

Complications of prosthetic heart valves

Morphologic mechanisms, clinical consequences, and interpretive pathways

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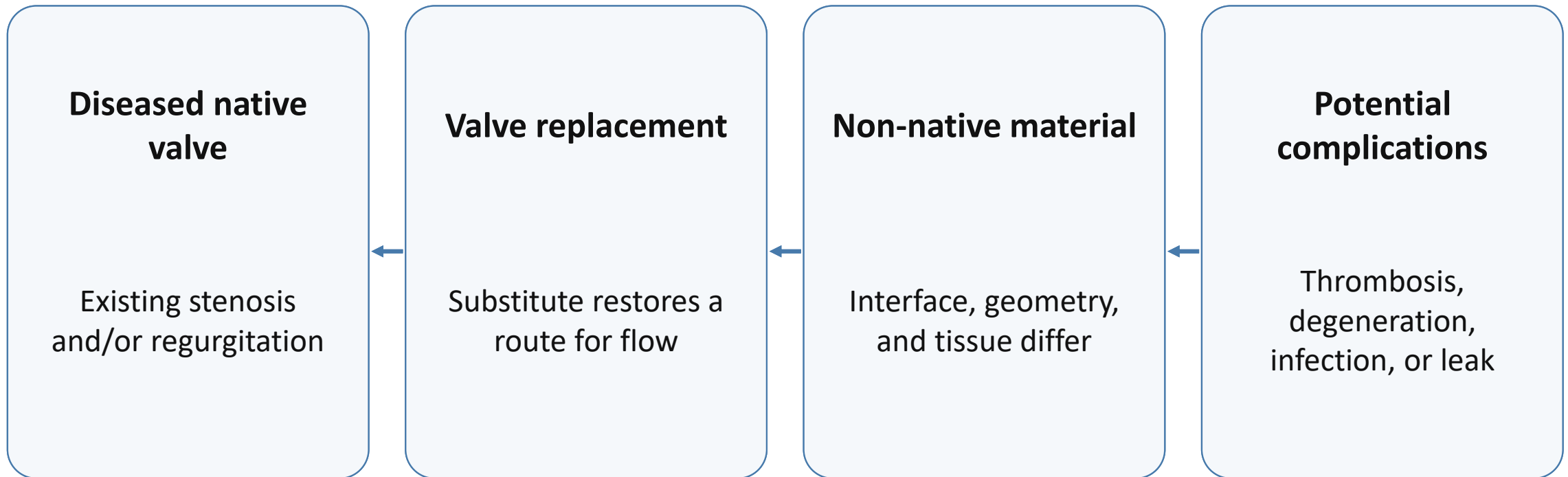
Learning outcomes

- Differentiate mechanical from tissue valve design and failure patterns.
- Explain how abnormal flow causes mechanical-valve thrombosis and why anticoagulation adds bleeding risk.
- Relate tissue-valve degeneration and prosthetic infection to structural dysfunction.
- Distinguish leaks, obstruction, orifice disproportion, and hemolysis using mechanisms.

Valve dysfunction has two fundamental expressions

Pathologic expression	What changes	Expected burden
Stenosis	Effective valve opening is reduced	Pressure overload upstream of the valve
Regurgitation	Closure fails; blood leaks backward	Volume overload involving the affected chambers
Both	Obstruction and incompetence coexist	Combined hemodynamic burden

Prostheses improve prognosis but are not native valves



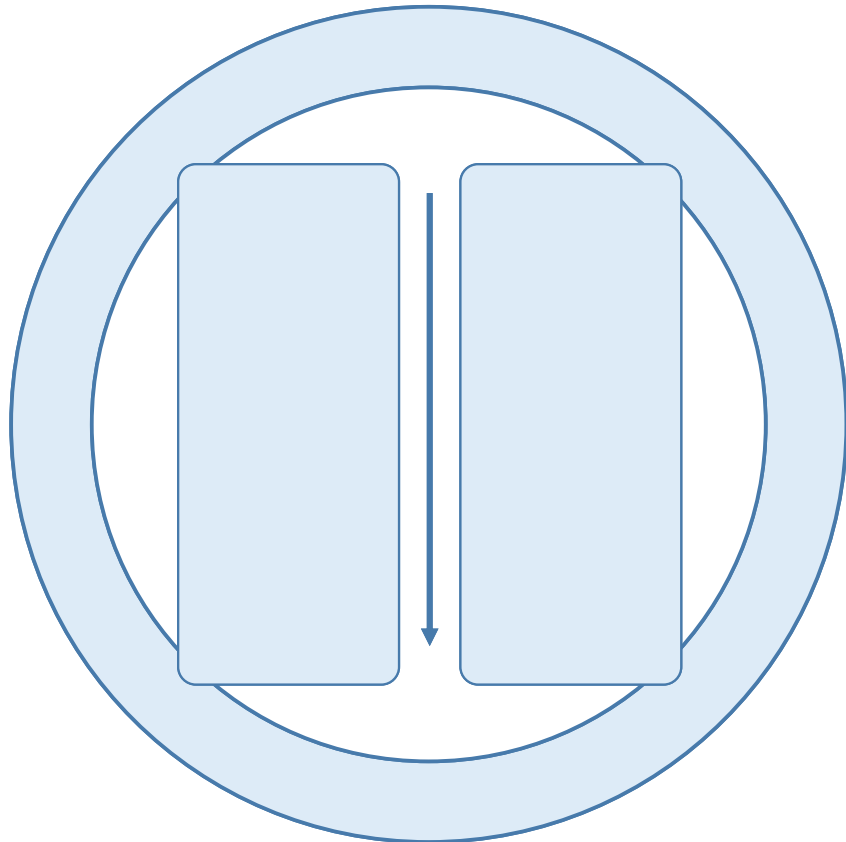
SCHEMATIC — causal / functional relationships

- The major prognostic benefit of replacement despite device-specific failure modes.

Two major categories of replacement valves

Characteristic	Mechanical valve	Tissue valve (bioprosthesis)
Primary material	Rigid, nonphysiologic materials	Human cadaveric or animal leaflet tissue
Typical architecture	Hinged semicircular flaps; bileaflet tilting disks	Flexible leaflets mounted on a frame
Major long-term liability	Thrombosis and anticoagulation-related bleeding	Calcification and/or tearing

Mechanical valves: Rigid bileaflet architecture



Conceptual schematic — bileaflet mechanical device

Rigid components

Nonphysiologic material

Hinged flaps

Semicircular opening elements

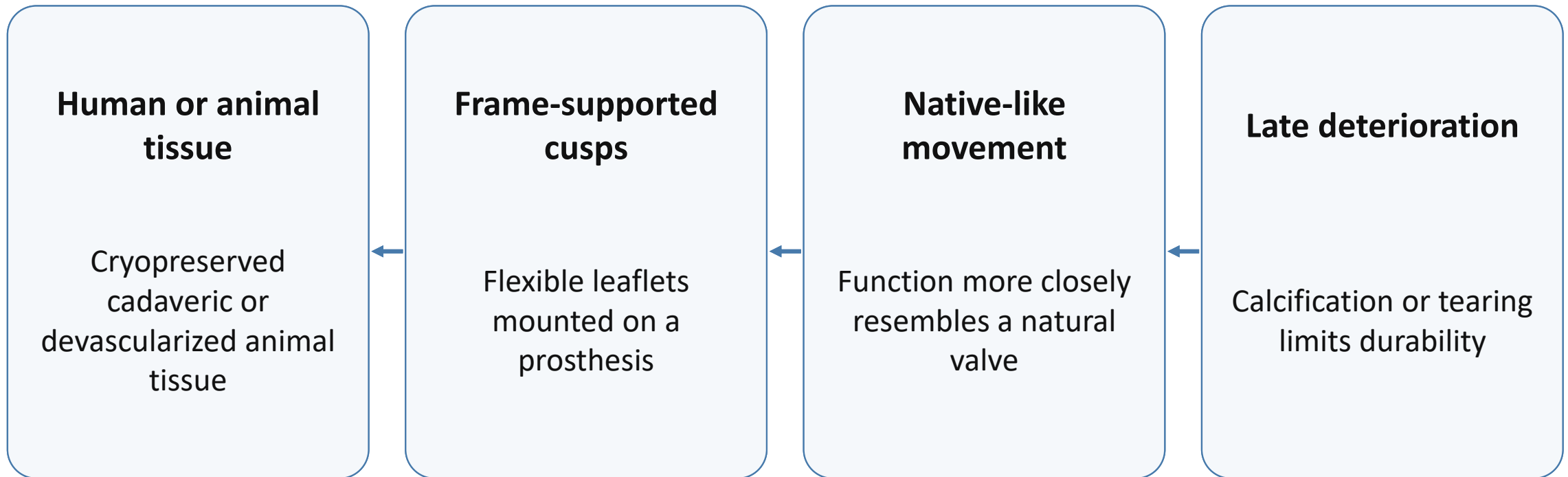
Bileaflet motion

Permits forward flow

Nonlaminar flow

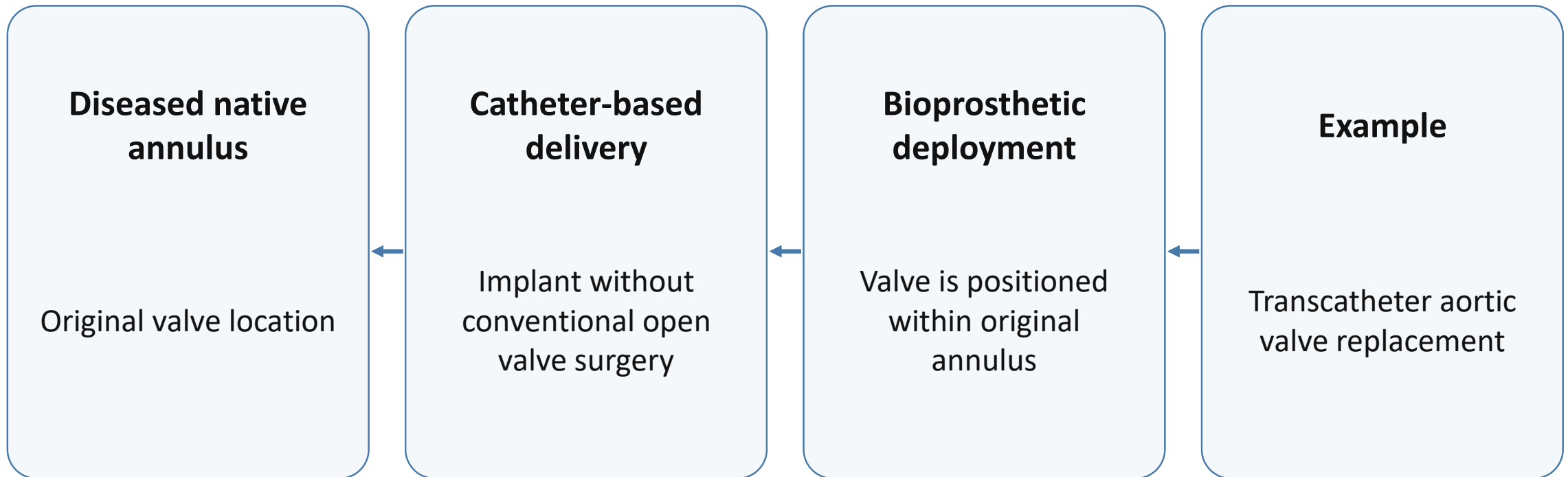
Turbulence and local stasis

Bioprostheses: Flexible nonviable leaflets



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Bioprostheses can be deployed by catheter



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- Increasing catheter-based use, especially where open surgery is unsuitable.

Valve design implies a different dominant trade-off

Dimension	Mechanical	Bioprosthetic
Durability of structure	Structural deterioration is rare with current devices	Long-term cusp calcification and/or tearing
Thrombotic liability	Major consideration	Not characterized as dominant in this section
Chronic anticoagulation	Required because of mechanical-valve thrombotic risk	No universal regimen specified by this source
Endocarditis	Potential complication	Potential complication

A prosthesis can fail without a fractured component

Functional failure	Possible pathology	Underlying problem
Obstruction	Thrombus; fibrous overgrowth; small effective orifice	Forward flow restricted
Regurgitation	Paravalvular leak; degenerated tissue leaflet	Backward blood flow
Systemic complication	Embolization; infection; bleeding; hemolysis	Damage beyond the valve itself

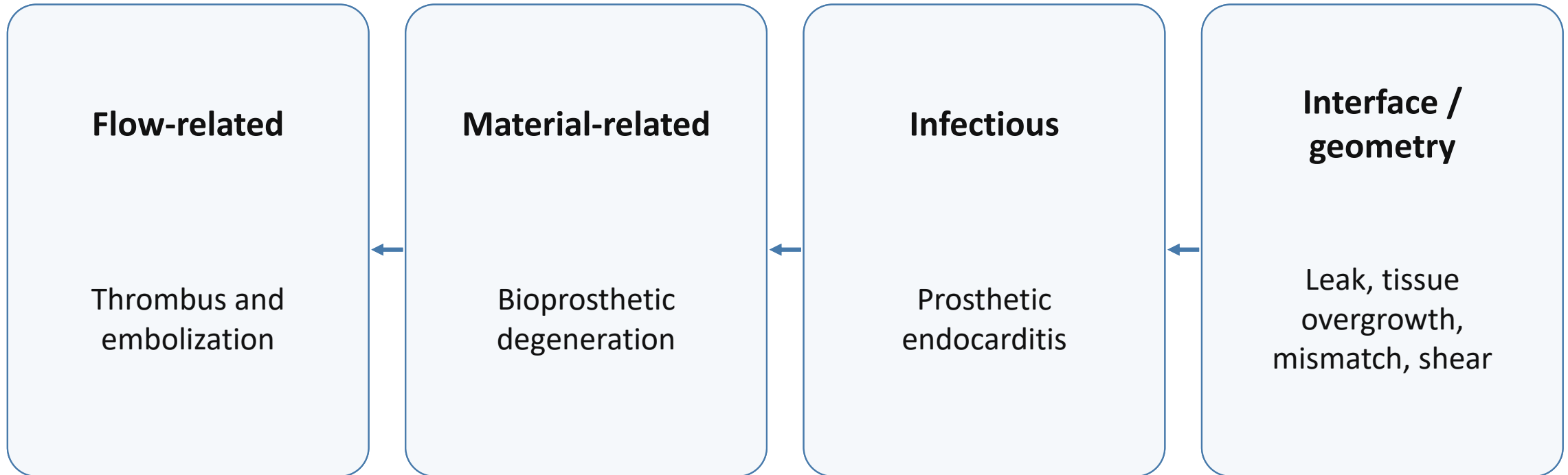
Ten-year burden: A substantial cumulative problem

≈60%

Within 10 years

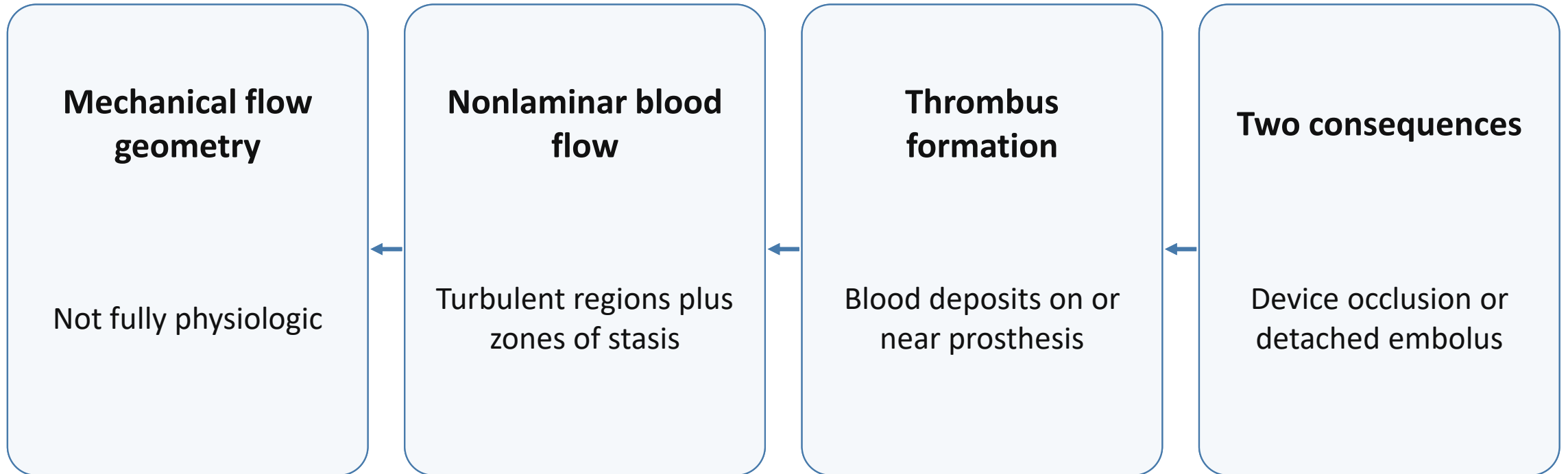
- That approximately 60% of valve recipients develop a serious prosthesis-related problem within 10 years after surgery.
- This is a composite statement about serious prosthesis-related problems—not a rate for any one specific mechanism.
- Risk differs by valve type; Do not interpret the figure as a head-to-head comparison of devices.

Taxonomy: Four major mechanisms plus related effects



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Mechanical-valve thromboembolism starts with flow



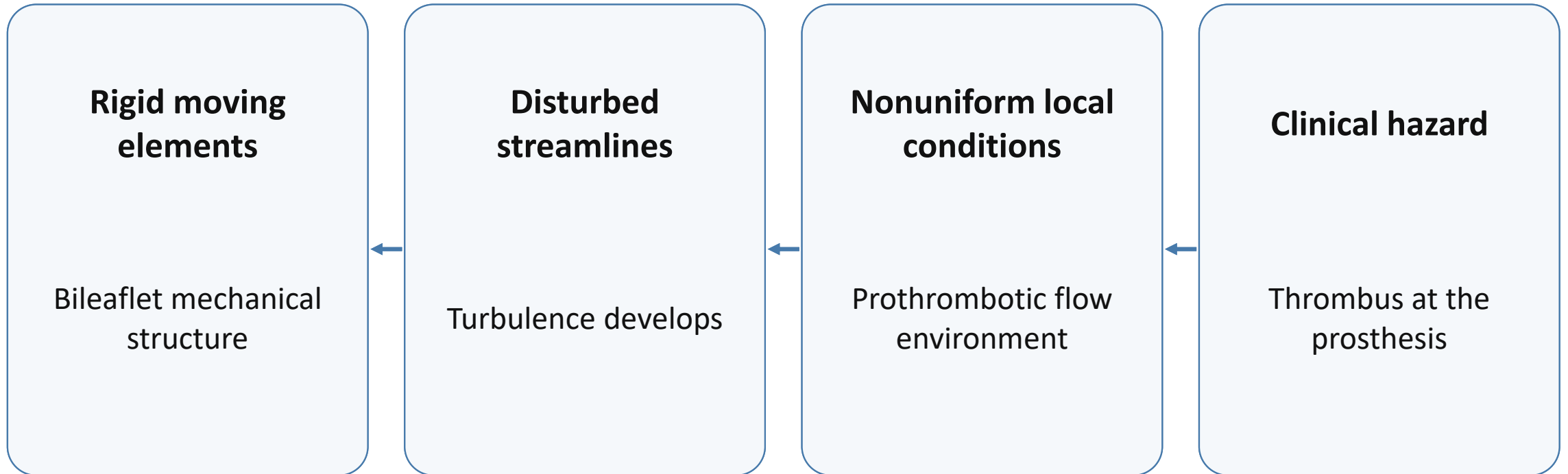
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Why nonlaminar flow matters

- Nonlaminar flow as a property of mechanical devices.
- A prosthesis creates focal regions of turbulence and stasis instead of uniformly streamlined flow.
- Those flow disturbances favor local thrombus formation.
- The resulting mass can remain at the valve or become a source of emboli.

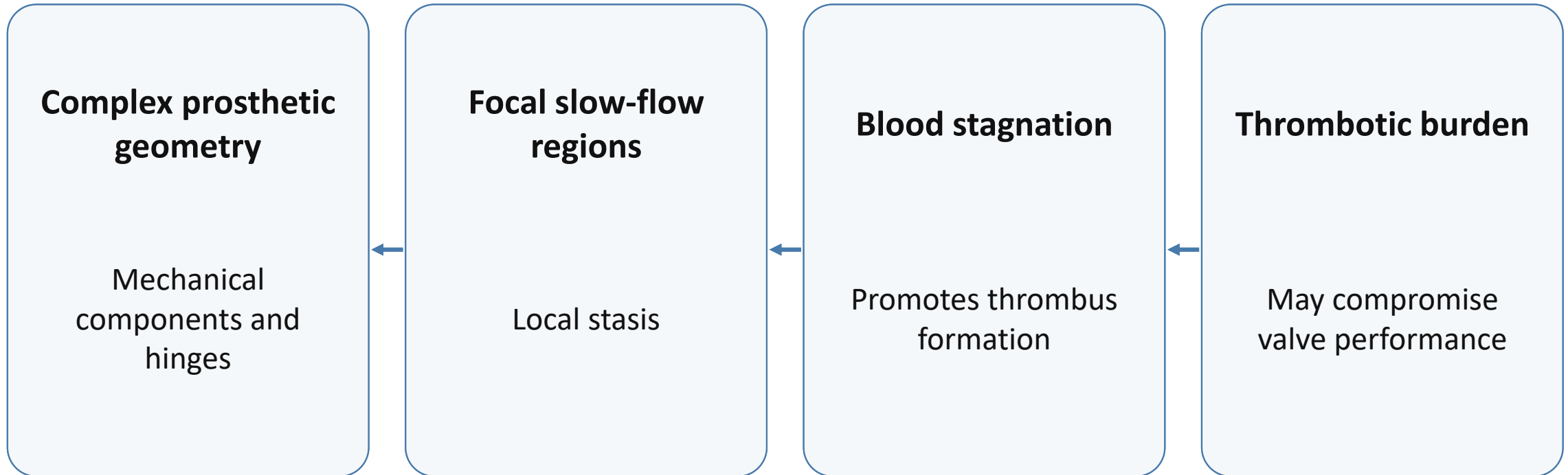
**Nonlaminar flow →
thrombogenic
conditions**

Turbulence: Disturbed flow near a rigid prosthesis



SCHEMATIC — causal / functional relationships

Stasis: Slower-flow regions add thrombotic risk



SCHEMATIC — causal / functional relationships

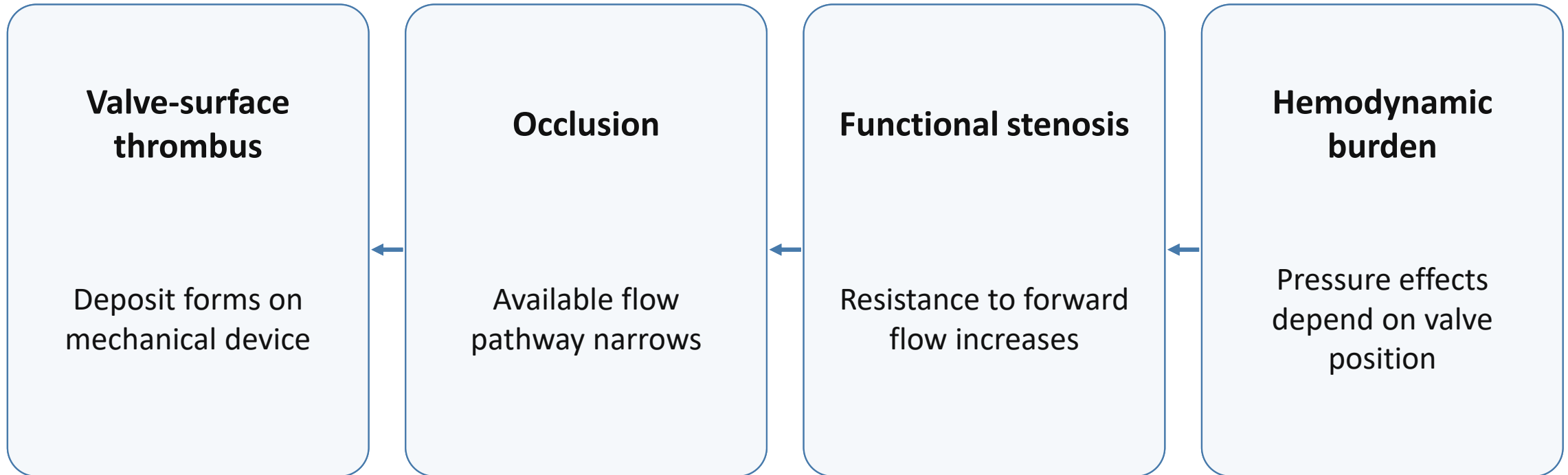
Turbulence and stasis act as paired contributors

Flow disturbance	Prosthetic setting	Consequential risk
Turbulence	Nonlaminar flow around a mechanical device	Thrombus formation
Stasis	Local slow-flow foci within disturbed circulation	Thrombus formation
Combined exposure	Both present in the same device	Thrombotic occlusion or embolization

Two clinically distinct thrombotic outcomes

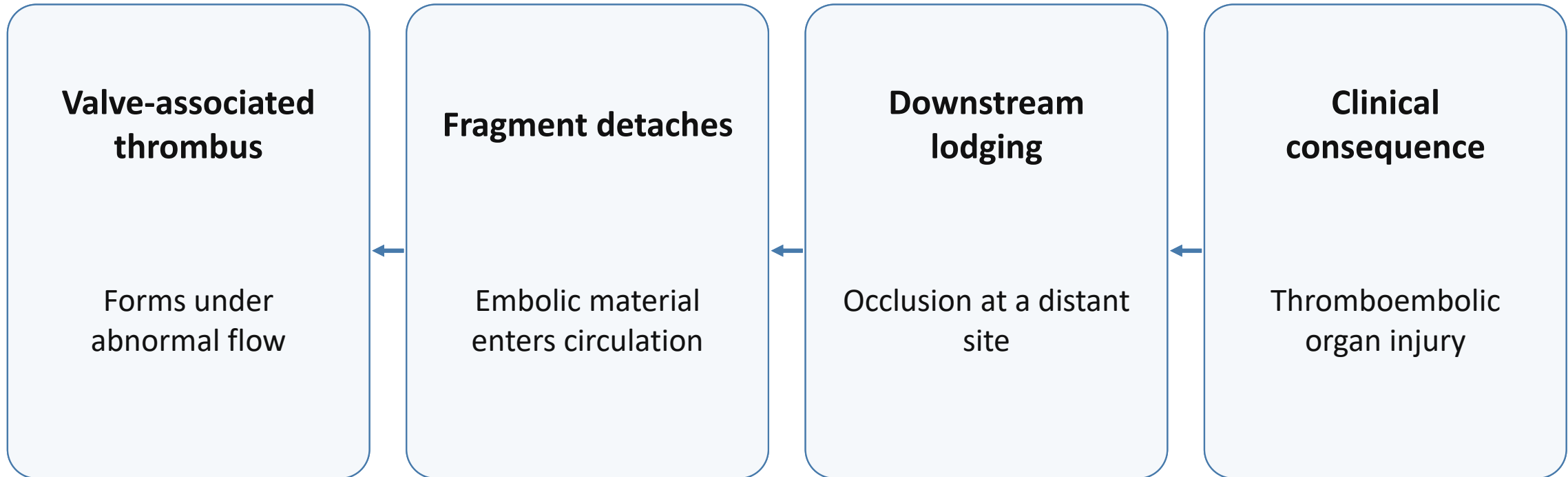
Outcome	Event at valve	Consequence
Prosthetic thrombosis	Thrombus occludes the valve orifice	Reduced forward flow; functional obstruction
Thromboembolism	Thrombus forms at valve then detaches	Embolus travels beyond original thrombus
Shared origin	Mechanical-valve thrombogenic conditions	Both motivate long-term anticoagulation

Thrombotic occlusion: The obstructive sequence



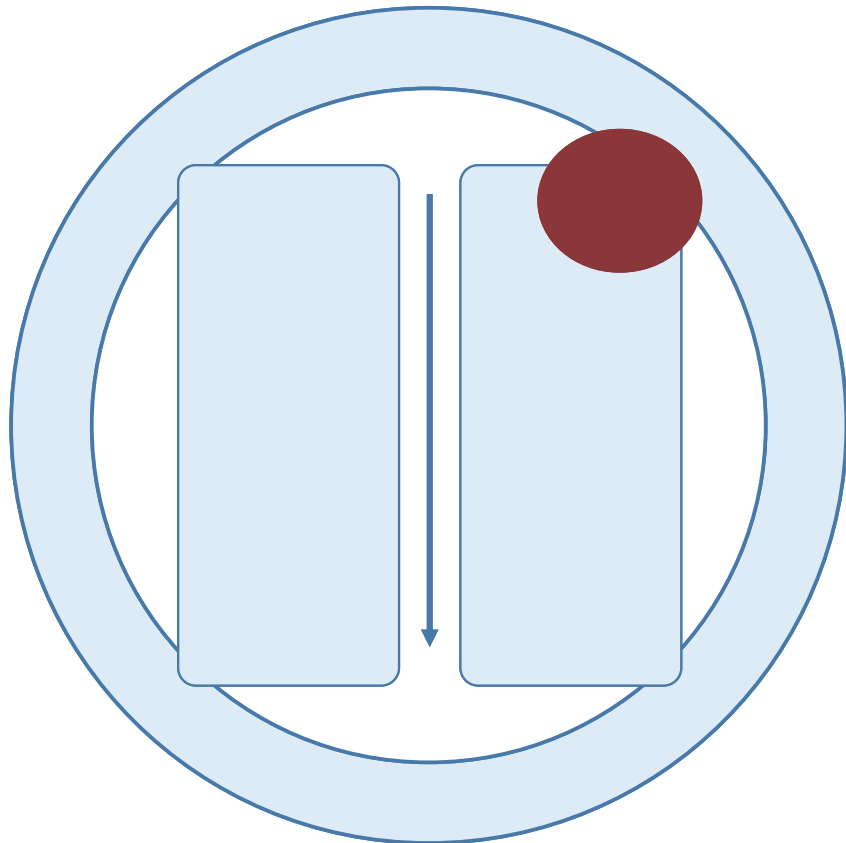
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Embolization: Injury remote from the prosthesis



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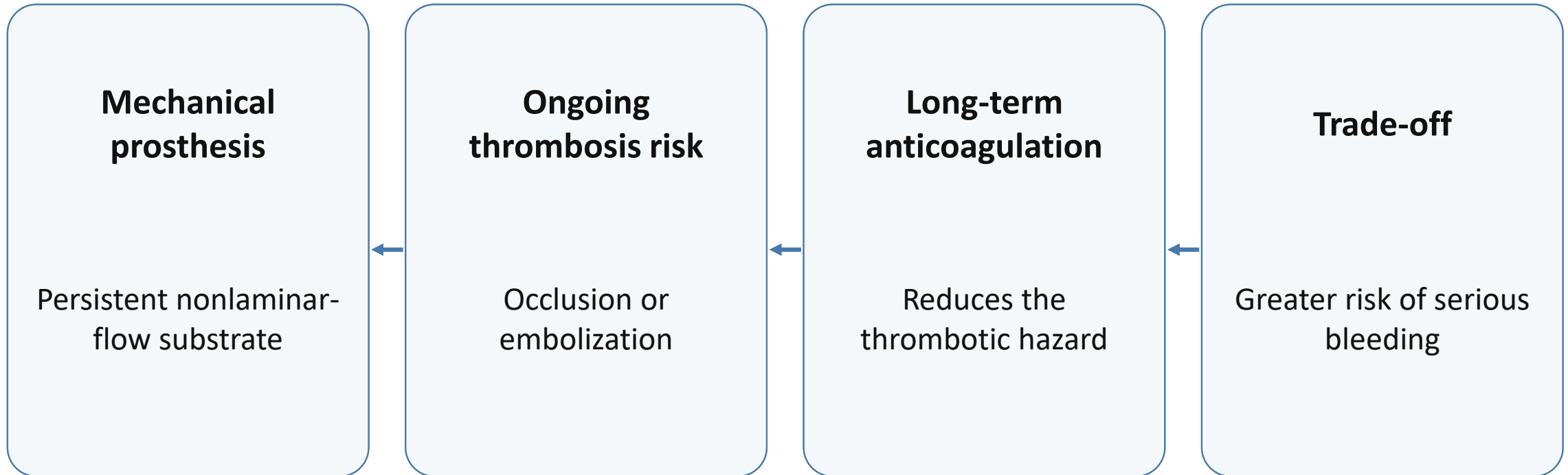
Mechanical-valve thrombosis: The source figure



Conceptual schematic — thrombus at the valve

- Robbins Figure 12.27A depicts thrombosis of a mechanical prosthetic valve.
- The morphologic point is thrombotic material involving an artificial valve, not native-leaflet calcification.
- The chapter describes device occlusion and valve-derived emboli as separate possible consequences.
- The published image illustrates valve-associated thrombosis; The teaching distinction is occlusion versus embolization.

Why chronic anticoagulation is required



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The anticoagulation trade-off is intrinsic

Without adequate thrombotic protection

Valve-associated thrombus remains a major concern

Prosthesis may become occluded

Thrombus may embolize

With anticoagulation exposure

Thrombotic risk is addressed but not cost-free

Serious hemorrhage becomes possible

Hemorrhagic stroke is a specific major concern

Hemorrhagic stroke is a serious treatment liability

- The long-term anticoagulation necessitated by mechanical valves carries bleeding hazards.
- Hemorrhagic stroke among the serious consequences.
- Other forms of serious bleeding also contribute to prosthesis-related morbidity.
- Bleeding is a treatment-associated complication, not mechanical structural failure.

**Device benefit ≠
absence of
treatment risk**

Assess the mechanism—not just the adverse event

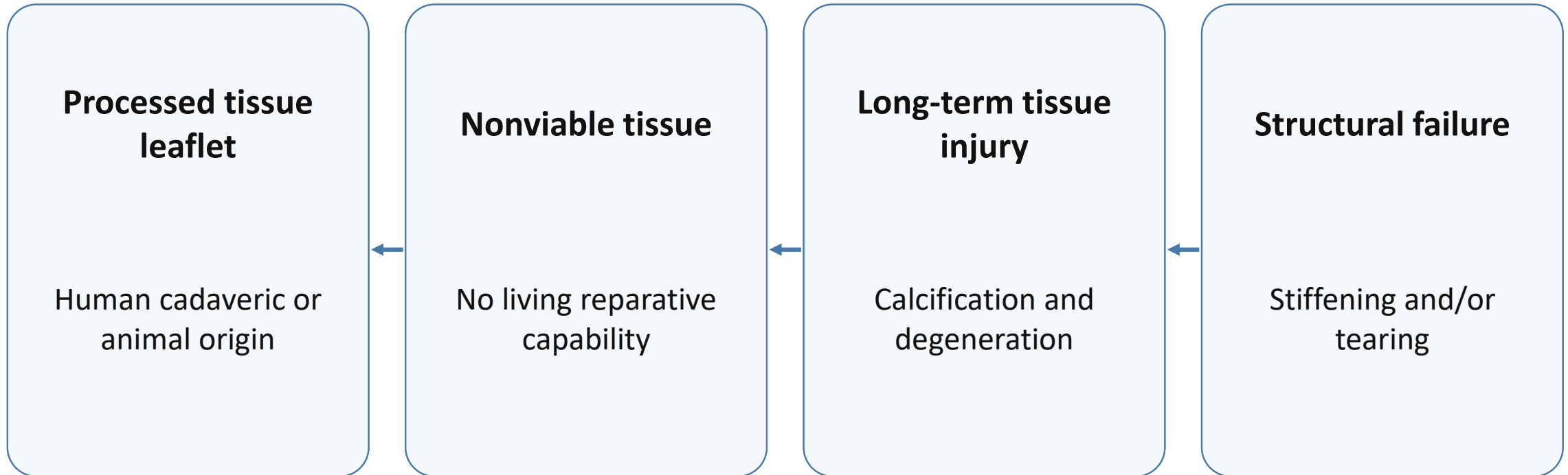
Observation	Mechanistic classification	What it does not prove
Occluded mechanical valve	Local prosthetic thrombosis is a consideration	Not automatically structural fracture
Embolic event	Valve-related thromboembolism is possible	Not proof of infection without evidence
Serious hemorrhage	Anticoagulation-related complication is possible	Not evidence of thrombotic occlusion

Mechanical structural breakage is uncommon

- Current mechanical valves rarely fail from structural deterioration.
- The predominant liabilities discussed by Robbins are thrombosis and hemorrhage.
- Material durability therefore should not be equated with freedom from complications.
- An abnormal mechanical valve should be assessed by the type of dysfunction present.

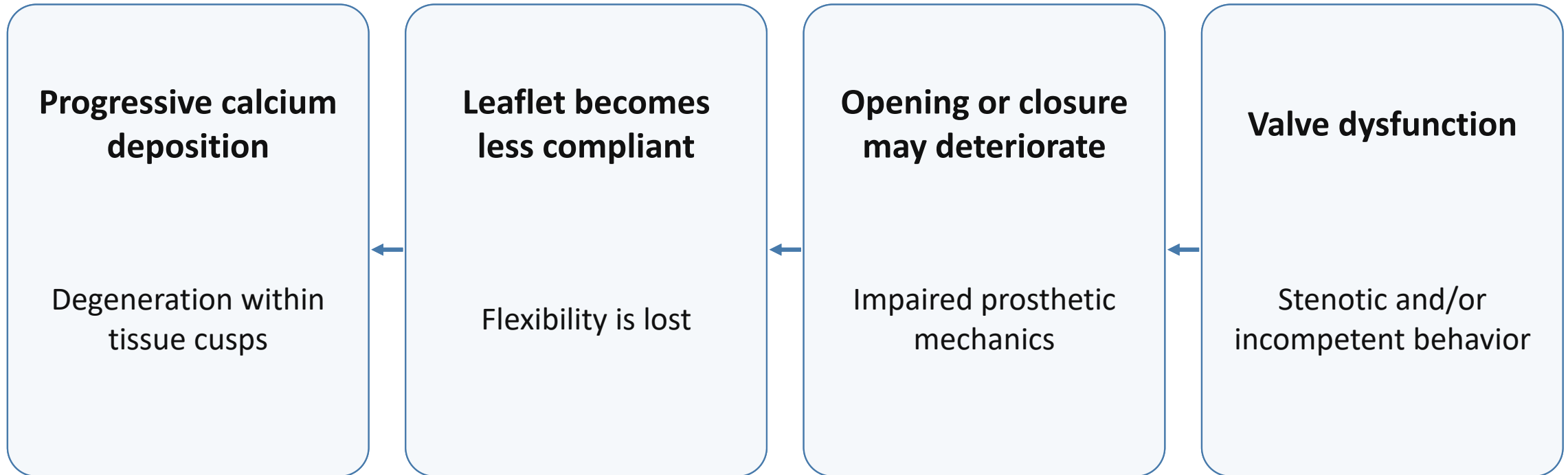
**Structural durability
≠ clinical
invulnerability**

Bioprosthetic tissue is nonviable



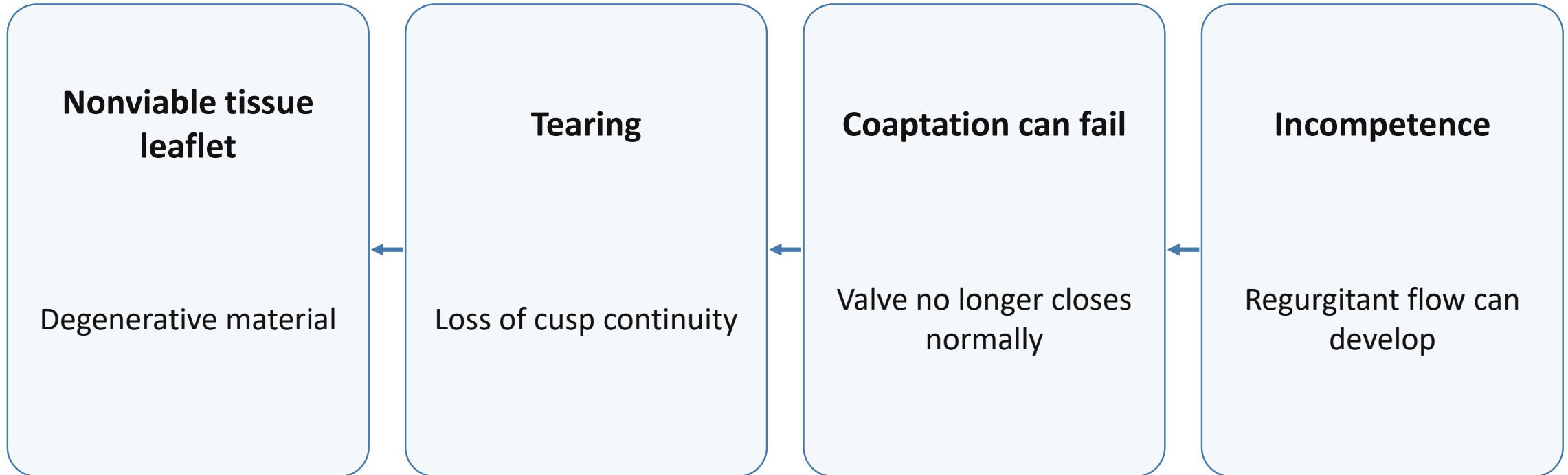
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Bioprosthetic calcification alters leaflet function



SCHEMATIC — causal / functional relationships

Bioprosthetic tearing is a structural lesion



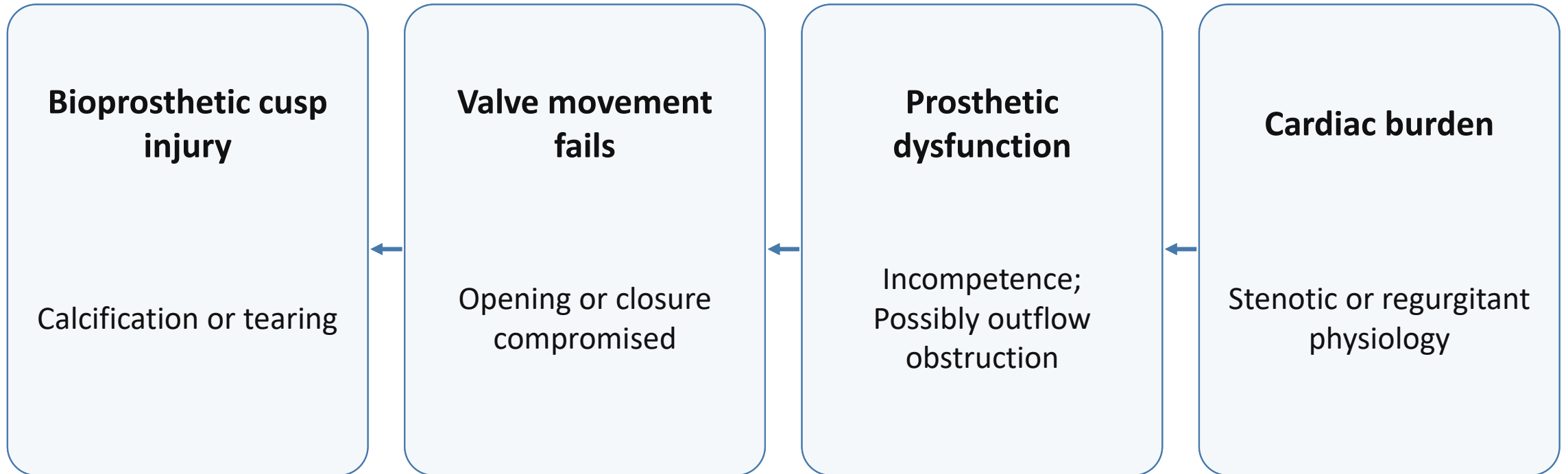
SCHEMATIC — causal / functional relationships

Calcification and tearing can coexist

- Failure of bioprostheses through calcification and/or tearing.
- Figure 12.27B specifically shows a porcine bioprosthesis with calcification and secondary tearing.
- The two lesions are not mutually exclusive causes of failure.
- A combined structural lesion can compromise leaflet mobility and integrity.

**Fig. 12.27B:
Calcification with
secondary tearing**

From cusp degeneration to incompetence



SCHEMATIC — causal / functional relationships

Late failure: Mechanical and tissue valves differ

Mechanism	Mechanical prosthesis	Bioprosthesis
Structural degeneration	Rare with current devices	Expected long-term vulnerability
Characteristic morphologic lesion	Thrombus shown in Fig. 12.27A	Calcium and leaflet tear in Fig. 12.27B
Major clinical consequence	Obstruction or embolus; bleeding under anticoagulation	Failure of valve competence
Shared hazard	Endocarditis possible	Endocarditis possible

Infection may target two prosthetic compartments

Prosthesis–tissue interface

Vegetations frequently develop where prosthesis joins host tissue.

Extension can produce an annular / ring abscess.

A ring abscess may progress to a paravalvular leak.

Bioprosthetic cusps

Vegetations can involve the tissue leaflets themselves.

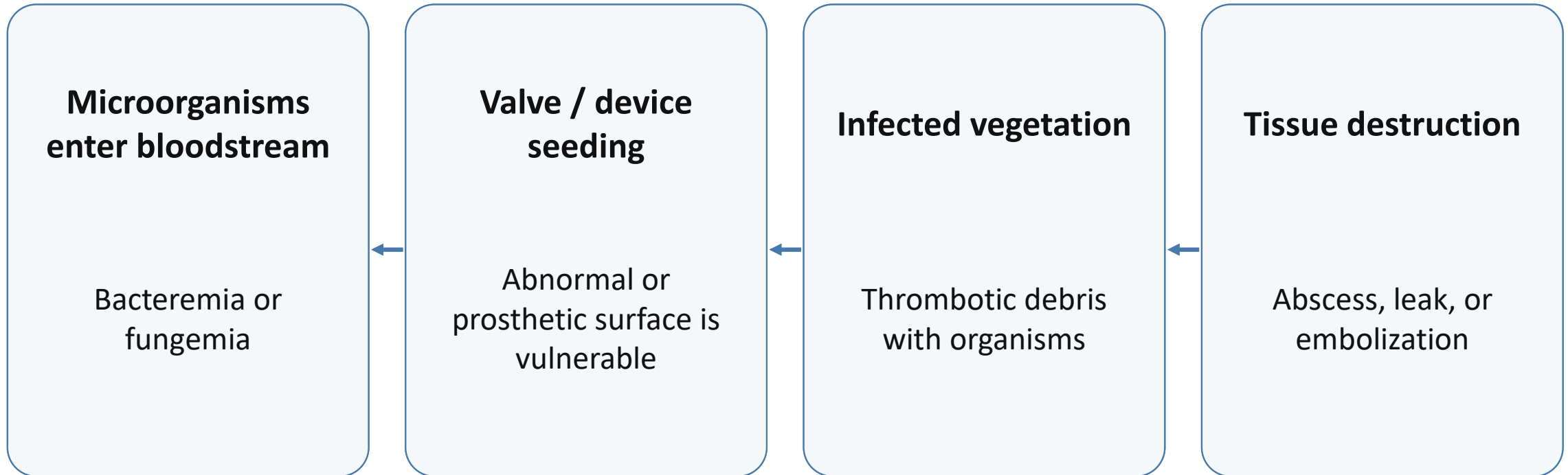
Direct leaflet infection adds tissue destruction.

The device may become incompetent through cusp involvement.

All valve replacements are susceptible to endocarditis

- Prosthetic-valve endocarditis is a serious complication of either valve type.
- Increased endocarditis risk in prosthetic valves compared with native valves.
- Infected vegetations are distinguished from sterile thrombotic deposits by their microbial nature.
- The anatomic distribution of infection helps explain subsequent destruction and leakage.

Bacteremia is the gateway to endocarditis



SCHEMATIC — causal / functional relationships

Early prosthetic endocarditis: Source-based pattern

Time context	Organisms emphasized in Robbins	Interpretation
First 1–2 months after surgical implantation	Staphylococcus aureus; Staphylococcus epidermidis	Skin flora are typical in this early setting
Not a universal rule	Other organisms are not logically excluded	Timing informs suspicion; it is not proof

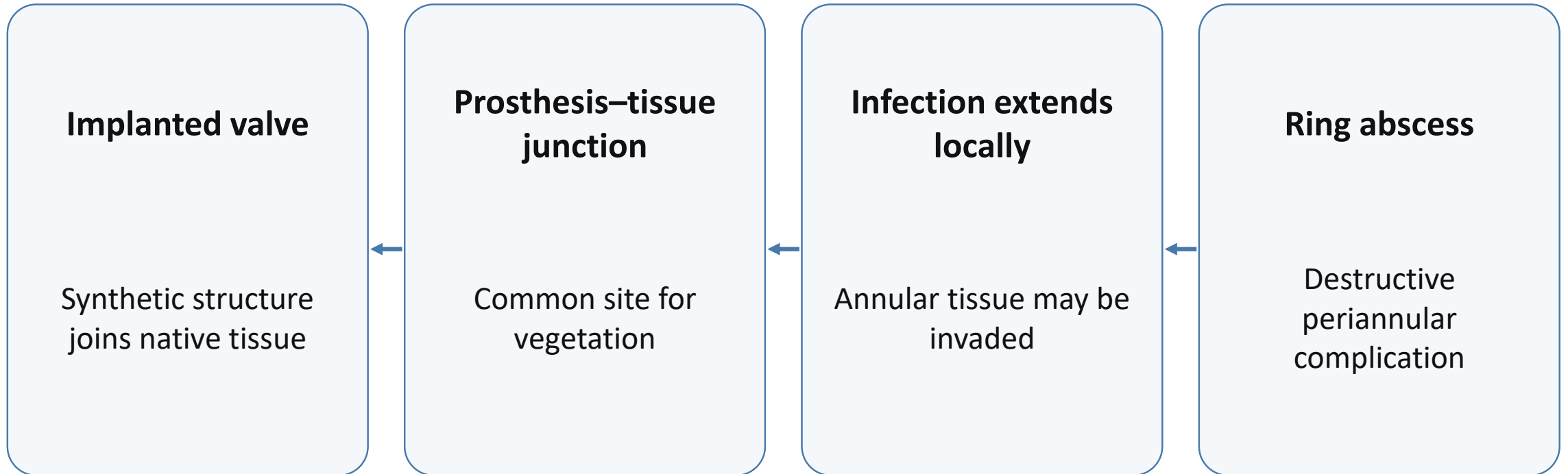
Late prosthetic endocarditis: A different pattern

Time context	Organisms emphasized in Robbins	Interpretation
At least 1 year after surgery	Streptococci; Staphylococcus aureus	Later microbiologic distribution differs from early cases
Between early and late windows	Not specifically categorized in the excerpt	Do not invent an intermediate-organism rule

Microbiologic patterns are conditional, not diagnostic

- Timing associations describe organisms that are typical—not organisms that must be present.
- Culture-negative endocarditis can follow prior antibiotic exposure or difficulty recovering microbes.
- Histology or culture of a vegetation or explanted prosthesis may provide pathologic evidence.
- Do not label a prosthetic mass infectious from device type or time since surgery alone.

Vegetations favor the prosthesis–tissue interface



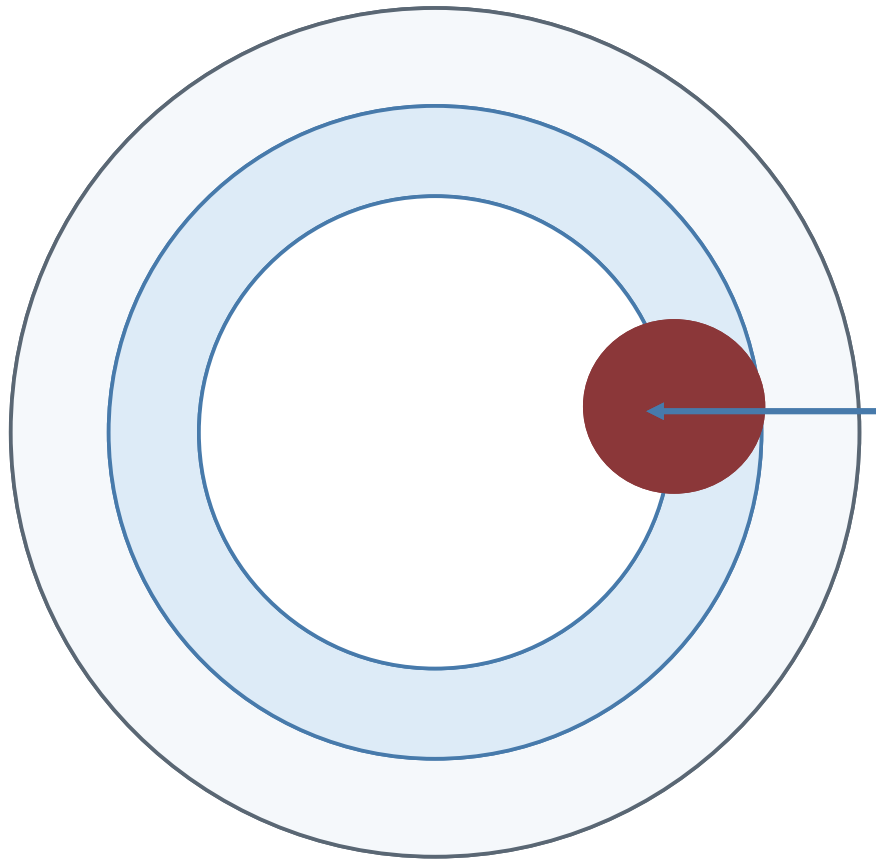
SCHEMATIC — causal / functional relationships

Ring abscess indicates extension beyond a vegetation

- A vegetation is infected material attached to an endocardial or valvular surface.
- At the prosthetic sewing interface, infection may penetrate adjacent tissue.
- Formation of an annular, or ring, abscess.
- This is destructive infection of supporting tissue—not merely a mass on the prosthesis.

**Vegetation →
periannular invasion
→ ring abscess**

From ring abscess to paravalvular regurgitation



Schematic — infected annulus / paravalvular pathway

Prosthetic vegetation

At tissue interface

Ring abscess

Periannular destruction

Loss of intact seal

Blood can traverse surrounding gap

Paravalvular leak

Regurgitation outside the intended orifice

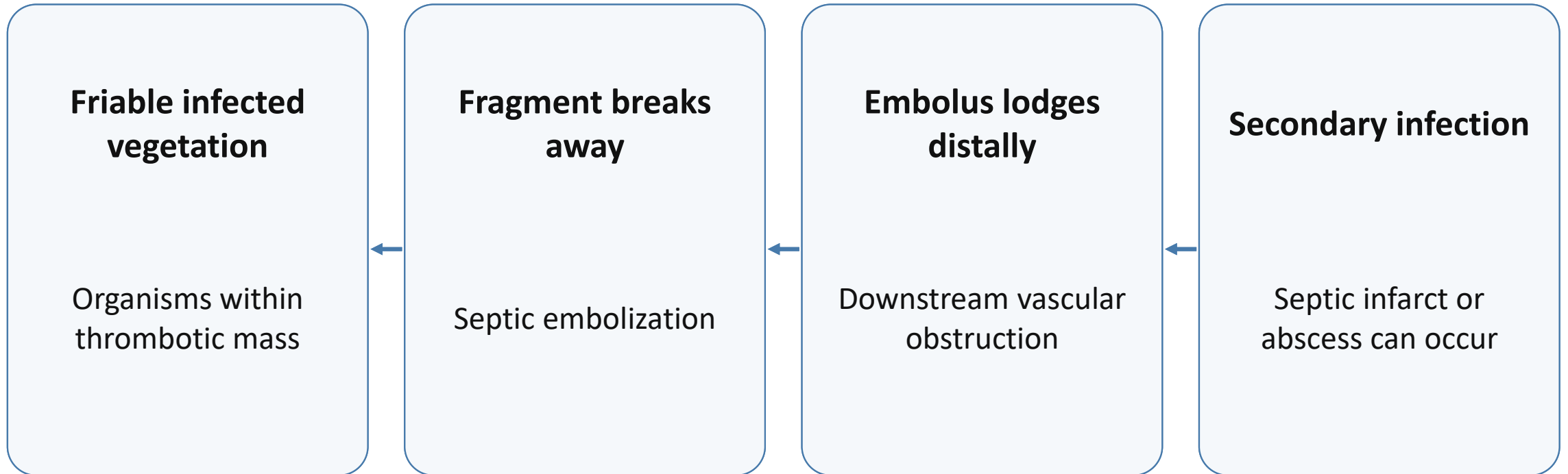
Bioprosthetic cusps can be directly infected

- The tissue of a bioprosthetic leaflet can carry an infective vegetation.
- This contrasts with the frequent prosthesis–tissue-interface distribution described for prosthetic endocarditis.
- Cusp involvement and periannular infection are anatomical patterns, not separate microbial diseases.
- Each pattern may adversely affect valve function through a different structural route.

What an infected vegetation contains

Component	Role in pathologic identification
Fibrin and thrombotic debris	Forms part of the vegetation mass
Inflammatory cells	Marks inflammatory response to infection
Bacteria or other microorganisms	Distinguishes microbial vegetation from bland thrombus
Potential adjacent destruction	Supports tissue-invasive endocarditis

Infected vegetations may embolize



SCHEMATIC — causal / functional relationships

Diagnostic evidence for prosthetic endocarditis

Source-supported evidence

What it contributes

Microbiology or tissue demonstration of organism

Supports pathologic/microbiologic infection

Imaging of oscillating mass, abscess or prosthesis separation

Suggests valve/device involvement

New regurgitation on imaging

Indicates new functional damage

Predisposing prosthesis, fever, vascular findings

Contributes to clinical criteria; not diagnostic alone

2023 Duke-ISCVID framework

- The chapter reproduces a 2023 modified Duke-ISCVID diagnostic criteria table.
- Clinical classification requires 2 major criteria, or 1 major plus 3 minor, or 5 minor criteria in the cited table.
- Prosthetic valve presence is a minor predisposing factor; It is not independently proof of infection.
- Pathologic demonstration of microorganisms in explanted prosthetic tissue is a distinct route of confirmation.

**Source: Robbins
Table 12.9; 2023
criteria**

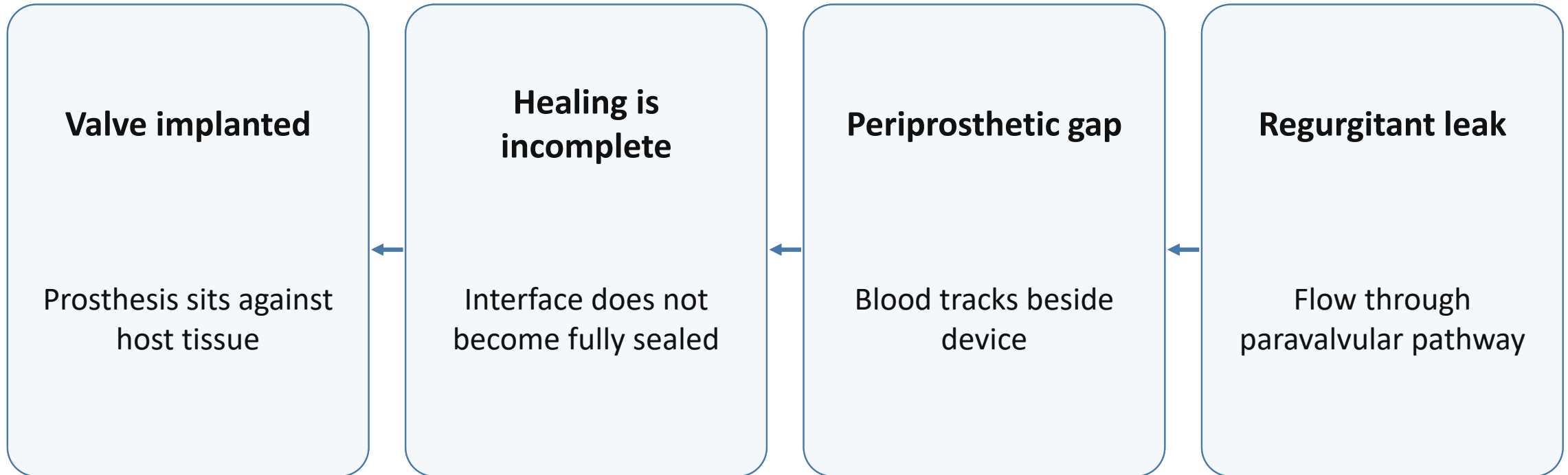
Prosthetic endocarditis is harder to eradicate

- The cure rate of endocarditis on prosthetic valves is worse than for native-valve infections.
- Valve re-replacement is commonly required account.
- Periannular infection and abscess provide a structural explanation for the difficulty.
- These are clinicopathologic observations—not a current treatment algorithm or indication list.

Paravalvular leak has more than one cause

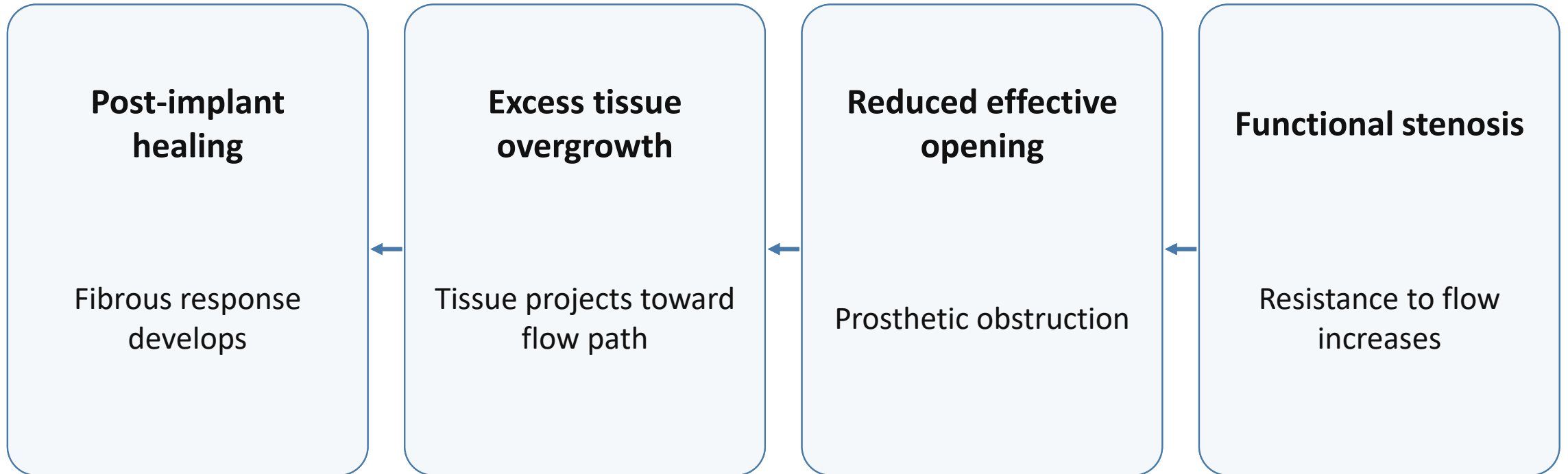
Pathway	Tissue-level mechanism	Distinguishing context
Infectious	Ring abscess damages periannular supporting tissue	Prosthetic endocarditis
Noninfectious	Inadequate healing leaves an imperfect seal	Healing-related prosthesis–tissue interface failure
Shared result	Blood bypasses the intended sealed boundary	Paravalvular regurgitation

Inadequate healing can cause a paravalvular leak



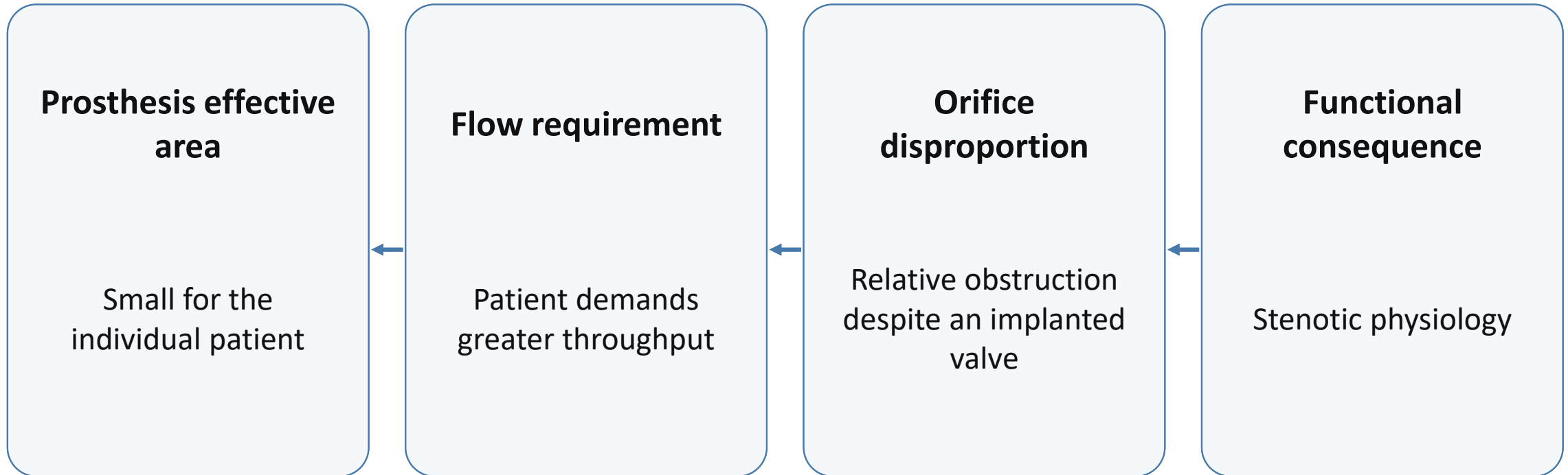
SCHEMATIC — causal / functional relationships

Fibrous tissue overgrowth can obstruct the valve



SCHEMATIC — causal / functional relationships

Valve–orifice disproportion is relative stenosis

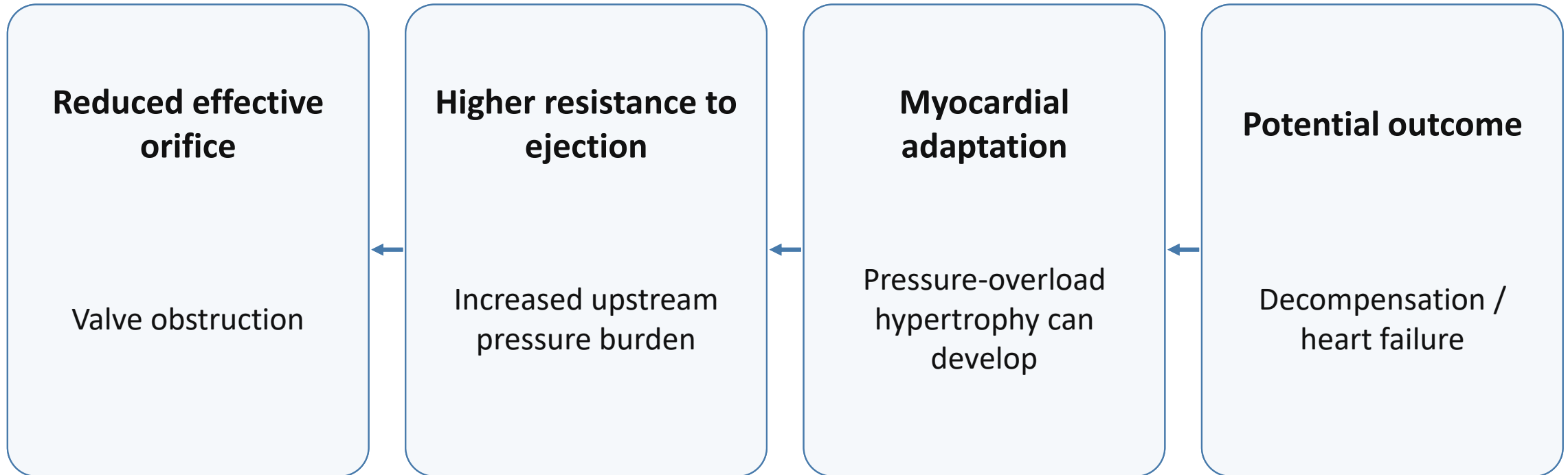


SCHEMATIC — causal / functional relationships

Obstruction has multiple distinct substrates

Source of obstruction	Physical / functional substrate	Category
Mechanical-valve thrombus	Blood clot physically occludes flow path	Thrombotic
Fibrous overgrowth	Healing tissue narrows the orifice	Healing-related
Valve–orifice disproportion	Effective orifice too small for patient needs	Geometric / hemodynamic
Calcified tissue leaflets	Degeneration reduces mobility	Structural degeneration

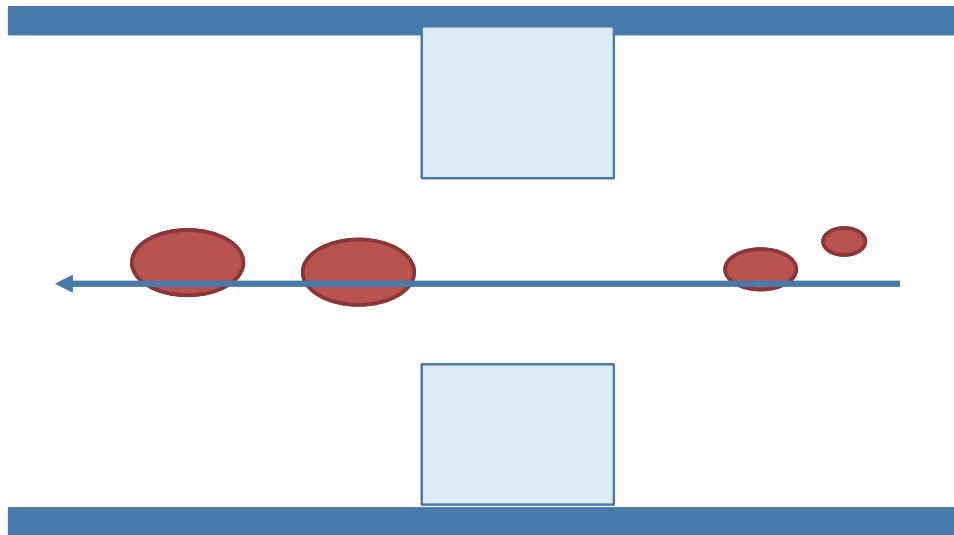
Obstruction produces pressure-overload physiology



SCHEMATIC — causal / functional relationships

Intravascular hemolysis reflects high shear

Narrow jet → elevated shear stress



Schematic — red-cell injury in abnormal flow; Not specimen morphology

Abnormal flow channel

Narrow or regurgitant path

High-velocity jet

Elevated shear stress

Red cell injury

Mechanical disruption of erythrocytes

Intravascular hemolysis

Blood destruction within circulation

Jet injury links valve dysfunction and hemolysis

- Intravascular hemolysis caused by high shear forces as a prosthetic-valve complication.
- In its parent discussion, a narrowed valve can generate a high-speed blood jet.
- A high-velocity jet may also injure endocardium where it strikes.
- Hemolysis therefore points to flow-related mechanical injury, not necessarily infection.

Mechanism-to-manifestation matrix

Mechanism	Valve / tissue consequence	Systemic or clinical consequence
Thrombus on mechanical valve	Occlusion	Embolization may occur
Bioprosthetic degeneration	Calcification and/or tearing	Valve incompetence
Prosthetic infection	Vegetation ± ring abscess	Leak, embolic infection
Interface / flow problems	Leak, overgrowth or mismatch	Regurgitation, stenosis, hemolysis

Failure pathways differ in timing and substrate

Process	Core substrate	Time statement justified by source
Implant/healing-related	Poor seal; fibrous overgrowth	Associated with healing; exact time not quantified
Mechanical thromboembolism	Disturbed flow	Long-term hazard warranting ongoing anticoagulation
Bioprosthetic degeneration	Nonviable tissue	Long-term calcification or tearing
Endocarditis	Microbial vegetation	Can occur after any valve replacement

Case 1: An obstructed mechanical prosthesis

A patient with a mechanical valve develops evidence of reduced prosthetic forward flow. A mass is identified at the valve. Which mechanism should be examined first, and what two outcomes can it produce?

Features to interpret

- Clue: Mechanical device
- Clue: Flow-path obstruction
- Distinguish material failure from blood-derived obstruction

Case 1: Mechanistic interpretation

- A mechanical-valve thrombus can produce direct prosthetic occlusion.
- The same valve-associated thrombotic process may release emboli into the circulation.
- Nonlaminar flow with turbulence and stasis explains the substrate.
- The anticoagulation requirement reflects ongoing thromboembolic risk.

Case 2: A degenerating tissue prosthesis

An explanted porcine bioprosthesis has hard deposits in the cusps and a leaflet tear. Which source-described failure mode links these observations to valve incompetence?

Features to interpret

- Clue: Bioprosthetic leaflet tissue
- Clue: Calcification plus loss of continuity
- Use the textbook Figure 12.27B description

Case 2: Mechanistic interpretation

- Calcification and tearing are the characteristic structural degenerative lesions of bioprostheses.
- Robbins specifically describes calcification with secondary tearing in a porcine valve.
- Degenerated cusps can lose their capacity for normal opening and closure.
- This is a material/tissue failure rather than the typical mechanical-valve thrombosis pathway.

Case 3: Infected prosthetic annulus

A prosthetic valve has a vegetation at its tissue interface, a periannular abscess, and new leakage around the device. Trace the causal sequence and name the alternative noninfectious cause of a similar leak.

Features to interpret

- Clue: Vegetation at the prosthesis–tissue interface
- Clue: Annular tissue destruction
- Clue: Paravalvular rather than transvalvular leak

Case 3: Mechanistic interpretation

- Infective vegetation may extend into periannular tissue and form a ring abscess.
- Destruction of the prosthesis–tissue seal permits a paravalvular regurgitant leak.
- Inadequate healing at the prosthesis–tissue interface is a separate, noninfectious cause.
- Microbiologic and other clinical evidence distinguishes infective disease from noninfective dehiscence.

A structured framework for an abnormal prosthesis

Question	Potential explanation	Next interpretive distinction
Obstructed?	Thrombus; overgrowth; disproportion; degenerated cusps	Which substrate compromises effective area?
Regurgitant?	Torn cusp or paravalvular gap	Through prosthesis or around it?
Infected?	Vegetation; abscess; microbial evidence	Infective versus sterile thrombus
Systemic injury?	Emboli, bleeding, or shear-related hemolysis	Device-related versus treatment-related

Pathology-oriented description of explanted valves

Record	Why it matters
Prosthesis type and involved material	Sets expected dominant failure mechanisms
Thrombus or vegetation; site of attachment	Separates thrombosis from possible infection
Calcification, tear, or fibrous overgrowth	Describes structural or healing-related obstruction
Prosthesis–tissue interface and abscess	Accounts for paravalvular leakage
Correlate with microbiology and clinical findings	Avoids attributing infection from gross appearance alone

Mechanism summary: A valve can fail in many ways

Major group	Archetypal lesion	Principal teaching distinction
Mechanical thromboembolism	Valve-associated clot	Obstruction versus embolization
Bioprosthetic degeneration	Calcification / tearing	Nonviable tissue deteriorates
Infection	Vegetation / ring abscess	Periannular leak and destructive disease
Other	Inadequate healing, overgrowth, mismatch, shear	Leak, stenosis, hemolysis without fracture

Essential points for clinical–pathologic reasoning

- First identify mechanical versus bioprosthetic construction; Dominant risks differ.
- For mechanical valves, link nonlaminar flow to clotting, emboli and the bleeding cost of anticoagulation.
- For tissue valves, connect calcification and tearing with structural failure.
- For either valve, investigate infection, prosthesis–tissue interface integrity, obstruction and high-shear hemolysis.

References

- Robbins, Cotran & Kumar: Pathologic Basis of Disease. 11th ed. (2026). Chapter 12, “The Heart.”
- Primary subsection: “Complications of Prosthetic Valves,” pp. 514–515; Figure 12.27 caption, p. 516.
- Parent context: “Valvular Heart Disease,” pp. 505–506; “Infective Endocarditis,” pp. 511–513; Table 12.9.
- The source discussion and figure descriptions frame the mechanisms reviewed in this lecture.