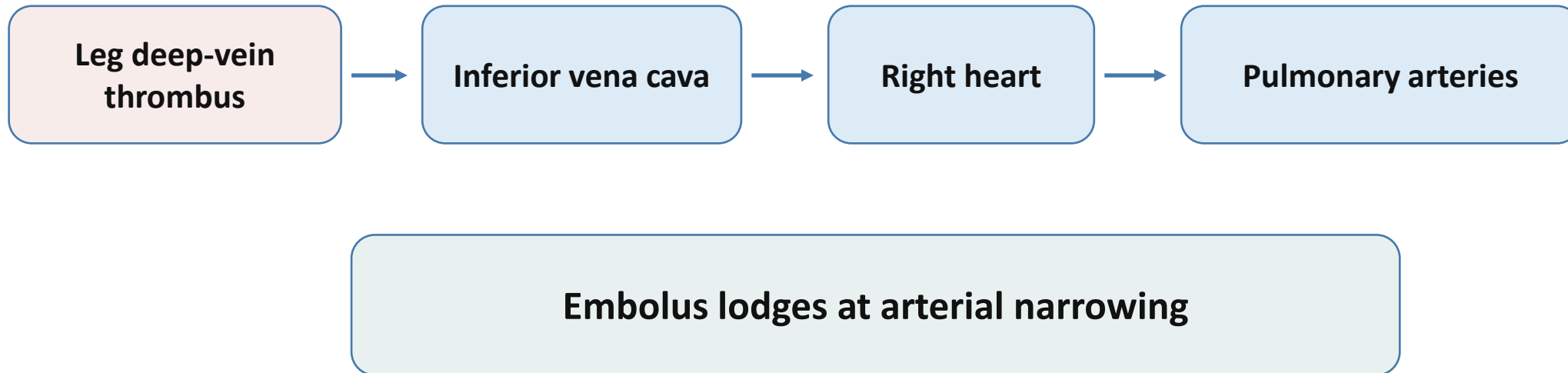
- A detached intravascular mass lodges within the pulmonary arterial circulation.
- Most clinically important pulmonary arterial occlusions arise from venous thrombi.
- Consequences depend on obstructed vascular territory and cardiopulmonary reserve.

Thrombus, embolus, and infarct are distinct



- Thrombus: Intravascular clot formed at its site of attachment.
- Embolus: Detached mass transported to a distant vascular territory.
- Infarct: Ischemic tissue necrosis; It does not follow every pulmonary embolus.

Why the deep veins matter



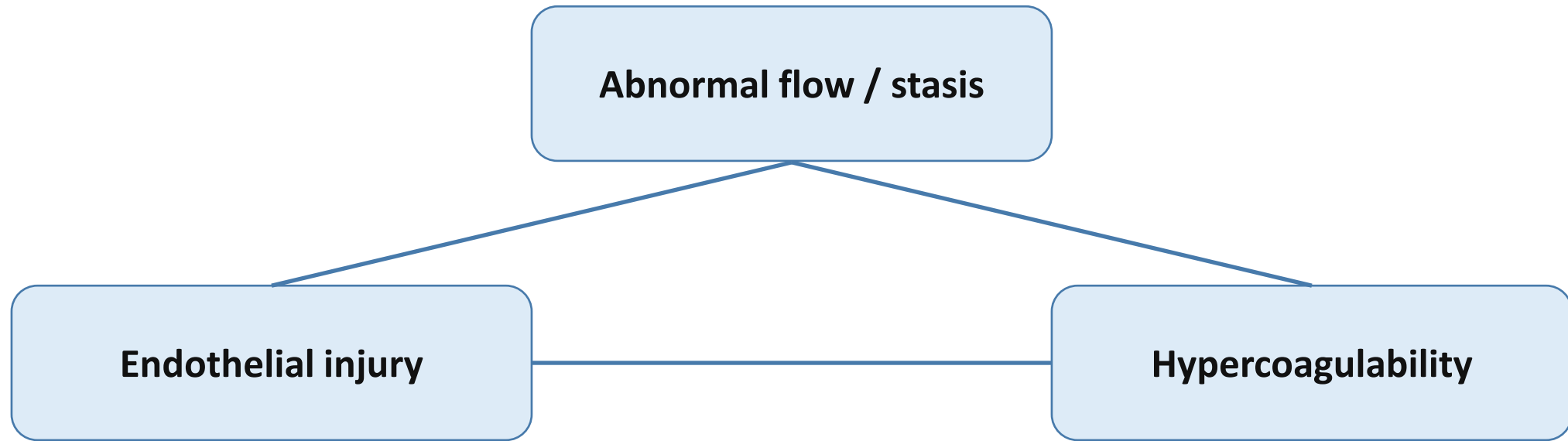
Schematic: Thrombus detachment → transport → pulmonary obstruction

- Leg deep venous thrombosis is the principal source of pulmonary thromboemboli.
- Thrombus fragments move through the vena cava and right heart into pulmonary arteries.
- Risk factors for pulmonary embolism largely overlap those for deep venous thrombosis.

Proximal deep venous thrombosis

- Popliteal, femoral, and iliac venous thrombi are particularly important embolic sources.
- Thrombi may propagate in the direction of venous blood flow.
- Proximal venous thrombi may fragment even when local leg symptoms are absent.

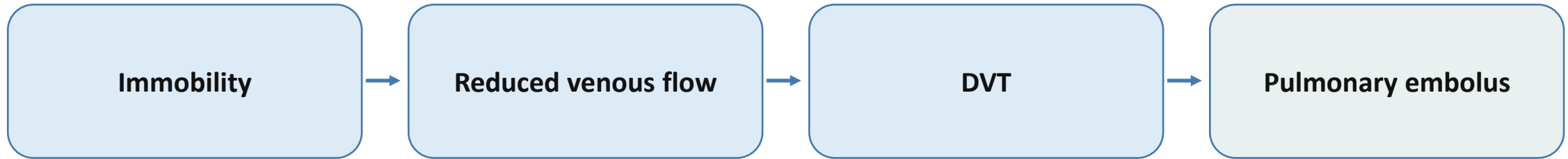
The Virchow triad explains susceptibility



Convergent mechanisms promoting venous thrombus formation

- Abnormal flow, vascular injury, and hypercoagulability contribute to thrombus formation.
- Clinical risk states often act through more than one mechanism.

Immobilility: Venous stasis



- Loss of the leg-muscle pump reduces venous return.
- Bed rest and prolonged immobilization favor lower-limb venous thrombosis.
- Hip fracture combines immobility with major tissue injury.

Trauma and surgery: Multiple mechanisms

- Vessel injury and tissue damage can favor coagulation.
- Postoperative immobility adds venous stasis.
- Trauma, fractures, severe burns, and major surgery are important precipitating contexts.

Inherited hypercoagulability

- Factor V Leiden is an inherited risk factor named in the pulmonary embolism section.
- Inherited antithrombin and other anticoagulant-pathway defects are discussed in Chapter 4.
- A hereditary predisposition may interact with acquired triggers.

Acquired hypercoagulability

- Cancer, pregnancy, oral contraceptive use, and obesity are relevant examples.
- Recent surgery and tissue injury can alter the coagulation balance.
- Cardiac failure contributes through impaired venous return and reduced reserve.

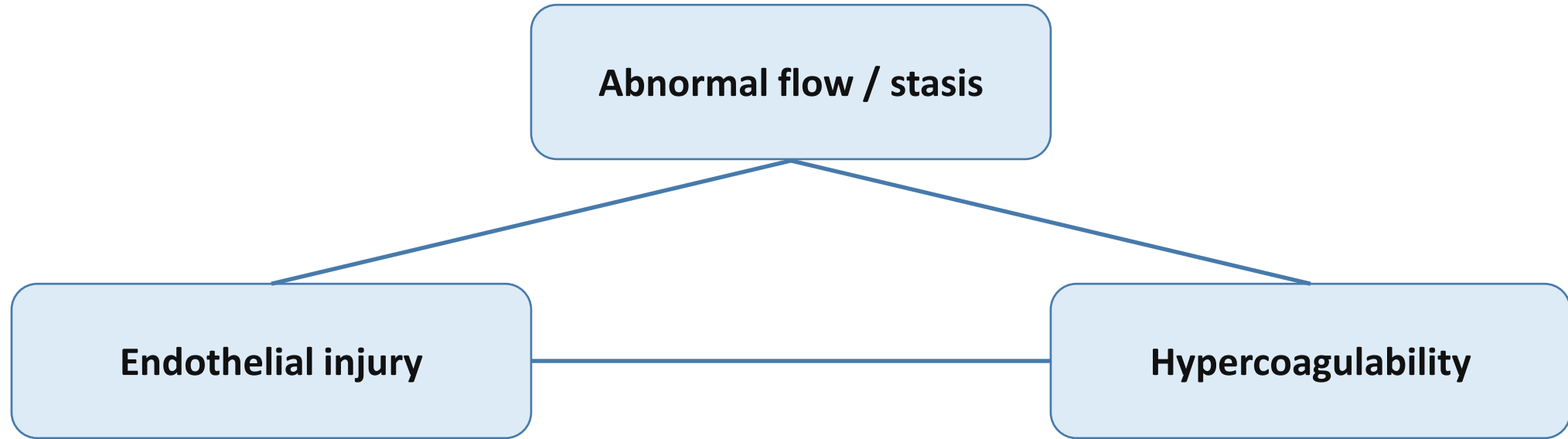
Malignancy-associated thrombosis

- Disseminated cancer is strongly associated with venous thromboembolic disease.
- Tumor-associated inflammation and procoagulant products contribute to thrombosis.
- An unprovoked pulmonary embolus may occur in patients with an underlying malignancy.

Pregnancy and the postpartum period

- Hormonal changes and increased coagulation potential increase thrombosis risk.
- Reduced return from lower-limb veins contributes to venous stasis.
- Early mobilization after delivery is one component of preventive care.

Risk factors often cluster



Convergent mechanisms promoting venous thrombus formation

- A hospitalized patient may have immobility, heart failure, recent surgery, and cancer.
- Each risk factor may affect stasis, vascular injury, or coagulation.
- Thromboembolic risk should be viewed as cumulative rather than single-cause.

Deep venous thrombosis may be silent

- Local collateral venous channels can prevent marked symptoms despite DVT.
- Chapter 4 reports that many deep venous thrombi are clinically unrecognized.
- Pulmonary embolism can therefore be the first manifestation of venous thrombosis.

Thromboembolism is the dominant subtype

Thromboembolus

Detached venous clot; Common

Nonthrombotic embolus

Fat, air, tumor or marrow;
Uncommon

Primary pulmonary thrombus

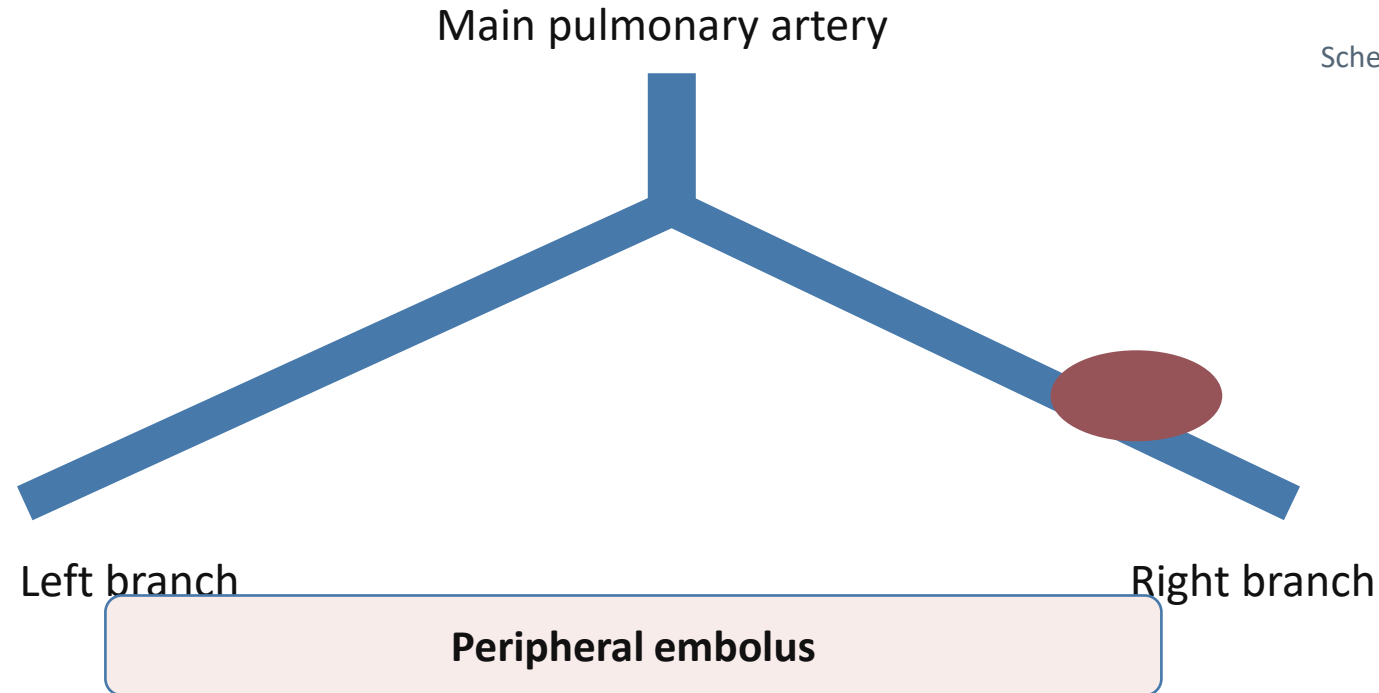
Forms in situ; Uncommon in
major vessels

- Blood-clot emboli account for the overwhelming majority of large pulmonary artery occlusions.
- Nonthrombotic emboli can comprise fat, air, bone marrow, or tumor material.
- Different embolus composition should not be confused with a routine venous thrombus.

Primary pulmonary arterial thrombosis

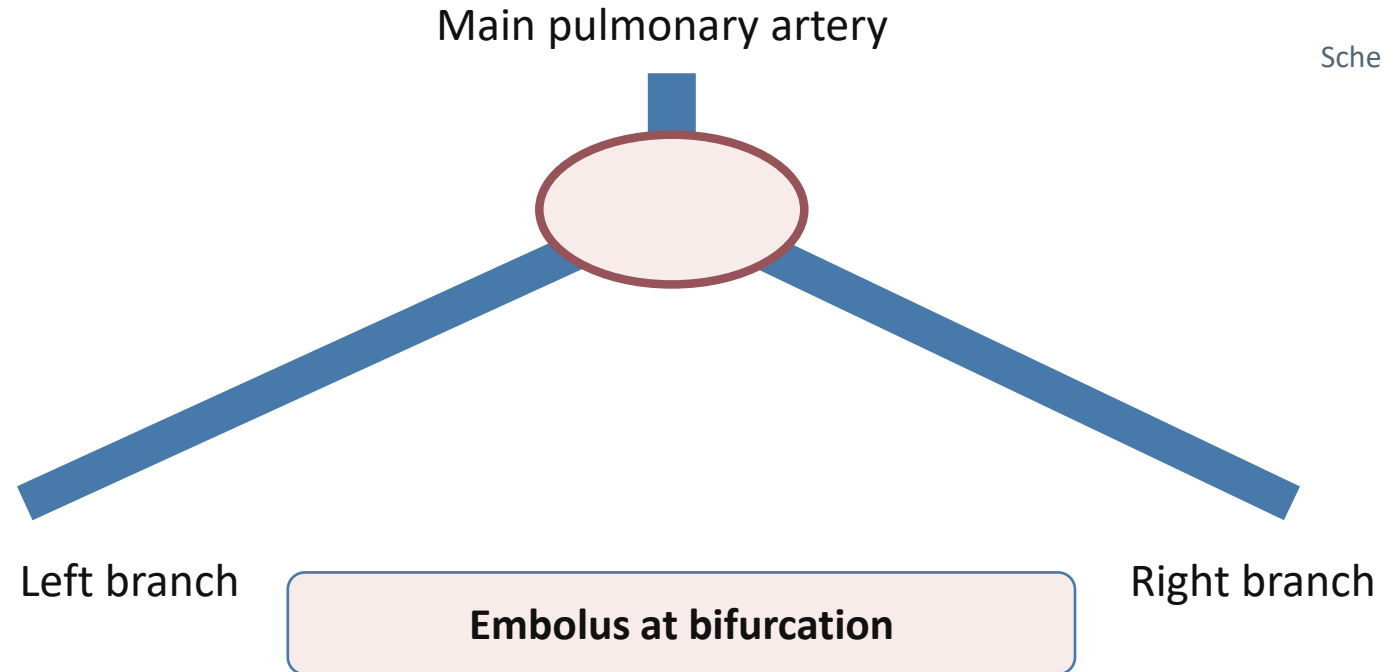
- Large-vessel pulmonary thrombosis formed in situ is unusual.
- The source associates it particularly with pulmonary hypertension and heart failure.
- An arterial filling defect should not automatically be labeled an in-situ clot.

Anatomy determines embolus lodging



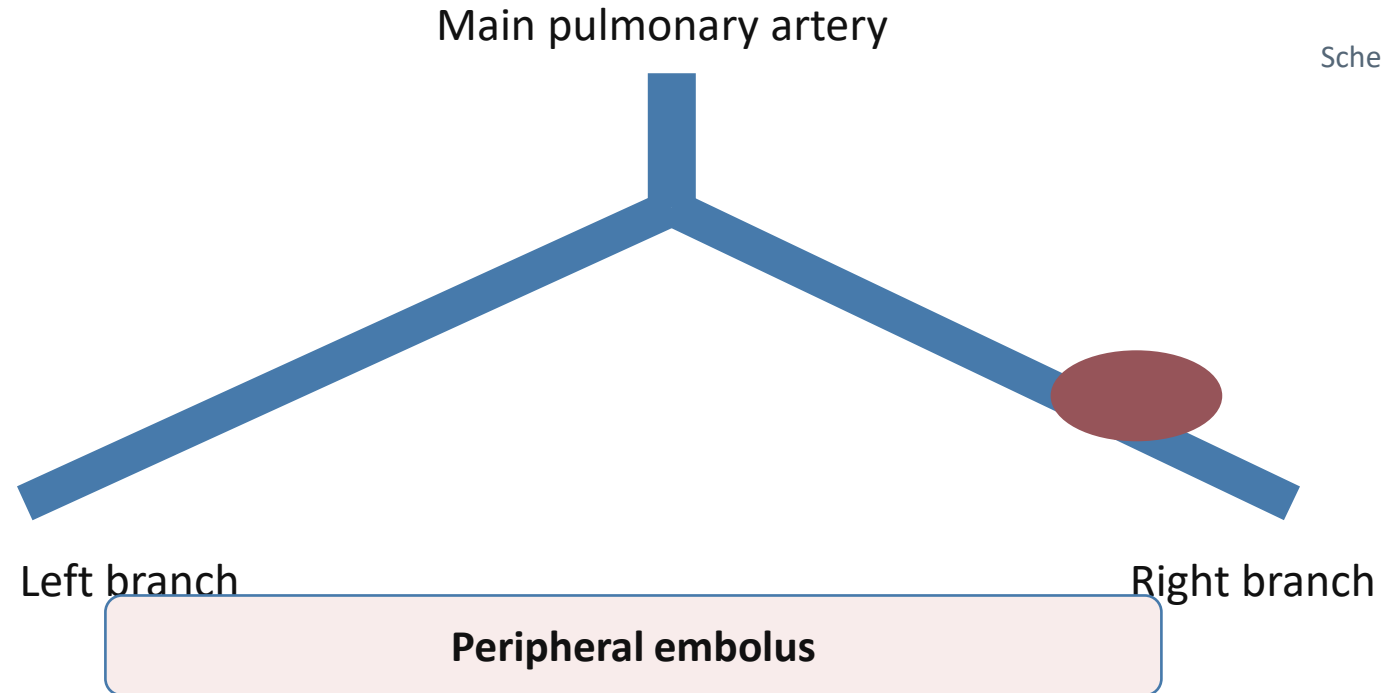
- A larger embolus can remain in a main pulmonary artery or lobar branch.
- A smaller embolus may pass distally into segmental and peripheral branches.
- The distal territory determines regional ischemic and hemorrhagic injury.

Saddle pulmonary embolus



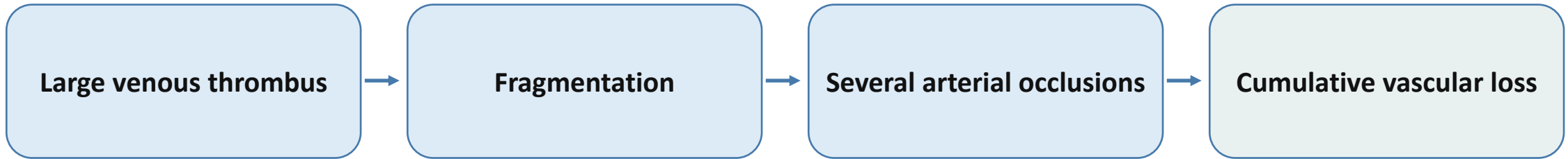
- A saddle embolus lies across the bifurcation of the main pulmonary artery.
- A large obstructive clot may severely compromise pulmonary blood flow.
- Anatomic location alone does not measure physiologic severity.

Peripheral arterial embolization



- Distal emboli obstruct smaller vessels supplying limited pulmonary territories.
- Peripheral embolization may produce focal hemorrhage or infarction.
- The accompanying pleural response helps explain pleuritic symptoms.

Emboli may be multiple

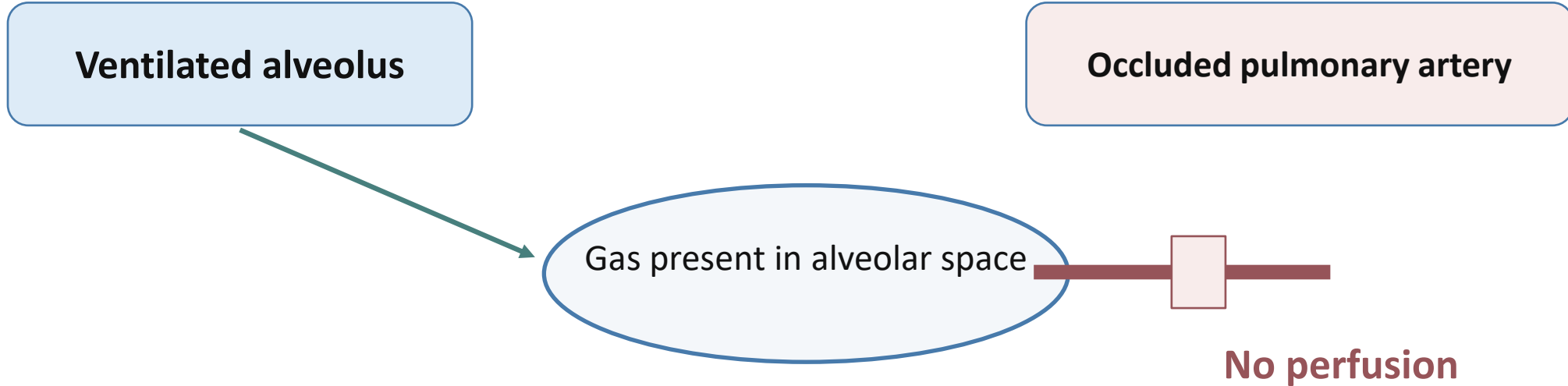


- A thrombus may fragment into several emboli.
- Emboli can arrive together or occur as repeated episodes.
- The aggregate obstruction can be more important than any single fragment.

Four determinants of physiologic severity

- Extent of pulmonary arterial blood-flow obstruction.
- Size and location of affected vessels.
- Number and timing of emboli.
- Pre-existing cardiopulmonary functional reserve.

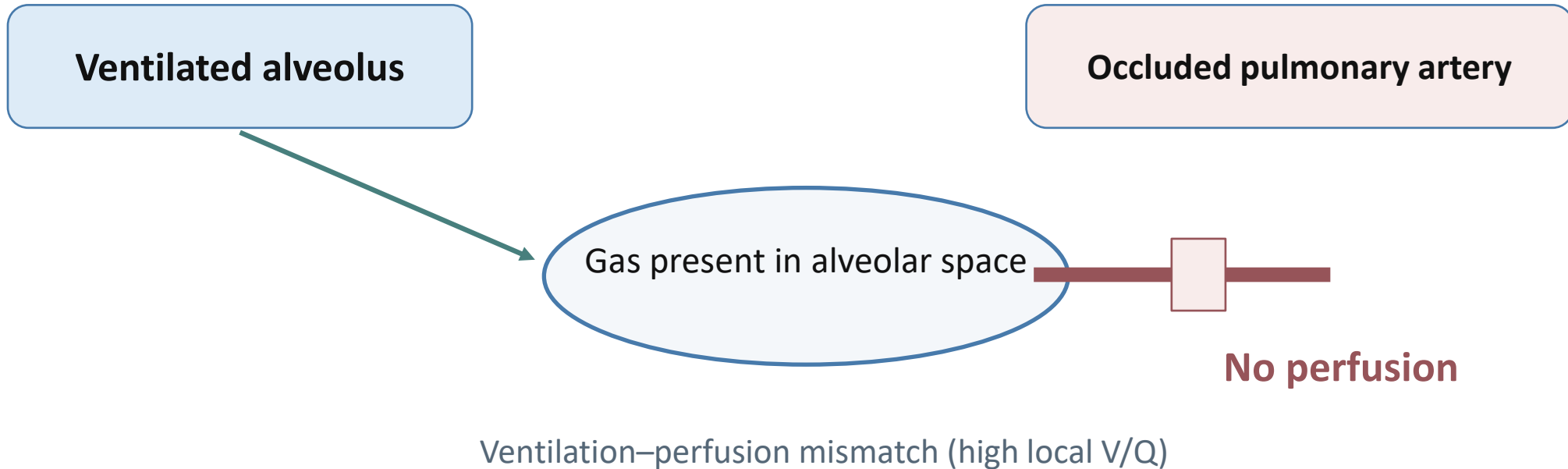
Ventilation–perfusion mismatch



Ventilation–perfusion mismatch (high local V/Q)

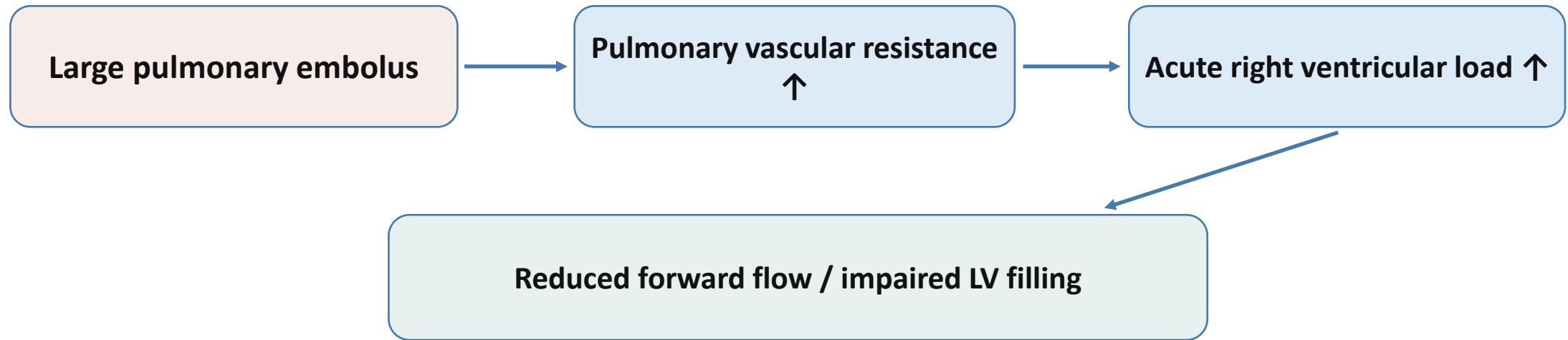
- Air may reach alveoli despite interruption of pulmonary blood flow.
- This generates ventilated but inadequately perfused lung units.
- The resulting respiratory impairment contributes to dyspnea.

The nonperfused alveolus



- Pulmonary arterial obstruction interrupts local capillary inflow.
- Alveolar ventilation alone cannot achieve normal oxygen transfer without perfusion.
- This explains a major respiratory consequence of pulmonary embolism.

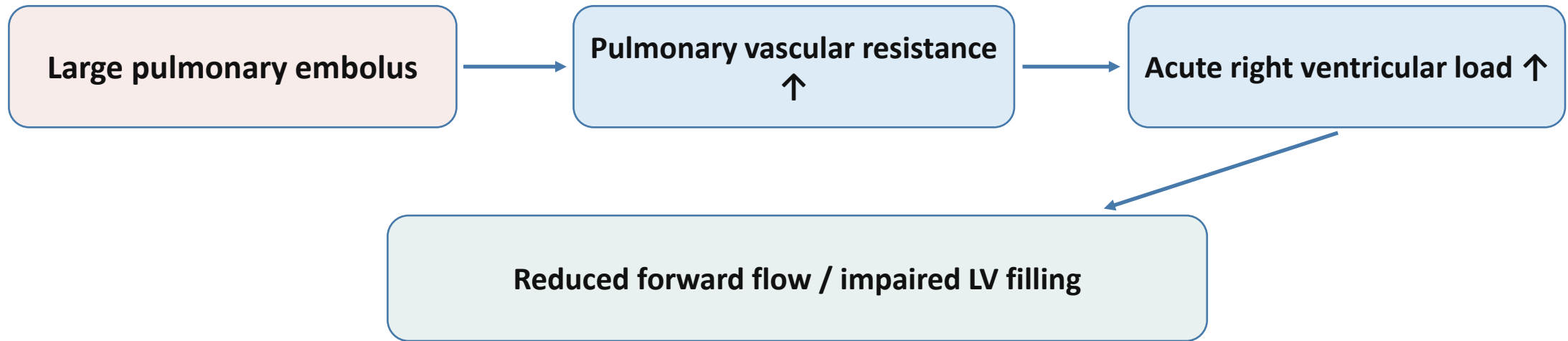
Hemodynamic effect: Increased resistance



Potential endpoint: Obstructive shock or acute cor pulmonale

- Embolic obstruction reduces the cross-sectional area available for pulmonary blood flow.
- Pulmonary vascular resistance increases.
- The right ventricle must eject against an acutely greater load.

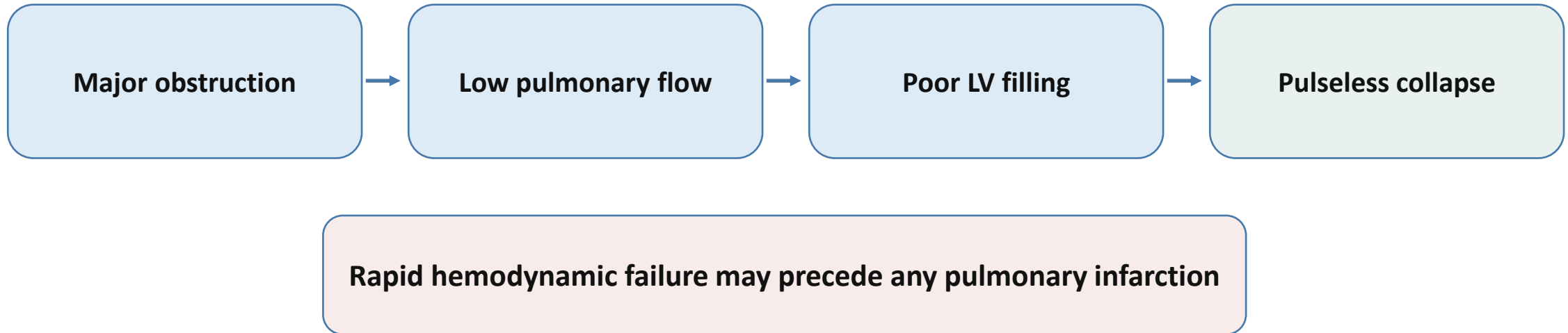
Acute right ventricular failure



Potential endpoint: Obstructive shock or acute cor pulmonale

- A large obstruction can overwhelm right ventricular reserve.
- Acute cor pulmonale denotes acute right-heart dysfunction due to pulmonary disease or vascular load.
- Decreased cardiac output may culminate in obstructive shock.

Mechanism of sudden collapse

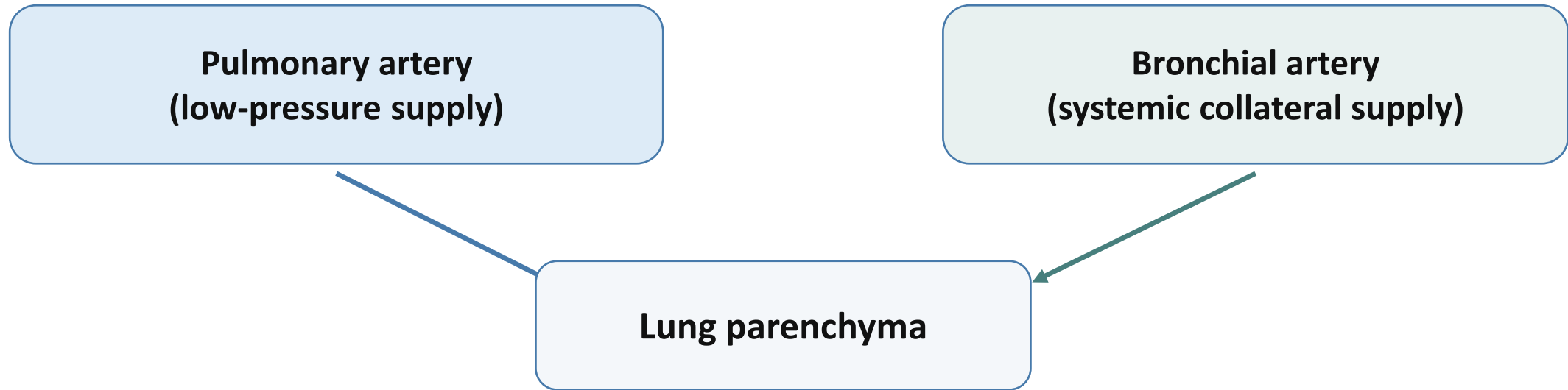


- Extensive pulmonary arterial blockage may abruptly curtail forward blood flow.
- A perfusing pulse can disappear even while organized electrical cardiac activity persists.
- The source describes this clinical scenario as electromechanical dissociation.

Cardiopulmonary reserve modifies injury

- A similar embolic burden may be tolerated differently across patients.
- Cardiac or pulmonary disease can magnify hemodynamic compromise.
- Reduced systemic perfusion also favors parenchymal infarction.

Why the lung resists infarction



Failure of one arterial inflow does not always produce tissue death

- Pulmonary parenchyma receives both pulmonary arterial and bronchial arterial inflow.
- The bronchial circulation can sustain tissue when a pulmonary arterial branch is blocked.
- Therefore pulmonary embolism can produce hemorrhage without necrosis.

Pulmonary hemorrhage $\neq$ pulmonary infarction

Pulmonary embolus

Arterial occlusion

Hemorrhage

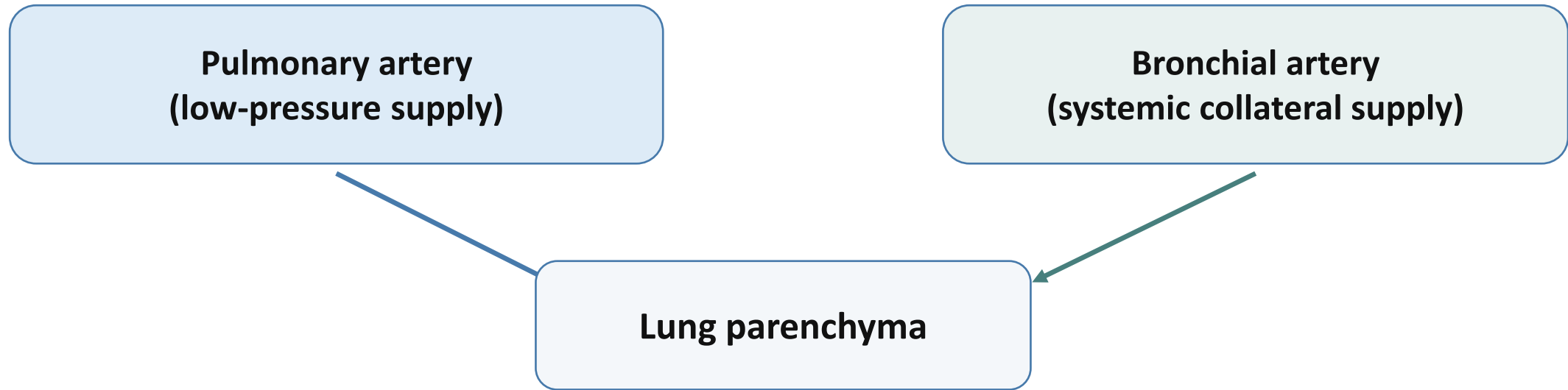
Blood extravasation; Tissue may survive

Infarction

Ischemic necrosis with hemorrhage

- Hemorrhage indicates escape of blood into pulmonary tissue or airspaces.
- An infarct requires ischemic necrosis of supporting structures.
- Collateral perfusion helps explain why these may occur separately.

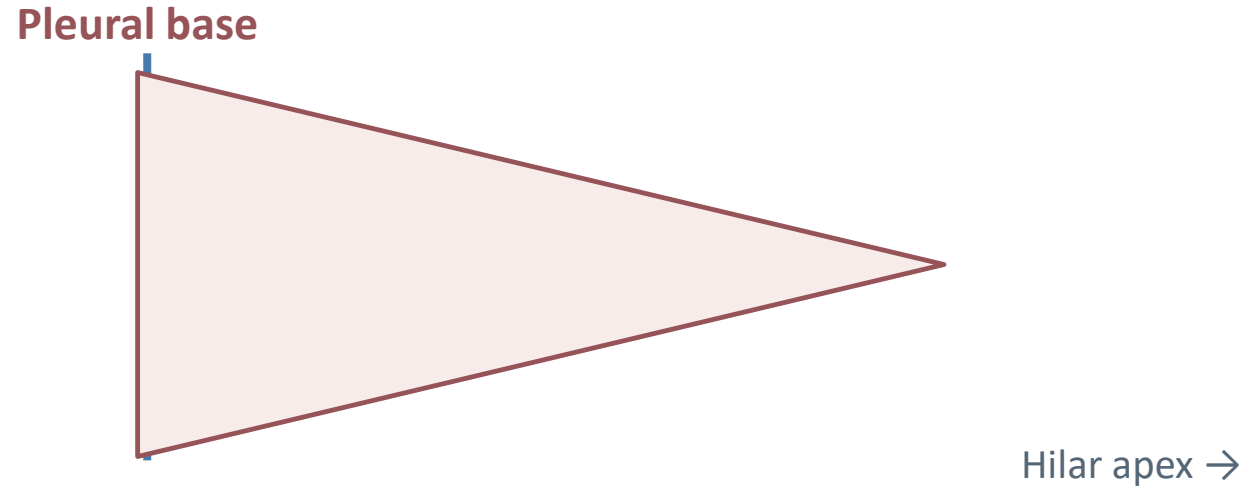
When does infarction become likely?



Failure of one arterial inflow does not always produce tissue death

- Pre-existing cardiovascular compromise reduces the compensatory supply.
- Heart and lung disease lower the ability to sustain the affected region.
- Smaller peripheral occlusions can produce localized infarction.

Gross pattern: Pleura-based wedge



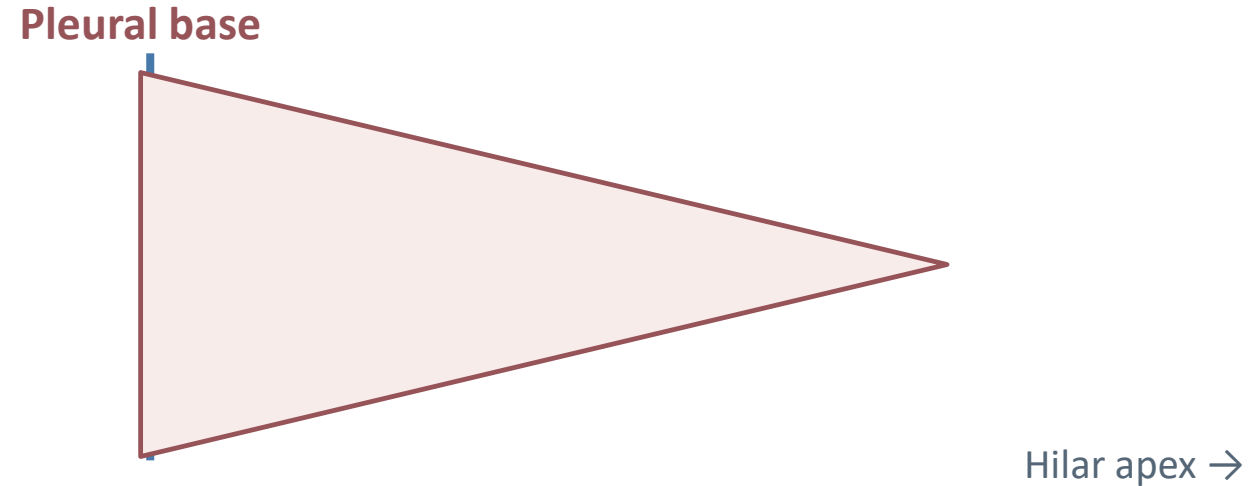
Peripheral, pleura-based wedge (schematic)

- The infarct classically extends to the pleura as a wedge.
- The apex points toward the pulmonary hilum and may contain an occluded vessel.
- The pleural surface may develop a fibrinous exudate.

Distribution and multiplicity

- The source describes a predilection for lower lobes.
- Multiple infarcts may be present in a single patient.
- Infarct size ranges from minute lesions to extensive lobar involvement.

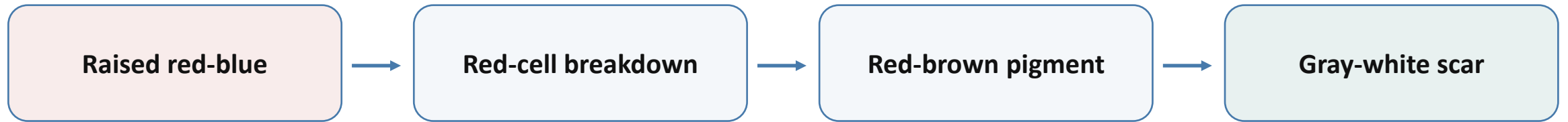
Early gross appearance



Peripheral, pleura-based wedge (schematic)

- An acute pulmonary infarct is typically hemorrhagic.
- It initially appears as a raised red-blue area.
- The gross appearance reflects ischemic tissue damage accompanied by hemorrhage.

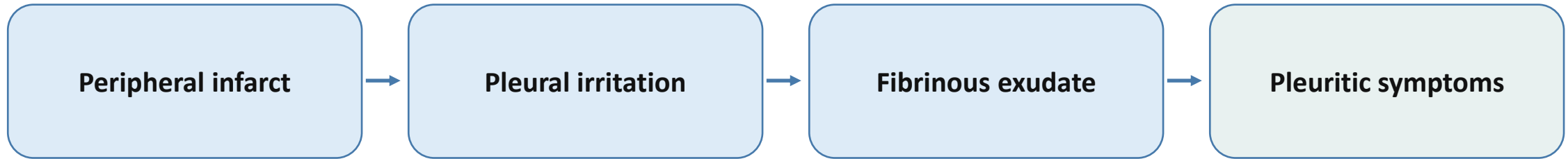
Evolution from hemorrhage to scar



The gross appearance evolves with clearance of erythrocytes and repair

- Erythrocytes begin to lyse and color becomes paler.
- Hemosiderin accumulation can produce a red-brown appearance.
- Peripheral fibrosis progresses toward a contracted scar.

Fibrinous pleuritis



- Pleura overlying an infarct can be coated by fibrinous inflammatory exudate.
- This accounts for pleuritic pain and sometimes a friction rub.
- Pleural inflammation is not an obligatory feature of every embolus.

Microscopy: Ischemic necrosis



Ischemic alveolar-wall necrosis

Associated vascular and bronchiolar injury

Conceptual tissue-architecture schematic; Not a histologic section

- Infarcted pulmonary tissue shows necrosis of alveolar walls.
- Bronchioles and small vessels within the region also undergo ischemic necrosis.
- Hemorrhage is a characteristic accompaniment of pulmonary infarction.

What distinguishes necrosis from hemorrhage?

Hemorrhage

Extravasated erythrocytes

True infarct

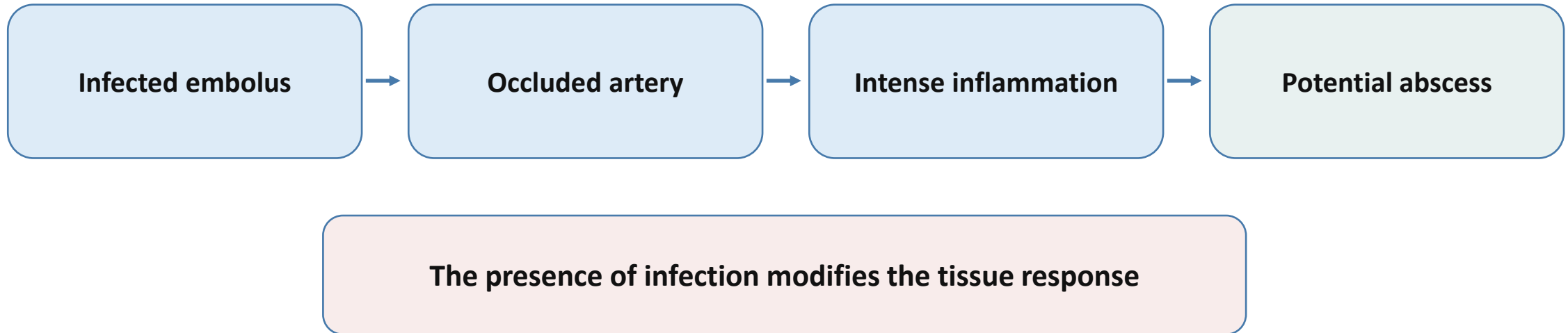
Necrotic alveolar walls and
vessels

Interpret together

Vascular blockage + tissue
response

- Blood in airspaces alone does not establish infarction.
- Loss of viable alveolar-wall architecture indicates ischemic tissue death.
- Assess vessel occlusion and the geographic relationship of tissue injury.

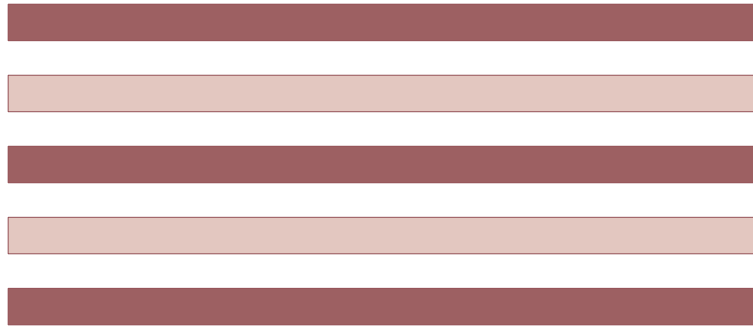
Infected emboli and septic infarcts



- An infected embolus can cause ischemic injury with marked neutrophilic inflammation.
- Such a lesion is described as a septic infarct.
- Some septic infarcts develop into pulmonary abscesses.

Antemortem embolus versus postmortem clot

Antemortem thrombus



Lines of Zahn: Laminated layers

Postmortem clot



Gelatinous; “chicken-fat” / red layering

- An antemortem thrombus can contain laminations known as lines of Zahn.
- Postmortem clots are typically gelatinous and exhibit dependent red-cell settling.
- Morphology helps distinguish a vital thrombotic event from a postmortem artifact.

Clinical manifestations follow clot burden

Uncomplicated / small embolus

Often silent or transient symptoms

Infarction

Pleuritic pain, hemoptysis, focal necrosis

Large obstruction

Shock, acute RV failure, sudden death

- Most small emboli are clinically silent or produce transient symptoms.
- Intermediate embolic events may cause dyspnea, cough, or pleuritic pain.
- Massive obstruction can produce shock, acute cor pulmonale, or sudden death.

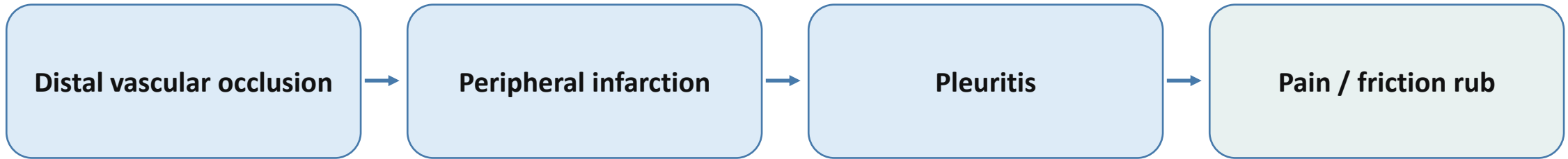
Small emboli: Clinically quiet events

- Small emboli may produce no recognizable clinical syndrome.
- Some cause transient chest pain or cough.
- Even a minor embolus may herald a later, more serious episode.

Symptomatic pulmonary embolism

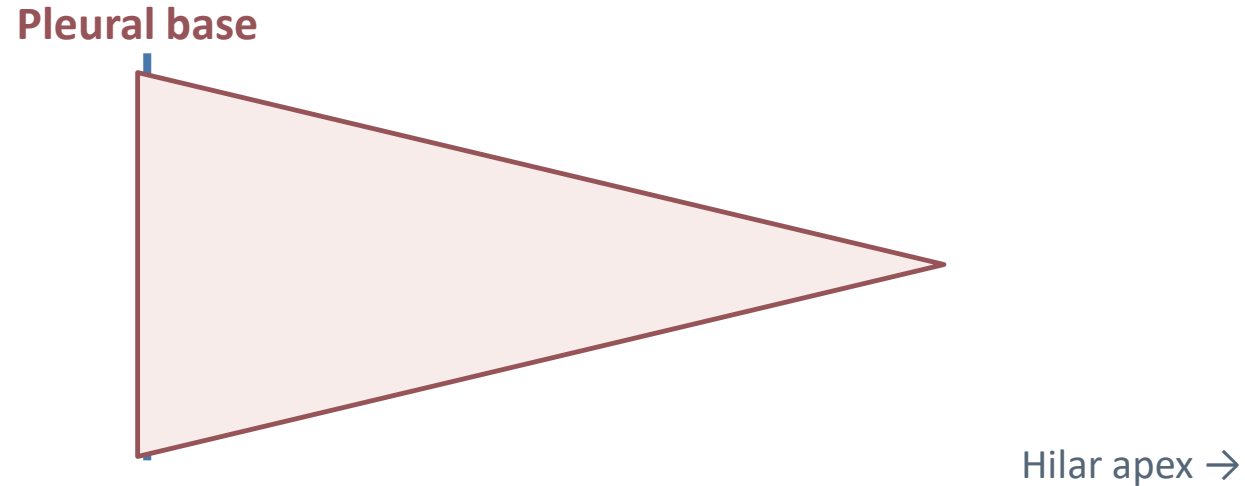
- Dyspnea is a leading presenting symptom.
- Pleuritic chest pain and cough are frequent associated complaints.
- Severity of symptoms does not by itself identify the anatomic size of the thrombus.

Pleuritic pain: Mechanism



- A peripheral infarct can inflame the adjacent pleura.
- Pleuritic pain is typically accentuated by respiration.
- A pleural friction rub may be audible when fibrinous pleuritis is present.

Why hemoptysis may occur



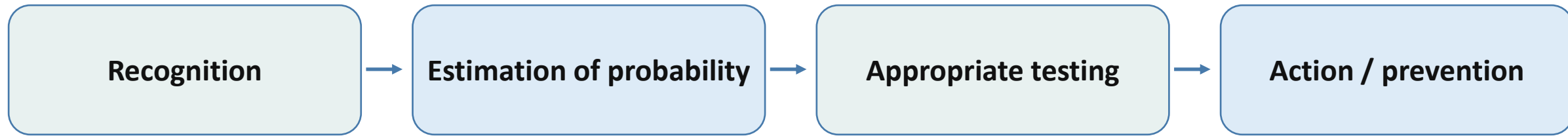
Peripheral, pleura-based wedge (schematic)

- Peripheral embolic injury often produces hemorrhage.
- Hemorrhagic infarction may release blood into the airways.
- Fever and hemoptysis support tissue injury but are not specific diagnostic tests.

Leg symptoms and source identification

- Calf or thigh swelling and pain can accompany pulmonary embolism.
- Absence of these symptoms does not exclude a deep venous source.
- Ultrasound can identify DVT when clinically indicated.

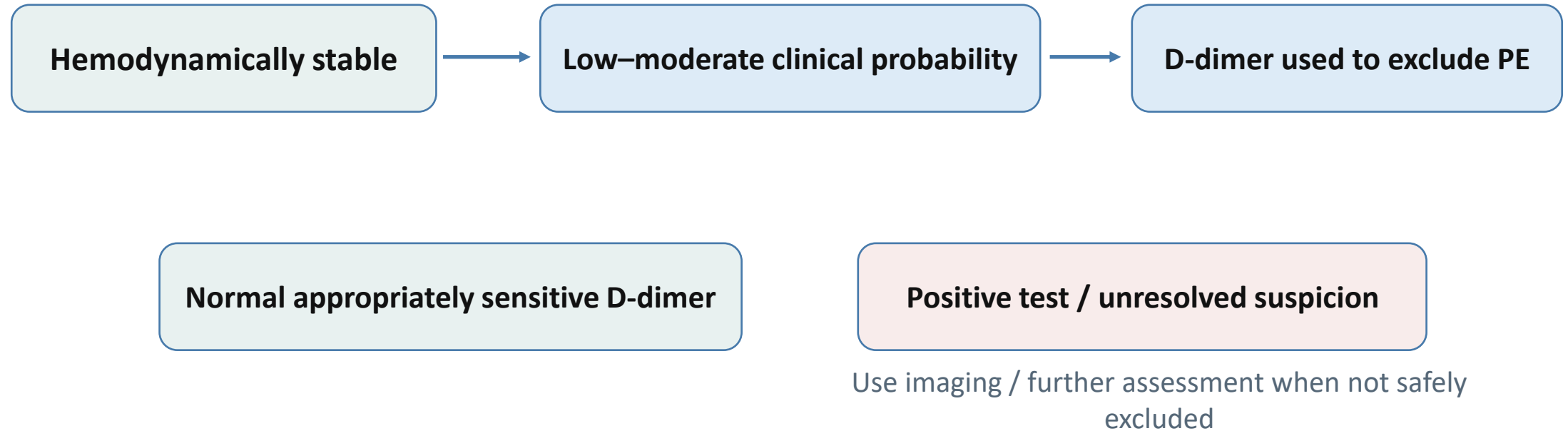
Diagnostic logic: Suspicion to confirmation



Clinical and morphologic information answer different questions

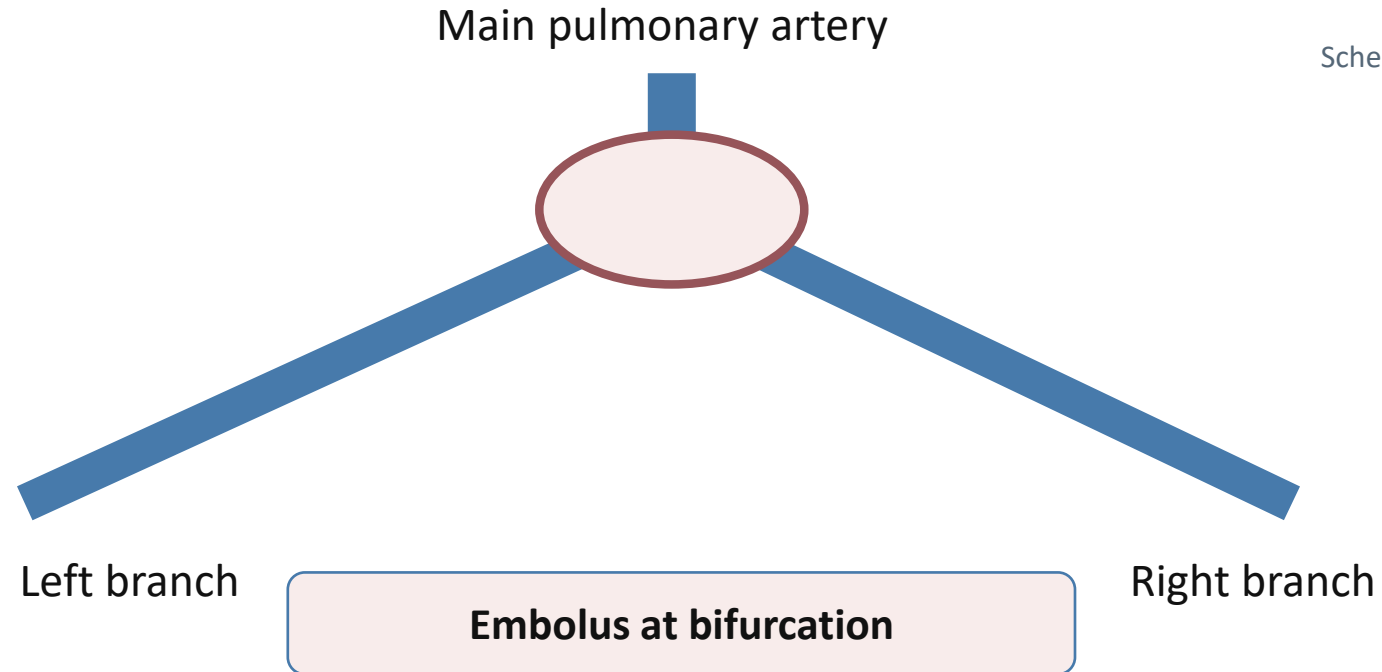
- Clinical risk and hemodynamic stability shape the testing pathway.
- D-dimer may help exclude disease in selected stable, lower-risk patients.
- CT pulmonary angiography usually demonstrates intraluminal arterial obstruction.

D-dimer: A contextual rule-out test



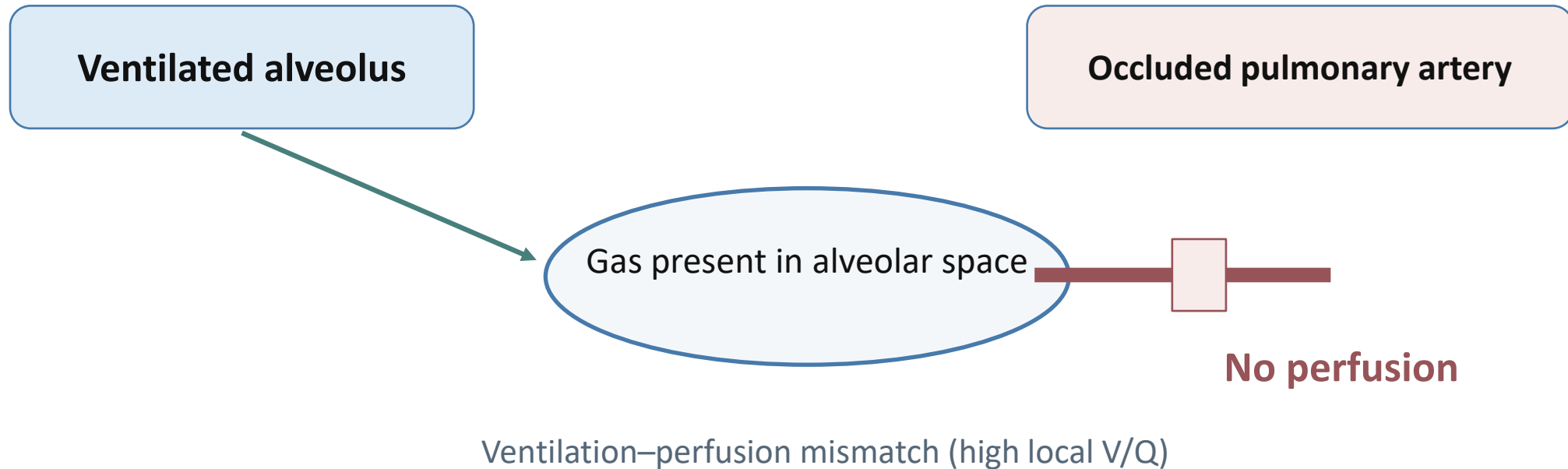
- The source emphasizes its use in hemodynamically stable, low-to-moderate-risk patients.
- A suitably normal result can make pulmonary embolism unlikely in that setting.
- An elevated D-dimer is not specific proof of pulmonary embolism.

CT pulmonary angiography



- The source describes CT pulmonary angiography as the usual definitive test.
- Imaging demonstrates filling defects or obstruction within pulmonary arteries.
- Assessing location and extent complements the clinical hemodynamic assessment.

Ventilation–perfusion imaging

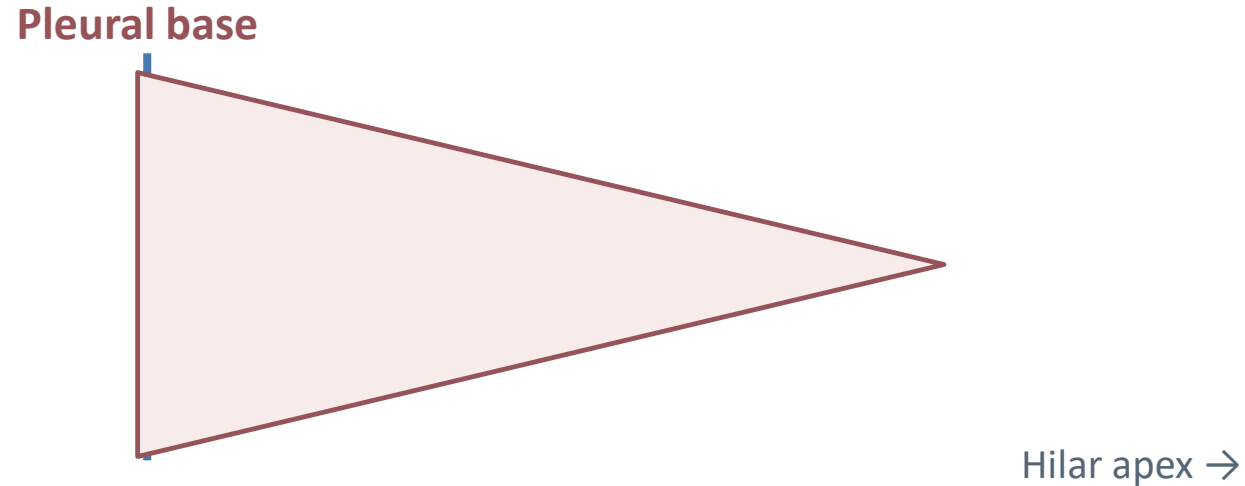


- Ventilation–perfusion scanning is an alternative diagnostic approach.
- Its central logic compares ventilation and regional pulmonary perfusion.
- The source notes that this is required only in a minority of diagnostic pathways.

Duplex ultrasonography

- Venous duplex ultrasound can identify a deep venous thrombus.
- It investigates a potential source of thromboembolism.
- A negative leg study alone should not be treated as proof that PE is absent.

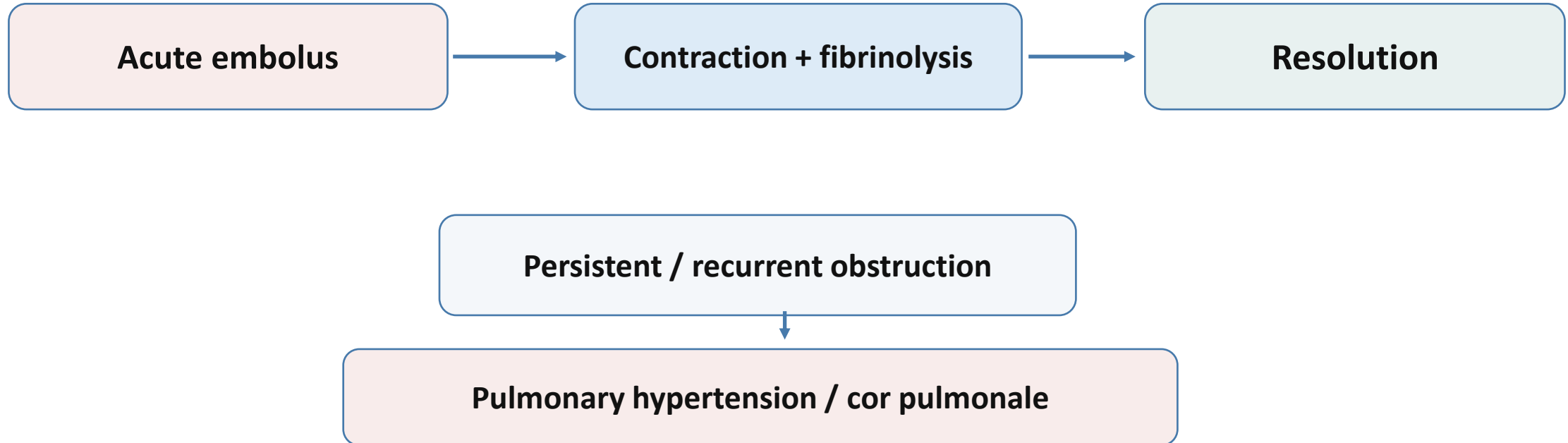
Chest radiography and pulmonary infarction



Peripheral, pleura-based wedge (schematic)

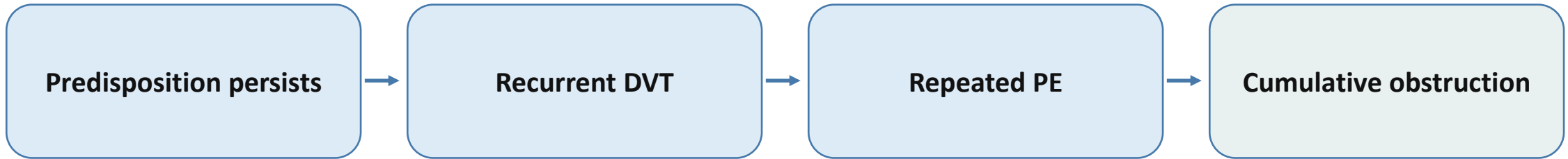
- Plain chest radiography can be normal in pulmonary embolism.
- An infarct may later appear as a peripheral, wedge-shaped infiltrate.
- This radiographic finding is less direct than imaging the obstructed artery.

Evolution of an embolus



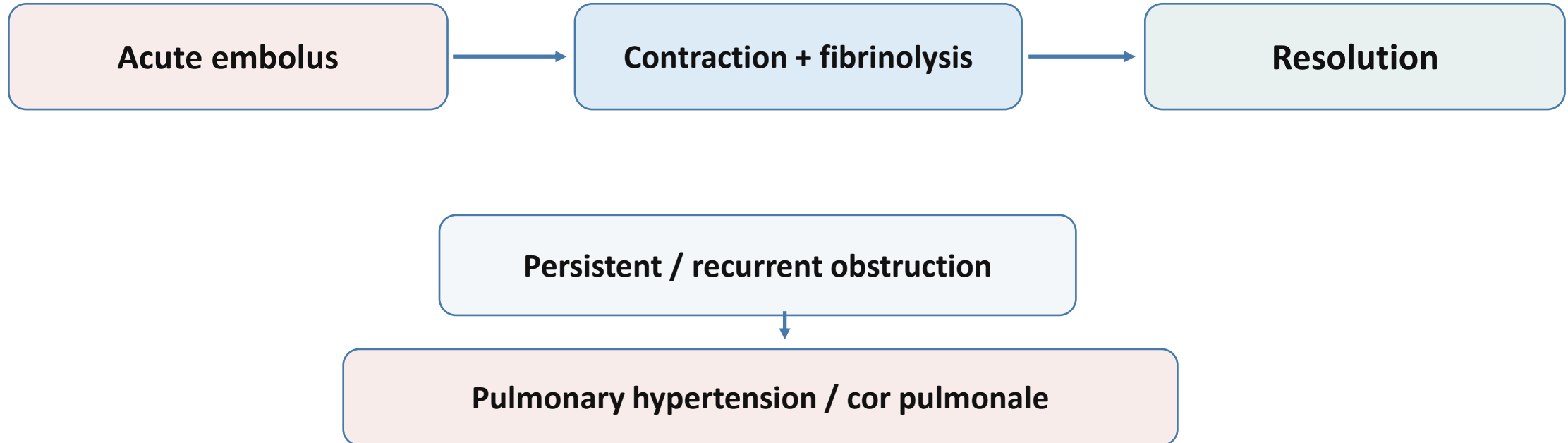
- After the acute event, emboli may contract and undergo fibrinolysis.
- Complete resolution is possible, particularly in younger patients.
- Persistent or repeated obstruction creates a different chronic vascular burden.

Recurrent embolization



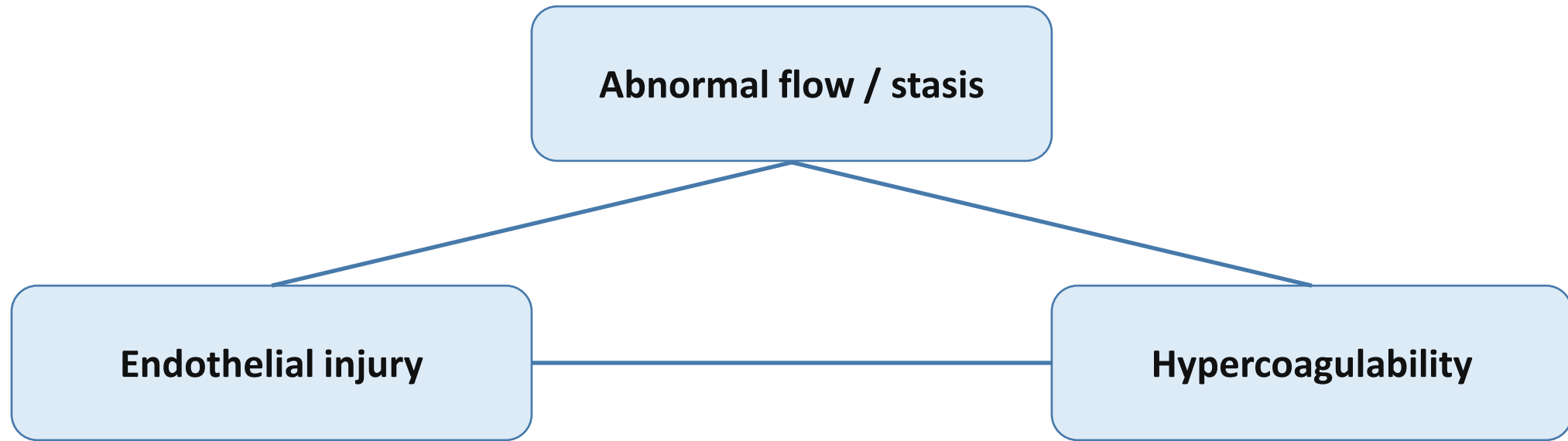
- A previous embolus indicates increased vulnerability when a provoking condition persists.
- Several small events can cumulatively reduce the pulmonary vascular bed.
- Recurrence prevention is important even after a relatively mild episode.

Chronic vascular sequelae



- Persistent and recurrent emboli may increase pulmonary vascular resistance.
- Chronic pulmonary hypertension can result.
- Advanced disease may cause chronic right-sided heart failure (cor pulmonale).

Primary prevention targets thrombosis



Convergent mechanisms promoting venous thrombus formation

- Early ambulation reduces venous stasis in relevant postoperative and postpartum settings.
- Mechanical compression is an option in selected immobilized patients.
- Anticoagulant prophylaxis can be used for individuals at increased thrombotic risk.

Anticoagulation and supportive care

- Anticoagulation is a central treatment component in the source.
- Supportive measures address respiratory and hemodynamic compromise.
- Anticoagulation primarily limits new clot propagation rather than immediately dissolving the embolus.

Thrombolysis: Benefit and harm

Clinical problem

Severe obstructive physiology

Potential benefit

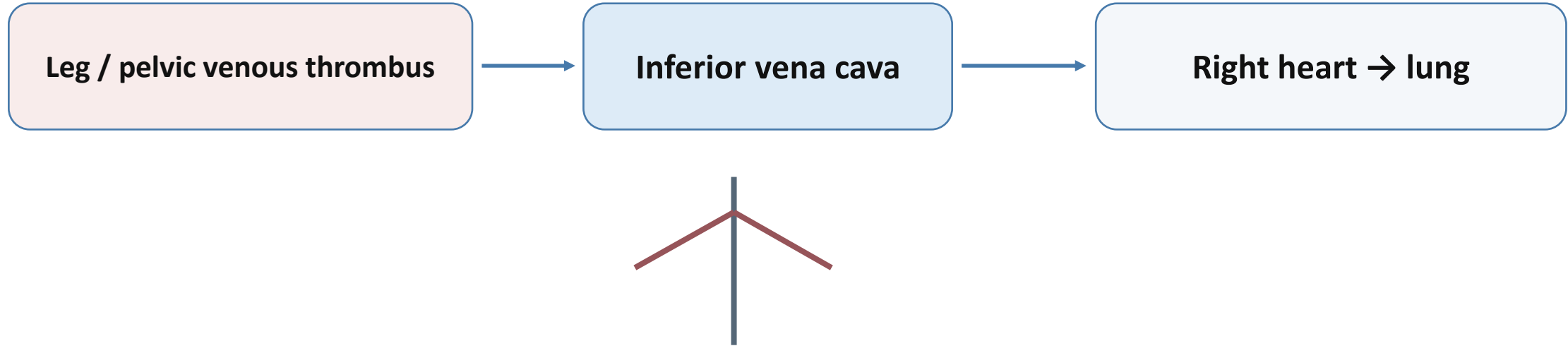
Rapid reduction of thrombus
burden

Major hazard

Serious hemorrhagic
complications

- Thrombolytic treatment may be considered in severe complications, such as shock.
- The intended effect is accelerated dissolution of obstructing thrombus.
- Major bleeding is a potentially serious adverse consequence.

Inferior vena cava filter: Restricted role



Filter intercepts embolizing thrombi; Does not treat the DVT itself

- A filter may intercept embolizing thrombi before they reach the pulmonary arteries.
- The text discusses its use when recurrent PE risk persists but anticoagulation is contraindicated.
- The device does not correct the underlying hypercoagulability or clear established emboli.

Three pathophysiologic patterns

Uncomplicated / small embolus

Often silent or transient symptoms

Infarction

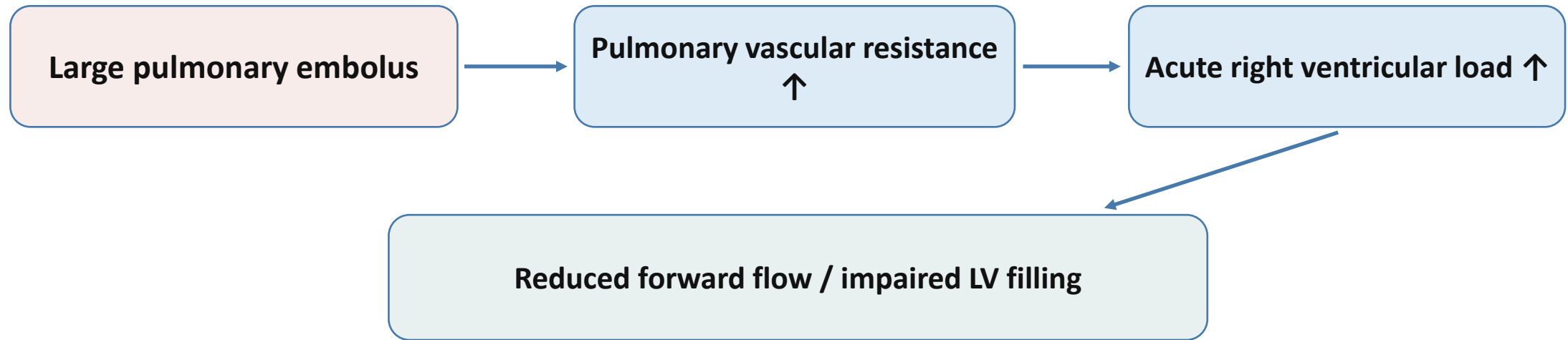
Pleuritic pain, hemoptysis, focal necrosis

Large obstruction

Shock, acute RV failure, sudden death

- Silent PE: Small embolic obstruction without major symptoms.
- Hemorrhagic infarction: Distal embolus plus insufficient collateral perfusion.
- Circulatory collapse: Extensive obstruction with acute RV failure or low pulmonary flow.

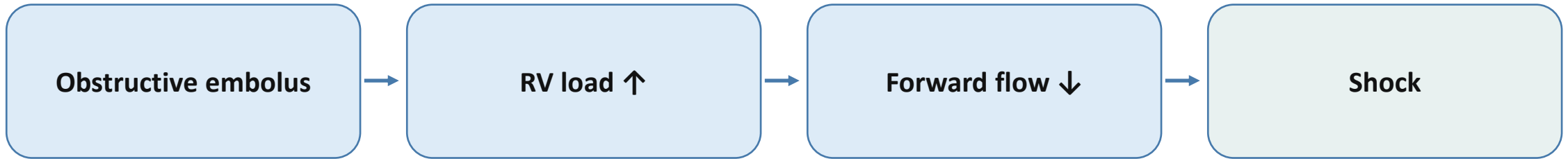
Clinicopathologic case: Sudden collapse



Potential endpoint: Obstructive shock or acute cor pulmonale

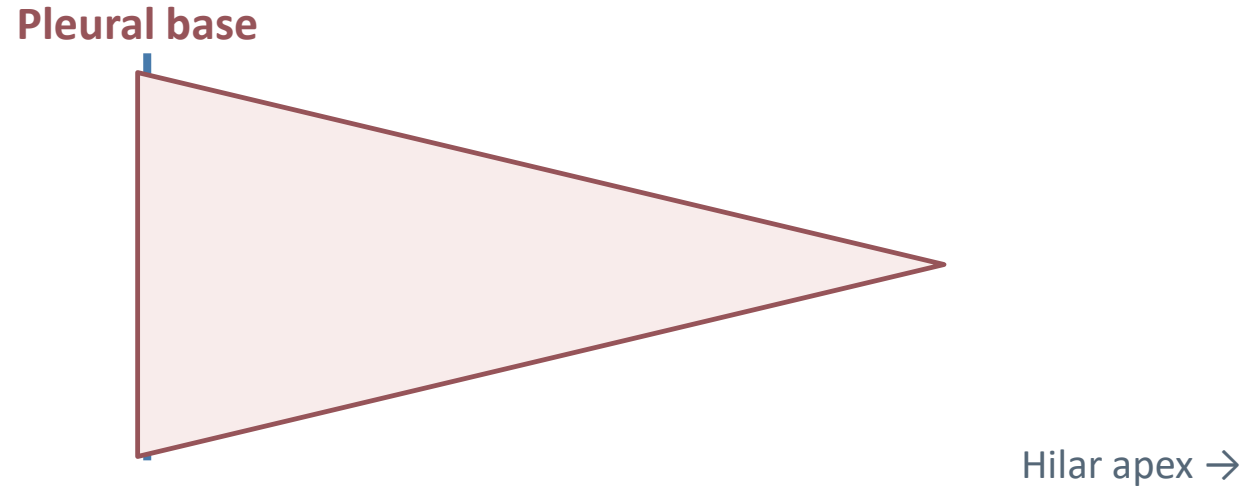
- A postoperative immobilized patient suddenly develops severe dyspnea and shock.
- Examination reveals profound circulatory failure without a preceding focal lung syndrome.
- Question: Which pathophysiologic consequence is most likely dominant?

Case resolution: Massive vascular obstruction



- Acute obstruction sharply increases pulmonary vascular resistance.
- Right-heart output and systemic circulation may fail within minutes.
- The fatal process may occur without time for morphologically evident infarction.

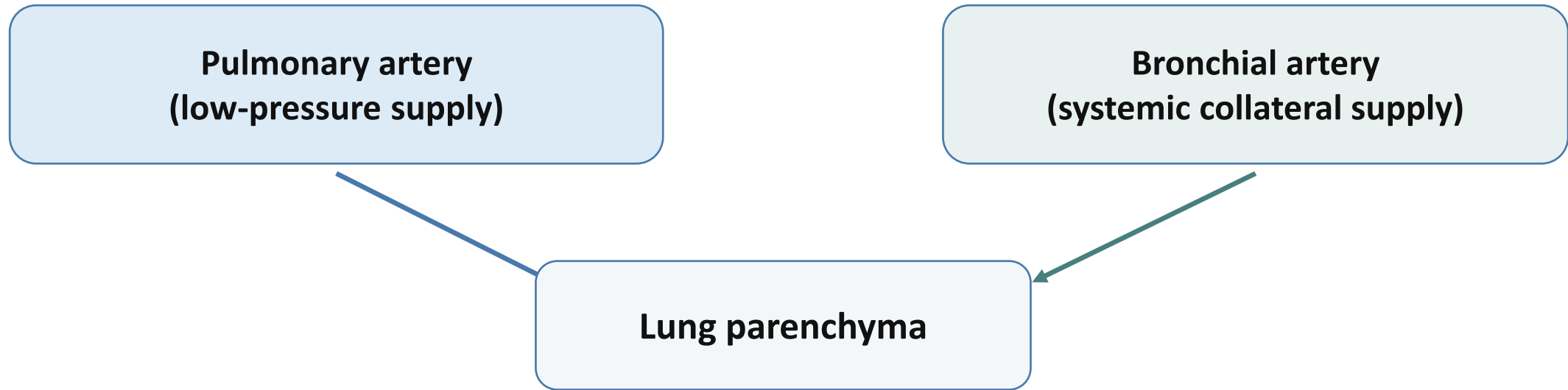
Clinicopathologic case: Pleuritic pain



Peripheral, pleura-based wedge (schematic)

- A patient with cardiac dysfunction develops pleuritic chest pain and hemoptysis.
- A peripheral pleura-based wedge-shaped pulmonary lesion is identified.
- Question: Why is tissue necrosis more likely in this patient?

Case resolution: Failed collateral protection



Failure of one arterial inflow does not always produce tissue death

- The pulmonary arterial branch is blocked by an embolus.
- Compromised cardiovascular function limits compensatory bronchial perfusion.
- Ischemic necrosis with hemorrhage explains the pleura-based wedge lesion.

Morphology checklist for an infarct

- Identify a peripheral hemorrhagic wedge with its apex toward the hilum.
- Assess a vessel near the apex for occluding thromboembolic material.
- Confirm ischemic necrosis of alveolar walls, bronchioles, or small vessels.
- Note overlying fibrinous pleuritis and evolution toward fibrosis when present.

High-yield clinicopathologic synthesis

Uncomplicated / small embolus

Often silent or transient symptoms

Infarction

Pleuritic pain, hemoptysis, focal necrosis

Large obstruction

Shock, acute RV failure, sudden death

- The primary source of thromboemboli is venous thrombosis.
- Obstruction produces both ventilation–perfusion mismatch and increased vascular resistance.
- Dual pulmonary/bronchial supply explains why infarction is not inevitable.
- Bleeding, necrosis, acute right-heart failure, and chronic pulmonary hypertension reflect distinct outcomes.

References and image provenance

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th edition (2026). Chapter 15: The Lung, pp. 639–640.
- Background: Chapter 4, Hemodynamic Disorders, Thromboembolic Disease, and Shock, pp. 112–115.
- All displayed vascular and tissue diagrams are original, editable PowerPoint schematics; no clinical images are presented as specimens.