

Chronic Arterial Occlusion

Fixed epicardial coronary stenosis in ischemic heart disease

Mechanism • morphology • coronary reserve • collateral perfusion

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Learning outcomes

- Explain coronary ischemia as a mismatch between perfusion and myocardial oxygen demand.
- Distinguish fixed epicardial stenosis, flow reserve, and collateral compensation.
- Interpret anatomic distribution and the clinical significance of severe chronic lesions.
- Differentiate chronic exertional ischemia from acute plaque complications.

Scope: A coronary lesion in ischemic heart disease

The indexed heading belongs to the heart chapter, under ischemic heart disease (IHD).

Fixed coronary atherosclerosis

Progressive flow limitation

Acute plaque disruption

Rupture / erosion $\pm$ thrombus

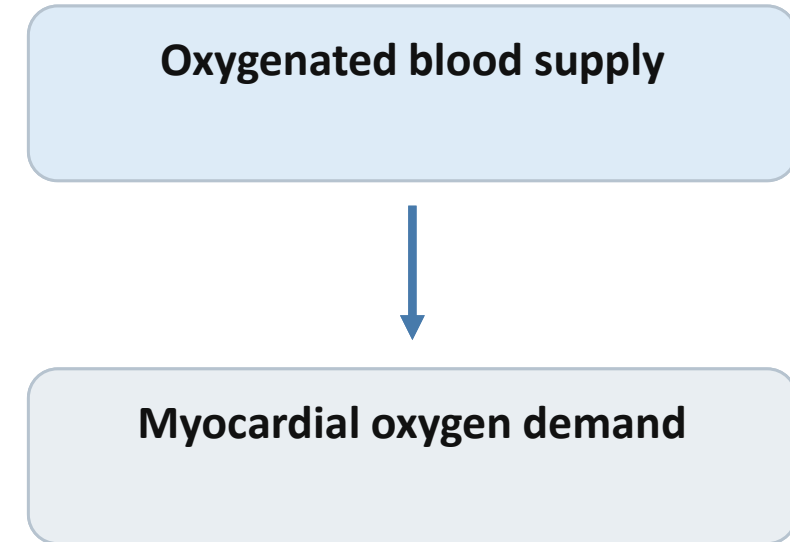
Vasospasm

Dynamic arterial constriction

Chronic fixed occlusion is one mechanism within the broader IHD spectrum.

Myocardial ischemia is a supply–demand imbalance

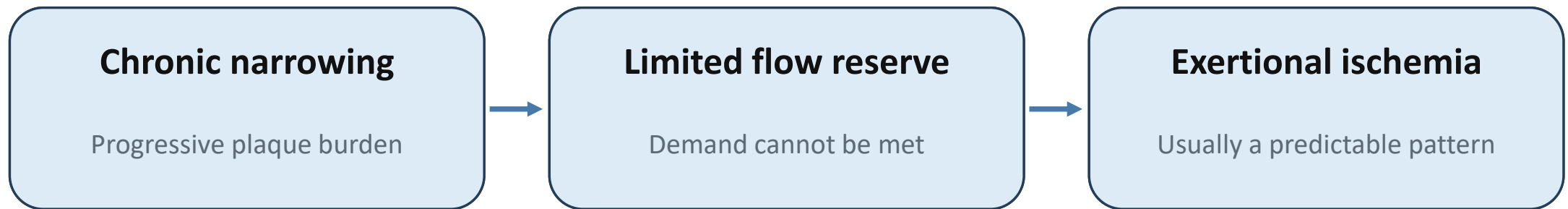
- IHD results when oxygenated coronary perfusion is insufficient for myocardial requirements.
- Fixed narrowing reduces capacity to augment flow when cardiac work rises.
- The same lesion may be tolerated at rest yet produce ischemia during activity.



Ischemia when perfusion is insufficient for demand

Schematic

Progressive narrowing: The route to demand ischemia



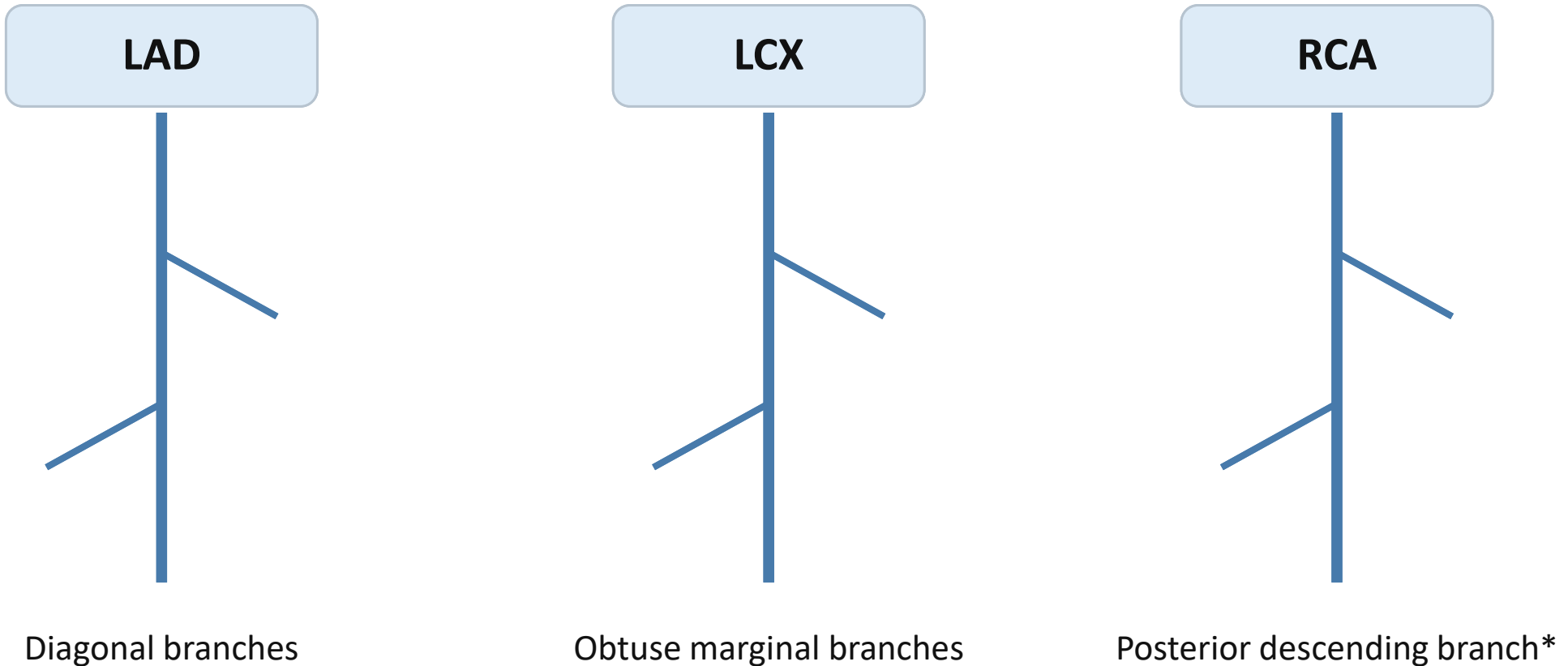
Schematic

Atherosclerosis predominates in IHD

- More than 90% of patients with IHD have atherosclerosis of ≥ 1 epicardial coronary artery.
- This is a statement about the patients with IHD—not the probability that any plaque causes symptoms.
- The functional consequences depend on stenosis, perfusion reserve, and collateral circulation.

Epicardial arteries are the main conduit targets

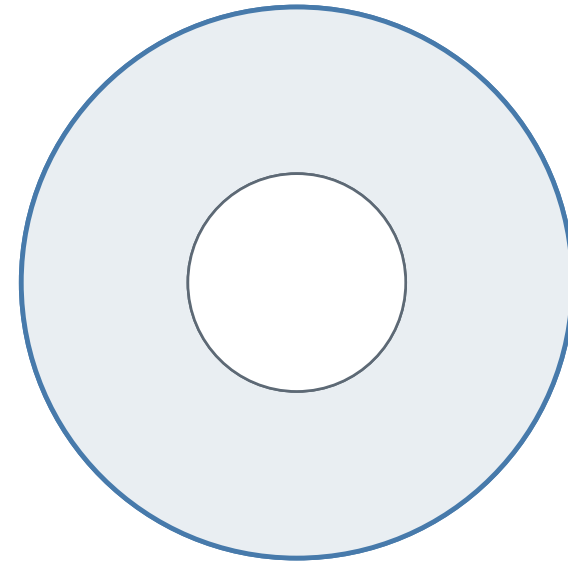
Large surface coronary arteries distribute blood to downstream myocardium.



Coronary branches shown as a conceptual tree, not to anatomical scale.

A plaque narrows the patent arterial lumen

- Atheroma forms within the arterial intima and displaces the blood-filled channel.
- Flow limitation arises when compensatory vessel enlargement is no longer sufficient.
- The relevant measurement is the residual lumen—not simply visible plaque size.

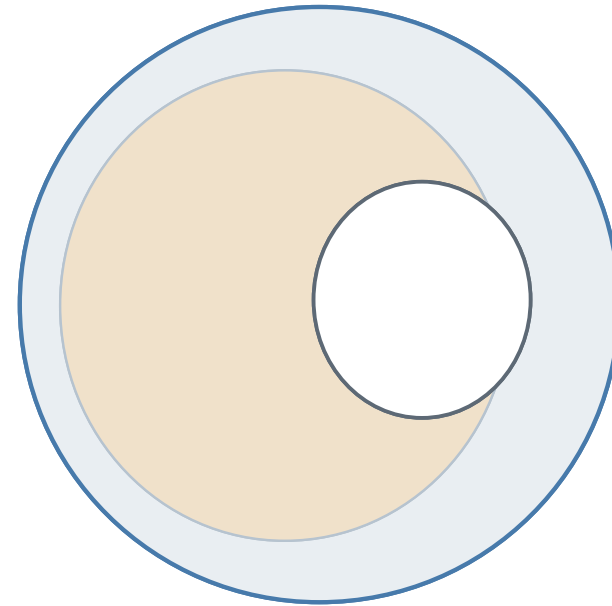


Remaining patent lumen

Schematic

An eccentric coronary atheroma

- An atheroma with a plaque-free wall segment and a compromised lumen.
- The cap overlies a lipid-rich necrotic core; The subjacent media may be attenuated.
- Eccentric geometry means that plaque burden can be uneven around the circumference.

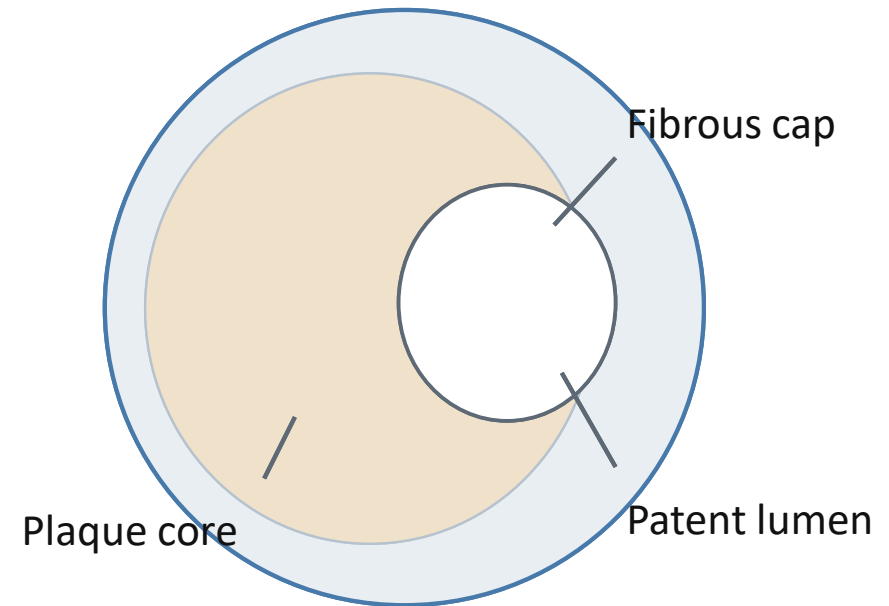


Eccentric plaque with lumen displacement

Schematic

Fibrous cap and atheromatous core

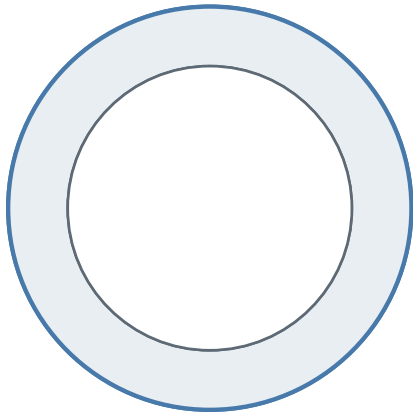
- A coronary atheroma contains a fibrous cap and an underlying lipid-rich necrotic core.
- The wall may show inflammatory cells, calcification, and neovascularization.
- Histologic architecture explains structural plaque behavior; It is not itself a flow measurement.



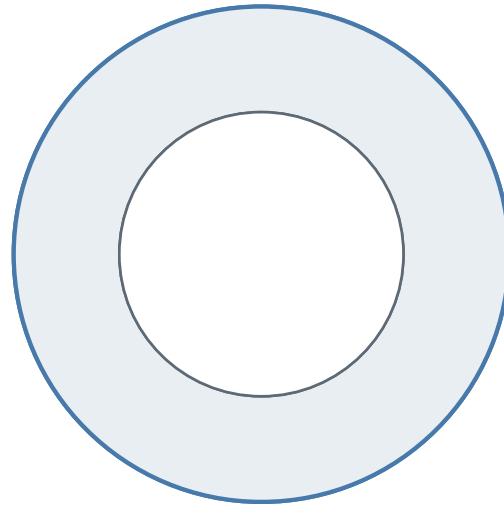
Schematic

Early outward remodeling preserves the lumen

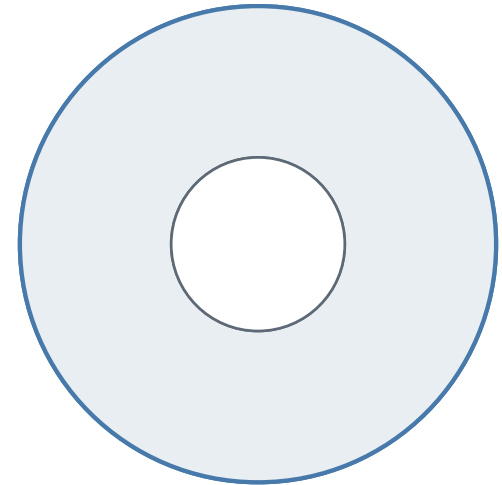
At early plaque stages, outward enlargement of the arterial wall can preserve luminal size.



Early plaque



Outward remodeling

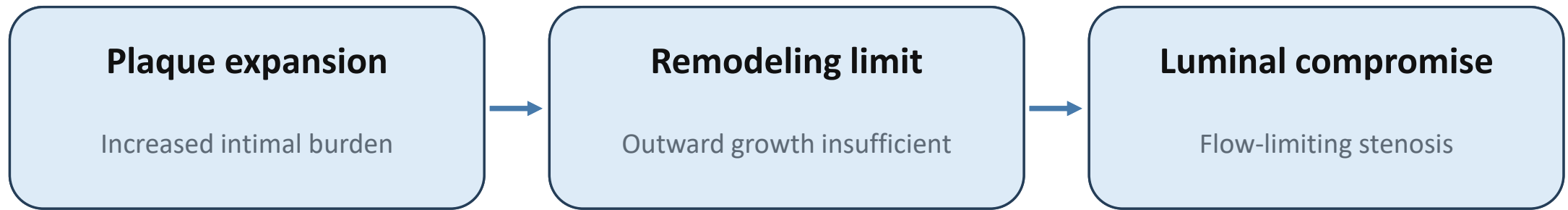


Remodeling limit

Initial outward vessel enlargement can preserve the lumen; Ultimately plaque growth narrows it.

Compensation by remodeling has a limit

As the plaque expands further, the artery can no longer accommodate it by outward expansion.



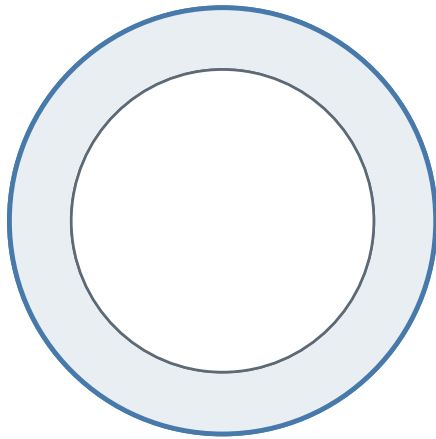
Schematic

A fixed stenosis is different from sudden closure

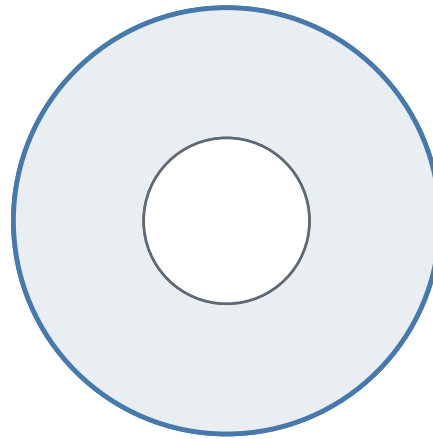
Characteristic	Chronic fixed lesion	Acute plaque complication
Time course	Develops gradually	Abrupt change may occur
Mechanism	Plaque-related narrowing	Disruption $\pm$ thrombosis
Typical consequence	Predictable demand ischemia	Acute coronary syndromes
Collateral response	May develop gradually	May be inadequate acutely

The 70% threshold refers to lumen AREA

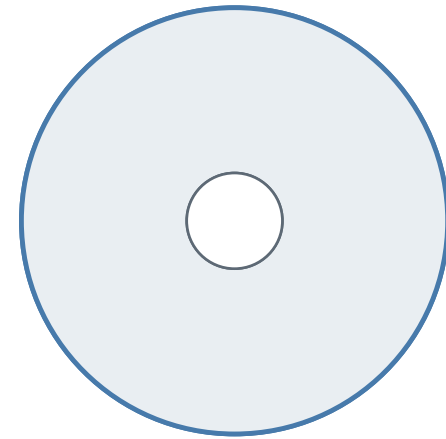
A reduction exceeding approximately 70% of lumen cross-sectional area is termed critical stenosis.



Unobstructed



70% area loss



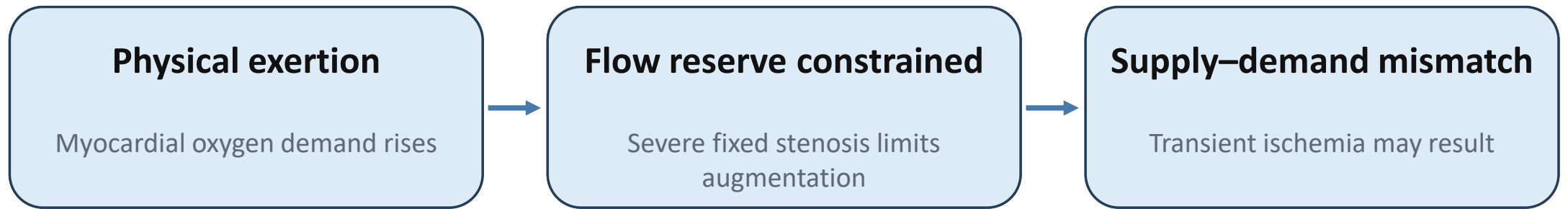
90% area loss

Idealized cross-sections: The percentages refer to lost luminal AREA, not diameter.

Schematic

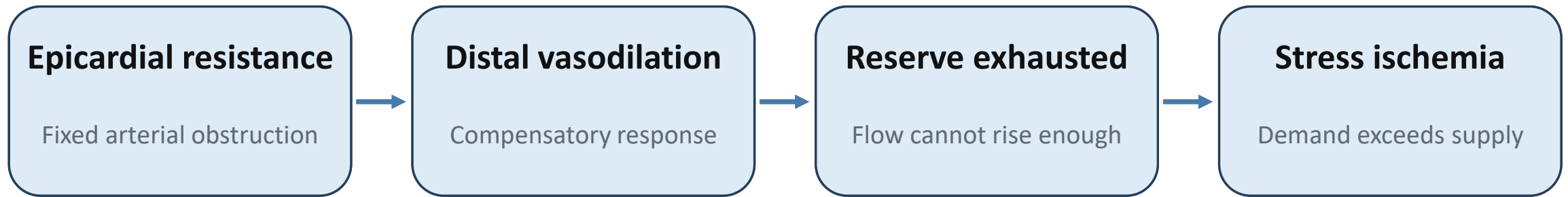
Critical stenosis: Exertional ischemia becomes likely

With a fixed obstruction greater than approximately 70% of luminal area, demand-induced ischemia often becomes symptomatic.



Functional limitation emerges when demand rises beyond available supply.

Why vasodilation can no longer compensate



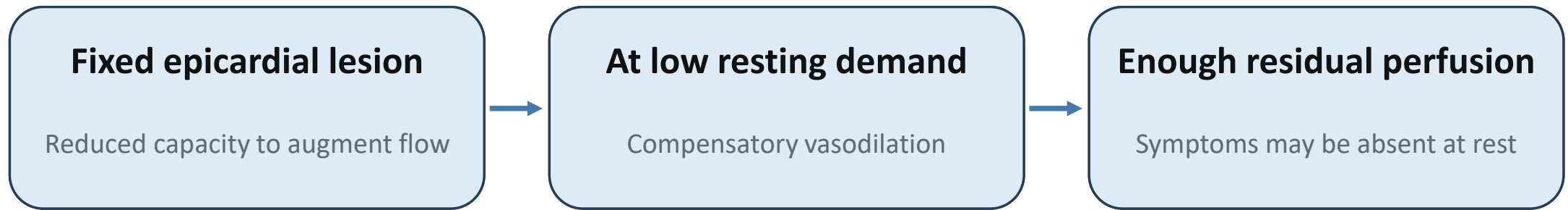
Schematic

Rest and exertion interrogate different reserves

Condition	Flow requirement	Significance of fixed lesion
Rest	Lower metabolic demand	Residual flow may remain adequate
Exertion	Higher myocardial demand	Need for additional perfusion
Severe fixed stenosis	Ability to augment flow restricted	Ischemia may occur before rest perfusion fails

At rest, severe stenosis can remain clinically quiet

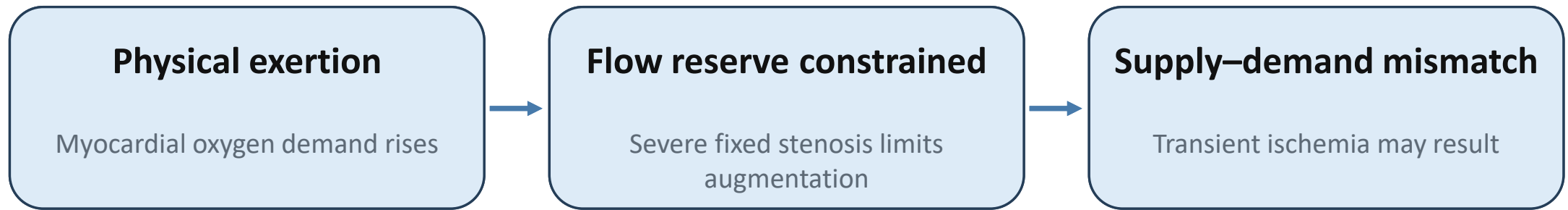
A fixed atherosclerotic lesion may not cause symptoms when demand is low.



Adequacy at rest does not establish normal reserve during exertion.

During exertion, flow reserve is tested

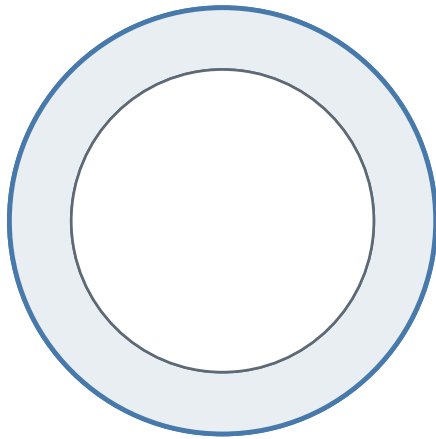
Physical activity increases myocardial oxygen demand.



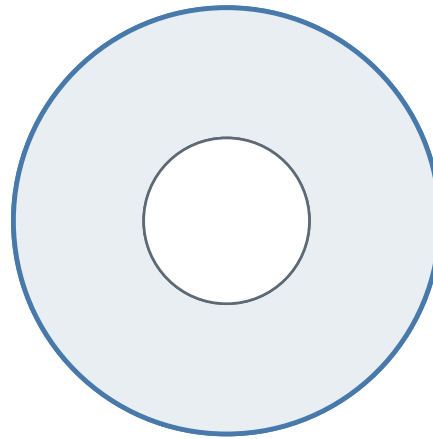
Functional limitation emerges when demand rises beyond available supply.

Severe fixed stenosis can compromise resting flow

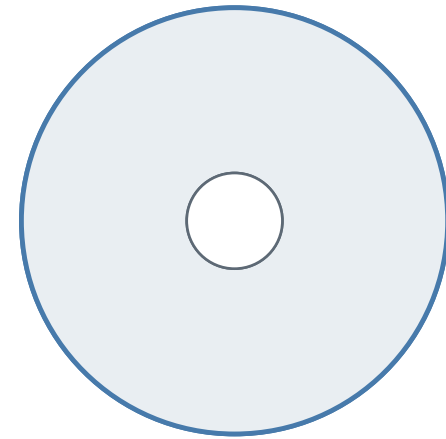
Obstruction of about 90% of luminal cross-sectional area generally reduces coronary flow even at rest.



Unobstructed



70% area loss



90% area loss

Idealized cross-sections: The percentages refer to lost luminal AREA, not diameter.

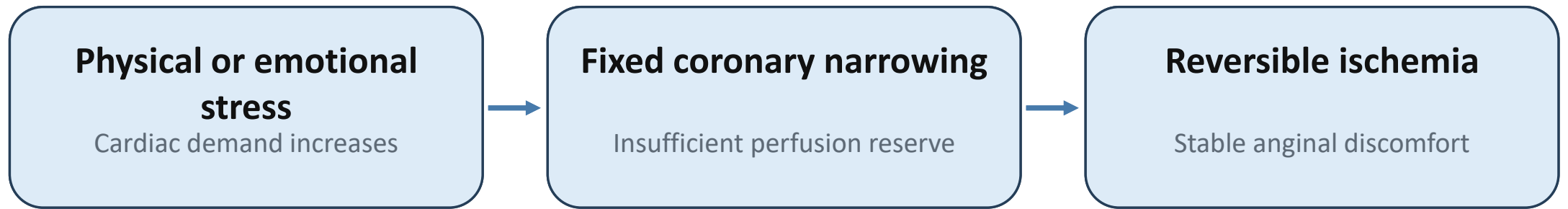
Schematic

Thresholds are functional tendencies, not absolutes

Approximate lumen area lost	Expected physiological context
<70%	May be asymptomatic from fixed obstruction
>70%	Exertional symptoms become typical
~90%	Inadequate flow may occur even at rest
At any severity	Collateral supply and multifocal disease modify expression

Stable exertional angina links anatomy to physiology

A chronic flow-limiting stenosis produces an imbalance between perfusion and oxygen demand.

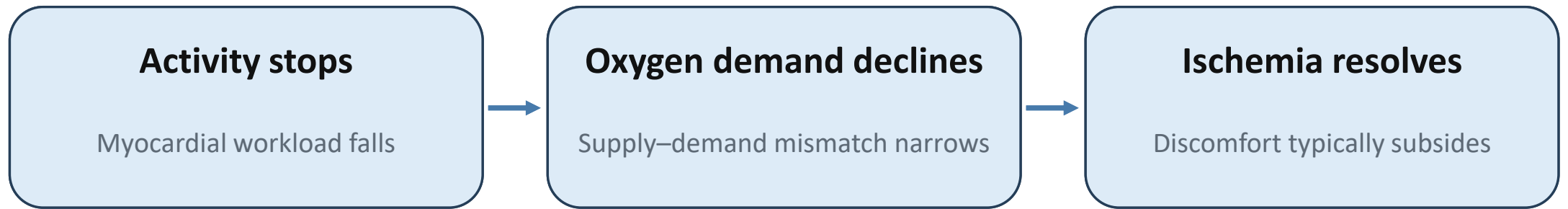


Schematic

A predictable activity threshold is informative

- In stable angina, a reproducible activity may consistently exceed available coronary reserve.
- The threshold reflects the interaction of myocardial demand and limited coronary perfusion.
- Changes in symptoms require careful interpretation; A new pattern is not simply “more of the same.”

Why rest often relieves stable angina



Schematic

Stable ischemia does not imply myocyte necrosis

- The adjacent angina section defines angina as transient ischemia insufficient to produce necrosis.
- A reversible supply–demand mismatch is therefore distinct from infarction.
- Do not equate severe stenosis, anginal pain, and myocardial cell death.

Symptoms need not be chest pain

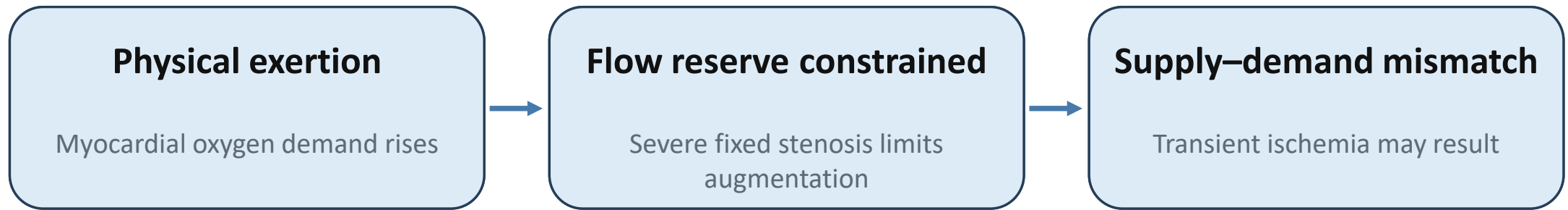
- Some myocardial ischemic episodes are silent.
- Atypical manifestations may include dyspnea, nausea, palpitations, diaphoresis, or fatigue.
- The absence of classic substernal discomfort does not prove adequate coronary perfusion.

Fixed stenosis has an anatomic and functional meaning

Anatomic assessment	Functional assessment
Where is the lesion?	How much does it limit flow?
How narrow is the lumen?	Can perfusion rise with demand?
How many lesions exist?	How do lesions interact across a vessel?
What territory is supplied?	Are alternate perfusion routes present?

Coronary flow reserve: The central physiological idea

A circulation requires the ability to augment flow above its resting state.



Functional limitation emerges when demand rises beyond available supply.

Schematic

Anatomic severity does not fully predict ischemia

- Comparable degrees of local narrowing can have different physiological consequences.
- Collateral circulation provides alternate delivery to some threatened territories.
- Additional stenoses can further restrict the available coronary flow response.

Serial and multifocal lesions complicate interpretation

- A single measured narrowing does not represent the total resistance of a diseased arterial tree.
- Multifocal disease can make the flow limitation greater than one local image suggests.
- This is why Robbins particularly emphasizes flow-reserve assessment in multifocal disease.

Sequential epicardial lesions



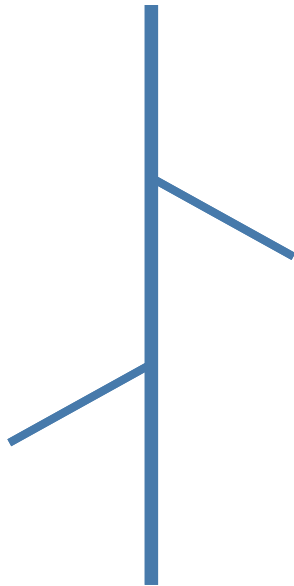
Serial narrowings

Schematic

Single-vessel disease is only one pattern

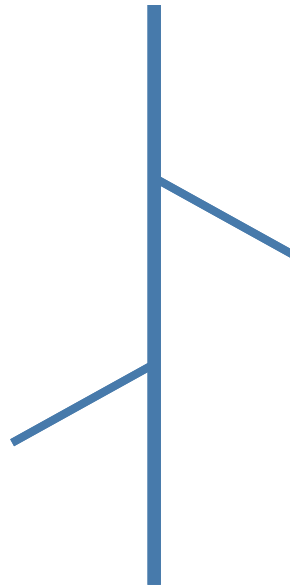
Atherosclerosis may involve only one major epicardial vessel.

LAD



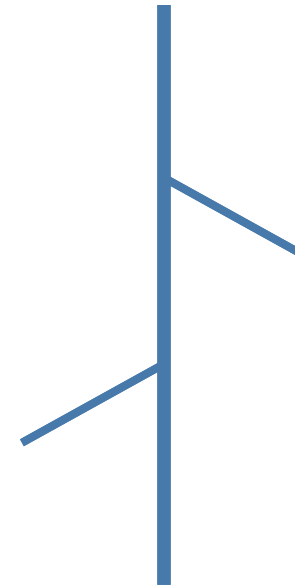
Diagonal branches

LCX



Obtuse marginal branches

RCA

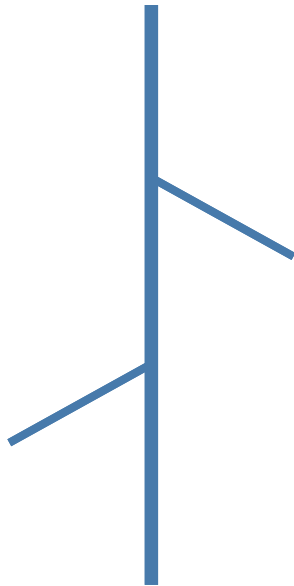


Posterior descending branch*

Coronary branches shown as a conceptual tree, not to anatomical scale.

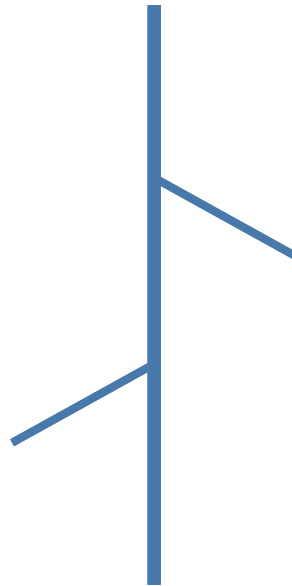
The three major named coronary arteries

LAD



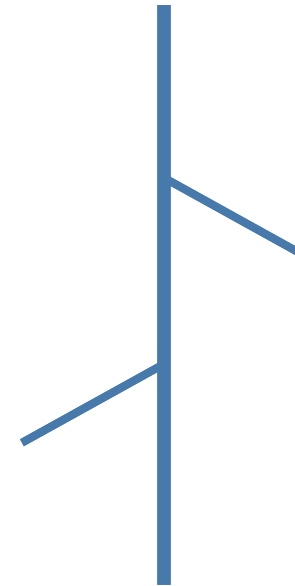
Diagonal branches

LCX



Obtuse marginal branches

RCA

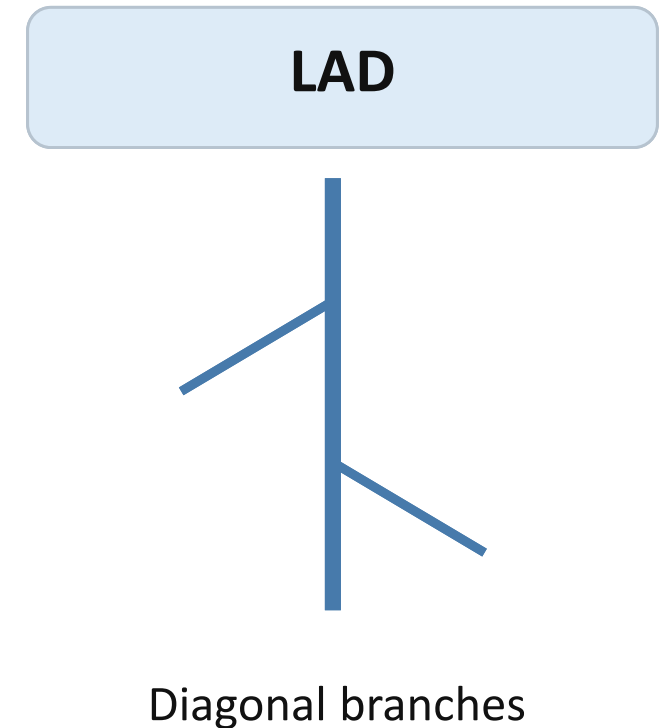


Posterior descending branch*

Coronary branches shown as a conceptual tree, not to anatomical scale.

Left anterior descending artery

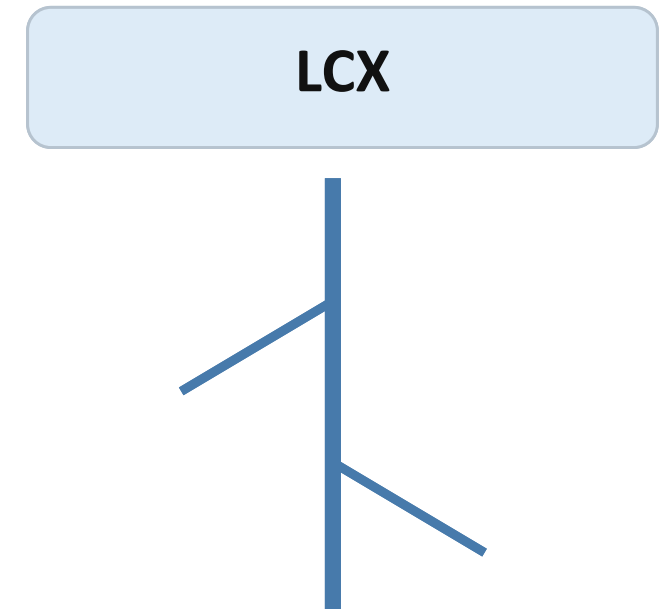
- The left anterior descending artery (LAD) is a major epicardial vessel commonly affected by atherosclerosis.
- Clinically significant plaque often occurs within its first several centimeters.
- Diagonal branches may also have atherosclerotic involvement.



Schematic

Left circumflex artery

- The left circumflex artery (LCX) is another major epicardial vessel.
- Atherosclerosis frequently affects proximal portions of the LCX.
- Obtuse marginal branches are among the larger epicardial branches that may also be involved.

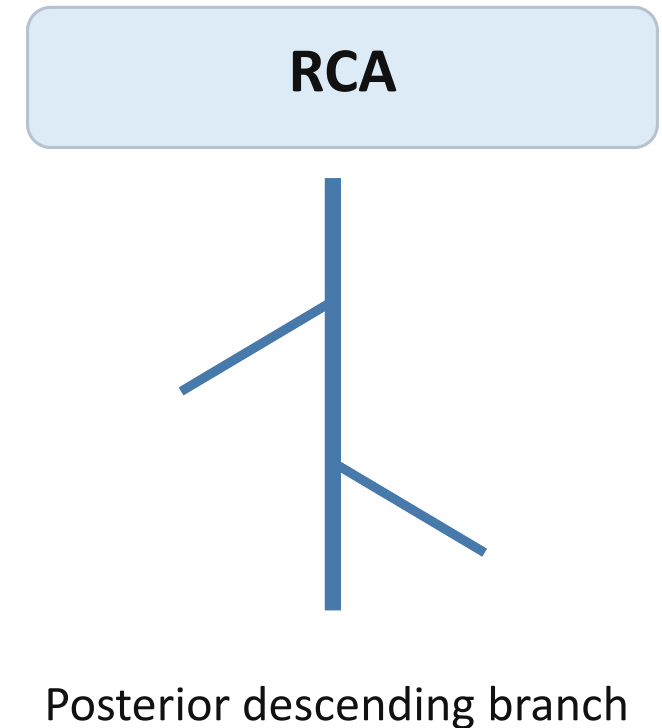


Obtuse marginal branches

Schematic

Right coronary artery

- The right coronary artery (RCA) is the third major named epicardial coronary vessel.
- RCA lesions can coexist with LAD or LCX disease.
- The posterior descending branch is among the named epicardial branches that may be involved.



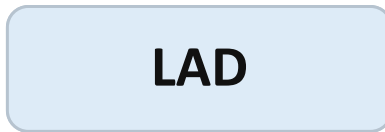
Schematic

Plaque can occur along the full epicardial course

- Clinically significant lesions may arise anywhere in a major coronary artery.
- Anatomic predilection does not make distal segments immune.
- A distributional assessment should consider the entire accessible epicardial tree.

Proximal LAD and LCX: Common plaque locations

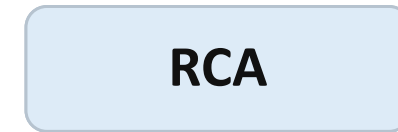
A concentration of significant plaques in the first several centimeters of the LAD and LCX.



Diagonal branches



Obtuse marginal branches



Posterior descending branch*

Coronary branches shown as a conceptual tree, not to anatomical scale.

Major branches may also be stenotic

Major artery	Named epicardial branch
LAD	Diagonal branches
LCX	Obtuse marginal branches
RCA	Posterior descending branch (as)

Intramyocardial penetrating branches are rarely involved

The supplied Robbins passage contrasts epicardial disease with infrequent atherosclerosis of penetrating branches.

Fixed coronary atherosclerosis

Progressive flow limitation

Acute plaque disruption

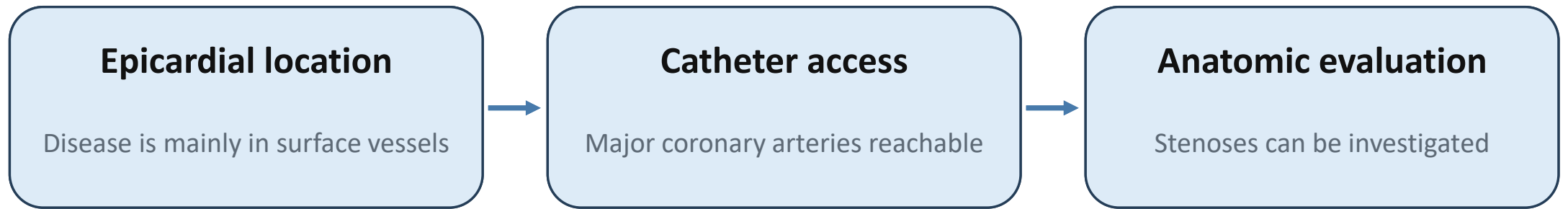
Rupture / erosion $\pm$ thrombus

Vasospasm

Dynamic arterial constriction

Chronic fixed occlusion is one mechanism within the broader IHD spectrum.

Why coronary catheterization is often possible



Schematic

Collateral vessels provide a bypass route

Gradual coronary narrowing can induce enlargement of alternate vascular channels.

Main epicardial pathway

Alternate channel carries flow around the obstruction



High-grade fixed stenosis

Collateralization can reduce—but does not guarantee elimination of—ischemia.

Schematic

Time course determines collateral opportunity

Slowly progressive stenosis	Abrupt vascular occlusion
Adjacent smaller vessels may enlarge over time	Little time for additional adaptation
Collateral supply may partially compensate	Residual supply may become insufficient
Severe local narrowing may be better tolerated	Acute ischemic consequences may be severe

Collaterals modify the stenosis–symptom relationship

A high-grade lesion does not produce identical symptoms in every individual.

Main epicardial pathway

Alternate channel carries flow around the obstruction



High-grade fixed stenosis

Collateralization can reduce—but does not guarantee elimination of—ischemia.

Schematic

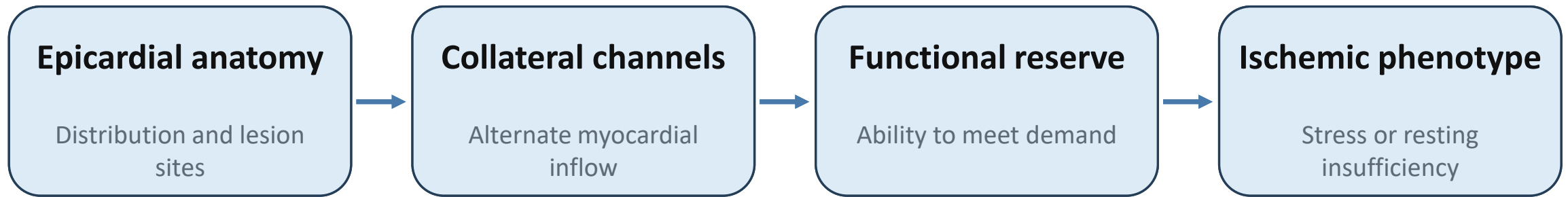
Collateral circulation is not unlimited

- Collateral supply can mitigate ischemia but may not fully satisfy increased demand.
- Its adequacy depends on the available alternate channels and the threatened tissue.
- Do not infer normal myocardial perfusion merely because collateralization is present.

Why two identical lesions may behave differently

Patient A	Patient B
Gradually progressive focal narrowing	Multifocal coronary involvement
Effective collateral channels	Limited alternate perfusion
Relatively preserved downstream flow	More restricted cumulative flow reserve
Symptoms may be delayed	Ischemia may arise at lower demand

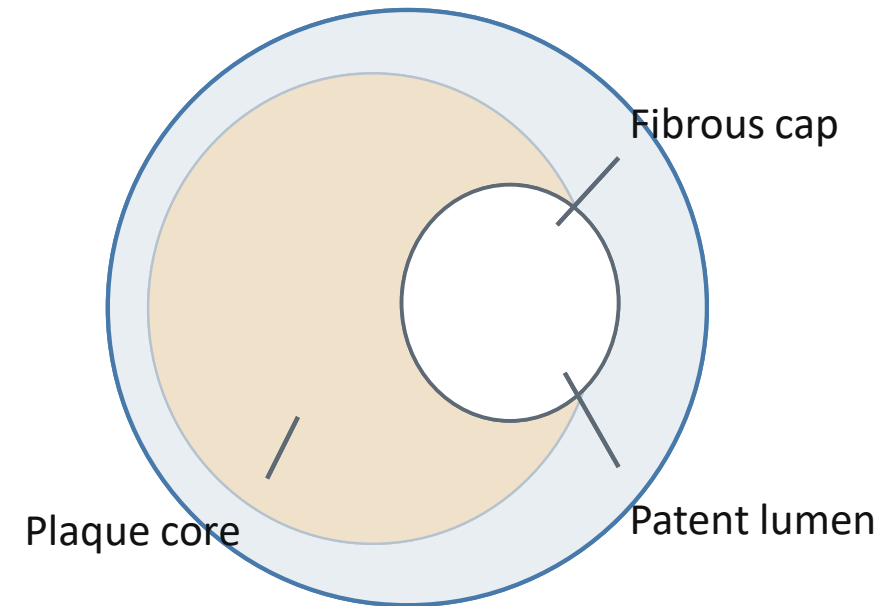
Coronary supply depends on a vascular network



Schematic

Stable plaque morphology and flow limitation

- A thick-cap fibrotic or fibrocalcific plaque can produce a persistent fixed obstruction.
- Progressive luminal compromise may cause exertional angina even without abrupt disruption.
- Structural stability and flow limitation describe different properties of a plaque.



Schematic

An acute event has a different pathological trigger

Abrupt plaque rupture or erosion may expose thrombogenic material.

Fixed coronary atherosclerosis

Progressive flow limitation

Acute plaque disruption

Rupture / erosion $\pm$ thrombus

Vasospasm

Dynamic arterial constriction

Chronic fixed occlusion is one mechanism within the broader IHD spectrum.

A noncritical plaque can still precipitate infarction

- The adjacent Robbins discussion stresses that many infarction culprit lesions were not critically stenotic beforehand.
- Plaque susceptibility to disruption is not the same as fixed obstruction severity.
- Chronic flow-limiting disease and acute plaque vulnerability must be assessed conceptually apart.

Distinguishing common ischemic presentations

Presentation	Dominant lesion concept	Temporal pattern
Stable angina	Chronic fixed stenosis with limited reserve	Exertional, typically reproducible
Unstable angina	Plaque change $\pm$ thrombus/spasm	Changing or resting symptoms
Myocardial infarction	Prolonged ischemia with myocyte necrosis	Acute injury event

A chronic lesion can underlie more than angina

- Long-standing flow-limiting coronary atherosclerosis contributes to chronic ischemic heart disease.
- The clinical consequences reflect myocardial perfusion, demand, collateralization, and other coronary events.
- A chronic stenosis by itself should not be presumed to specify the entire clinical syndrome.

Assessment: Do not read lumen narrowing in isolation

- Identify the epicardial location and whether disease is focal or multifocal.
- Interpret area narrowing alongside capacity to augment coronary blood flow.
- Consider the possible effect of collateral circulation.
- Relate the physiology to activity-induced or resting ischemic symptoms.

Case 1: Reproducible exertional discomfort

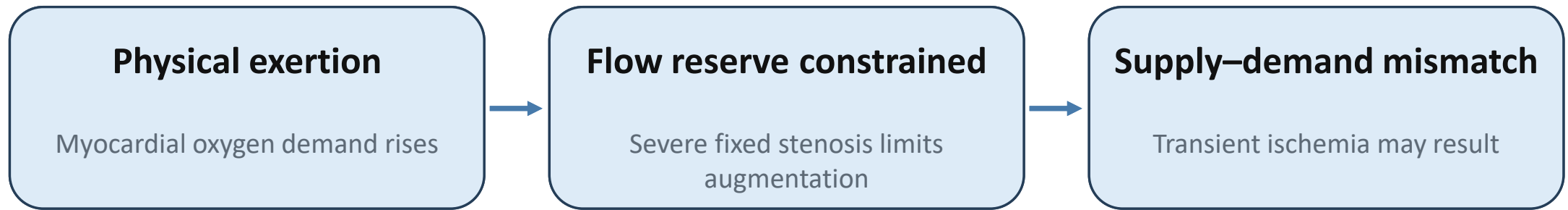
A patient reports substernal discomfort during a similar level of exertion on repeated occasions. The discomfort subsides with rest. No resting symptoms are described.

Interpretation prompts

- What mechanism best links a fixed epicardial lesion to this pattern?
- Why can perfusion be adequate at rest but insufficient during activity?

Case 1: Mechanism and interpretation

A severe fixed lesion limits the increase in coronary flow when oxygen demand rises.



Functional limitation emerges when demand rises beyond available supply.

Schematic

Case 2: Major stenosis but modest symptoms

A patient has gradually progressive severe narrowing of a major epicardial coronary artery but reports fewer exertional symptoms than the local anatomic lesion alone might suggest.

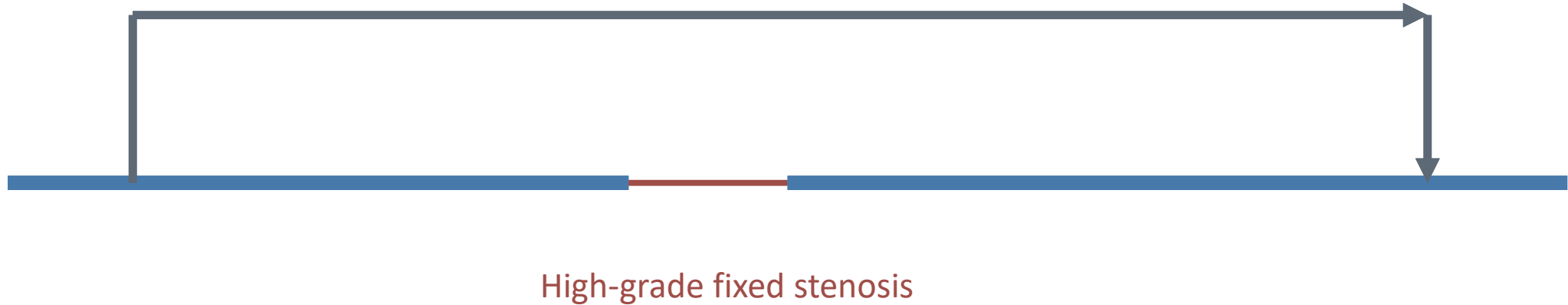
Interpretation prompts

- What physiological adaptation could help explain this?
- Why is stenosis severity alone an incomplete measure?

Case 2: Collateral compensation

Slow progression can permit collateral channels to enlarge.

Main epicardial pathway Alternate channel carries flow around the obstruction



Collateralization can reduce—but does not guarantee elimination of—ischemia.

Schematic

Case 3: Several moderate lesions

Coronary assessment identifies multiple atherosclerotic narrowings rather than one isolated lesion. Symptoms appear during modest physical activity.

Interpretation prompts

- Why might focusing on only the single most obvious lesion mislead?
- Which physiological concept does Robbins recommend considering?

Case 3: Multifocal disease and reserve

- Several fixed lesions can collectively limit the coronary flow response.
- Local anatomy does not reliably describe cumulative perfusion consequences.
- Robbins specifically favors evaluating flow reserve when occlusions are multifocal.

Sequential epicardial lesions



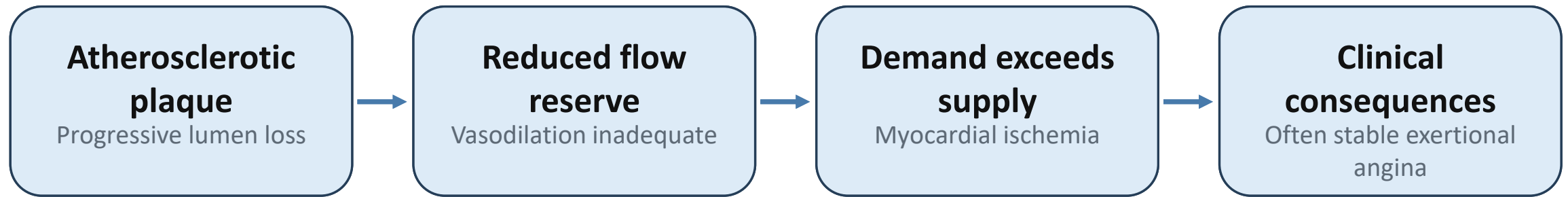
Serial narrowings

Schematic

Interpretation pitfalls

Incorrect shortcut	More accurate interpretation
“70% means 70% diameter loss”	Robbins threshold is luminal cross-sectional AREA
“No rest pain means normal coronary arteries”	Fixed severe lesions can be silent at rest
“Most severe lesion dictates all flow”	Multifocal stenoses and collaterals matter
“Only critical plaques cause MI”	Acute culprit plaques may be noncritical beforehand

The full chronic-occlusion mechanism



Schematic

Five clinicopathologic conclusions

- Epicardial coronary atherosclerosis accounts for the dominant substrate of IHD.
- Critical narrowing is described by luminal area loss and constrained flow reserve.
- Approximately 70% area stenosis commonly unmasks demand ischemia; ~90% may affect resting flow.
- Distribution, serial lesions, and collateralization modify functional effects.
- Fixed stenosis and sudden plaque disruption are distinct, although related, pathological problems.

Selected reading

- Robbins, Cotran & Kumar. Pathologic Basis of Disease, 11th edition (2026).
- Ischemic Heart Disease: chronic arterial occlusion and stable angina.
- Atherosclerotic Stenosis: plaque morphology and vessel remodeling.
- The original diagrams are instructional schematics, not clinical specimens.