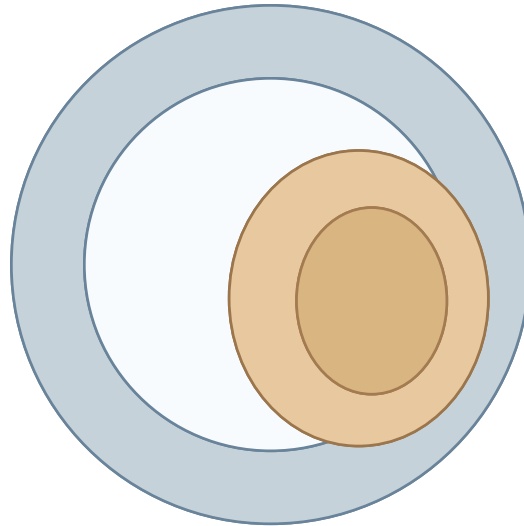


ATHEROSCLEROTIC STENOSIS



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Schematic arterial cross-section
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Learning objectives

- Trace the progression from intimal atheroma to fixed stenosis.
- Explain outward arterial remodeling and its eventual failure.
- Relate lesion architecture and stenosis to chronic tissue ischemia.
- Distinguish fixed flow limitation from sudden plaque disruption.

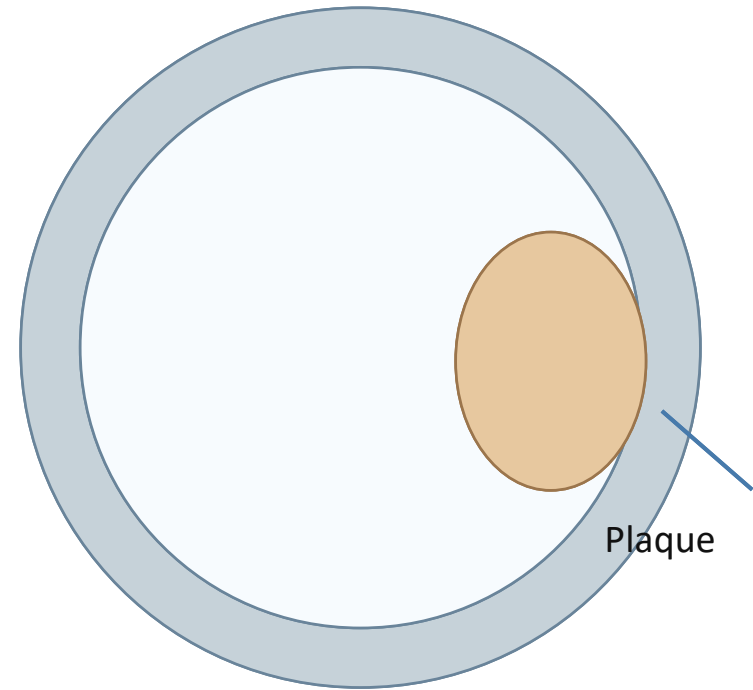
Stenosis is a hemodynamic consequence of plaque growth

- An atheroma occupies the arterial intima and can encroach on the lumen.
- Progressive narrowing can reduce distal blood supply.
- When perfusion becomes inadequate for tissue demand, ischemic injury develops.



Atherosclerosis is an intimal-based arterial disease

- Atheromas contain cells, matrix, lipid, and often calcification.
- Enlarging plaques distort the vessel wall and eventually the blood channel.
- Stenosis is one outcome; Rupture, thrombosis, and aneurysm are others.



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Major arterial beds affected

- Extensive disease involves lower abdominal aorta/iliac, coronary, and popliteal arteries.
- Internal carotid arteries and vessels of the circle of Willis are also involved.
- Mesenteric and renal arterial disease is relatively concentrated at ostia.

Aortoiliac

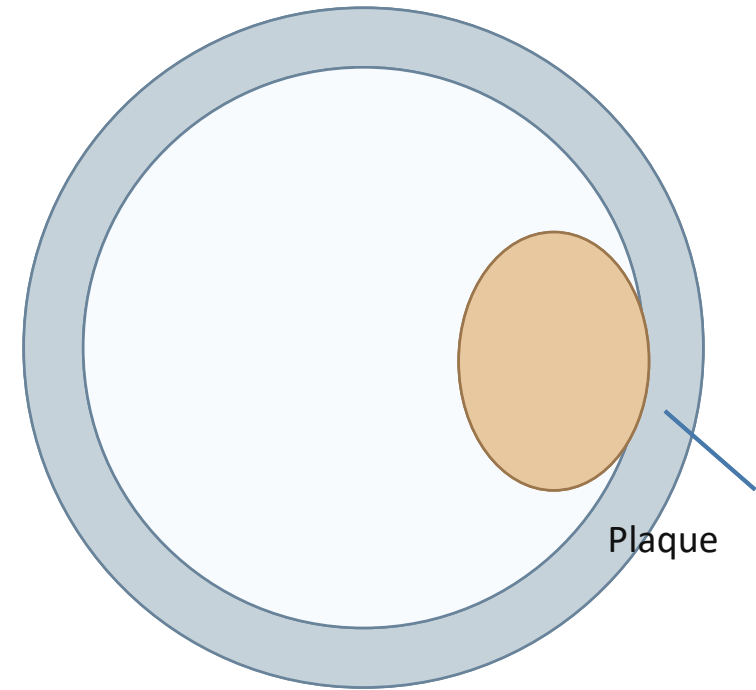
Coronary

Popliteal

Carotid / cerebral

Lesions are patchy rather than uniformly circumferential

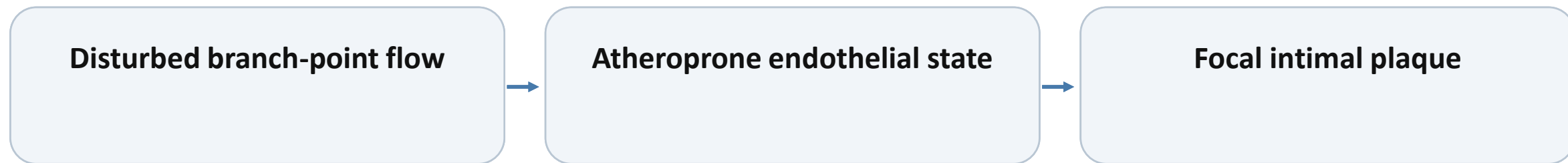
- Most plaques occupy only part of the vessel perimeter.
- Cross-sections therefore commonly show eccentric luminal narrowing.
- Mixed stages of plaque development can coexist along one arterial tree.



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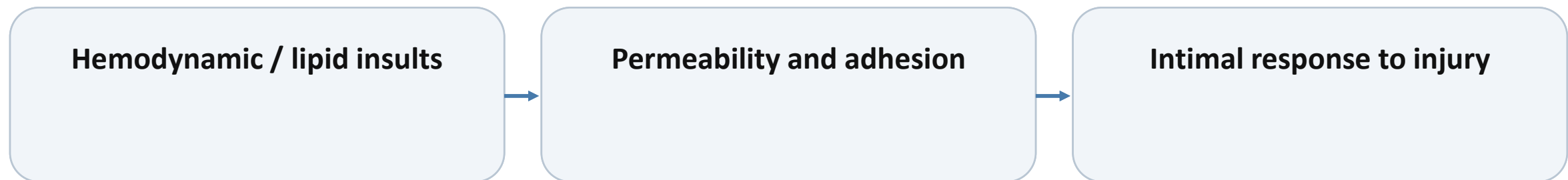
Branch points are plaque-prone

- Hemodynamic flow disturbances localize plaques to ostia and branching sites.
- Nonlaminar flow changes endothelial behavior toward an atheroprone state.
- This helps explain focal disease despite systemic exposure to risk factors.



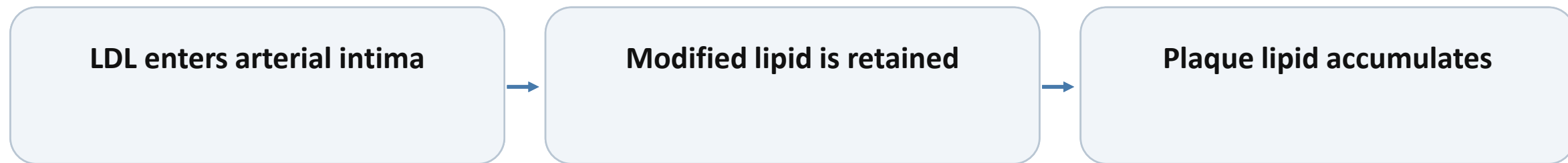
Endothelial dysfunction initiates the response-to-injury

- Endothelium may be morphologically intact but functionally abnormal.
- Increased permeability and leukocyte adhesion favor plaque development.
- Hemodynamic disturbances, hypercholesterolemia, and inflammation are major drivers.



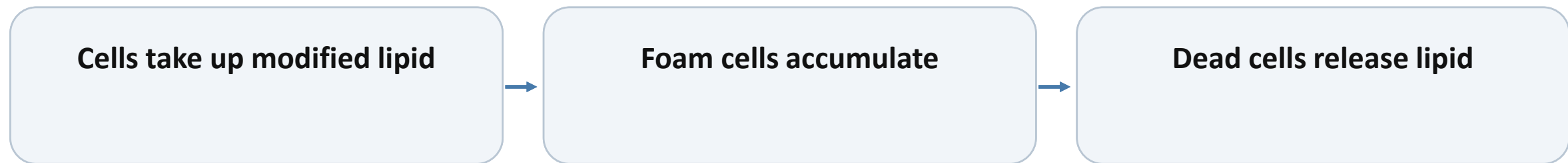
Intimal lipid accumulation sustains lesion growth

- Lipoproteins accumulate within the vessel wall, especially modified LDL.
- Cholesterol influx and insufficient removal favor lipid retention.
- Intracellular and extracellular lipid become structural constituents of atheroma.



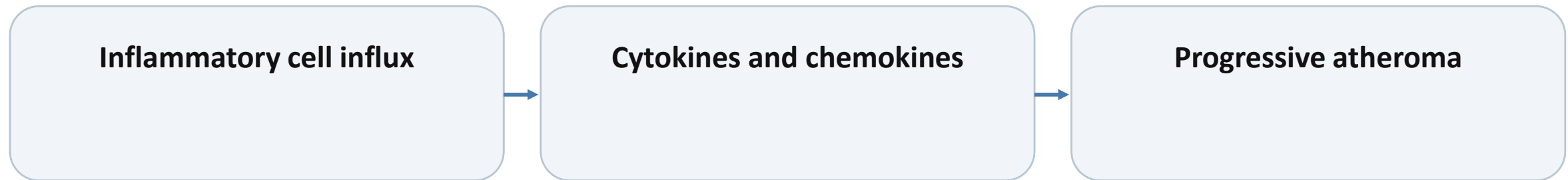
Foam cells contribute to early plaque formation

- Macrophages and smooth muscle cells can accumulate modified lipids.
- Lipid-laden cells are termed foam cells.
- Cell death releases lipid and necrotic debris into the developing plaque.



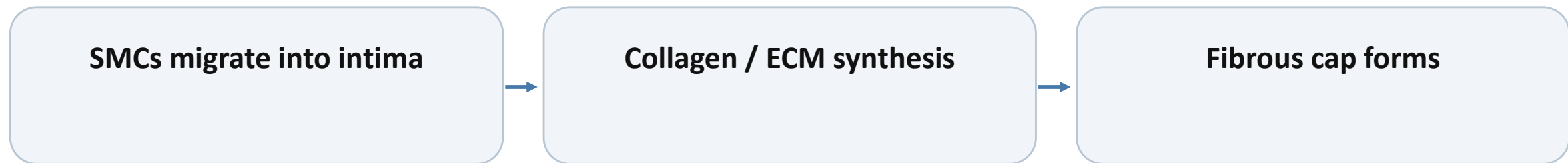
Inflammation sustains atheroma progression

- Monocytes and T lymphocytes enter the atherosclerotic lesion.
- Cytokines and chemokines shape leukocyte and smooth muscle cell activity.
- The process links lipid accumulation with cellular repair and injury.



Smooth muscle cells build a fibrous matrix

- Medial smooth muscle cells migrate into the intima and proliferate.
- They produce extracellular matrix, notably collagen and proteoglycans.
- Matrix synthesis helps generate the fibrous plaque architecture.



Fatty streaks precede some advanced plaques

- Early lesions contain lipid-filled macrophages and appear as yellow streaks.
- They produce little or no important flow disturbance.
- Not every fatty streak progresses to a mature atheroma.



Possible, not inevitable, progression • schematic

Plaque progression is not a single inevitable pathway

- Lesions change as lipid, cells, and matrix accumulate.
- Some become fibrotic and remain comparatively stable.
- Others acquire features that favor sudden plaque disruption.

Intimal lesion

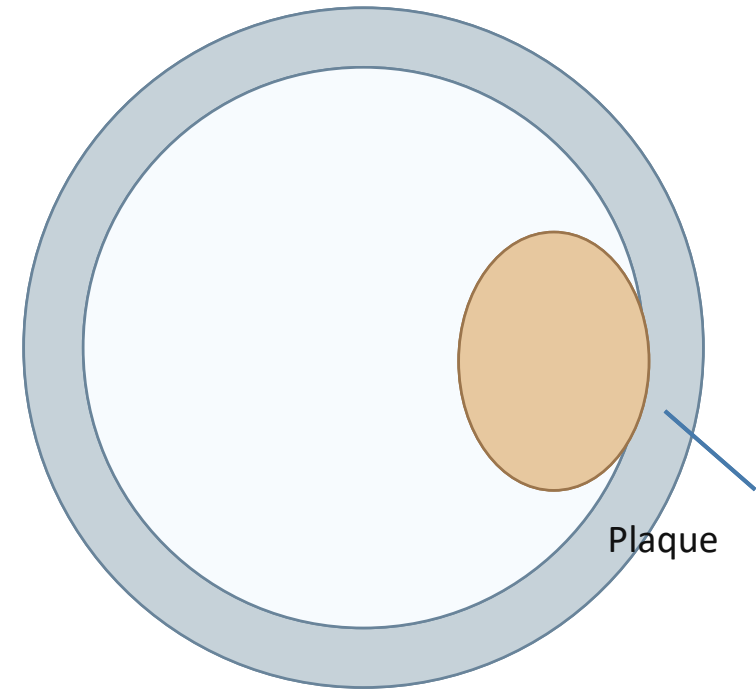
Fibrous plaque

Thin-cap lesion

Alternative plaque outcomes • no necessary sequence

A mature plaque is an intimal mass

- Gross atheromas are yellow-tan and raised above the arterial surface.
- They may coalesce into larger lesions.
- Expansion of this mass is the structural basis for later stenosis.



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Four components define plaque composition

- Cells: Smooth muscle cells, macrophages, and T lymphocytes.
- Extracellular matrix: Collagen, elastic fibers, proteoglycans.
- Intracellular/extracellular lipid and, in later lesions, calcification.

Cells

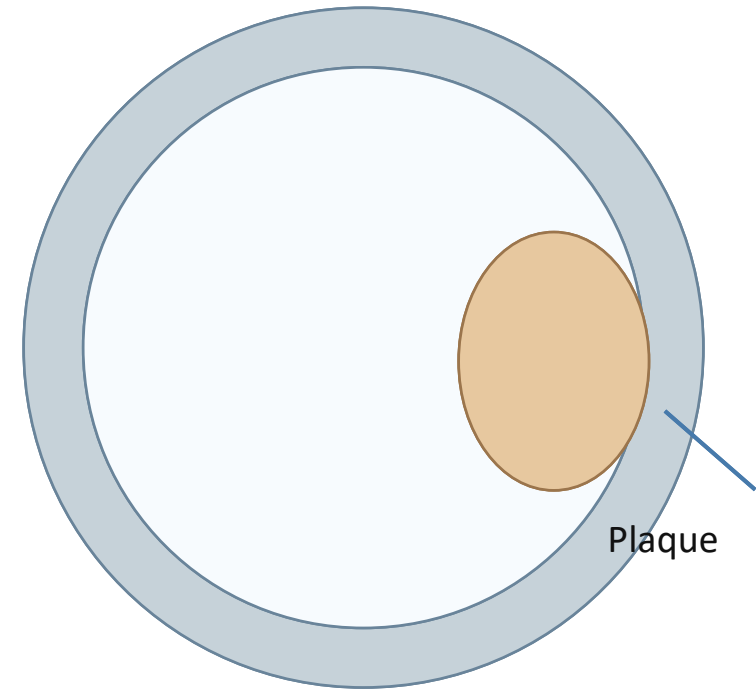
Matrix

Lipids

Calcification

The fibrous cap faces the blood lumen

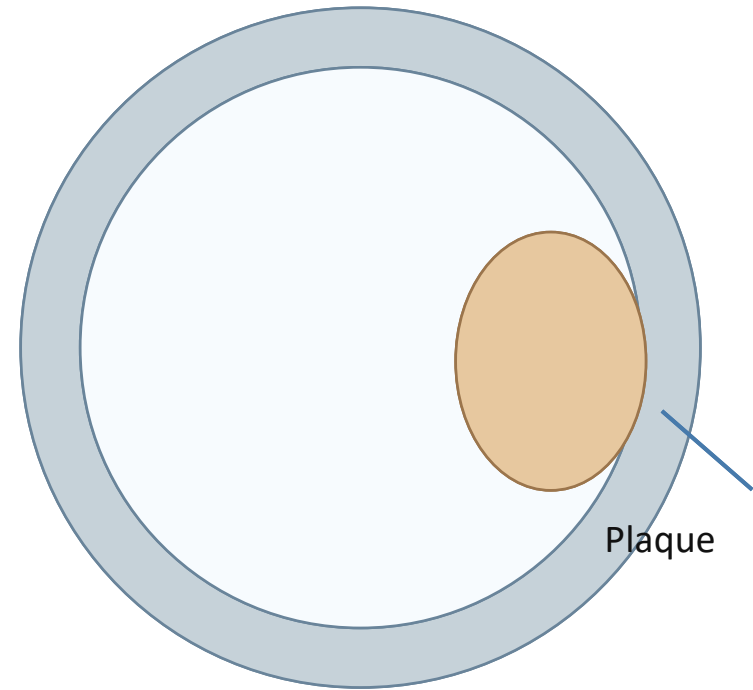
- A superficial layer rich in smooth muscle cells and dense collagen.
- Separates circulating blood from the underlying necrotic core.
- Its matrix content helps determine the mechanical integrity of the plaque.



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The necrotic core lies deep to the cap

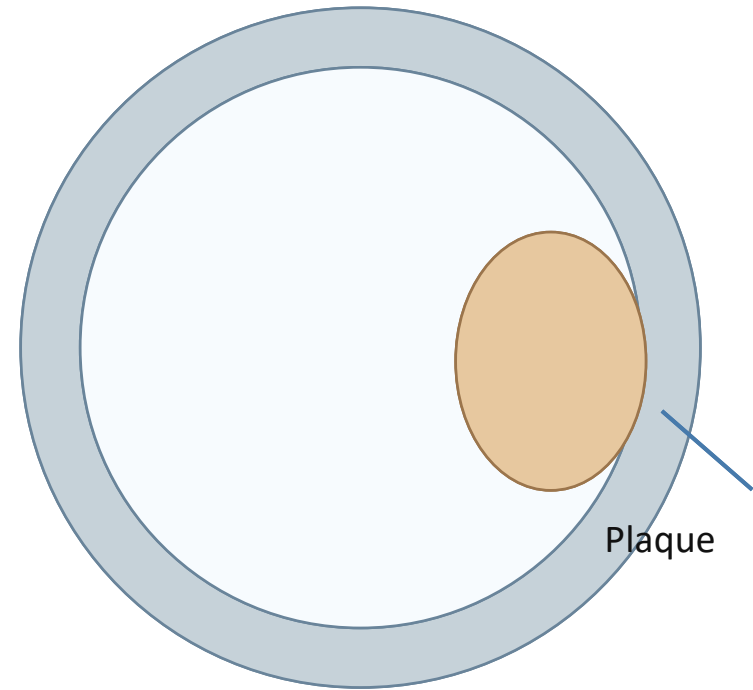
- Contains cholesterol, cholesterol esters, dead-cell debris, and foam cells.
- Fibrin and thrombus-related material may be incorporated.
- Lipid-rich debris occupies intimal space that can expand over time.



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Plaque shoulders are relatively cellular

- Lateral regions near the fibrous cap often contain inflammatory cells.
- Macrophages, T lymphocytes, and smooth muscle cells intermingle there.
- Plaque composition is therefore spatially heterogeneous.



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Cholesterol clefts are a characteristic histologic clue

- Cholesterol can form crystalline aggregates within the necrotic core.
- Routine processing dissolves lipids and leaves empty cleft-like spaces.
- Clefts are morphological evidence of prior plaque lipid accumulation.

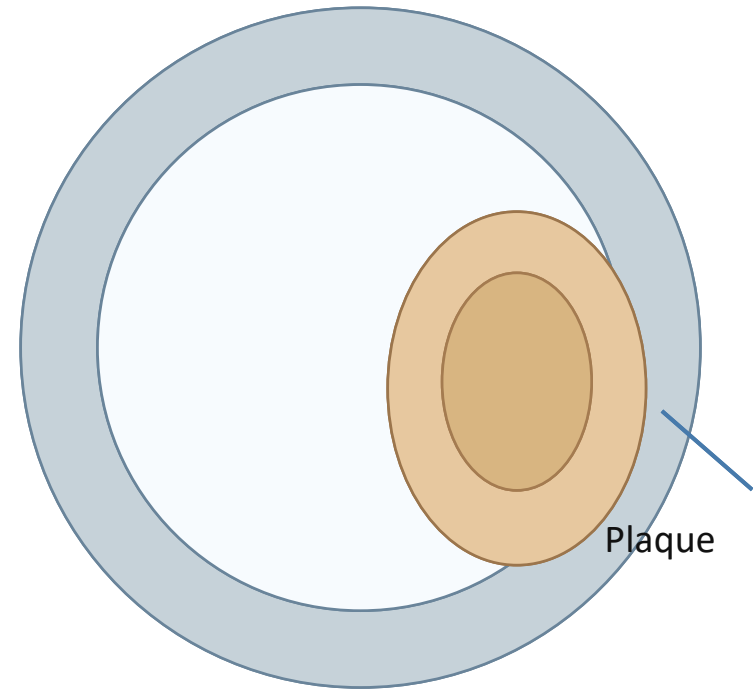
Core lipid

Processing

Empty clefts

Underlying media can become attenuated

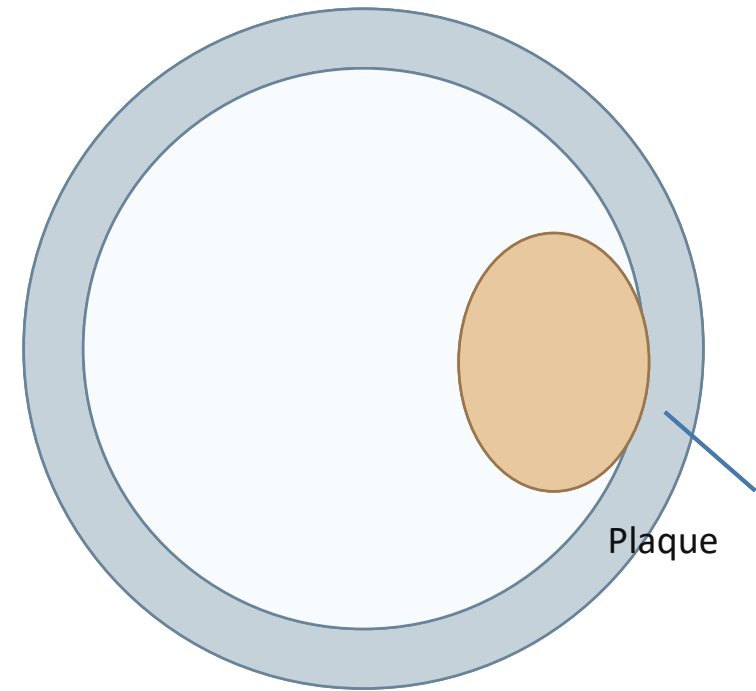
- The expanding plaque can compress the media.
- Smooth muscle cell loss and elastic membrane attenuation may develop.
- Plaque-related wall remodeling affects both lumen and vessel architecture.



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Later plaques may become fibrocalcific

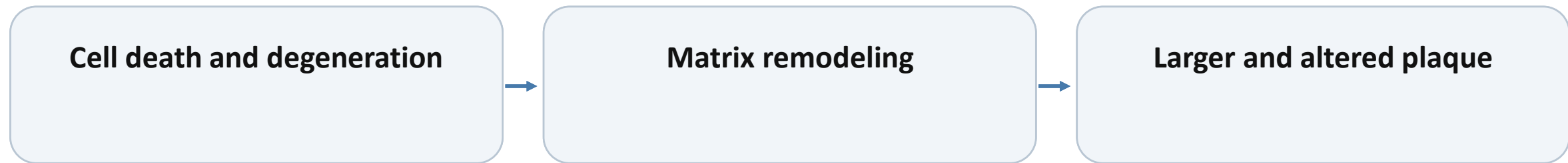
- Calcification can accompany progressive extracellular matrix remodeling.
- Some plaques consist predominantly of smooth muscle cells and fibrous tissue.
- Fibrosis and calcification are not equivalent to complete luminal closure.



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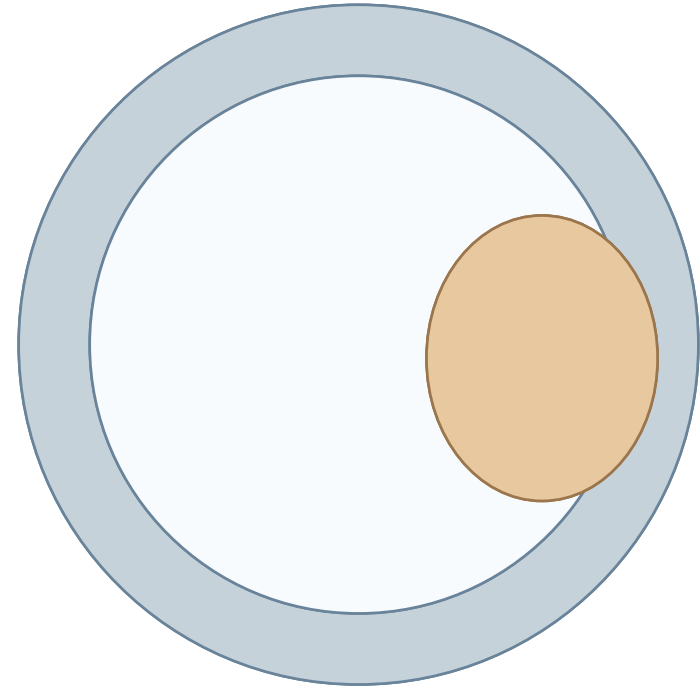
Plaques enlarge through several interacting processes

- Cell death and degeneration add necrotic material.
- Synthesis and breakdown of matrix alter plaque size and texture.
- Organized thrombus and secondary calcification may add to the lesion.



Early plaque enlargement can spare the lumen

- Outward remodeling expands the arterial wall around an enlarging plaque.
- This compensation helps preserve luminal dimensions early.
- The source names this adaptive process the Glagov phenomenon.



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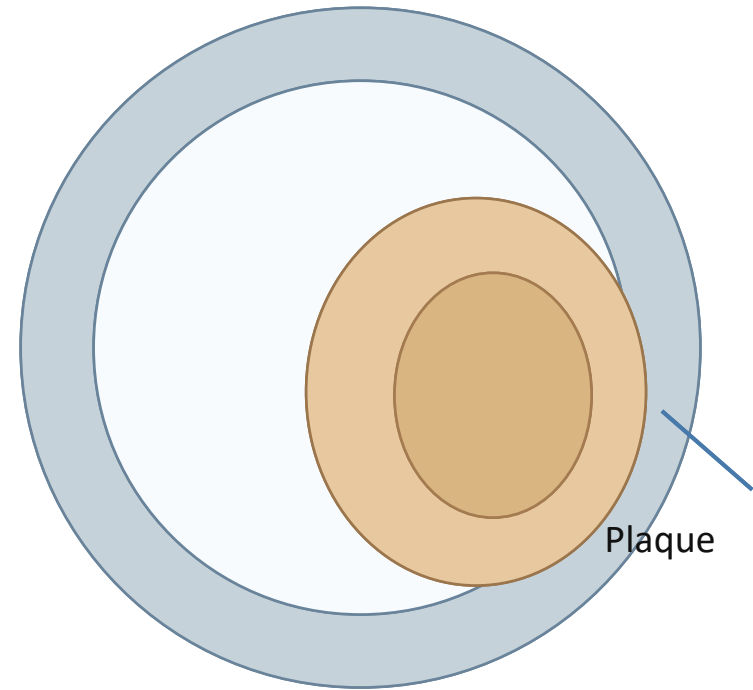
Outward remodeling creates a temporary reserve

- Plaque accumulation initially enlarges the vessel outer boundary.
- The inner channel may appear relatively unchanged despite plaque burden.
- Luminal caliber alone may therefore underrepresent early wall disease.

Feature	Compensated remodeling	Advanced uncompensated plaque
Wall response	Outward expansion	Limited further expansion
Luminal effect	Initially preserved	Progressive encroachment
Perfusion	Relatively maintained	May become inadequate

Remodeling has finite capacity

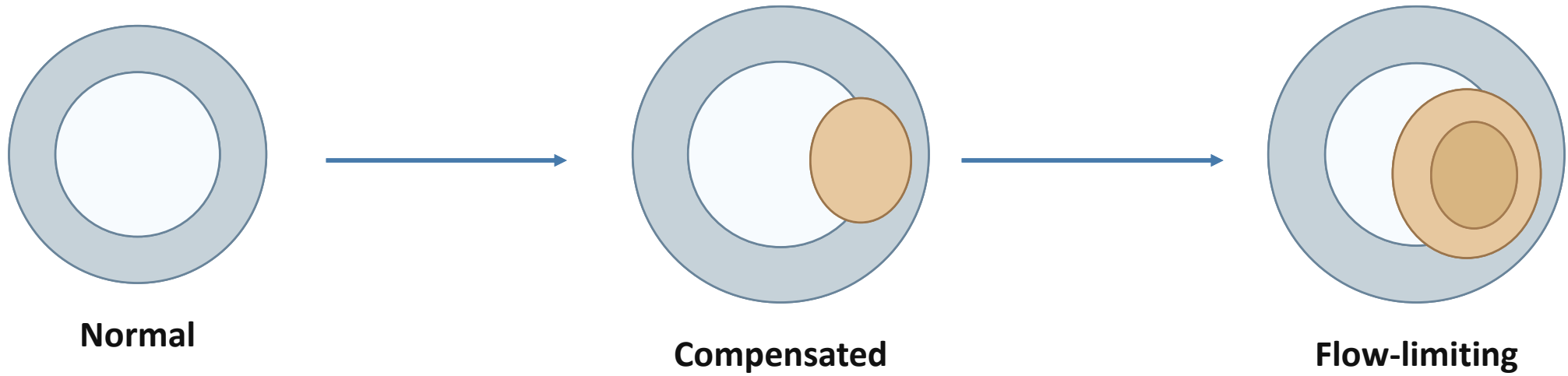
- Continued growth eventually exhausts outward enlargement.
- Further plaque expansion projects into the blood channel.
- The resulting stenosis is accompanied by reduced perfusion reserve.



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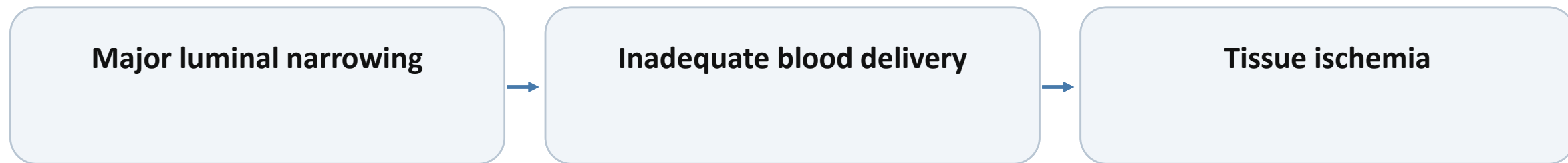
Lumen and wall burden need not change together

- A large plaque can coexist with comparatively preserved lumen early.
- Later, even additional plaque growth can sharply reduce open area.
- The site and stage of remodeling alter the anatomical appearance.



Critical stenosis produces tissue ischemia

- Critical stenosis denotes narrowing sufficient to compromise oxygen delivery.
- It is a physiologic consequence of an anatomical obstruction.
- The threshold depends on vascular territory, collateral flow, and demand.



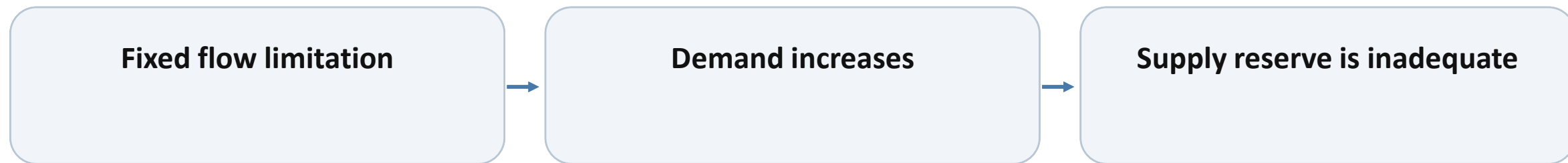
A typical critical area reduction

- In coronary and other circulations, the text gives approximately 70%–75% loss of luminal cross-sectional area.
- This is a textbook contextual approximation, not a universal patient-specific cutoff.
- Do not confuse area reduction with percentage diameter reduction.

Measure	Meaning
Luminal area	Cross-sectional open blood-channel area
Area loss	Reduction relative to an unobstructed reference
Clinical interpretation	Requires flow demand / collateral context

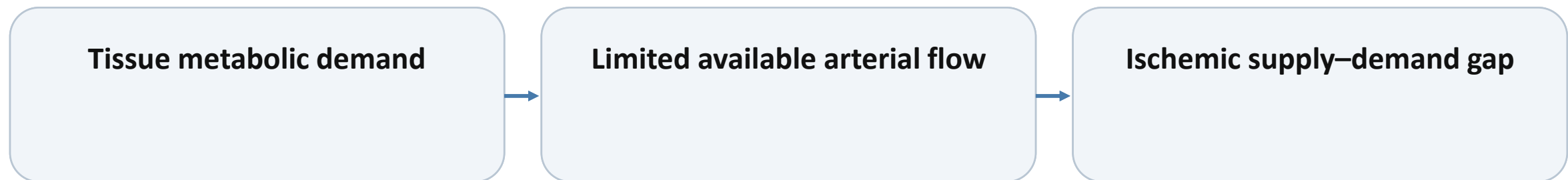
Narrowing matters because it limits flow reserve

- Obstruction restricts the delivery of blood downstream.
- Symptoms are particularly evident when tissue demand increases.
- Baseline perfusion can be relatively adequate before exertion.



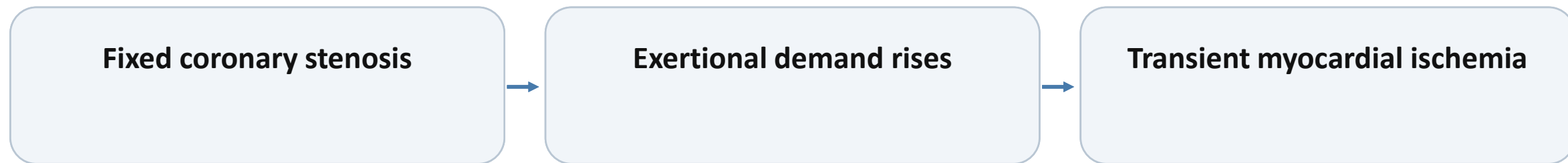
Supply–demand mismatch is the immediate mechanism

- Nutrient and oxygen supply depend on arterial perfusion.
- Metabolic demand changes with organ activity.
- Ischemia occurs when demand exceeds the supply the stenosed circulation can deliver.



Coronary stenosis may present as exertional angina

- Coronary flow-limiting plaques may supply sufficient blood at rest.
- Exercise increases myocardial metabolic requirements.
- Transient mismatch can produce stable angina.



Stable angina links obstruction with reproducible stress

- A fixed lesion limits augmentation of coronary flow.
- Provocation occurs when myocardial demand increases.
- The symptom reflects ischemia, not necessarily an acute plaque rupture.

Feature	Resting state	Exertional state
Demand	Lower	Increased
Flow limitation	Can be compensated	May become apparent
Consequence	Often no pain	Possible angina

Chronic cardiac ischemia can accumulate damage

- Progressive stenosis causes chronically diminished myocardial perfusion.
- The source lists chronic ischemic heart disease as a consequence.
- This is distinct from acute thrombotic infarction after plaque change.

Plaque

Narrowing

Hypoperfusion

Chronic ischemia

The coronary bed is only one target

- Atherosclerotic stenoses can compromise multiple arterial circulations.
- Symptoms depend on the tissues supplied by the obstructed artery.
- The same vascular mechanism yields different organ manifestations.

Heart

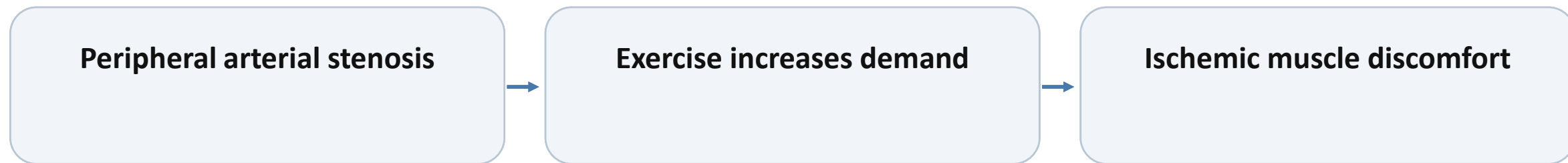
Brain

Bowel

Limbs

Intermittent claudication reflects limb flow limitation

- Peripheral arterial stenosis reduces perfusion to working limb muscles.
- Walking increases oxygen demand relative to available flow.
- Ischemic discomfort is a clinical consequence of this mismatch.



Atherosclerosis can compromise cerebral perfusion

- Cerebral arterial supply is a major site of symptomatic disease.
- Persistent flow limitation is associated with ischemic encephalopathy.
- Local distribution and available collaterals influence tissue effects.

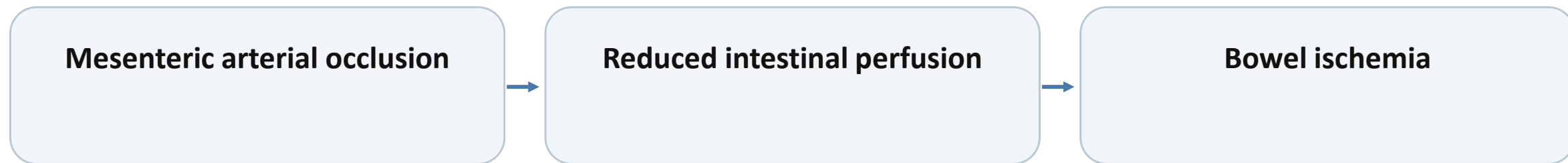
Arterial lesion

Reduced supply

Cerebral ischemia

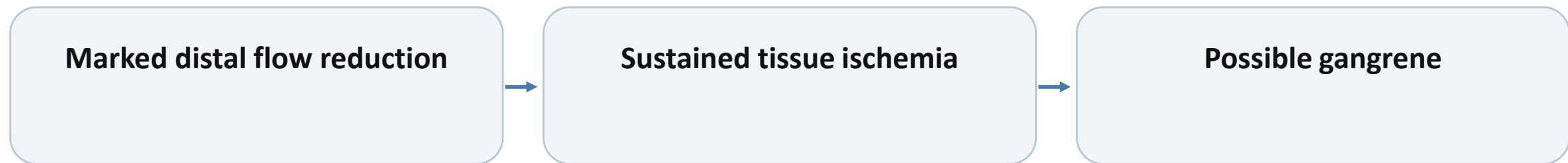
Mesenteric disease can produce bowel ischemia

- Mesenteric arterial occlusion is among the consequences described.
- Bowel injury reflects inadequate arterial delivery to intestinal tissue.
- The degree of collateralization modifies severity.



Peripheral disease can progress to tissue loss

- Severe arterial insufficiency may result in ischemic injury of the extremity.
- The parent section identifies gangrene of the legs as a major complication.
- Extent of injury depends on residual supply and tissue requirements.



Renal arteries are not uniformly involved

- Symptomatic atherosclerotic disease can involve the renal circulation.
- Relative sparing of renal arteries except at their origins.
- An ostial lesion illustrates why anatomical site matters.

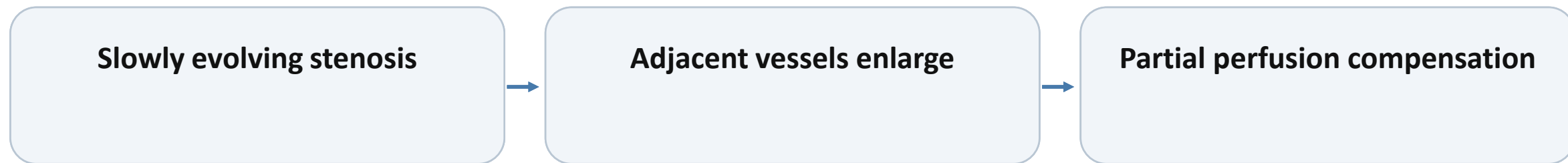
Aortic branch

Renal ostium

Renal perfusion

Collateral arteries modify the consequences

- When a stenosis develops slowly, adjacent smaller arteries may enlarge.
- Newly effective collateral routes partially preserve distal perfusion.
- Collateral compensation is limited and varies between organs.



Rate of narrowing affects the adaptive response

- Gradual obstruction can permit recruitment and enlargement of collateral pathways.
- Sudden occlusion may leave less opportunity for compensation.
- Chronology of vessel obstruction is clinically important.

Feature	Slow stenosis	Acute occlusion
Time course	Gradual	Sudden
Adaptation	Collateral enlargement possible	Limited time to adapt
Possible effect	Chronic ischemia	Acute ischemic injury

Vessel anatomy changes the effect of equal narrowing

- The territory supplied by the artery determines exposed tissue.
- Existing alternative routes determine collateral supply.
- Organ metabolic demand determines whether residual supply is sufficient.

Supply territory

Collateral routes

Metabolic demand

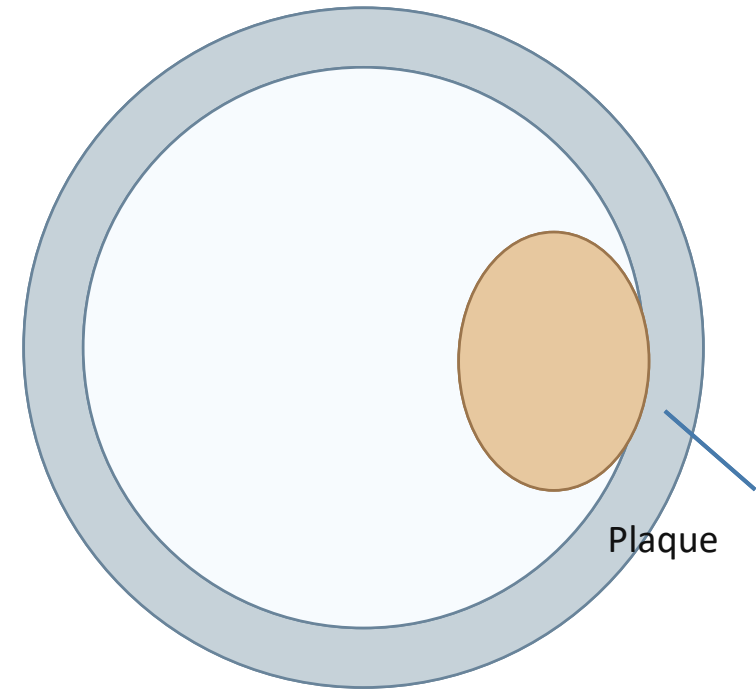
Stenosis and plaque instability are distinct properties

- Plaque volume and luminal encroachment govern fixed obstruction.
- Fibrous cap composition and inflammation influence rupture propensity.
- A noncritical lesion may still undergo sudden destabilization.

Feature	Fixed stenosis	Plaque vulnerability
Principal property	Degree of narrowing	Cap structure / inflammation
Common effect	Chronic hypoperfusion	Sudden plaque change
Relationship	Can coexist	Not defined by stenosis alone

Stable plaques are not necessarily harmless

- A relatively thick fibrous cap may make rupture less likely.
- The same lesion can progressively occupy the vessel lumen.
- A stable, fibrotic plaque may therefore still cause critical stenosis.



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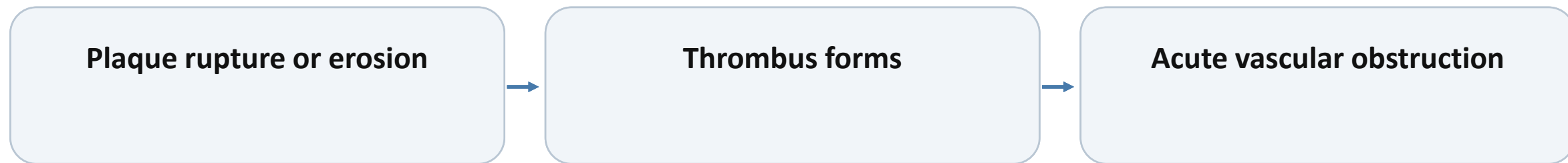
The fibrous cap controls a different clinical risk

- Collagen-rich caps increase mechanical integrity.
- Thin caps over lipid-rich cores are more prone to sudden rupture.
- Neither feature alone directly measures the severity of luminal narrowing.

Feature	Thick fibrous cap	Thin fibrous cap
Structural feature	More collagen / matrix	Less supporting matrix
Clinical tendency	Greater stability	Greater rupture susceptibility
What is not implied	Normal lumen	Critical stenosis

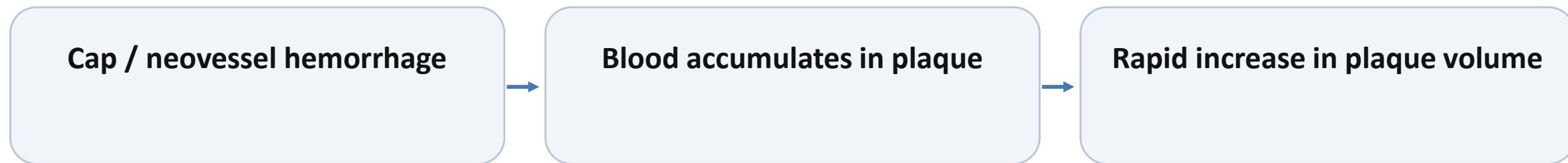
Acute plaque change is a different pathway to occlusion

- Rupture or erosion exposes thrombogenic plaque constituents.
- Superimposed thrombus can rapidly obstruct an artery.
- This abrupt event differs from slow luminal loss due to plaque enlargement.



Intraplaque hemorrhage can suddenly expand a lesion

- Bleeding may arise from the cap or neovessels within the plaque.
- A contained hematoma adds volume to the atheroma.
- This secondary change is not synonymous with gradual fibrotic stenosis.



Chronic ischemia versus acute infarction

- Fixed stenosis mainly restricts sustained or demand-related flow.
- Plaque rupture with thrombosis can cause acute arterial obstruction.
- Organ injury may be chronic, episodic, or abrupt depending on mechanism.

Feature	Progressive narrowing	Thrombotic plaque event
Mechanism	Plaque encroachment	Rupture / erosion plus thrombosis
Typical tempo	Gradual	Abrupt
Illustrative outcome	Stable angina / claudication	Acute tissue infarction

A modest lumen defect does not rule out acute risk

- The adjacent source notes many abruptly complicated coronary plaques were previously noncritical.
- Thus luminal stenosis and risk of rupture cannot be treated as identical.
- A purely geometric interpretation of atherosclerosis is incomplete.



Morphology and clinical consequences must be paired

- Macroscopic plaque burden establishes chronic arterial disease.
- Cross-sectional architecture demonstrates eccentric narrowing and media remodeling.
- Functional effects require considering the downstream organ and supply reserve.

Plaque morphology

Lumen geometry

Distal demand

Case exercise: Exertional chest discomfort

CLINICOPATHOLOGIC APPLICATION

- A patient develops chest discomfort during walking uphill.
- At rest, myocardial demand is lower and symptoms may resolve.
- Explain how a fixed coronary atheroma can account for this pattern.

Reason through the mechanism; Answer in presenter notes.

Case analysis: What remodeling explains

CLINICOPATHOLOGIC APPLICATION

- An eccentric coronary plaque is large, but the lumen is initially relatively preserved.
- Later sections show a smaller lumen despite only incremental plaque growth.
- Identify the compensatory process and why it becomes inadequate.

Reason through the mechanism; Answer in presenter notes.

Case exercise: Exercise-related calf discomfort

CLINICOPATHOLOGIC APPLICATION

- A patient experiences calf pain during walking that is absent at rest.
- The popliteal artery has marked plaque-related narrowing.
- Connect the clinical pattern to muscle oxygen demand and blood supply.

Reason through the mechanism; Answer in presenter notes.

Case exercise: Chronic mesenteric flow limitation

CLINICOPATHOLOGIC APPLICATION

- A patient has extensive atherosclerotic disease involving mesenteric vessel origins.
- A distal intestinal territory receives limited arterial perfusion.
- Predict the central tissue-level consequence without assuming acute plaque rupture.

Reason through the mechanism; Answer in presenter notes.

Case analysis: Narrowing is not the whole story

CLINICOPATHOLOGIC APPLICATION

- Two patients have similar anatomical stenoses but different degrees of ischemia.
- One has slowly enlarging collateral routes around the obstruction.
- What additional variables should be evaluated conceptually?

Reason through the mechanism; Answer in presenter notes.

Case analysis: Stable plaque versus acute event

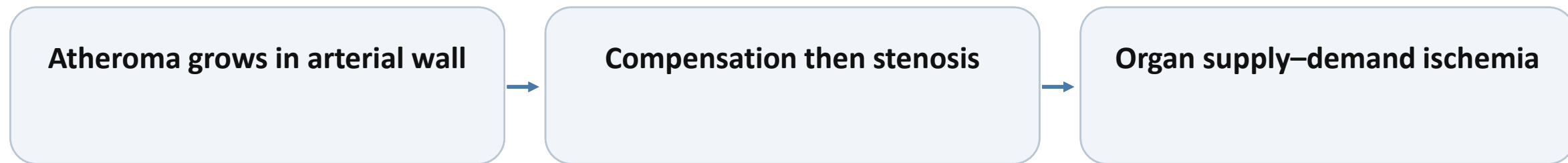
CLINICOPATHOLOGIC APPLICATION

- One lesion causes exertional angina through longstanding fixed narrowing.
- Another previously noncritical plaque ruptures and becomes thrombosed.
- Explain the different mechanisms rather than judging both by stenosis alone.

Reason through the mechanism; Answer in presenter notes.

Interpret the lesion from wall to organ

- Endothelial dysfunction, lipid retention, inflammation, and matrix deposition build an atheroma.
- Outward remodeling masks early luminal effects; Later growth causes obstruction.
- Clinical injury emerges according to vascular supply, collaterals, and demand.



Three distinctions prevent common errors

- Plaque burden is not identical to luminal stenosis.
- Anatomic stenosis is not identical to tissue ischemia.
- Plaque instability is not identical to critical fixed narrowing.

Burden $\neq$ narrowing

Narrowing $\neq$ ischemia

Instability $\neq$ stenosis

Reference and source boundaries

- Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th ed. (2026), Ch. 11.
- Core: “Atherosclerotic Stenosis,” pp. 456–458; parent morphology and pathogenesis, pp. 452–456.
- Adjacent “Acute Plaque Change,” p. 458, used only for mechanism contrasts.
- Anatomical drawings are editable schematics, not diagnostic photomicrographs.