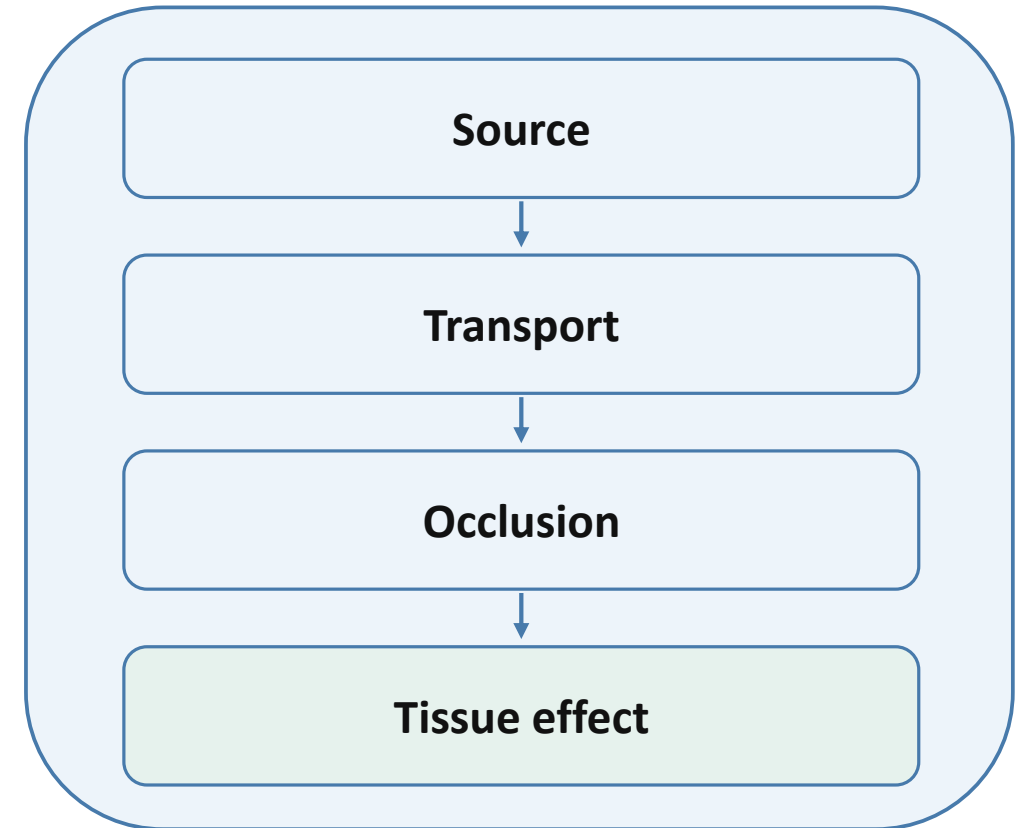


Embolism: pathologic transport and vascular injury

A detached intravascular mass travels to an anatomically distant vascular bed.

- Most emboli are thrombotic fragments, but solid, liquid, and gaseous emboli also occur.
- Clinical injury reflects the destination, vessel caliber, and remaining perfusion.



Dr Abu Suraih Sakhri

MBBS, MD (Pathology)

Schematic: source → transport → vascular lodging → downstream injury.
www.abusuraihsakhri.com

Learning objectives

By the end, distinguish routes, compositions, and consequences of embolization.

- Explain the difference between thrombosis, embolization, and infarction.
- Trace venous, systemic arterial, and paradoxical embolic routes.
- Connect embolus type to its pathogenesis, pathology, and clinical presentation.
- Interpret focused clinicopathologic scenarios.

Mechanism

**Vascular
destination**

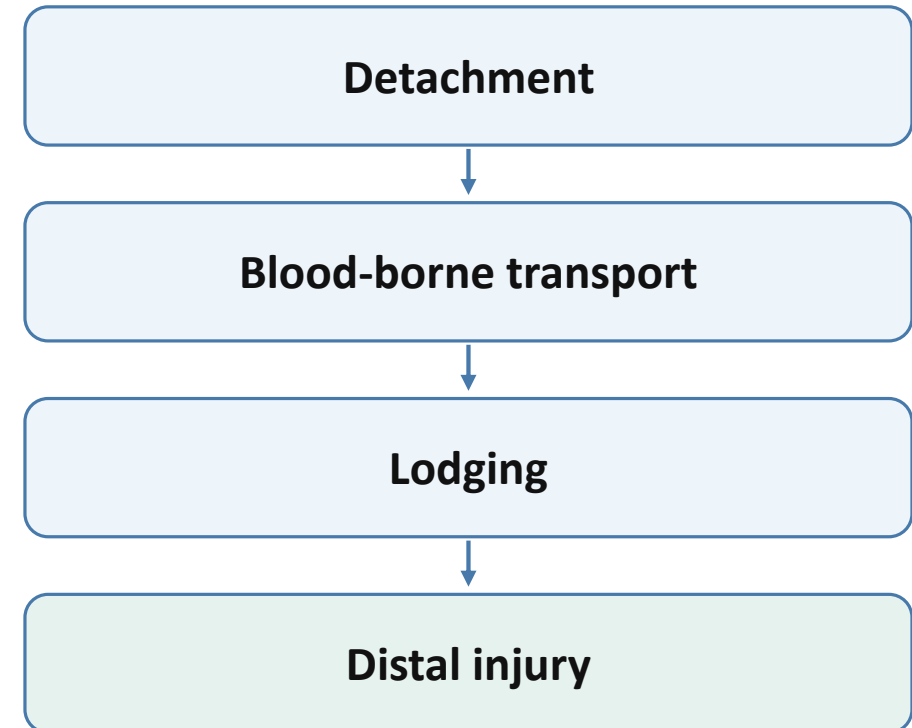
Morphology

**Clinical
consequence**

An embolus is defined by displacement

The defining event is transport away from the original location.

- The embolic material is intravascular before transport.
- Blood flow carries it from origin to a remote vessel.
- Arrest usually occurs where the vascular lumen becomes too narrow.
- Downstream consequences range from none to infarction.



Thrombus versus embolus

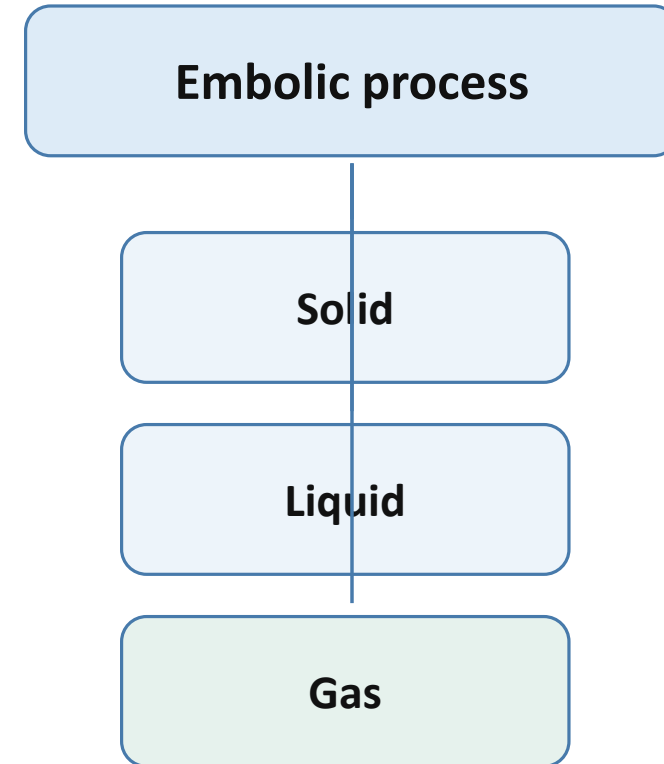
Local formation and distal transport are different events.

Feature	Thrombus	Embolus
Defining process	Forms locally	Travels from origin
Location	At formation site	Distant site of lodging
Relationship	Can embolize	May be thrombotic or not

Three physical states can embolize

The definition accommodates material beyond coagulated blood.

- Solid: Thrombi, plaque debris, tumor fragments, marrow, foreign bodies.
- Liquid: Fat droplets and amniotic fluid.
- Gas: Air entry or nitrogen coming out of solution.



Composition is not the same as destination

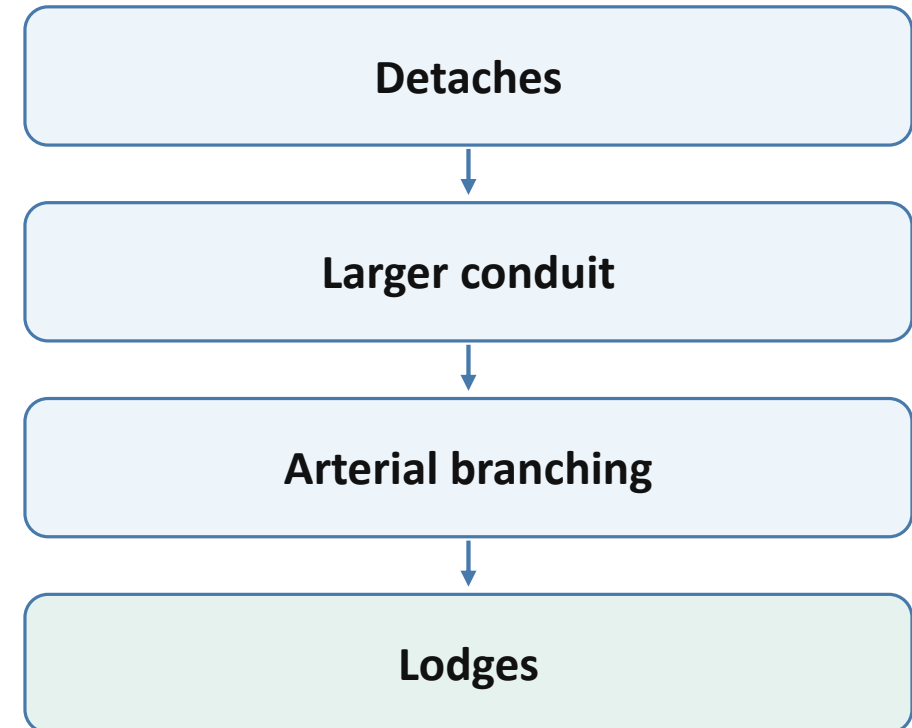
Two independent classification axes organize embolic disease.

Composition	Example	Potential destination
Solid	Venous thrombus	Pulmonary artery
Solid	Cardiac mural thrombus	Systemic artery
Liquid	Marrow fat droplets	Pulmonary microvessels
Gas	Nitrogen bubbles	Blood and tissues

Embolic material follows the blood stream

Flow anatomy determines the likely point of arrest.

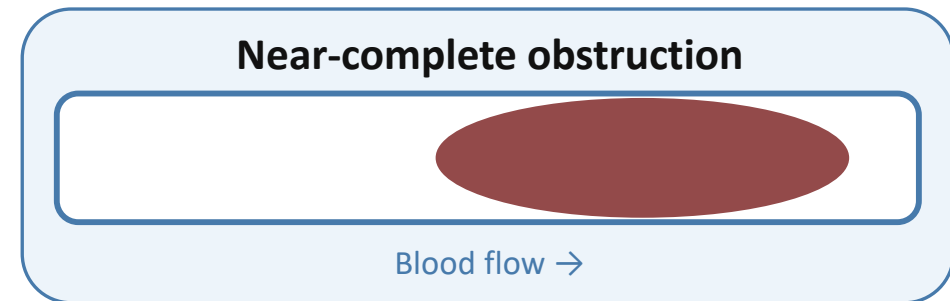
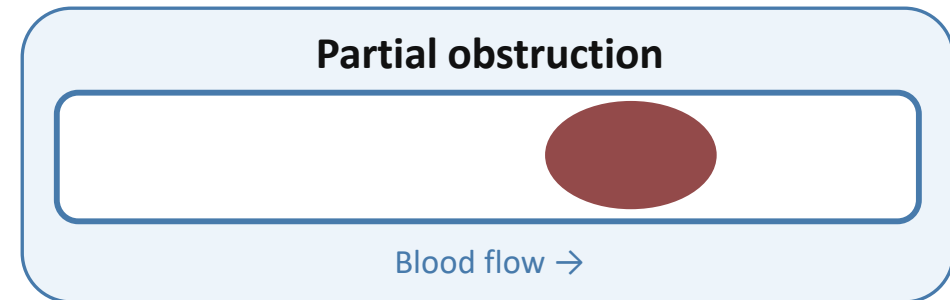
- A fragment enters progressively larger upstream channels after detachment.
- Beyond the heart, it encounters successively smaller arterial branches.
- It lodges when size and geometry prevent further passage.



Partial and severe vascular obstruction

An embolus may restrict rather than entirely abolish perfusion.

- Partial obstruction reduces cross-sectional area available for flow.
- A completely blocked channel cannot conduct flow; Parallel supply may persist.
- Organ function depends on the territory and reserve circulation.



Schematic longitudinal vessel views (not histology).

Four determinants of embolic damage

The same material can have very different clinical consequences.

- Size of the embolus relative to the recipient vessel.
- Location within the vascular tree and downstream tissue served.
- Available collateral or dual blood supply.
- Intrinsic susceptibility of the target tissue to ischemia.

Embolus size

Location

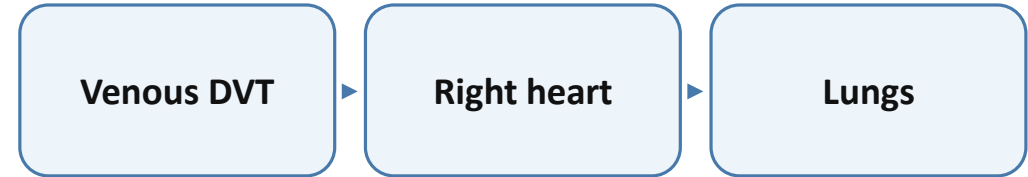
Collateral supply

Tissue vulnerability

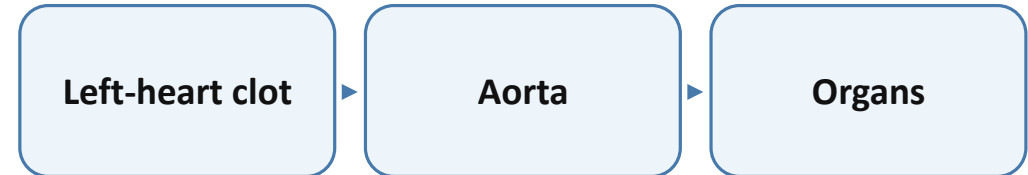
Major routes of thromboembolism

Venous and arterial emboli enter different circulatory circuits.

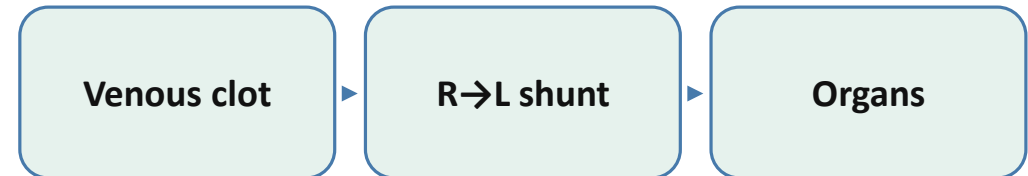
- Systemic venous thrombus → right heart → pulmonary arteries.



- Left-sided cardiac or aortic thrombus → systemic arterial branches.



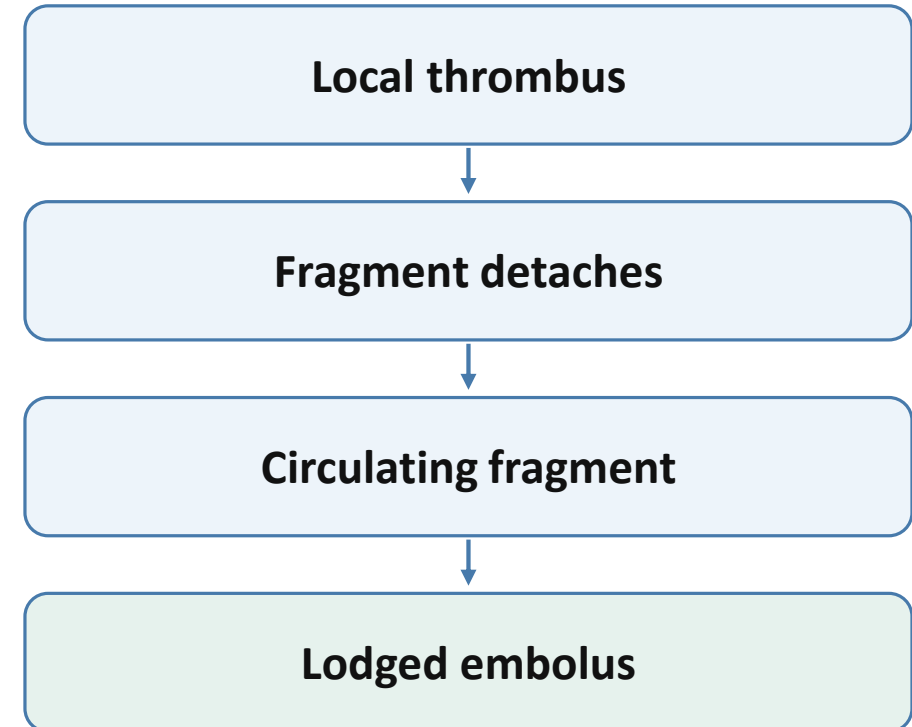
- Rare right-to-left shunt permits paradoxical systemic embolization.



Thromboemboli predominate

Detachment of pre-existing thrombi accounts for most embolic events.

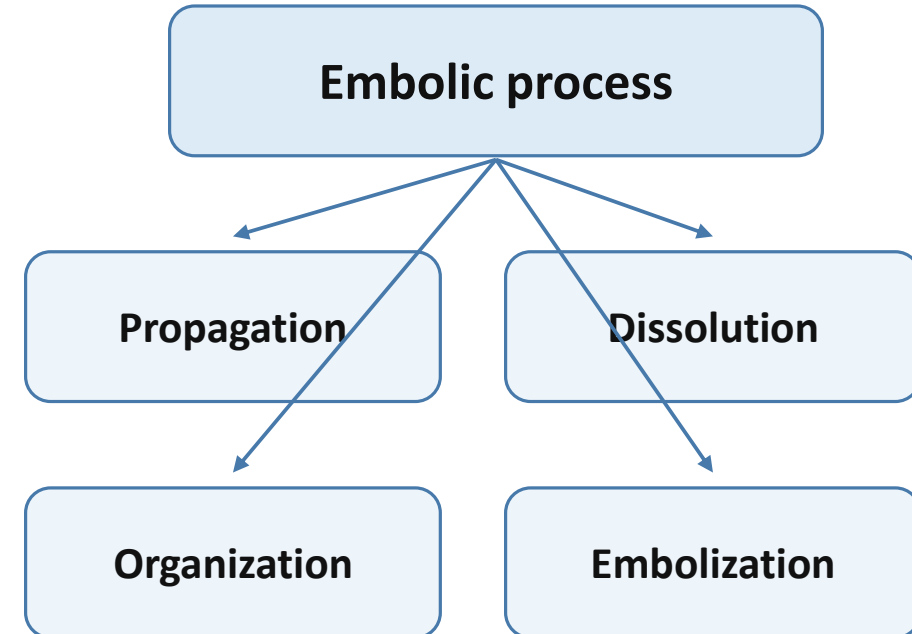
- The parent thrombus remains attached locally until a fragment dislodges.
- A propagating portion may be poorly attached and prone to fragment.
- Embolization is one of four recognized thrombus fates.



Fates of an established thrombus

Embolization competes with growth, lysis, and organization.

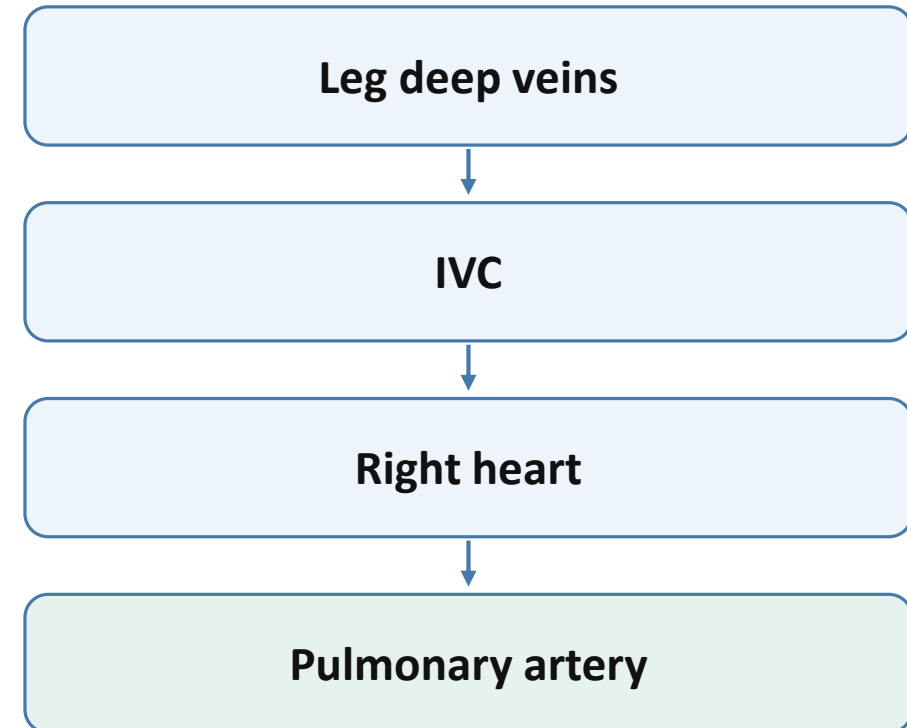
- Propagation adds platelets and fibrin to the original thrombus.
- Dissolution results from fibrinolysis, particularly in recent thrombi.
- Organization and recanalization develop as tissue grows into older thrombi.
- Embolization carries detached fragments downstream.



Deep venous thrombosis is the main PE source

Lower-extremity deep venous thrombi are the classic precursors of pulmonary emboli.

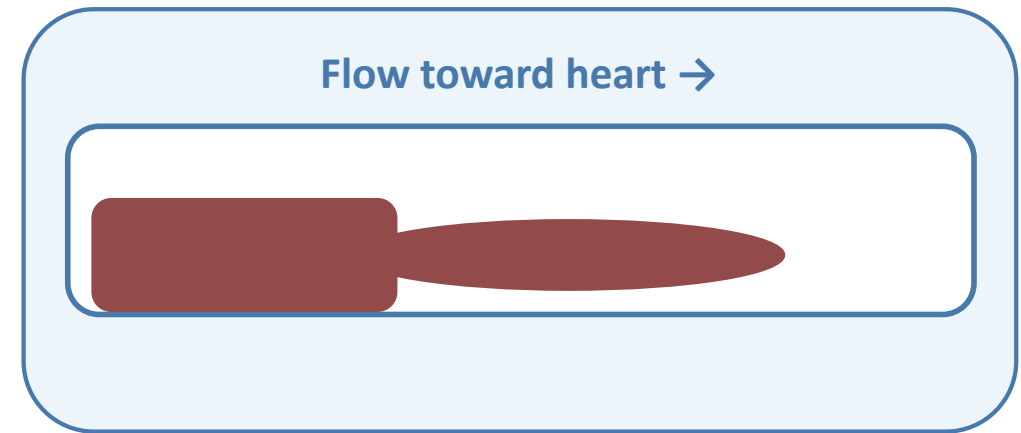
- Proximal leg veins are clinically important sources.
- Stasis and hypercoagulability are frequent contributors to leg DVT.
- Venous thrombi may be clinically silent until embolization.



Why venous thrombi embolize

Thrombus architecture helps explain fragmentation.

- Venous thrombi form in low-flow settings and may extend as long luminal casts.
- Attachment is often most secure at the point of initiation.
- The expanding tail may fragment and enter venous return.



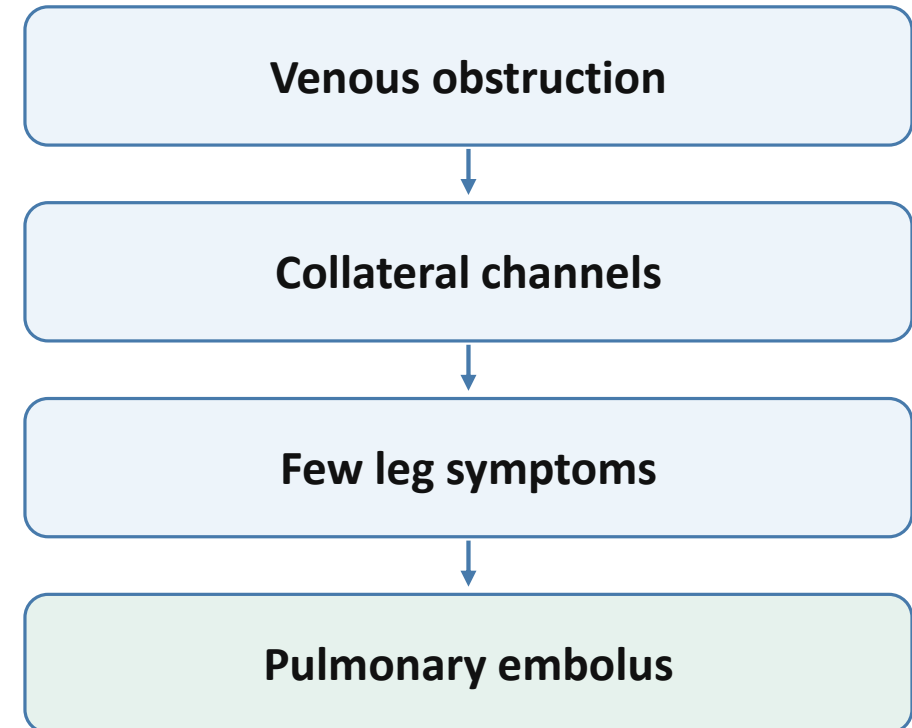
**Attached point of
origin**

**Propagating portion: More
prone to detach**

A silent DVT can have a major consequence

Absence of local symptoms does not exclude an embolic source.

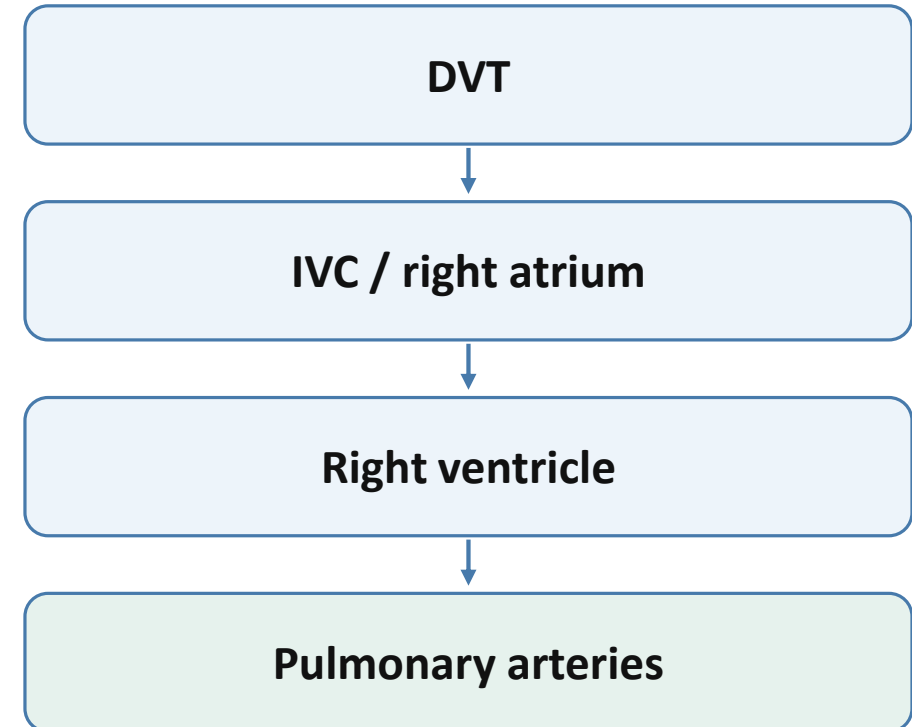
- Collateral venous drainage may blunt pain and edema.
- Embolization can be the first clinically recognized manifestation.
- Source assessment therefore extends beyond the symptomatic organ.



The anatomic route to the pulmonary arteries

Venous fragments traverse the right-sided cardiac circulation.

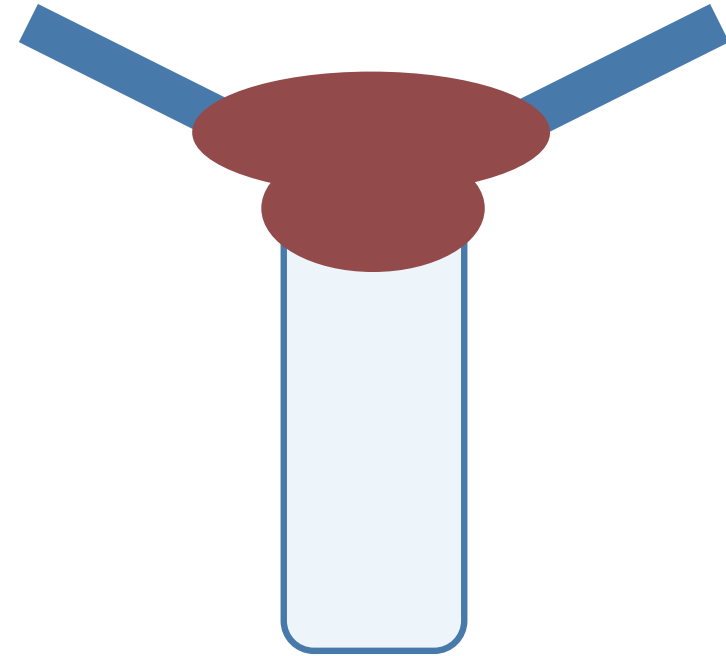
- Lower-extremity vein → inferior vena cava.
- Right atrium → right ventricle.
- Pulmonary trunk → right or left pulmonary arterial branches.



Pulmonary emboli lodge at branching points

Large emboli may obstruct central pulmonary arterial segments.

- Central occlusion may involve the main pulmonary trunk.
- A saddle embolus straddles the bifurcation of the pulmonary trunk.
- Smaller fragments reach segmental and distal branches.



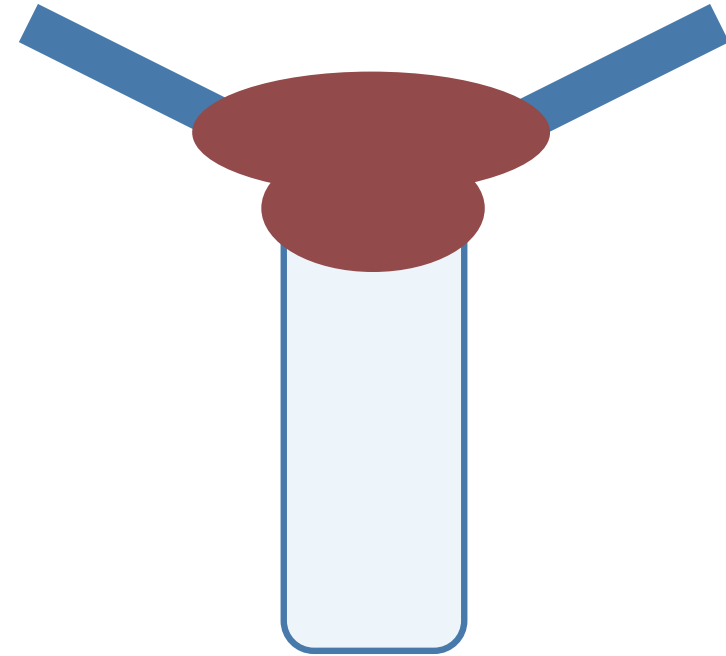
Trunk

Schematic central embolus; Not a gross specimen or angiographic image.

Saddle pulmonary embolism: Morphology

“Saddle” describes anatomic position, not a separate embolus composition.

- The embolus bridges the major pulmonary arterial bifurcation.
- It may compromise flow into both lungs.
- Severity still depends on total obstruction and hemodynamic response.



Main pulmonary artery

Schematic central embolus; Not a gross specimen or angiographic image.

Single versus multiple pulmonary emboli

Embolic burden can accumulate through repeated events.

- One large fragment can obstruct a proximal vessel.
- A shower of smaller emboli can lodge in multiple distal vessels.
- Sequential events help explain recurrence and chronic vascular consequences.

Embolic process

Large central embolus

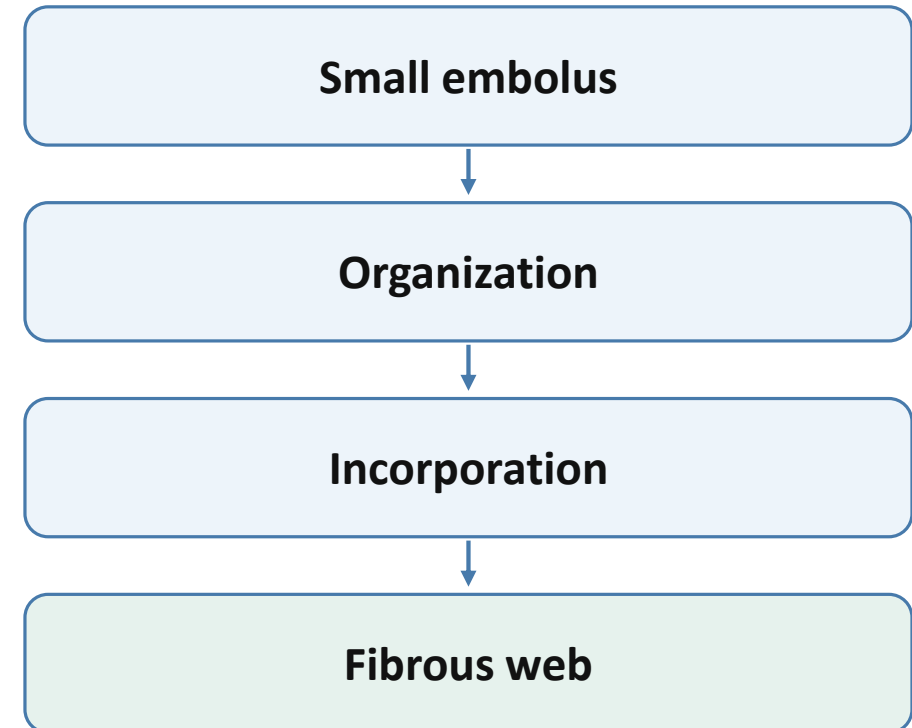
Multiple distal emboli

Recurrent episodes

Small pulmonary emboli may be silent

Small emboli can resolve without a clinically apparent episode.

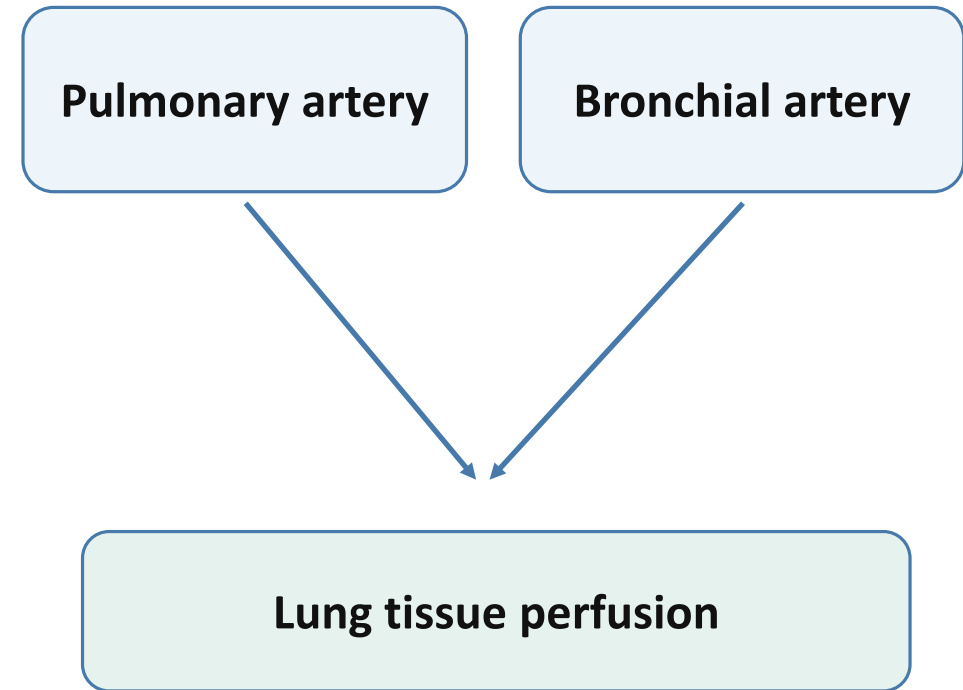
- The Robbins text describes many small PE as clinically silent.
- Organization may incorporate the thromboembolus into the vessel wall.
- Residual thin fibrous webs may persist after organization.



Why PE does not always cause lung infarction

The lung has two arterial systems with partially overlapping perfusion.

- Pulmonary arteries provide the low-pressure gas-exchange circulation.
- Bronchial arteries provide a systemic arterial contribution.
- Preserved bronchial circulation can prevent tissue necrosis after PE.



Alternative bronchial flow can help preserve viability

Pulmonary hemorrhage is not synonymous with infarction

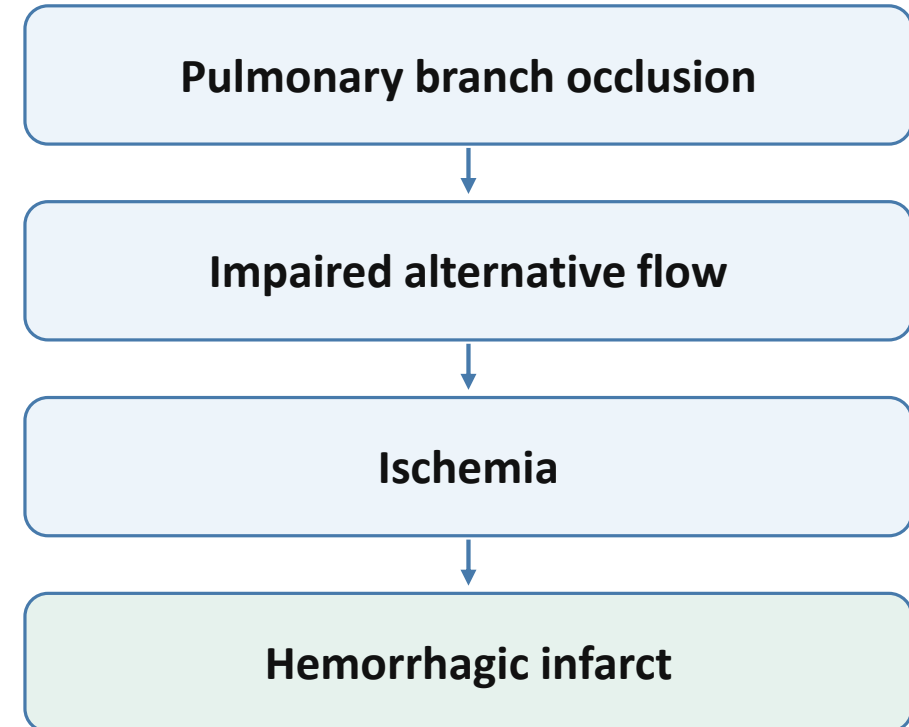
Vascular rupture may allow blood into tissue without irreversible ischemic necrosis.

Finding	Hemorrhage	Infarction
Mechanism	Blood extravasation	Ischemic necrosis
Necrosis required?	No	Yes
PE setting	Possible with viable tissue	Requires insufficient perfusion

When pulmonary infarction becomes more likely

Dual circulation is protective but not absolute.

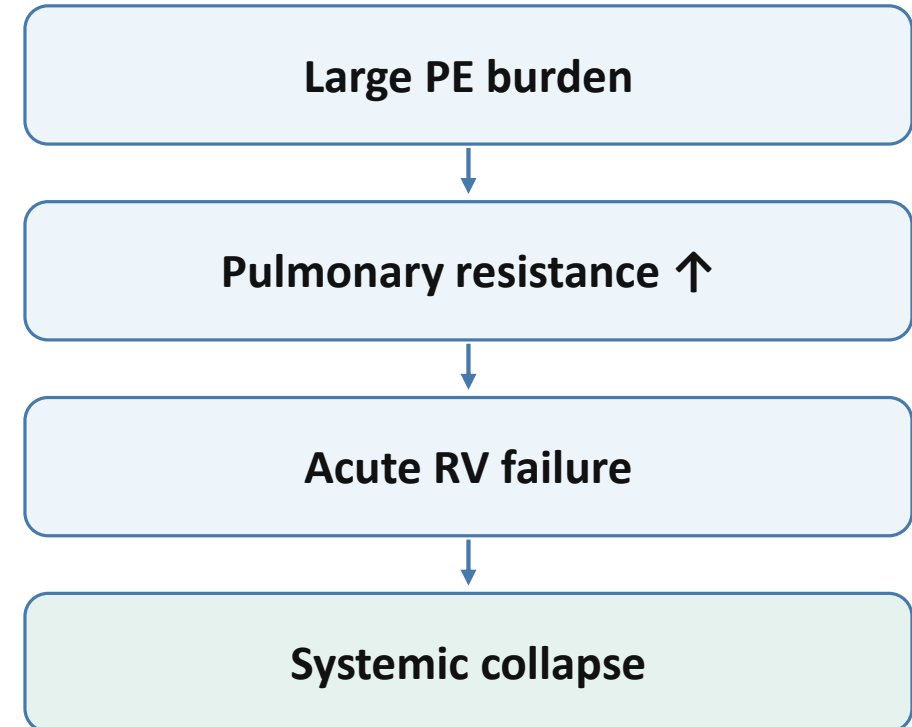
- Distal small-branch occlusions may produce hemorrhage or infarction.
- Compromised bronchial perfusion reduces protection.
- The involved territory and collateral reserve determine the outcome.



Massive PE produces acute right-heart stress

A large obstructed vascular bed abruptly increases pulmonary vascular resistance.

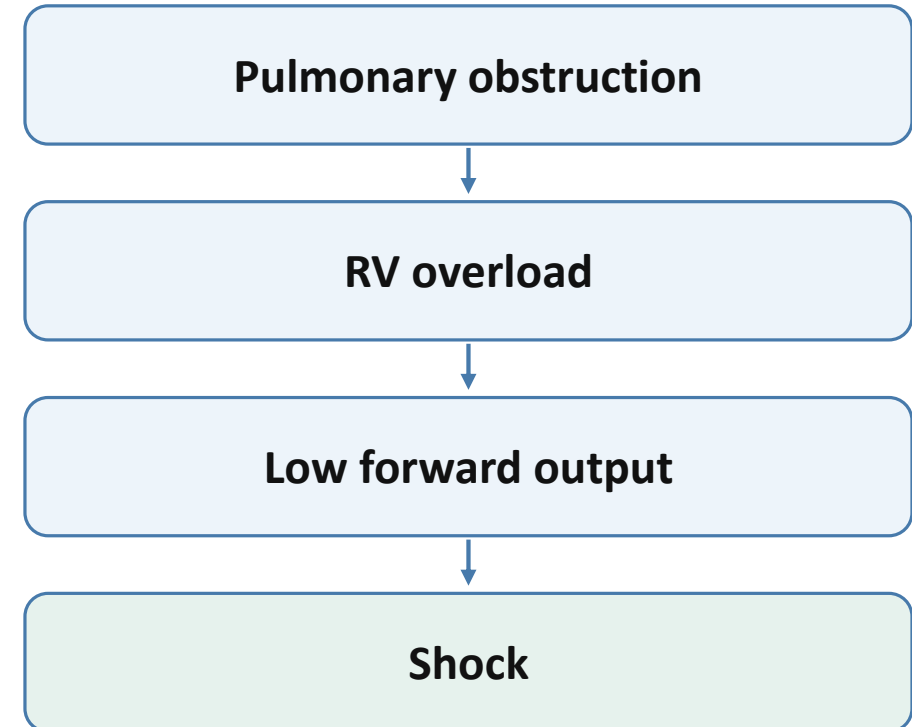
- The right ventricle faces an acute increase in afterload.
- Right ventricular failure can limit left ventricular filling.
- Severe episodes may cause cardiovascular collapse or sudden death.



Pulmonary vascular obstruction and shock

Mechanical occlusion has systemic hemodynamic effects.

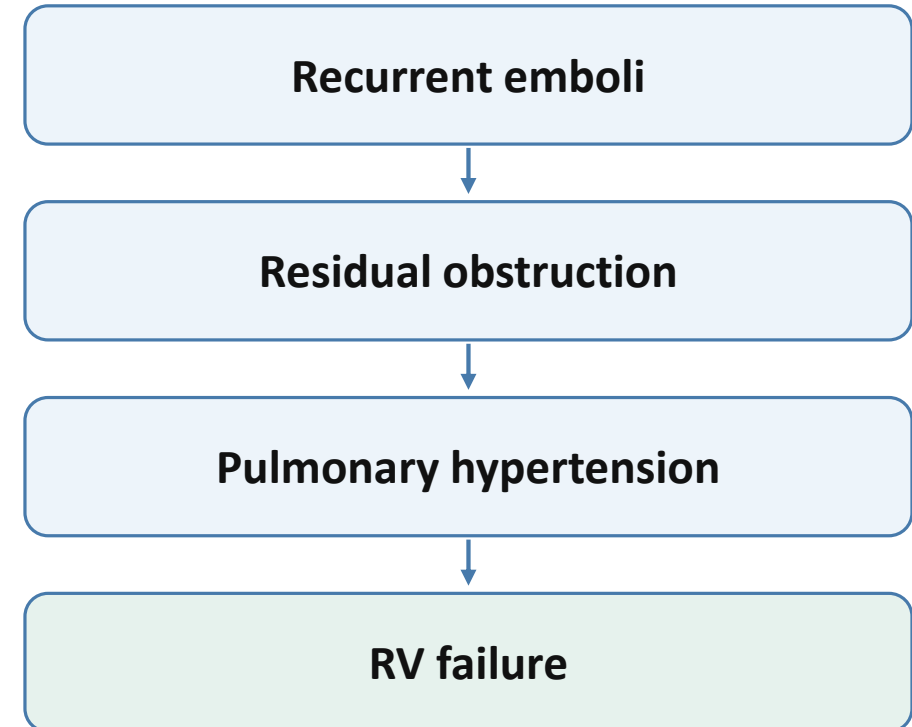
- Reduced pulmonary flow limits delivery to the left heart.
- Acute RV dilation and dysfunction can aggravate low cardiac output.
- Death may occur without a pulmonary infarct.



Recurrent emboli can produce chronic strain

Embolization is not limited to isolated acute events.

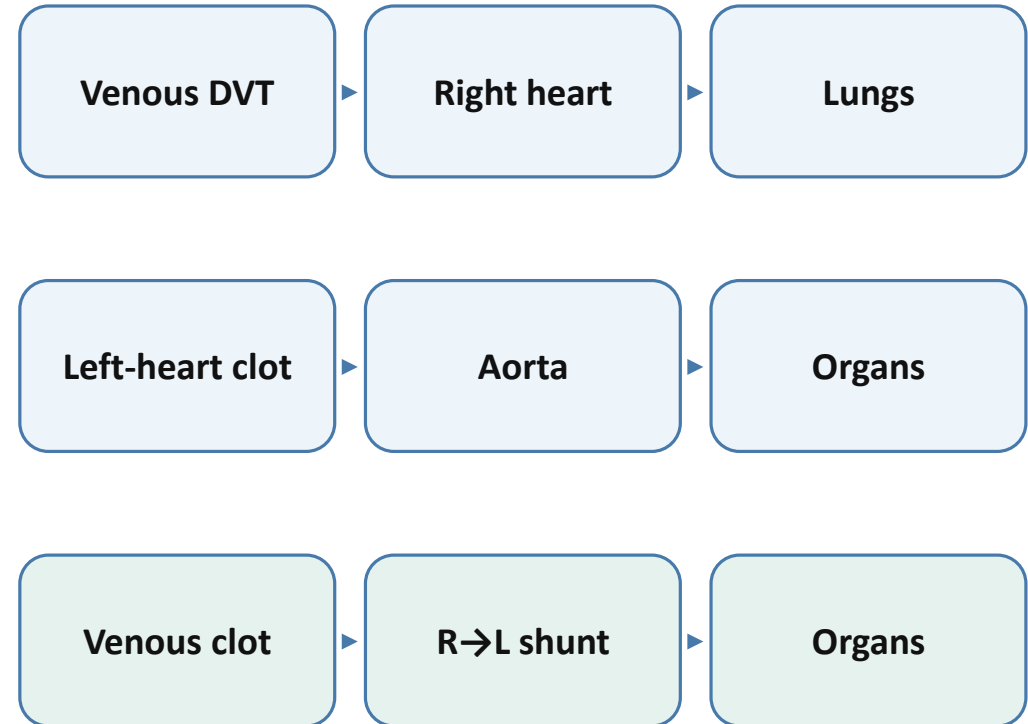
- Multiple emboli over time may increase pulmonary vascular resistance.
- Persistent obstruction and organization can contribute to pulmonary hypertension.
- Prolonged pressure burden can culminate in right ventricular failure.



Paradoxical embolism crosses circulatory circuits

A venous embolus rarely reaches the systemic arterial circulation.

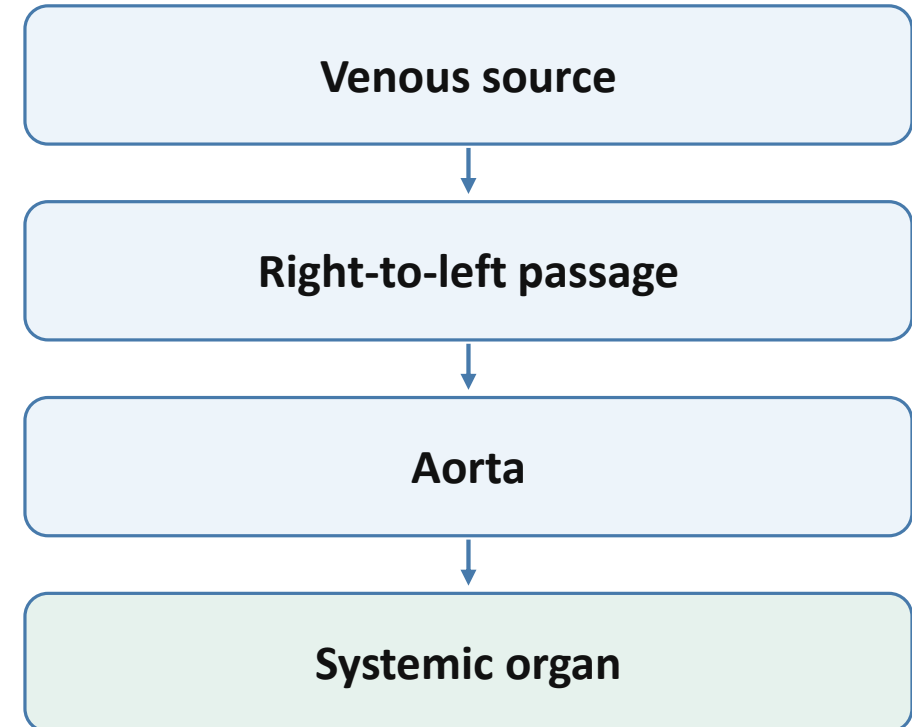
- An interatrial or interventricular defect can provide a conduit.
- Right-to-left passage bypasses the pulmonary arterial filter.
- The embolus then threatens a systemic organ.



Paradoxical embolism: Causal sequence

Both a venous source and an appropriate intracardiac route are required.

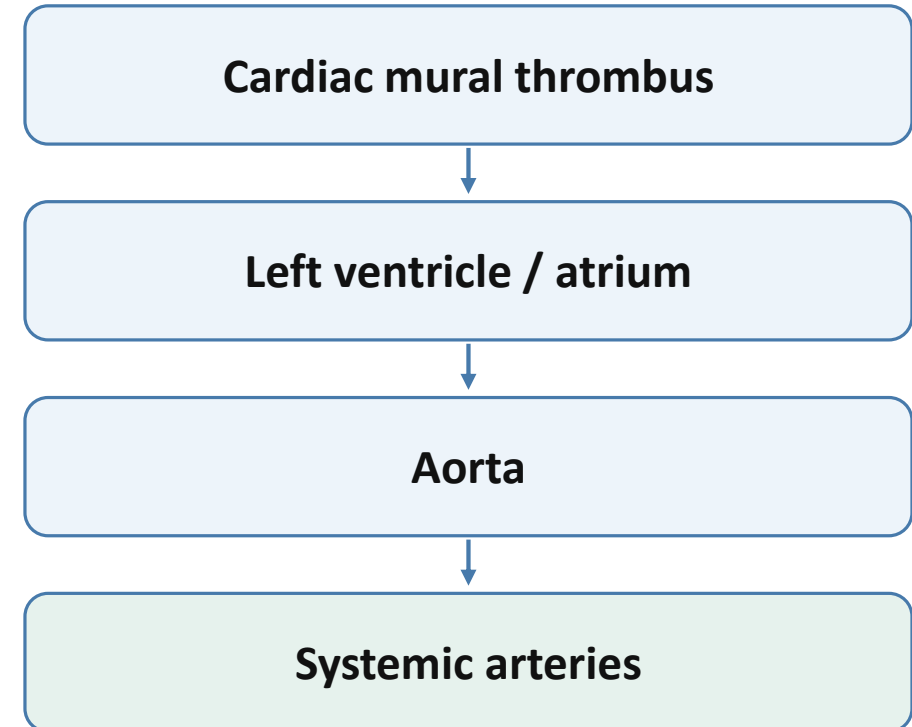
- A thrombus detaches into systemic venous blood.
- The embolus traverses a right-to-left intracardiac communication.
- It enters left-sided outflow and reaches an arterial organ bed.



Systemic emboli usually arise in the left heart

The left ventricle and atrium are frequent origins of arterial thromboemboli.

- Left ventricular mural thrombus can follow myocardial infarction.
- Atrial enlargement and fibrillation favor atrial mural thrombus.
- Detached material enters the aorta and systemic arteries.



Cardiac mural thrombi: Why they form

Abnormal flow and damaged endocardial surfaces promote local thrombosis.

- Postinfarction dyskinesia or akinesia creates abnormal ventricular flow.
- Atrial fibrillation reduces effective atrial emptying.
- Embolization converts a cardiac source into distant organ ischemia.

Wall injury

**Abnormal
contraction**

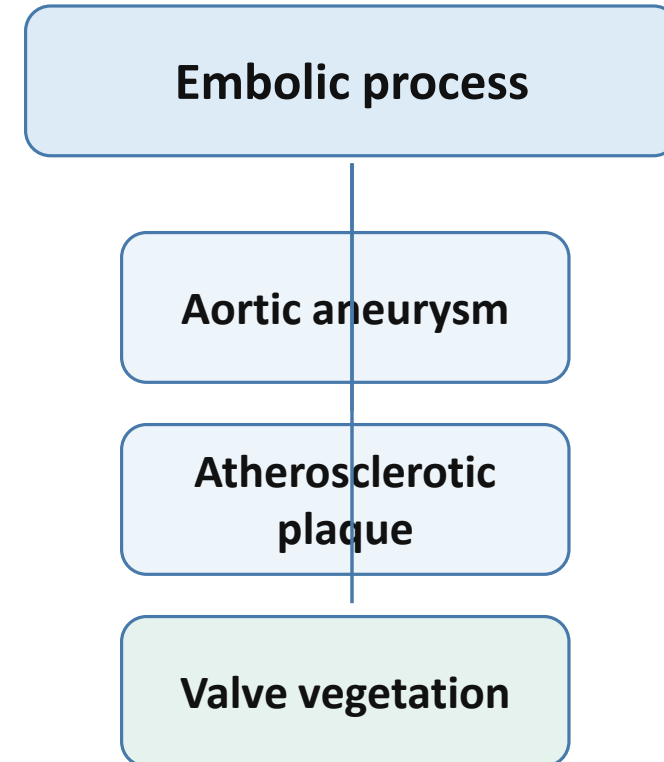
Blood stasis

Distal embolism

Aortic and valvular sources of systemic emboli

Not all systemic arterial emboli originate in the ventricular cavity.

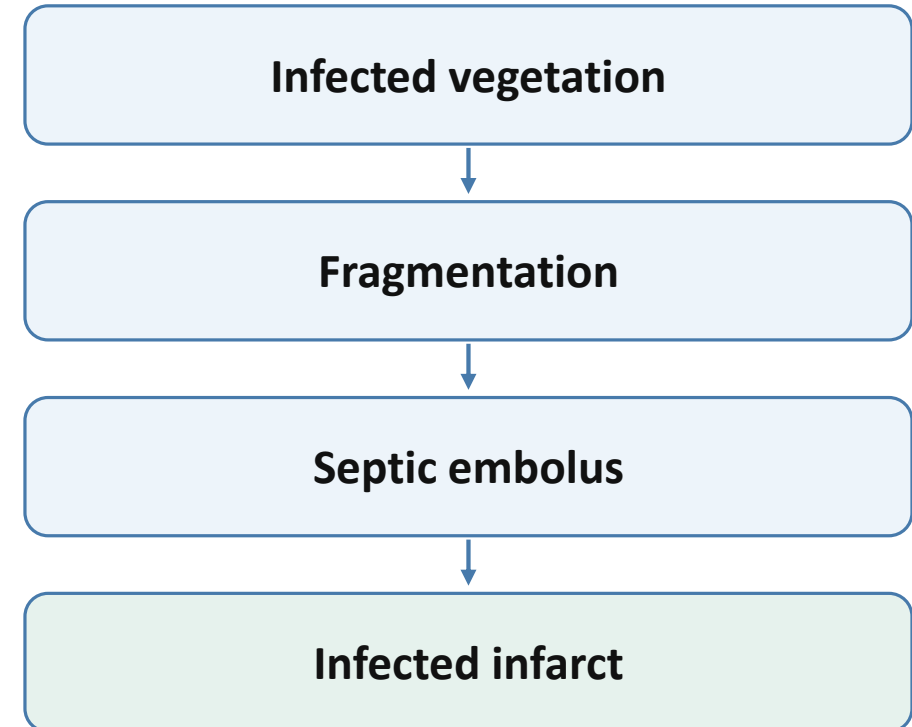
- Aortic aneurysms can harbor emboligenic thrombi.
- Atherosclerotic plaques can release debris or associated thrombus.
- Valvular vegetations can break off and enter the systemic circulation.



Septic emboli add an infectious mechanism

Infected valvular vegetations can spread microbes with the embolic fragment.

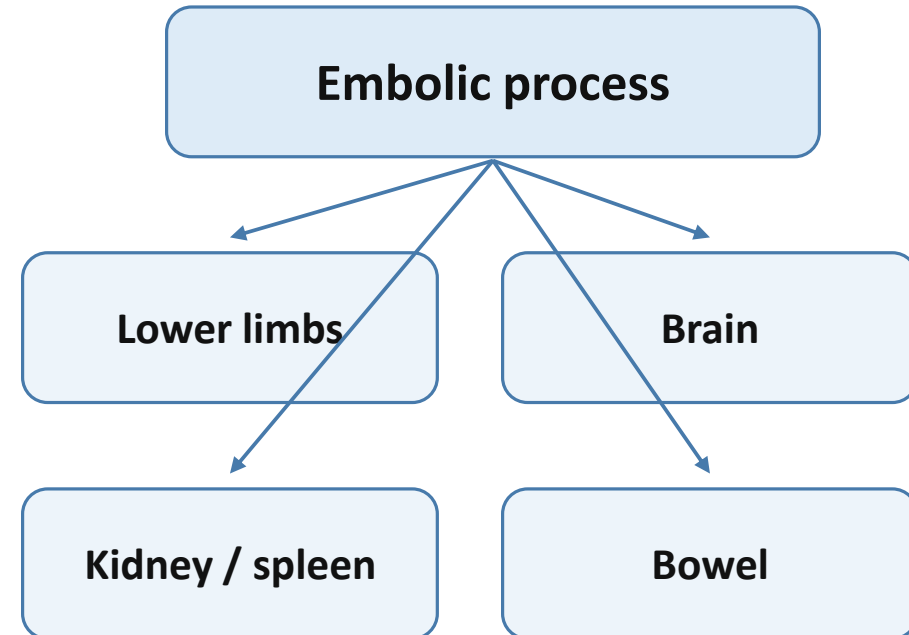
- An embolus may occlude a vessel and seed bacteria or fungi.
- Infection can intensify tissue destruction beyond ischemia alone.
- This differs from sterile mural-thrombus embolization.



Arterial destinations depend on blood flow

Systemic emboli reach organs according to source anatomy and downstream flow.

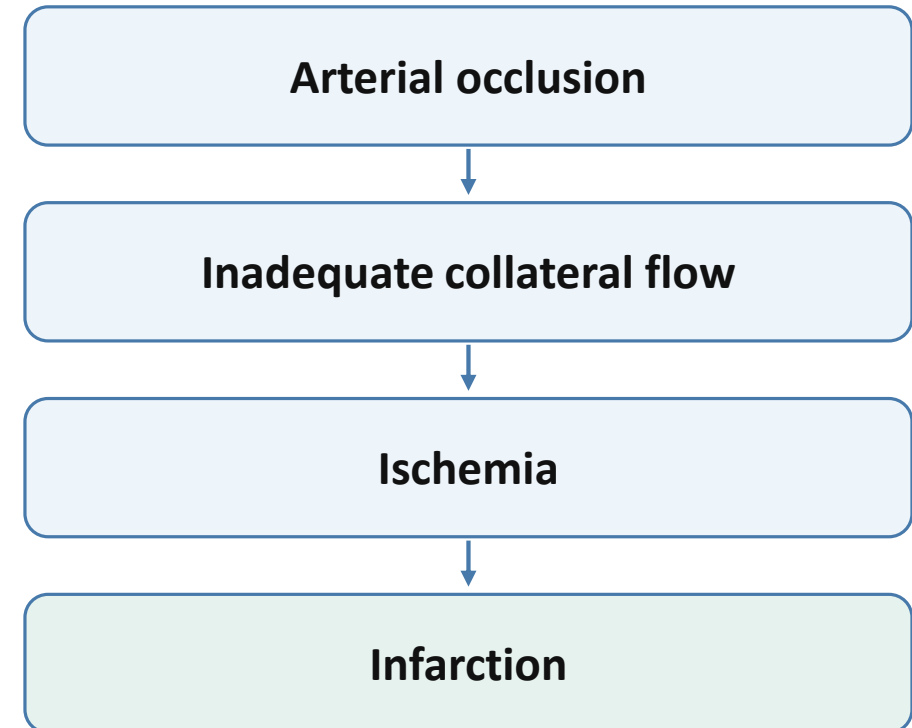
- The lower extremities and brain are common destinations.
- Kidneys, spleen, intestines, and upper extremities are also vulnerable.
- Distribution is not determined by embolus composition alone.



Why an arterial embolus causes infarction

Acute reduction in flow may outpace local collateral compensation.

- End-arterial organs are especially vulnerable to acute obstruction.
- Small arterial occlusions may be tolerated where collaterals are adequate.
- Tissue-specific ischemic sensitivity determines the injury threshold.



Contrast: Renal versus pulmonary embolic injury

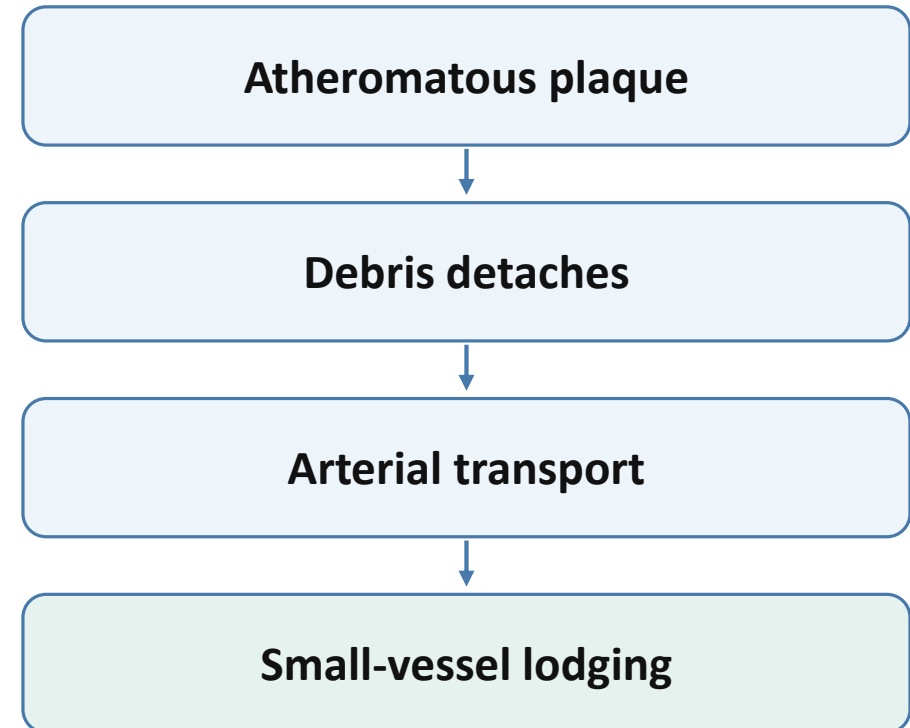
Perfusion architecture helps explain differences in tissue outcome.

Feature	Kidney	Lung
Dominant architecture	End-arterial	Dual arterial supply
Typical embolic threat	Renal infarction	PE with variable tissue necrosis
Alternative perfusion	Limited	Bronchial circulation

Cholesterol and atheromatous debris can embolize

Not every solid arterial embolus resembles a fibrin-rich thrombus.

- Plaque material can detach from diseased large arteries.
- Cholesterol-rich debris can obstruct small downstream vessels.
- Recognition requires linking vascular source to microvascular injury.



Rare solid emboli broaden the differential

Pathologic transport can involve malignant tissue, marrow, or foreign matter.

- Tumor fragments are an example of nonthrombotic solid emboli.
- Marrow fragments may accompany fat embolization after skeletal trauma.
- Foreign-body emboli arise from intravascular introduction of material.

Embolic process

Tumor fragments

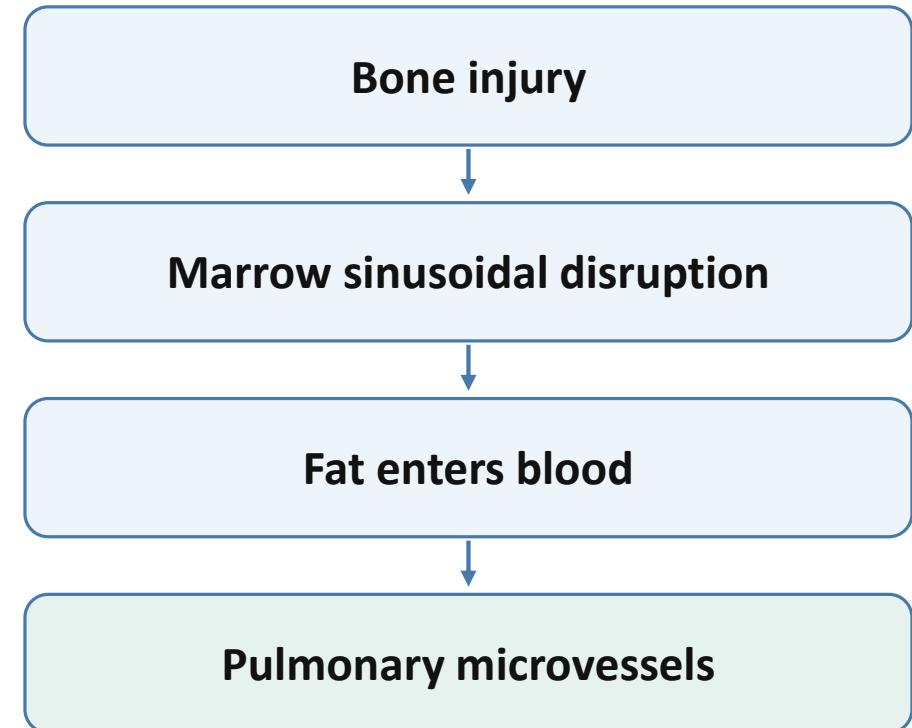
Bone marrow

Foreign bodies

Fat embolism follows major skeletal injury

Marrow fat can enter injured small venules and reach the circulation.

- Long-bone fractures and severe skeletal trauma are classic settings.
- Traumatic rupture of marrow vascular channels permits entry.
- Some cases follow burns or soft-tissue trauma.



Fat embolism is not fat embolism syndrome

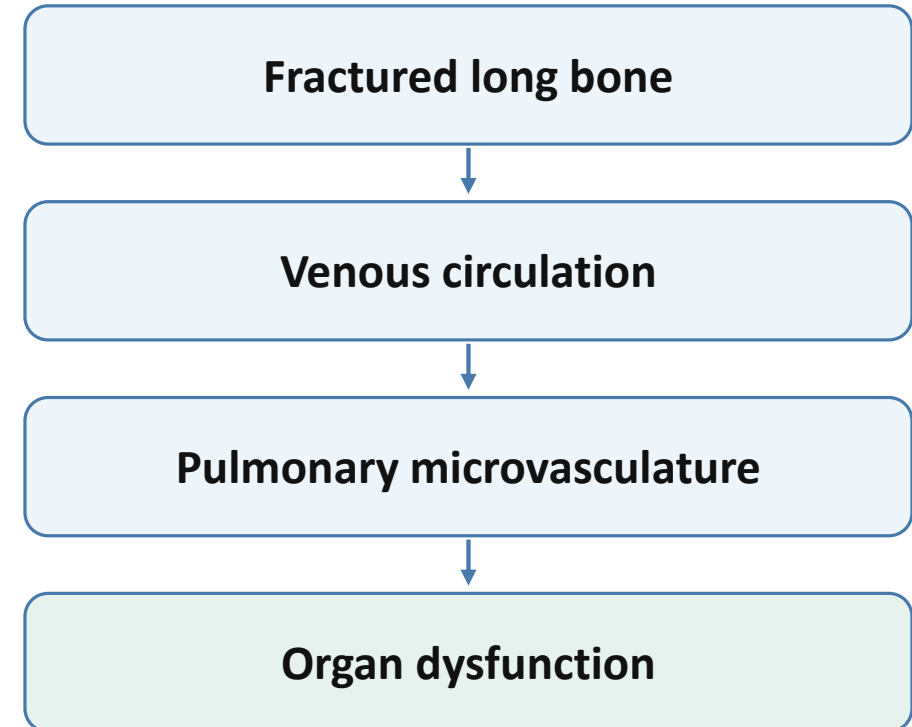
Microscopic fat emboli are more frequent than the symptomatic syndrome.

Term	Fat embolism	Fat embolism syndrome
Core definition	Fat in vessels	Systemic clinical illness
Symptoms required	No	Yes
Clinical focus	Distribution	Pulmonary + neurologic signs

Fat droplets reach the lung from marrow

Marrow fragments and fat globules travel through the venous circuit.

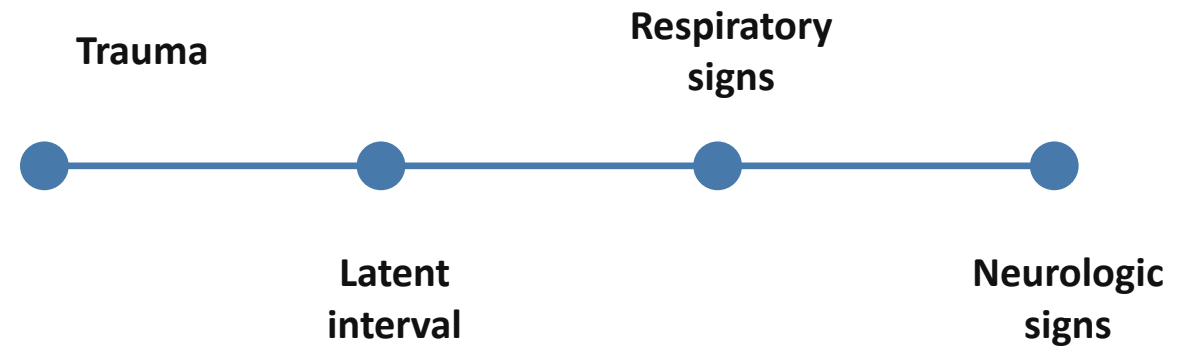
- Skeletal trauma creates access to venous blood.
- Microemboli lodge in pulmonary microcirculation.
- Systemic manifestations also implicate the cerebral circulation.



The clinical delay is diagnostically useful

Fat embolism syndrome often manifests after an interval following injury.

- Onset typically 1–3 days after injury.
- Early features include tachypnea, dyspnea, and tachycardia.
- Neurologic irritability or restlessness may progress to coma.



Fat embolism syndrome: Characteristic cluster

Pulmonary, neurologic, and hematologic abnormalities reflect diffuse microvascular injury.

- Respiratory insufficiency or hypoxemia.
- Neurologic dysfunction, from restlessness to delirium or coma.
- Thrombocytopenia and anemia.
- Petechial rash may provide an additional diagnostic clue.

Respiratory

Neurologic

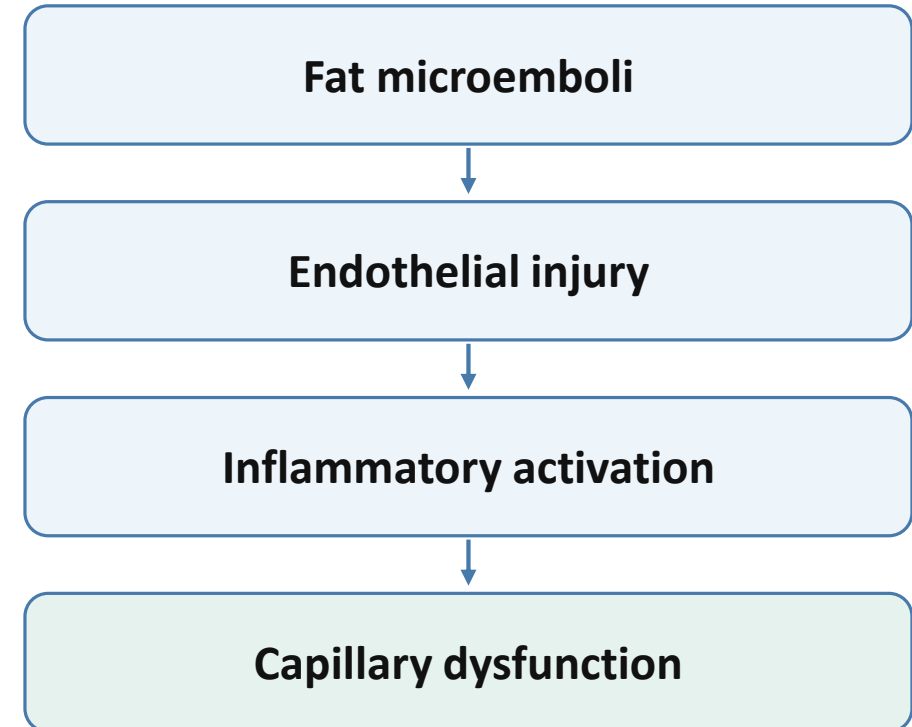
Hematologic

Petechiae

FES injury is mechanical and biochemical

Vessel blockage alone does not account for its systemic effects.

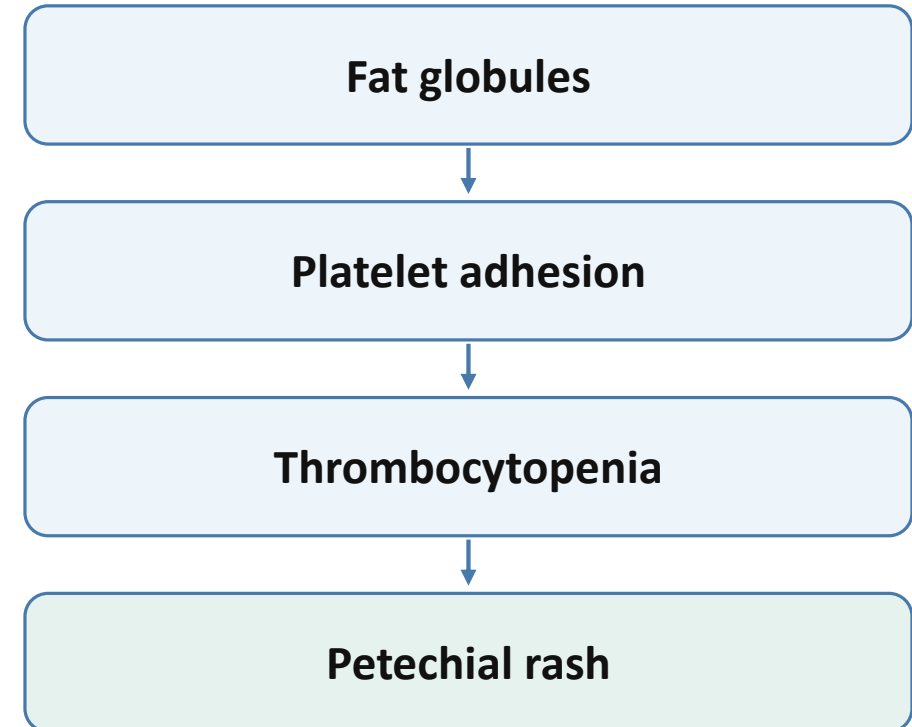
- Fat droplets plus cellular aggregates occlude small vessels.
- Free fatty acids exert endothelial toxicity.
- Activated platelets and recruited granulocytes amplify injury.



Fat-associated platelet changes

Hematologic findings emerge from circulating cell interactions.

- Platelets adhere to fat globules and may aggregate or be sequestered.
- Anemia may follow red-cell aggregation or hemolysis.
- Petechiae are associated with acute thrombocytopenia.



Histologic pitfall: Routine processing removes fat

A negative-appearing fat space is not an intrinsically stained droplet.

Technique	Paraffin H&E	Frozen-section fat stain
Effect on lipid	Usually extracted	Can preserve lipid
Finding	Empty spaces / marrow cells	Fat-positive globules
Interpretive caution	Not definitive alone	Requires context

Fat embolism versus thrombotic PE

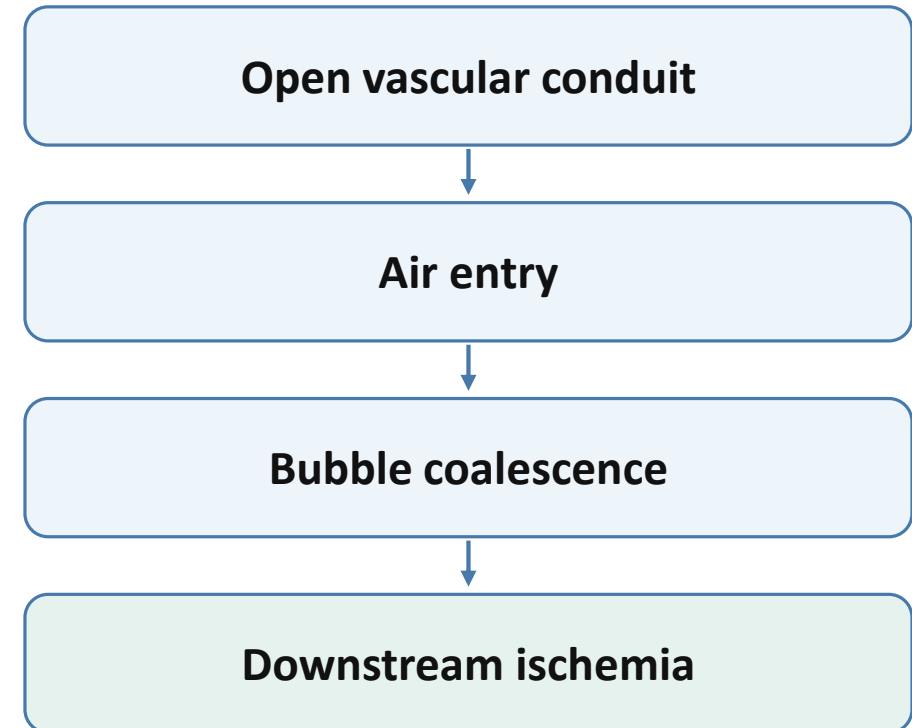
Both may impair pulmonary circulation but have different pathogenesis.

Dimension	Thrombotic PE	Fat embolism syndrome
Source	Deep venous thrombus	Bone / soft-tissue injury
Vessels	Central to peripheral pulmonary	Predominantly microvascular
Distinctive effects	RV overload or infarction	Neurologic / petechial / cytopenic

Air embolism requires gas to enter circulation

An air–blood interface and favorable pressure gradient can enable entry.

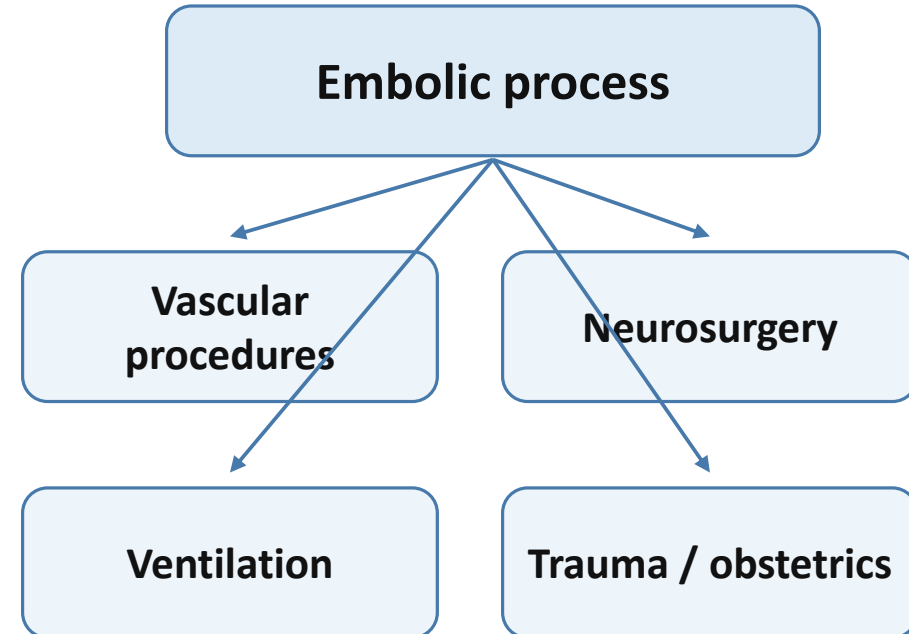
- Air entry occurs when a vessel communicates with outside air.
- Pressure gradients influence whether gas is aspirated into vessels.
- Intravascular bubbles may obstruct flow and trigger inflammation.



Clinical settings of iatrogenic air entry

Procedures can inadvertently create access for ambient gas.

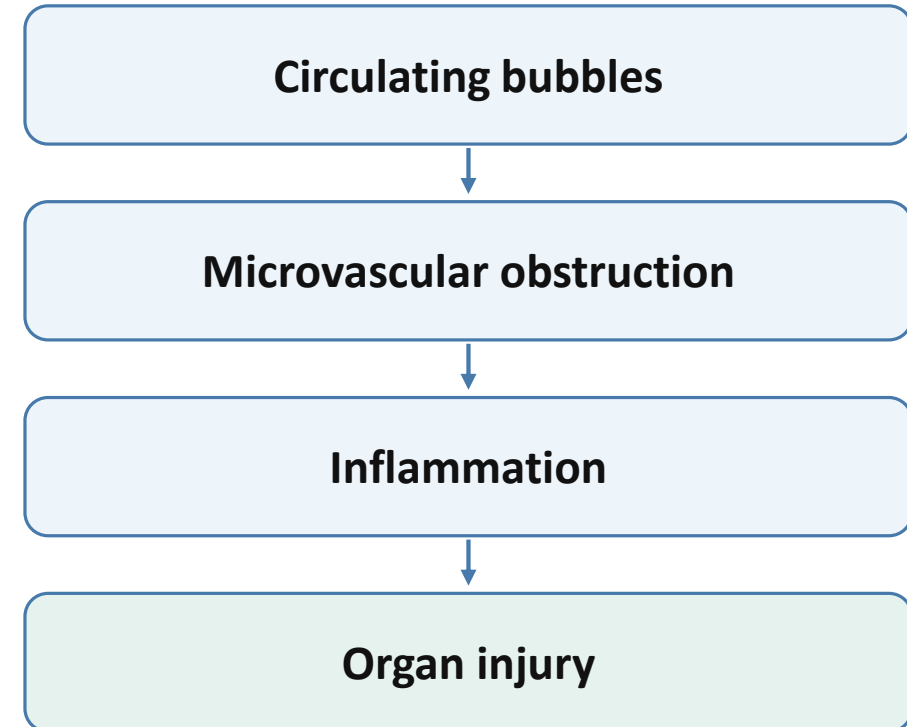
- Endovascular and other interventional procedures.
- Certain neurosurgical positions and procedures.
- Mechanical ventilation, obstetric or laparoscopic interventions.
- Chest wall injury may also allow air entry.



Gas bubbles cause more than simple plugging

Mechanical obstruction is amplified by host responses.

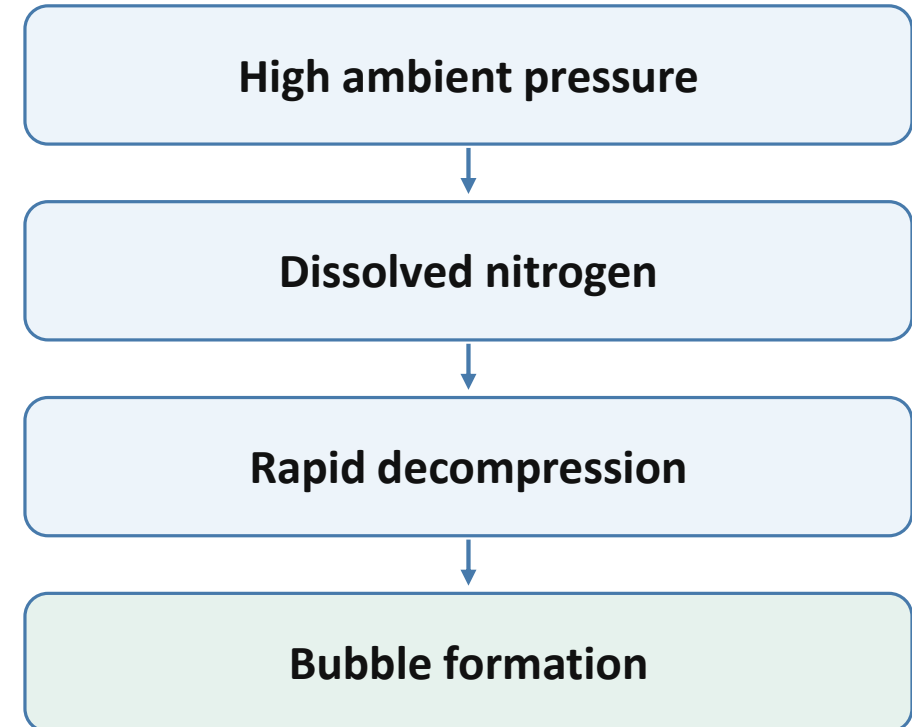
- Bubbles coalesce and reduce flow in receiving vessels.
- Inflammatory responses may injure pulmonary alveoli.
- Gas bubbles reaching the CNS can cause acute neurologic dysfunction.



Decompression sickness: A distinct gas source

Dissolved nitrogen can form bubbles after abrupt pressure reduction.

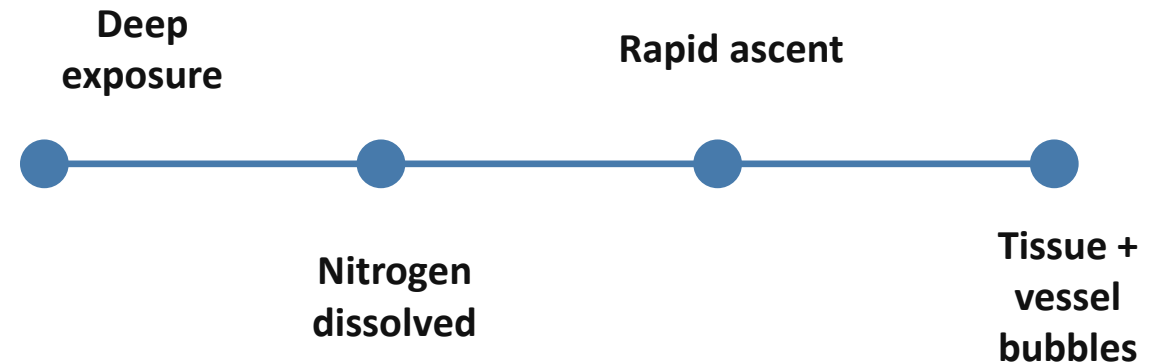
- High ambient pressure increases dissolved nitrogen in blood and tissues.
- Rapid ascent lowers pressure before nitrogen can be eliminated.
- Gas leaves solution and expands into bubbles.



Nitrogen comes out of solution during rapid ascent

The critical issue is the kinetics of pressure reduction.

- With gradual ascent, gas can be eliminated over time.
- With rapid ascent, tissue nitrogen becomes supersaturated.
- Bubbles can form in vessels and supporting tissues.



The bends and the chokes

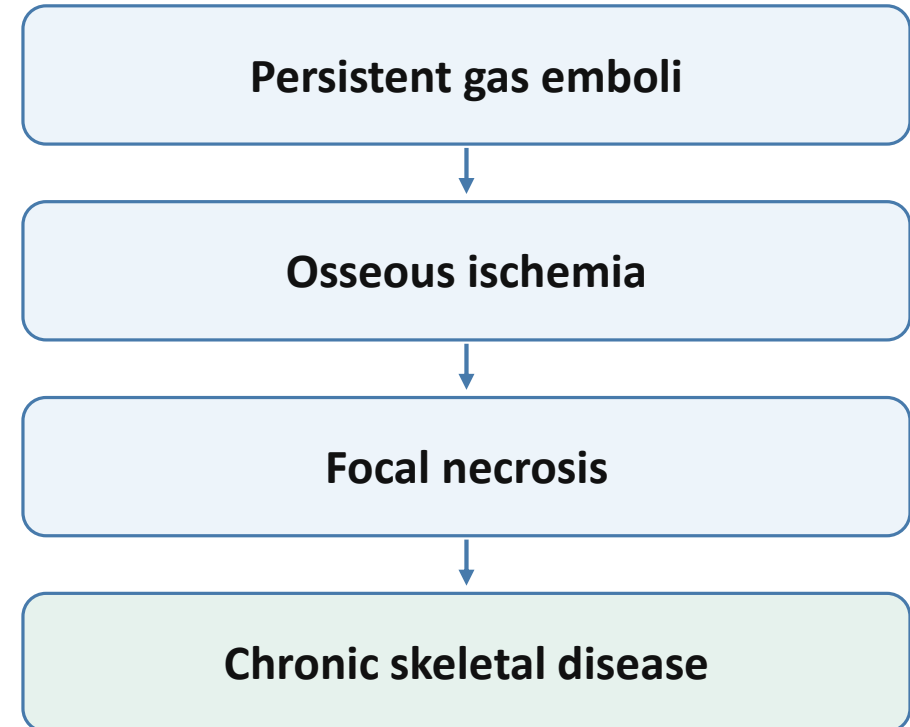
Symptoms reflect where the gas bubbles accumulate.

Syndrome	Bends	Chokes
Principal site	Joints / supporting tissue	Pulmonary vasculature
Typical effect	Severe pain	Respiratory distress
Shared mechanism	Decompression bubbles	Decompression bubbles

Caisson disease: Chronic skeletal injury

Persistent gas emboli may produce repeated ischemic bone damage.

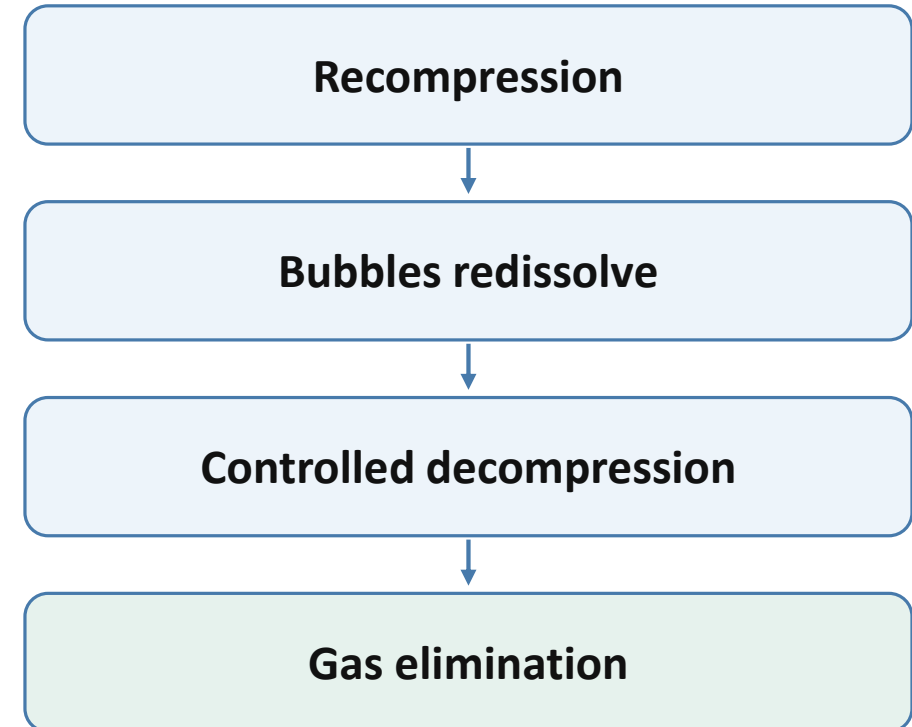
- A chronic form of decompression disease affects the skeletal system.
- Ischemic necrosis can occur in femoral heads, tibiae, and humeri.
- This differs from an acute isolated episode of the bends.



Why controlled recompression works

The treatment principle reverses bubble formation rather than removing a solid embolus.

- Increased pressure drives gas bubbles toward dissolution.
- Slow subsequent decompression enables gradual gas elimination.
- Rapid decompression again risks recurrent bubble formation.



Air entry versus decompression gas embolism

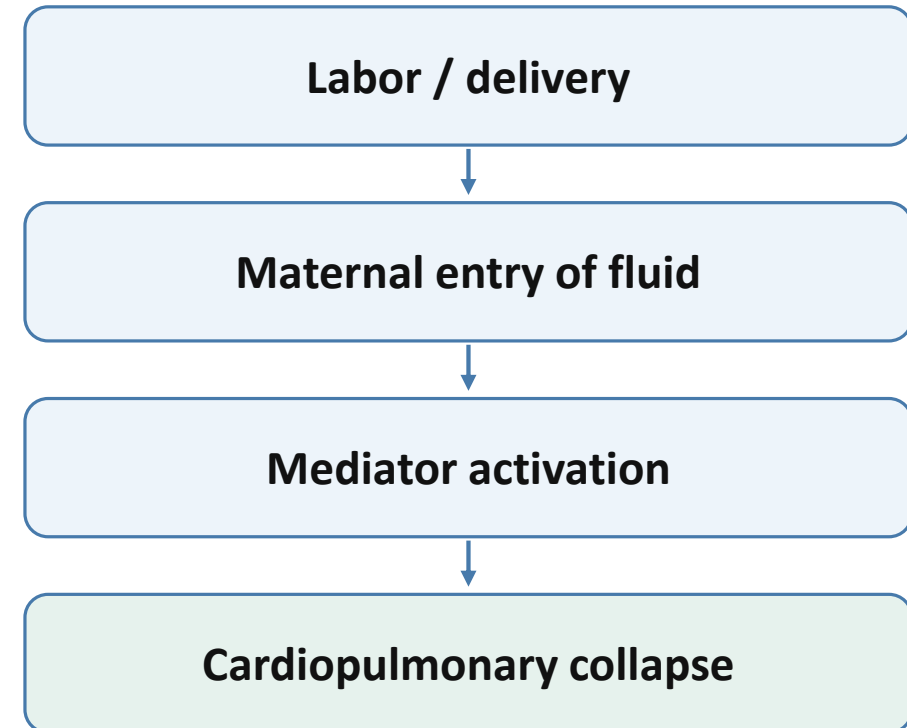
Both generate bubbles, but the gas enters by different routes.

Feature	Air entry	Decompression sickness
Gas source	Outside atmosphere	Dissolved tissue nitrogen
Trigger	Vascular–air conduit	Rapid pressure reduction
Typical clue	Procedure / trauma	Rapid ascent

Amniotic fluid embolism is an obstetric emergency

It usually occurs during labor or in the immediate postpartum period.

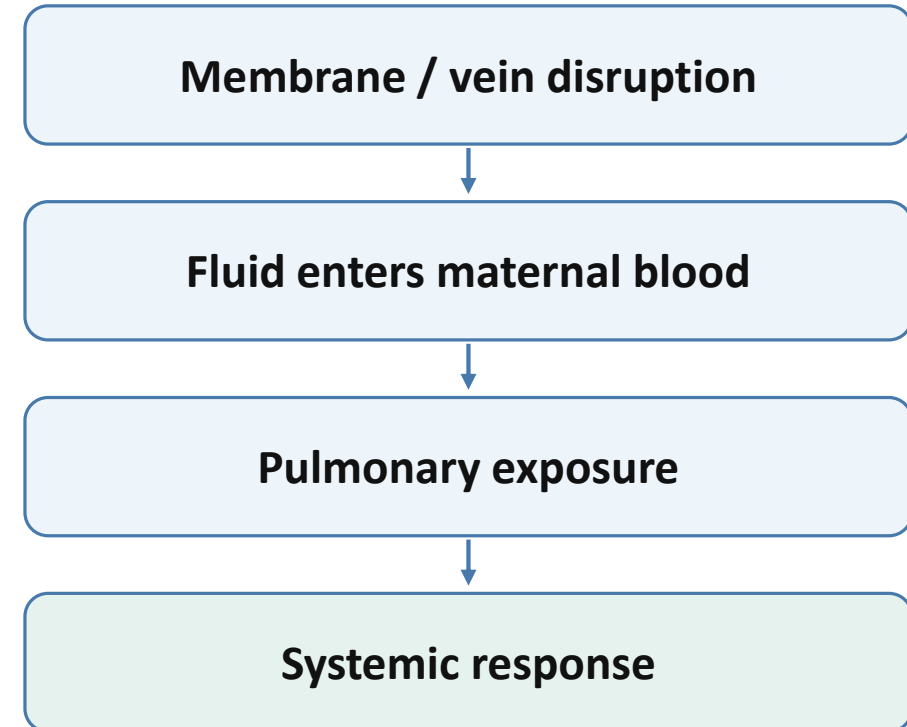
- Amniotic contents gain access to the maternal circulation.
- The ensuing syndrome involves pulmonary, hemodynamic, and coagulation abnormalities.
- Mechanisms include immune and mediator responses, not only physical blockage.



How fetal material reaches maternal blood

Placental membrane tears or uterine venous disruption can create an entry route.

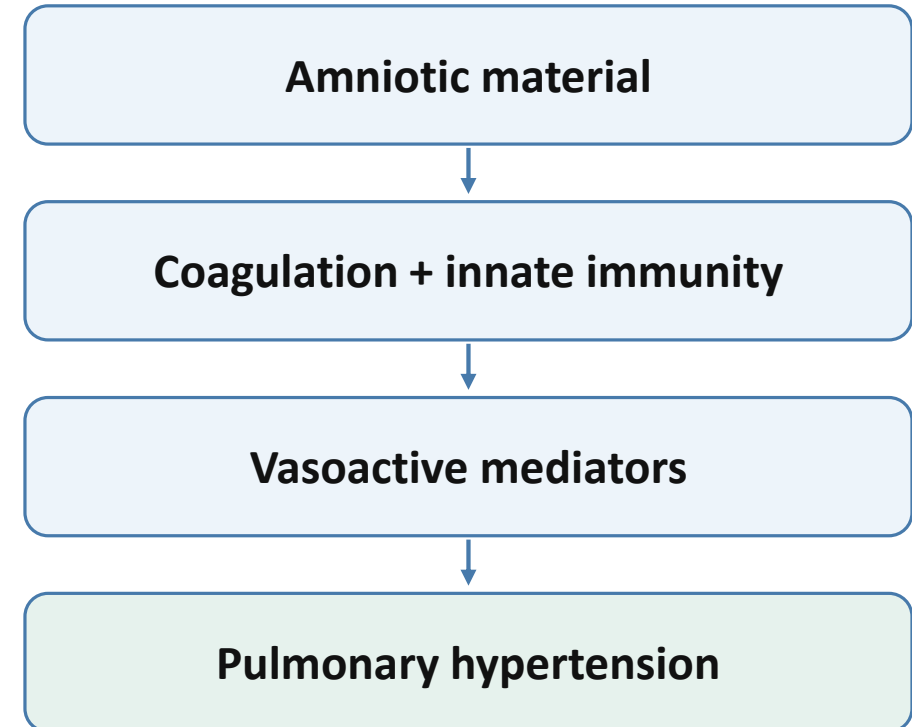
- Amniotic fluid and fetal tissue enter the maternal vascular compartment.
- Pulmonary microvasculature may contain fetal-origin components.
- Their presence supports mechanism but does not reduce the syndrome to plugging alone.



Amniotic fluid triggers mediator-driven injury

Circulatory compromise is disproportionate to simple embolic obstruction.

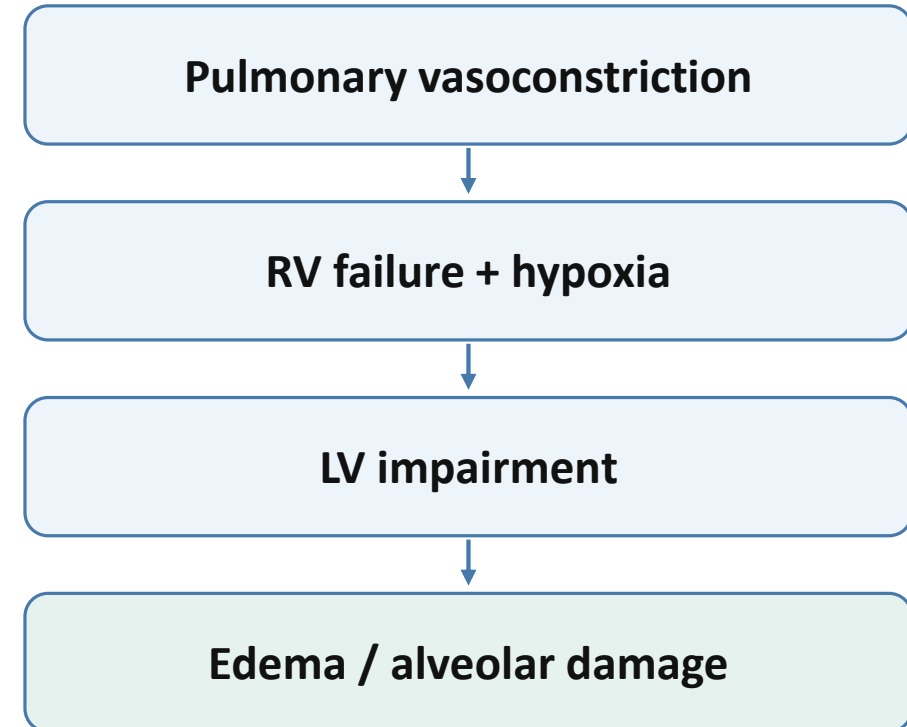
- Amniotic material contains tissue factor and activated coagulation factors.
- Coagulation and innate immune activation release vasoactive mediators.
- Pulmonary hypertension and RV dysfunction can rapidly develop.



From maternal pulmonary hypertension to edema

Right- and left-sided dysfunction may occur in sequence.

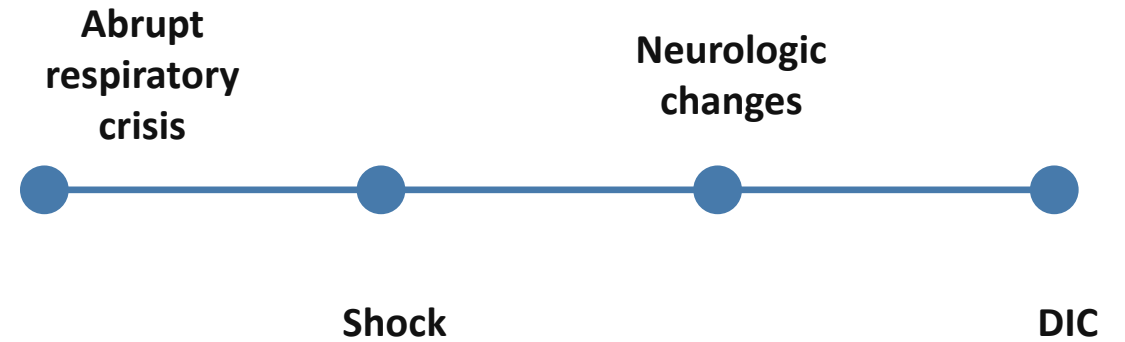
- A sudden increase in pulmonary vascular resistance stresses the right ventricle.
- Hypoxia and left-heart dysfunction may follow.
- Pulmonary edema and diffuse alveolar damage contribute to respiratory failure.



The clinical pattern includes DIC

Coagulopathy distinguishes amniotic fluid embolism from many ordinary thrombotic PE cases.

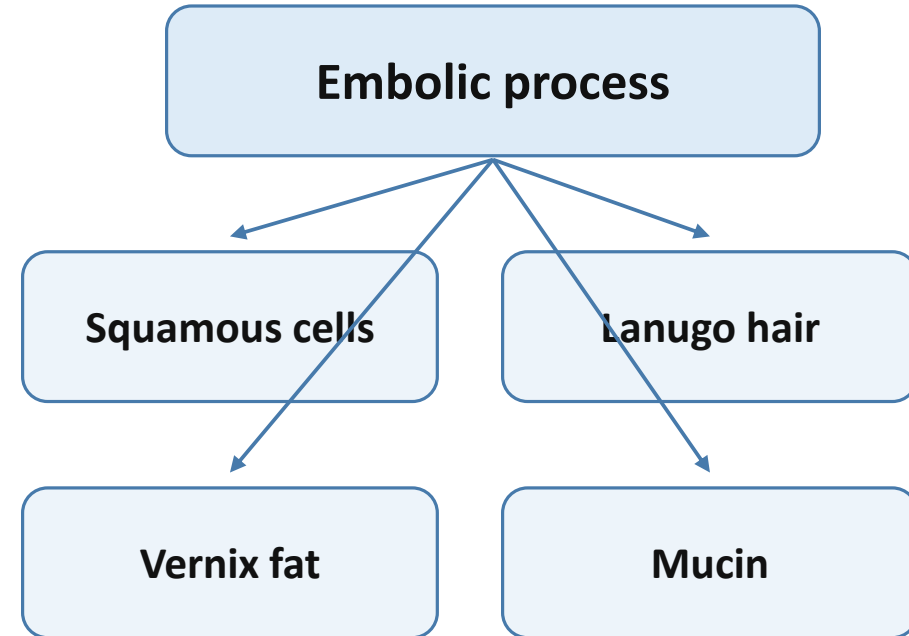
- Abrupt dyspnea, cyanosis, and shock are early manifestations.
- Neurologic impairment may range from headache to seizures or coma.
- Disseminated intravascular coagulation may rapidly emerge.



Fetal components in pulmonary vessels

Autopsy findings can support exposure to amniotic constituents.

- Squamous cells shed from fetal skin.
- Lanugo hair and fat derived from vernix caseosa.
- Mucin derived from fetal respiratory or gastrointestinal tract.
- Interpret tissue findings with the characteristic clinical syndrome.



Amniotic embolism versus thrombotic PE

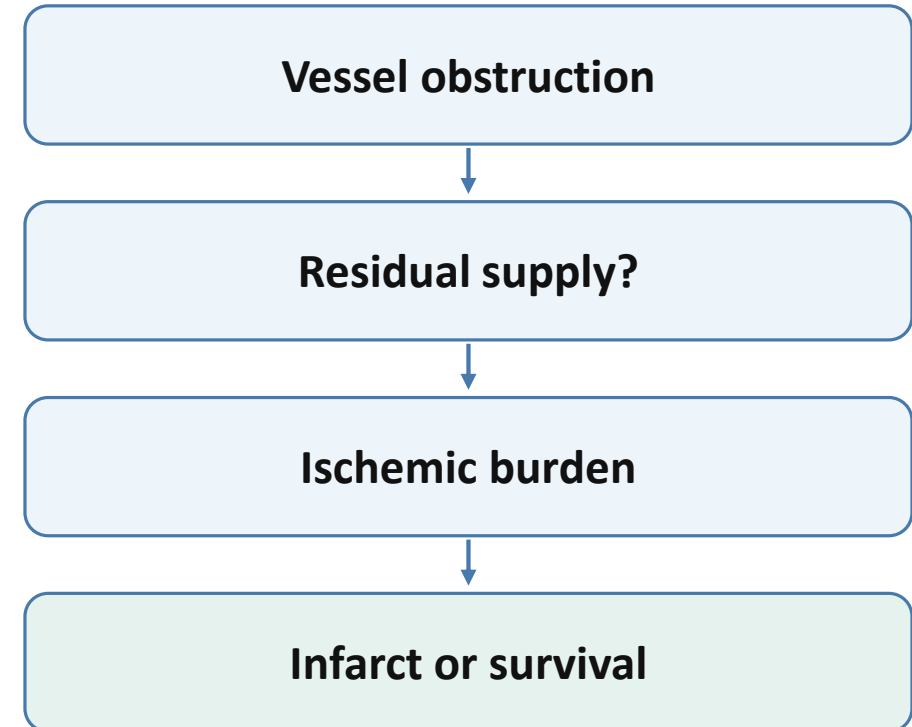
Similar abrupt respiratory compromise can have distinct underlying mechanisms.

Feature	Thrombotic PE	Amniotic fluid embolism
Usual setting	DVT / VTE risk	Labor / early postpartum
Dominant injury	Vascular obstruction	Mediator + coagulation injury
Associated finding	RV strain / infarction	DIC, neurologic dysfunction

Embolism can cause infarction—but not always

Vascular occlusion and tissue necrosis are distinct events.

- Infarction requires ischemia sufficient to cause tissue death.
- Available collaterals and rate of obstruction modify outcome.
- A pulmonary thromboembolus can be clinically silent or cause shock without infarction.



Compare mechanisms across embolic materials

The injury pathway differs by the constituent.

Type	Dominant pathway	Characteristic outcome
Thrombotic	Mechanical obstruction	PE / systemic infarction
Fat	Microvascular + biochemical	Respiratory + neurologic syndrome
Gas	Bubble blockage / inflammation	Ischemia / decompression illness
Amniotic	Coagulation + vasoactive	Shock / DIC

Case: Postoperative sudden dyspnea

A patient develops abrupt dyspnea after prolonged immobilization following surgery.

- A swollen calf had been noticed earlier but was not investigated.
- What is the likely origin of the embolic material?
- Trace the route from the originating vessel to the affected organ.

Interpret the mechanism

Suspected origin

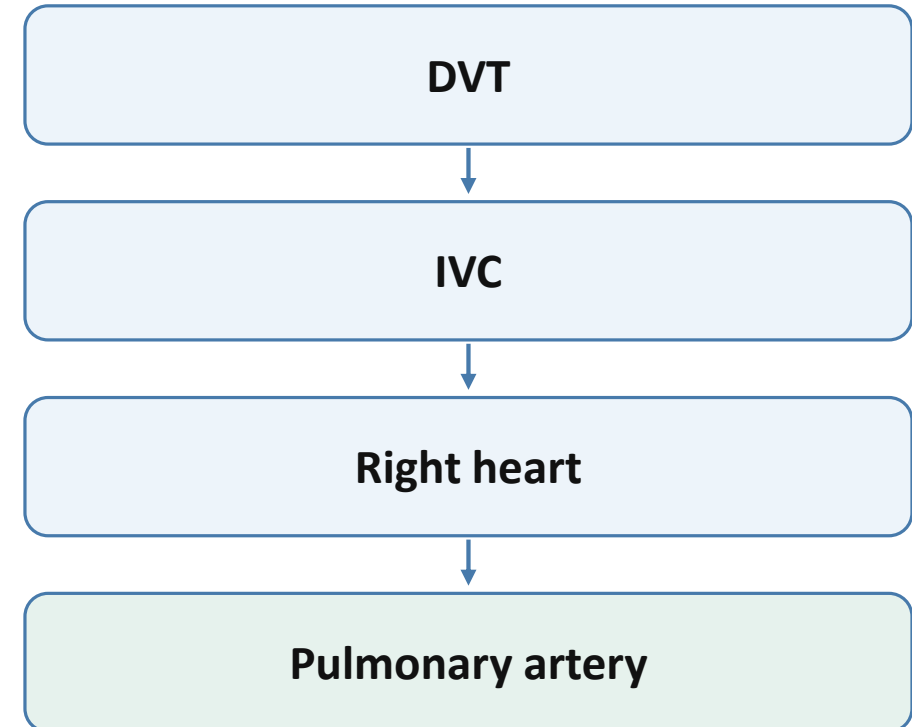
Route

Expected destination

Case interpretation: Venous thromboembolism

The likely mechanism is a fragment from a lower-extremity DVT.

- Venous stasis supports formation of a deep-vein thrombus.
- Embolus traverses IVC → right heart → pulmonary artery.
- Severity depends on pulmonary vascular burden and RV response.



Case: Fracture followed by systemic signs

Two days after a long-bone fracture, a patient develops respiratory distress and confusion.

- Thrombocytopenia and petechiae accompany the deterioration.
- What embolic syndrome best unifies the findings?
- Which two injury pathways are involved?

Interpret the mechanism

Time course

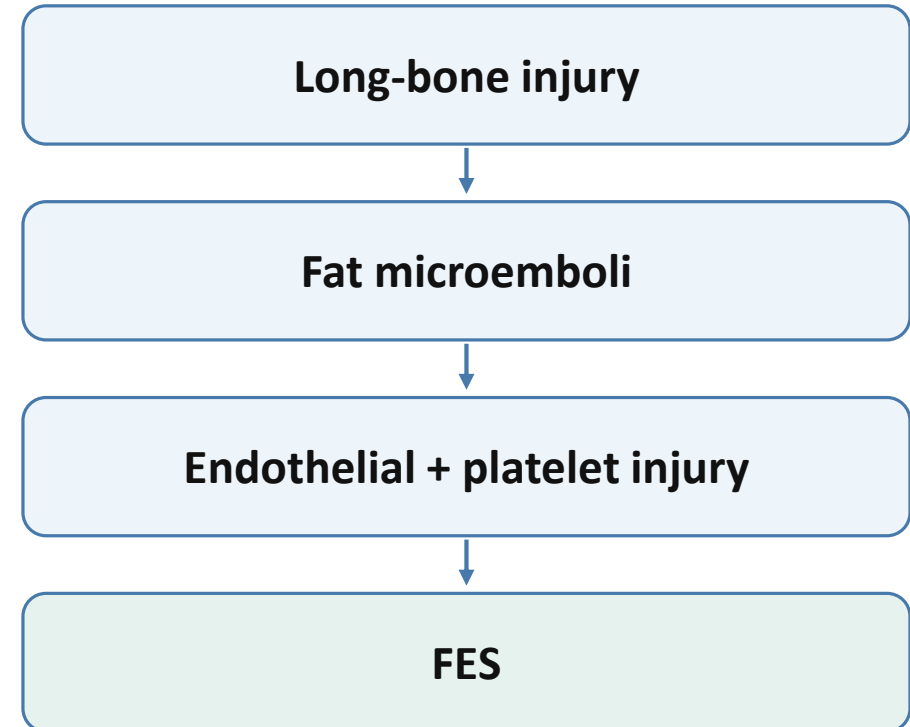
Multiorgan pattern

Mechanism

Case interpretation: Fat embolism syndrome

Marrow fat enters damaged venules and injures the microcirculation.

- The 1–3-day latency is characteristic of the textbook description.
- Mechanical occlusion is supplemented by fatty-acid toxicity.
- Respiratory and cerebral abnormalities combine with cytopenias.



Case: Neurologic symptoms after rapid ascent

A diver ascends quickly and develops joint pain and neurologic disturbances.

- No thrombus formation is required to explain this embolic syndrome.
- Identify the source of intravascular gas.
- Explain why recompression and slow decompression are effective.

Interpret the mechanism

Pressure change

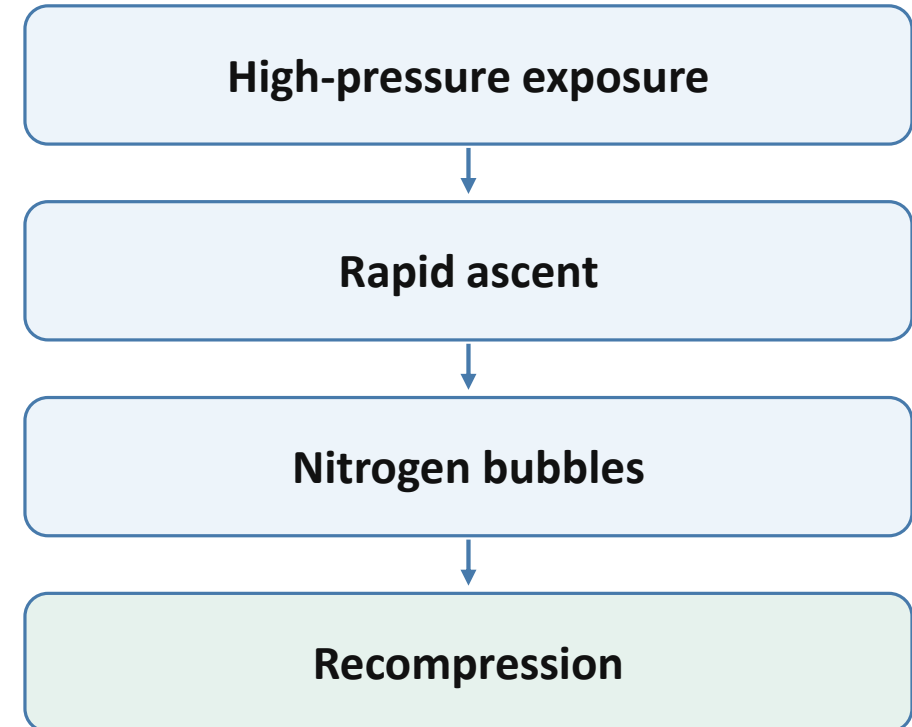
Bubble source

Treatment principle

Case interpretation: Decompression sickness

Nitrogen bubbles emerge as ambient pressure falls too rapidly.

- High-pressure exposure increased dissolved nitrogen.
- Rapid ascent created tissue supersaturation and bubbles.
- Recompression supports redissolution; Controlled ascent permits elimination.



Case: Collapse immediately after delivery

A postpartum patient acutely develops cyanosis, shock, and a coagulopathy.

- Consider a mechanism beyond pulmonary vascular plugging alone.
- What embolic syndrome is characteristic of this setting?
- What accounts for DIC and severe cardiovascular dysfunction?

Interpret the mechanism

Obstetric context

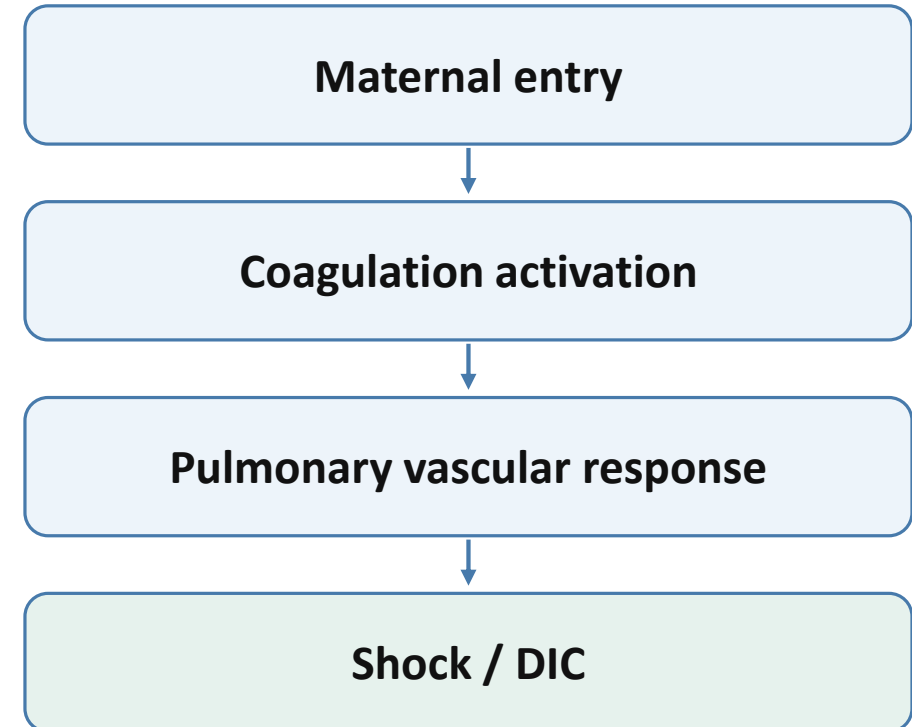
Pulmonary crisis

Coagulopathy

Case interpretation: Amniotic fluid embolism

Amniotic material triggers severe vascular and coagulation responses.

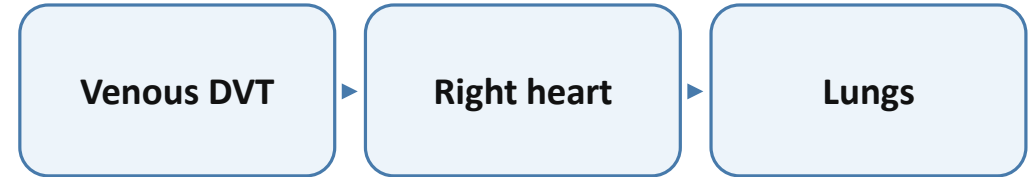
- Entry into maternal blood follows placental or uterine vascular disruption.
- Tissue factor and immune-mediated responses contribute to DIC.
- Vasoactive mediators can induce pulmonary hypertension and RV failure.



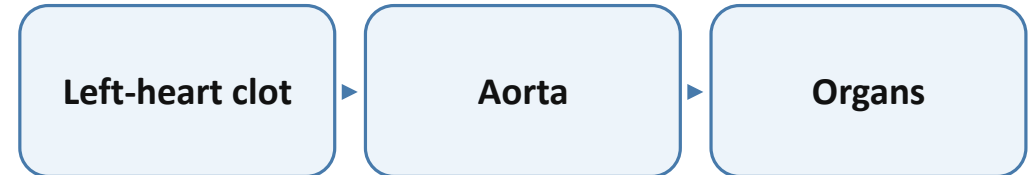
High-yield classification by origin and target

Identify the source before assigning a destination or an outcome.

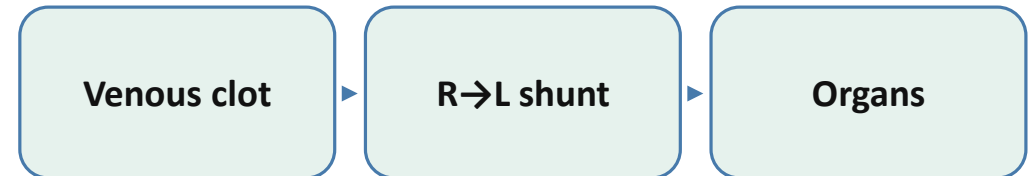
- Venous thrombus → pulmonary artery.



- Left cardiac or aortic source → systemic organ.



- Right-to-left conduit → paradoxical arterial embolus.

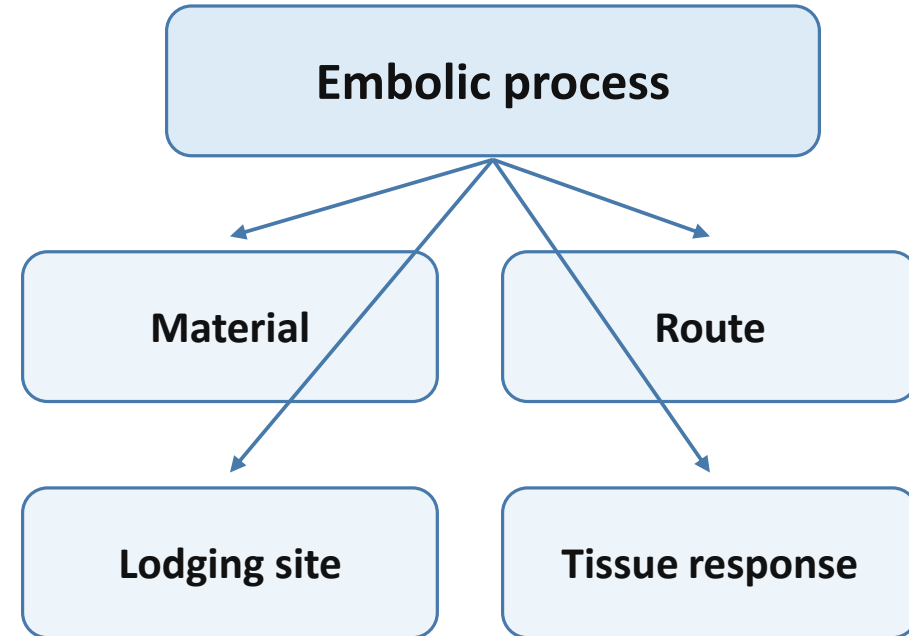


- Fat, gas, and amniotic material demand context-specific pathogenesis.

Five statements to retain

Every embolism case can be reduced to material, route, and injured vascular bed.

- An embolus is detached material transported in blood.
- Most emboli are thromboemboli; Nonthrombotic types matter.
- Anatomic routing predicts the principal organ at risk.
- Collateral circulation explains why occlusion may not cause infarction.
- Fat, gas, and amniotic emboli add mechanisms beyond simple obstruction.



References

Primary textbook reading on embolic pathology.

- Robbins, Cotran & Kumar: Pathologic Basis of Disease. 11th ed. 2026.
- Chapter 4: Hemodynamic Disorders, Thromboembolic Disease, and Shock.
- Sections: thrombosis; embolism; pulmonary and systemic emboli.
- Additional sections: fat, gas, amniotic fluid embolism, and infarction.