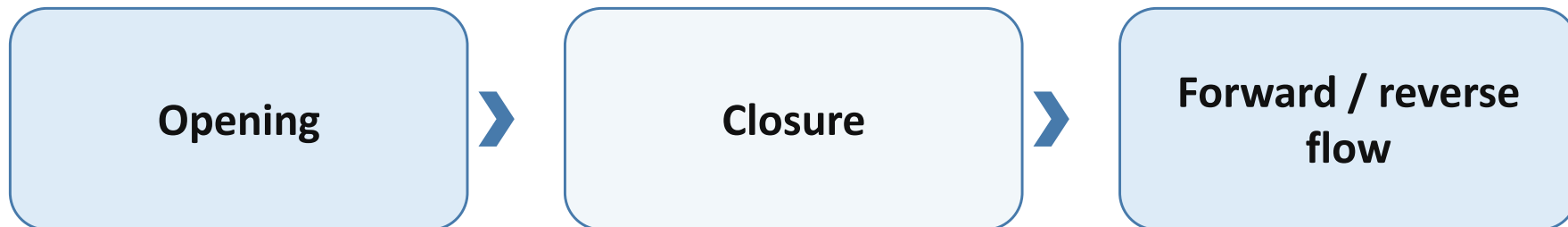


Valvular heart disease

Mechanisms, consequences and acquired causes



Dr Abu Suraih Sakhri

MBBS, MD (Pathology)

www.abusuraihsakhri.com

The teaching scope: Mechanism before lesion name

- Identify stenosis, insufficiency, and coexistence of both lesions.
- Localize disease to leaflets or valve-supporting structures.
- Relate valve dysfunction to murmurs, remodeling and heart failure.
- Compare the acquired causes of major mitral and aortic lesions.

**Valve
function**

**Anatomic
substrate**

**Clinical
effects**

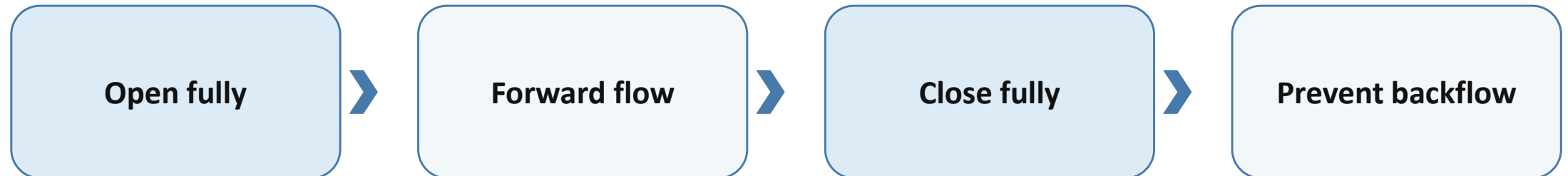
Two fundamental valve failure modes

- Stenosis: Incomplete opening obstructs forward blood flow.
- Insufficiency: Incomplete closure permits backward blood flow.
- The same valve can exhibit stenosis and insufficiency together.

OPENING FAILURE	CLOSURE FAILURE
Restricted forward flow	Backward regurgitant flow

Valve competence requires two distinct actions

- During forward flow, adequate valve opening avoids mechanical obstruction.
- When the valve closes, leaflet coaptation prevents backflow.
- An opening defect and a closure defect therefore produce different lesions.



Stenosis is failure of complete opening

- Obstruction arises at the narrowed valvular orifice.
- The primary flow defect is reduced forward passage through the valve.
- This mechanism is distinct from leakage through an incompletely closed valve.

Incomplete opening



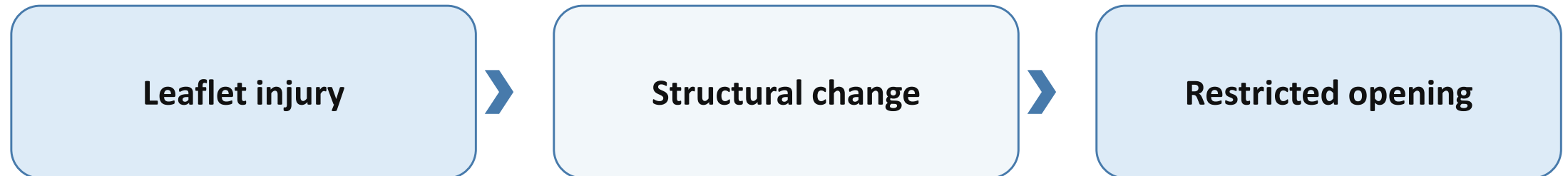
Outflow obstruction



Pressure overload

Acquired stenosis is generally leaflet-based

- Acquired stenosis is almost always associated with a primary leaflet abnormality.
- The lesion is typically a chronic process, not an abrupt failure of support.
- Calcification and valve scarring are representative leaflet mechanisms.



Calcification can restrict leaflet mobility

- Calcific change is identified as a cause of acquired valve narrowing.
- Calcific aortic disease can involve anatomically normal or congenitally bicuspid valves.
- The detailed pathology of calcific aortic stenosis belongs to a separate disease topic.

Calcific leaflet change



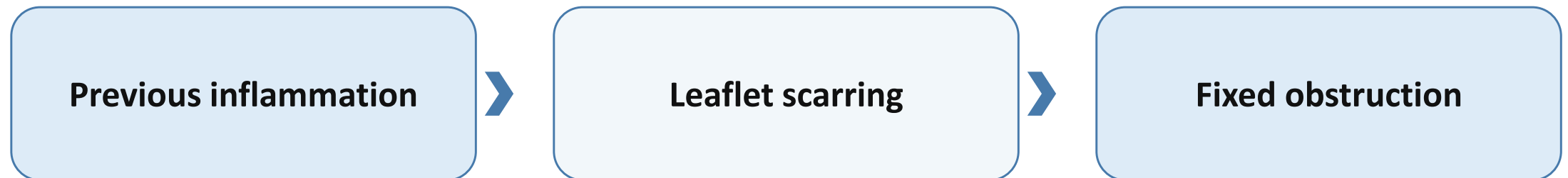
Reduced excursion



Stenosis

Scarring can also restrict leaflet opening

- Postinflammatory scarring is a major acquired cause of mitral stenosis.
- Rheumatic heart disease is the named example in the etiology table.
- Scarring illustrates delayed stenosis after previous valve injury.



Stenosis: Obstruction rather than backflow

- A stenotic valve does not achieve full excursion during opening.
- The consequence is impeded forward movement of blood.
- Any coexisting regurgitation is a second, separately described functional defect.

STENOSIS

Opening incomplete → forward obstruction

REGURGITATION

Closure incomplete → reverse flow

Insufficiency is failure of complete closure

- Insufficiency, regurgitation and incompetence are synonymous terms in this context.
- Failure of coaptation permits flow in the reverse direction.
- Closure failure can develop from intrinsic leaflet injury or an altered support apparatus.

Incomplete closure



Regurgitant flow



Volume overload

Primary leaflet injury can cause insufficiency

- Damage to the valve leaflets may directly impair closure.
- Infective endocarditis is an example of intrinsic leaflet disease.
- Inflammatory scarring can also cause leaflet retraction and progressive incompetence.

Intrinsic leaflet lesion



Failed coaptation



Regurgitation

The support apparatus matters even with intact leaflets

- Regurgitation does not require a primary lesion of the leaflet tissue.
- Abnormalities may involve the aorta, annulus, chordae, papillary muscles or ventricle.
- The site of disruption must be identified separately from the direction of blood flow.

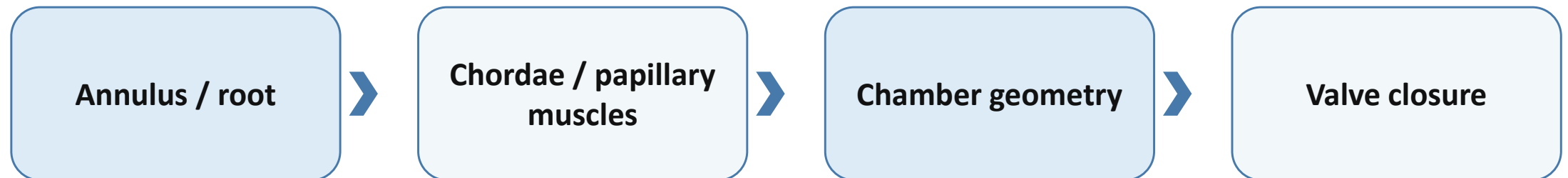
Leaflets

Supporting structures

Regurgitant flow

Supporting structures form a functional system

- The mitral annulus provides a structural attachment for the valve.
- Tendinous cords and papillary muscles are part of the valve tensor apparatus.
- The ventricular free wall and aortic root can affect valve competence.



Chordal rupture: Abrupt mechanical incompetence

- Tendinous cord rupture is explicitly cited as an acute cause of insufficiency.
- The initiating defect lies in the tensor apparatus rather than necessarily in a leaflet.
- The important mechanistic feature is sudden loss of competent closure.

Ruptured chordae



Unstable leaflet support



Acute regurgitation

Papillary muscle pathology can impair valve closure

- Rupture of a papillary muscle appears among causes of mitral regurgitation.
- Papillary muscle dysfunction from fibrosis is another listed mechanism.
- Both illustrate how tensor-apparatus damage creates functional insufficiency.

STRUCTURAL DISRUPTION	FUNCTIONAL DYSFUNCTION
Papillary muscle rupture	Papillary muscle fibrosis

Ventricular geometry can alter valve competence

- Left ventricular enlargement is a cause of mitral regurgitation.
- The table associates enlargement with myocarditis and dilated cardiomyopathy.
- Thus, a myocardial disorder can produce valvular incompetence without primary leaflet injury.

**Left ventricular
enlargement**



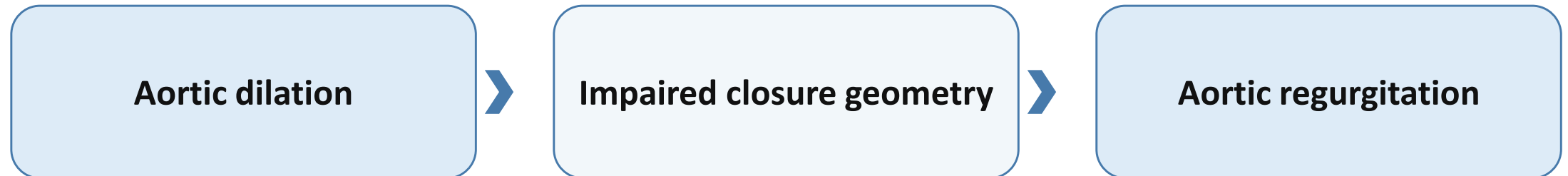
Distorted support / annulus



Mitral insufficiency

Aortic disease can produce valve insufficiency

- The aorta is listed among structures capable of causing valve incompetence.
- Ascending aortic dilation is a frequent setting for aortic insufficiency.
- This is an extravalvular mechanism, distinct from intrinsic cusp damage.



Leaflet lesions and support lesions are distinct

- Intrinsic valve disease modifies the leaflets themselves.
- Support failure alters the structures needed for effective closure.
- Both pathways can converge on insufficiency despite different morphologic targets.

INTRINSIC PATHWAY

Leaflet disease → incomplete closure

EXTRINSIC PATHWAY

Support distortion → incomplete closure

Regurgitation may start abruptly or insidiously

- Insufficiency can be acute after structural failure.
- It may emerge gradually after chronic valve scarring and retraction.
- Rate of onset is a major determinant of clinical consequences.

ABRUPT	GRADUAL
Chordal rupture	Scarring and retraction

Acute mechanical failure: Loss of closure at once

- Chordal rupture represents a sudden disturbance of support.
- The regurgitant lesion may appear with little opportunity for prolonged adaptation.
- Such abrupt lesions with slowly developing valvular disease.



Chronic leaflet scarring: Progressive incompetence

- Scarring and retraction can gradually undermine leaflet closure.
- The change can evolve over time rather than presenting as a sudden lesion.
- Slow tempo permits a different degree of compensation than acute valve injury.

Prior leaflet injury



Fibrosis / retraction



Progressive leak

Stenosis and insufficiency may coexist

- The two defects are not mutually exclusive within a diseased valve.
- A valve can both open inadequately and close incompletely.
- Describing a lesion requires specifying each present functional abnormality.

Restricted opening

Same diseased valve

Incomplete closure

Disease may affect one valve or several

- Valvular disease may involve an isolated valve.
- Alternatively, more than one valve can be abnormal.
- Evaluation should record both the number of affected valves and their lesion types.

SINGLE VALVE

One valve with stenosis and/or insufficiency

MULTIPLE VALVES

More than one diseased valve

The mitral valve is a frequent target

- The general Robbins discussion identifies the mitral valve as the most commonly affected.
- Its lesions include stenosis and regurgitation arising from different etiologies.
- Frequency of involvement does not identify the cause in an individual lesion.

Mitral stenosis

Mitral regurgitation

Varied underlying causes

Describe a valve disorder on three axes

- Functional axis: Stenosis, insufficiency or both.
- Anatomic axis: Leaflet versus support apparatus / adjacent tissue.
- Distribution axis: A single valve versus multivalvular disease.

Function	Opening / closure
Substrate	Leaflet / support
Distribution	Single / multiple

Abnormal valve flow commonly creates a murmur

- Diseased valves generate abnormal patterns of blood flow.
- The resulting abnormal heart sounds are termed murmurs.
- The clinical examination therefore reflects a functional disturbance in flow.

Abnormal valve flow



Audible turbulence



Murmur

A thrill is a palpable manifestation of severe disturbance

- Severe valvular lesions may produce externally palpable thrills.
- A thrill is a physical finding in addition to an audible murmur.
- The text describes this as a feature of severe lesions, not a property of every valve disease.

Severe valve lesion



Marked flow disturbance



Palpable thrill

Murmur location depends on the valve

- Murmurs are not equally well heard at all chest-wall locations.
- The optimal area varies according to the involved valve.
- This localization contributes to bedside characterization of the lesion.

Valve involved

Chest-wall position

Best auscultation area

Murmur quality reflects the lesion

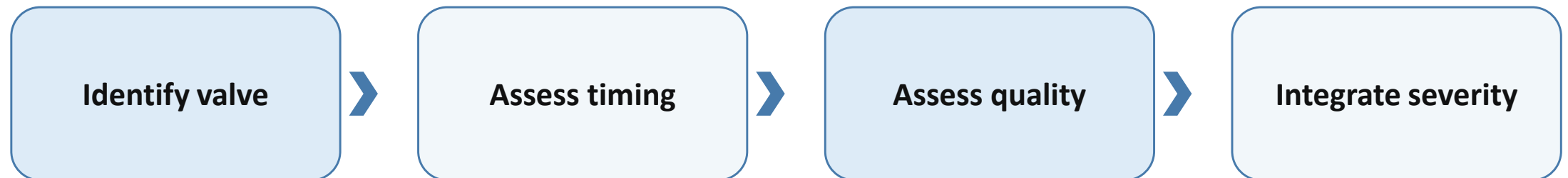
- The nature of valve dysfunction influences the sound generated.
- Robbins explicitly contrasts harsh and soft murmur qualities.
- The severity of dysfunction also contributes to the auscultatory character.

LESION TYPE
Stenosis versus regurgitation

LESION SEVERITY
Influences murmur quality

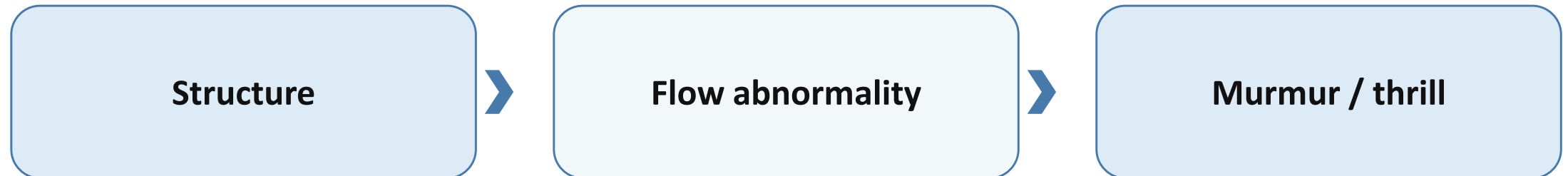
Murmur timing is clinically informative

- The text distinguishes systolic from diastolic murmurs.
- Valve involved, type of dysfunction and severity determine the murmur pattern.
- Timing and quality must be interpreted in relation to the suspected valvular lesion.



Physical findings reflect flow, not just anatomy

- A morphologic lesion can have an audible hemodynamic expression.
- Stenosis and regurgitation generate distinct types of flow disturbance.
- Murmurs and thrills are clues that require anatomical and functional interpretation.



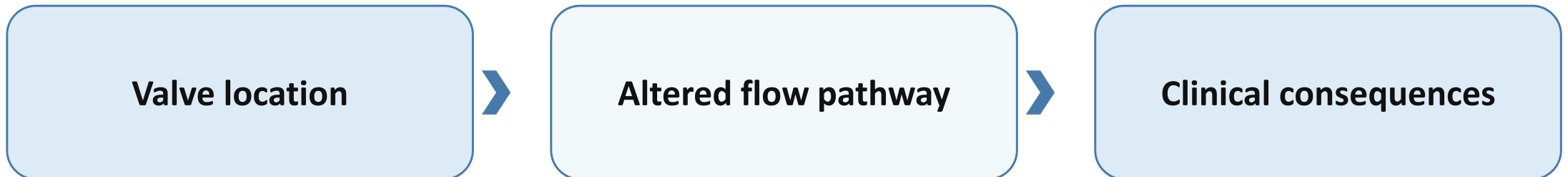
Clinical impact has four principal determinants

- Which valve is diseased.
- How severely the valve function is impaired.
- How quickly the lesion develops.
- How effectively and how rapidly the heart compensates.

Site	Affected valve
Magnitude	Degree of impairment
Tempo	Rate of onset
Adaptation	Quality of compensation

Which valve is involved changes the consequences

- Different valves occupy different positions within cardiac blood flow.
- The clinical effects therefore vary by the valve involved.
- A mechanism-based diagnosis first localizes the abnormal valve.



Degree of impairment influences clinical effects

- Valvular lesions range in the magnitude of functional obstruction or leak.
- Impairment severity as a determinant of clinical outcomes.
- Physical findings and secondary cardiac consequences must be considered together.

Degree of dysfunction



Altered cardiac load



Clinical consequence

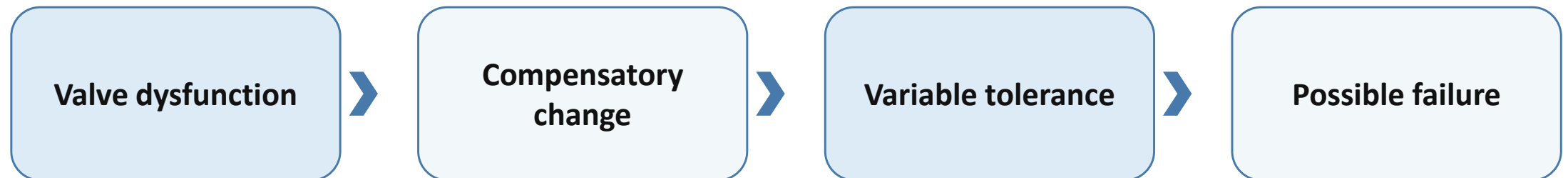
Tempo: Rapid and gradual lesions behave differently

- Abrupt valve failure may cause major consequences before meaningful adaptation.
- Gradual disease may be tolerated over extended intervals.
- The same functional category can therefore vary in its immediate clinical expression.

RAPID ONSET	SLOW ONSET
Little time for adaptation	Longer period for compensation

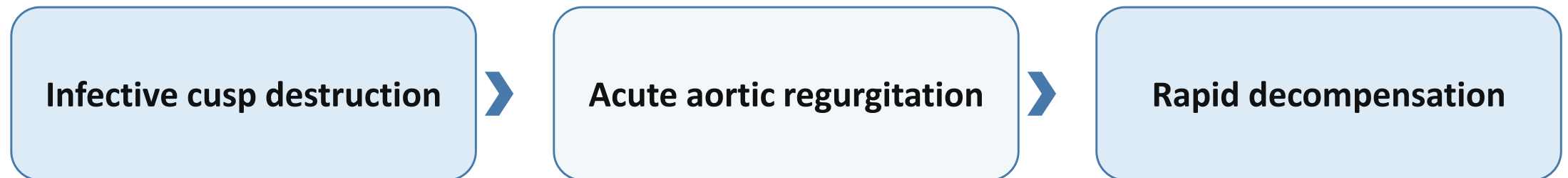
Compensation changes the clinical course

- Clinical effects depend on both rate and effectiveness of compensation.
- The heart may remodel in response to abnormal valvular loading.
- However, compensatory changes do not guarantee that failure will be avoided.



Acute valve destruction can be catastrophic

- Destruction of an aortic cusp by infection as an example.
- Acute, massive aortic regurgitation can follow abrupt cusp damage.
- The described outcome may be rapidly fatal.



Rheumatic mitral stenosis exemplifies slow disease

- Rheumatic mitral stenosis commonly develops gradually over years.
- Its clinical effects can be tolerated for prolonged periods.
- This example contrasts with sudden destructive valve incompetence.

Remote inflammation



Progressive stenosis



Prolonged tolerance

Compare clinical tempo, not just lesion labels

- A rapidly destructive lesion may have immediate severe consequences.
- Chronic obstruction may allow a period of adaptation.
- Tempo and compensation are essential elements of clinicopathologic interpretation.

ACUTE CUSP INJURY

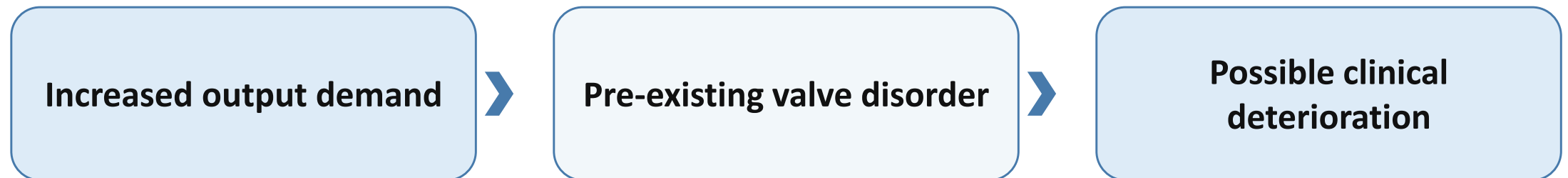
Abrupt severe regurgitation; Potentially fatal

CHRONIC RHEUMATIC INJURY

Slow mitral stenosis; May be tolerated

Pregnancy can unmask limited cardiac reserve

- Pregnancy increases the output demands placed upon the heart.
- These demands can exacerbate pre-existing valve dysfunction.
- The source notes potential adverse maternal or fetal outcomes.



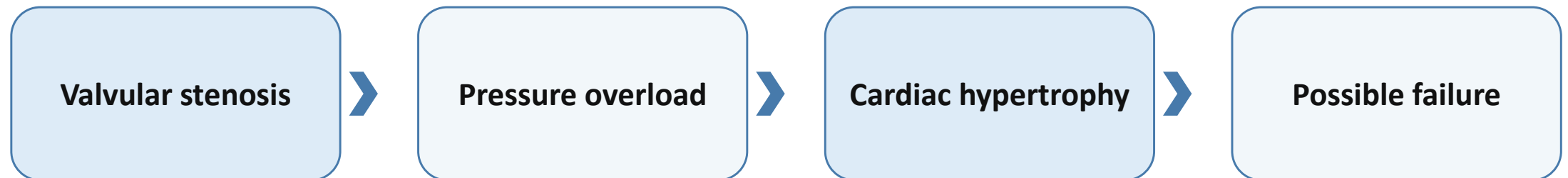
Valve disease can remodel regions on both sides

- Secondary changes may occur proximal to the affected valve.
- Changes may also occur distal to the valve.
- Myocardial remodeling is particularly important in clinically significant valvular disease.



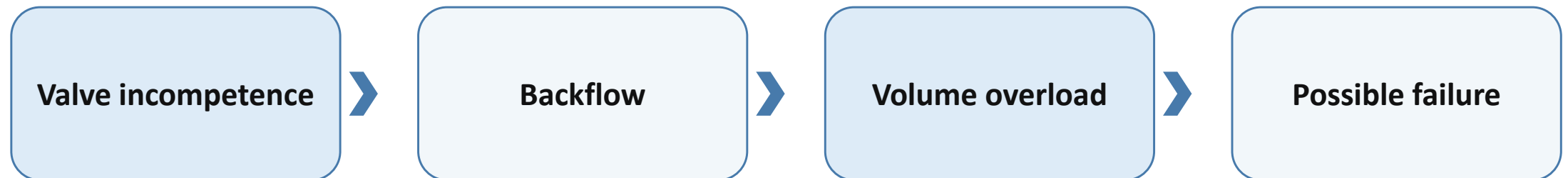
Stenosis produces pressure overload

- A narrowed orifice creates resistance to forward ejection or passage.
- The source associates valvular stenosis with pressure-overload hypertrophy.
- Continued loading can ultimately contribute to heart failure.



Insufficiency produces volume overload

- Regurgitation returns blood in the reverse direction.
- The source associates valvular insufficiency with volume overload.
- Progressive loading may culminate in cardiac failure.



Pressure and volume overload are not interchangeable

- Stenosis primarily changes pressure loading conditions.
- Insufficiency primarily creates volume loading.
- Both can remodel myocardium and converge on heart failure.

STENOSIS

Pressure overload → hypertrophy

INSUFFICIENCY

Volume overload → remodeling

Heart failure is a shared downstream outcome

- Both stenotic and regurgitant disease may progress to heart failure.
- Initial compensatory responses can precede decompensation.
- The presence of a murmur alone does not describe the full myocardial consequence.

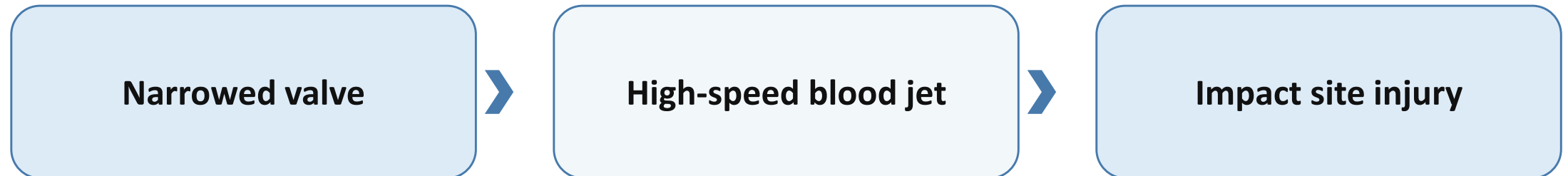
Pressure overload

Myocardial response

Heart failure

Stenotic jets are a second source of tissue injury

- Narrowed valves can create very high-speed streams of blood.
- These jets may strike the endothelium or endocardium.
- Mechanical impact at the site of contact may injure these surfaces.



High-speed jets can also damage erythrocytes

- Robbins explicitly identifies significant hemolysis as a consequence of narrowed-valve jets.
- This is a blood-cell consequence distinct from endothelial or endocardial injury.
- It underscores that abnormal flow can injure tissue and circulating cells.

SURFACE EFFECT	BLOOD EFFECT
Endothelial / endocardial damage	Hemolysis

Valve abnormalities may be congenital or acquired

- The source distinguishes congenital lesions from acquired ones.
- Congenital valve anatomy can predispose to later functional disease.
- The present general lecture emphasizes mechanisms of acquired dysfunction.

CONGENITAL	ACQUIRED
Abnormal valve from development	Later injury or support alteration

Acquired stenosis often declares itself years later

- Remote or chronic leaflet injury may remain subclinical for an extended period.
- Progressive anatomic restriction eventually produces stenosis.
- This is why the history of injury and time course both matter.

Remote/chronic injury



Progressive leaflet damage



Delayed clinical stenosis

Acquired insufficiency has several anatomic origins

- Leaflet damage and supporting-structure dysfunction are both possible.
- The aorta and mitral annulus are named supporting sites.
- Chordae, papillary muscles and ventricular wall are additional sites.

Leaflet	Intrinsic tissue injury
Root / annulus	Altered attachment geometry
Tensor apparatus	Chordal / papillary damage
Ventricle	Distorted chamber structure

Four common acquired lesion–cause pairs

AORTIC STENOSIS

Calcific changes of normal or congenitally bicuspid valves

AORTIC INSUFFICIENCY

Aortic dilation; Intrinsic cusp lesions

MITRAL STENOSIS

Postinflammatory rheumatic scarring

MITRAL INSUFFICIENCY

Myxomatous prolapse; Ventricular dilation;
Other leaflet/support causes

Acquired aortic stenosis: Etiologic map

- Calcific change may affect an anatomically normal aortic valve.
- A congenitally deformed / bicuspid valve can also calcify.
- The etiology table additionally lists postinflammatory scarring.

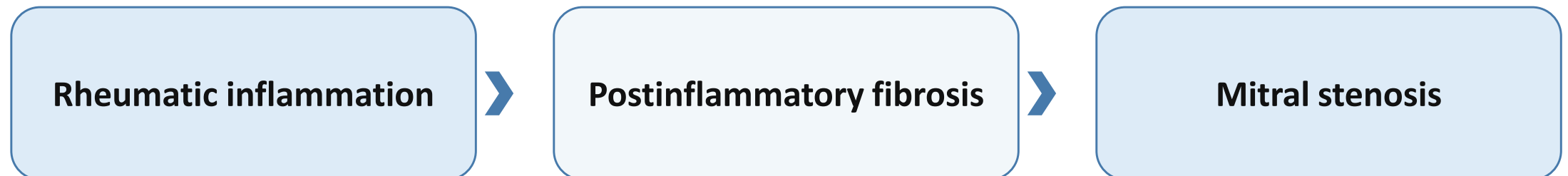
Previously normal valve

Congenital valve deformity

Postinflammatory scar

Acquired mitral stenosis: Inflammatory scarring

- The etiology table identifies postinflammatory scarring as its mechanism.
- Rheumatic heart disease is the disease example.
- Mitral stenosis provides the benchmark for chronic, slow valve disease in the general text.



Aortic insufficiency: Intrinsic cusp disease

- Postinflammatory scarring is an intrinsic valvular cause.
- Infective endocarditis is another intrinsic valvular cause.
- The same functional lesion can alternatively arise from aortic disease.

VALVE LEAFLETS	AORTIC STRUCTURE
Rheumatic injury; Endocarditis	Dilation and other aortic disorders

Aortic insufficiency: Disease of the aorta

- Degenerative aortic dilation is included in the etiologic table.
- Syphilitic aortitis is separately named.
- Ankylosing spondylitis, rheumatoid arthritis and Marfan syndrome are also listed.

Degenerative	Aortic dilation
Infectious	Syphilitic aortitis
Inflammatory	Ankylosing spondylitis / rheumatoid arthritis
Connective tissue	Marfan syndrome

Mitral regurgitation: Leaflet and commissural disease

- Postinflammatory scarring and infective endocarditis are listed.
- Mitral valve prolapse represents another leaflet-related cause.
- Drug-associated valvular fibrosis (fenfluramine–phentermine) appears in the source table.

Postinflammatory / infective

Mitral valve prolapse

Drug-associated fibrosis

Mitral regurgitation: Tensor apparatus injury

- Papillary muscle rupture can produce regurgitation.
- Papillary muscle dysfunction from fibrosis is another listed cause.
- Ruptured chordae tendineae can also cause mechanical valve incompetence.

Papillary rupture	Loss of tissue integrity
Papillary fibrosis	Muscle dysfunction
Chordal rupture	Loss of tethering

Mitral regurgitation: Chamber and annular causes

- Left ventricular enlargement is a listed cause.
- Associated conditions include myocarditis and dilated cardiomyopathy.
- Calcification of the mitral ring is separately listed in this etiologic category.

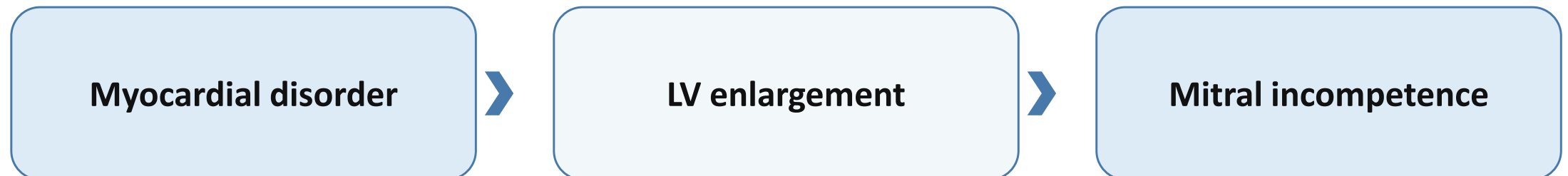
Left ventricular enlargement

**Myocarditis / dilated
cardiomyopathy**

Mitral annular calcification

Ventricular failure can generate a valve lesion

- The etiologic table links mitral insufficiency with left ventricular enlargement.
- The ventricular abnormality is the upstream structural condition.
- This example prevents assuming that every regurgitant valve has primary leaflet disease.



Mitral annular calcification is etiologic context

- Calcification of the mitral ring appears in the mitral regurgitation causes table.
- This abnormality is categorized with chamber and annular mechanisms.
- Its full morphology and clinical profile belong to a separately indexed topic.

Mitral ring calcification



Annular abnormality



Potential regurgitation

The four major lesions have different dominant causes

AORTIC STENOSIS

Calcific changes of normal or congenitally bicuspid valves

AORTIC INSUFFICIENCY

Aortic dilation; Intrinsic cusp lesions

MITRAL STENOSIS

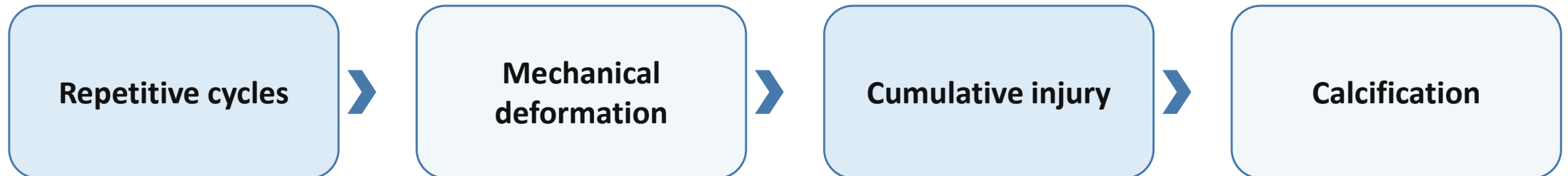
Postinflammatory rheumatic scarring

MITRAL INSUFFICIENCY

Myxomatous prolapse; Ventricular dilation;
Other leaflet/support causes

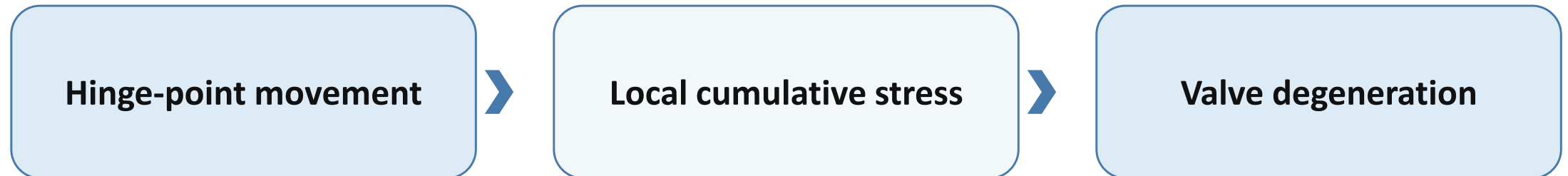
Calcific degeneration begins with repetitive stress

- Heart valves undergo millions of cycles of opening and closing each year.
- Cusp tissue deforms substantially during each contraction.
- Cumulative stress can contribute to damage and calcific degeneration.



Valve hinge points are stress-sensitive

- Mechanical stress is especially prominent at leaflet hinge points.
- Repetitive motion concentrates deformation at these attachments.
- The source links cumulative injury to clinically important dysfunction.



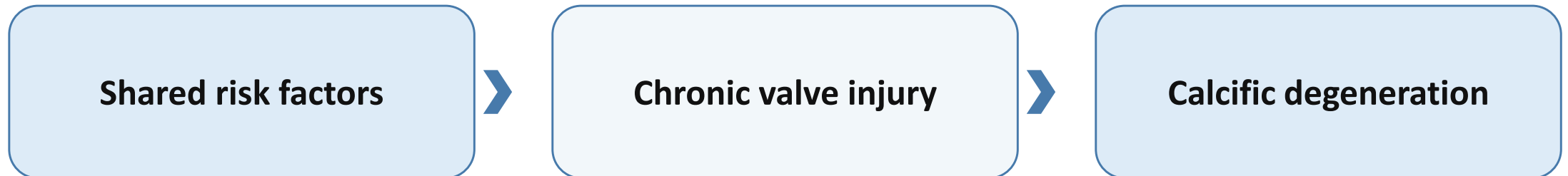
Closed valves withstand substantial gradients

- The source reports a transvalvular closed-phase gradient of approximately 120 mm Hg for the mitral valve.
- The corresponding aortic-valve estimate is approximately 80 mm Hg.
- These source figures illustrate the repetitive loading borne by delicate valvular tissue.

MITRAL VALVE	AORTIC VALVE
≈120 mm Hg in the closed phase	≈80 mm Hg in the closed phase

Calcific degeneration and vascular risks overlap

- Atherosclerotic risk factors are strongly associated with calcific valvular degeneration.
- The association partly to shared aspects of injury.
- This general relationship should not be mistaken for a complete account of calcific aortic stenosis.



Separate mechanism, anatomic site and etiologic name

- A functional label (regurgitation) does not identify the injured tissue.
- An anatomic label (papillary muscle or cusp) does not by itself specify the clinical tempo.
- A disease label (rheumatic or infective) provides causal context, not the whole hemodynamic description.

Function

Anatomic substrate

Etiology / tempo

Clinical exercise: Intact cusps, significant regurgitation

Question: Can significant valve insufficiency occur without primary leaflet damage?

- Identify at least three nonleaflet structures mentioned by Robbins.
- Explain the shared consequence of their failure.

Intact leaflets



Support apparatus failure



Regurgitant flow

Clinical exercise: Mechanistic resolution

- Yes. The aorta, annulus, chordae, papillary muscles and ventricle are all possible sites.
- Disruption or distortion can prevent complete leaflet closure.
- This produces backward flow, even if primary leaflet injury is absent.

Nonleaflet lesion



Failure of coaptation



Insufficiency

Clinical exercise: Two lesions, two loading states

Stenosis: Identify the initial flow defect and the dominant loading response.

- Insufficiency: Identify the initial flow defect and the dominant loading response.
- State the shared potential downstream outcome.

STENOSIS	INSUFFICIENCY
Incomplete opening → ?	Incomplete closure → ?

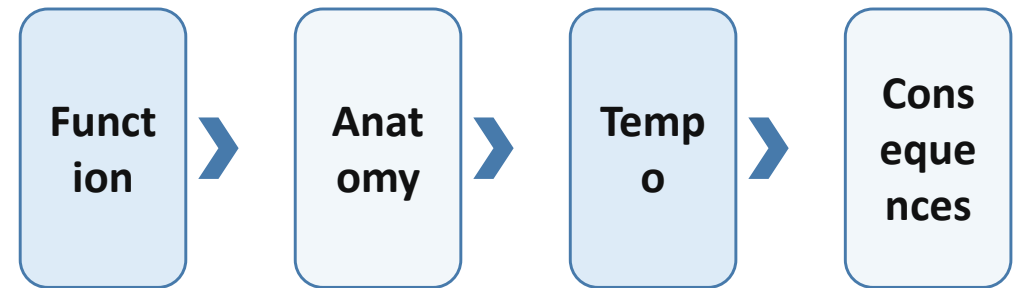
Clinical exercise: Mechanistic resolution

- Stenosis obstructs forward flow and creates pressure overload.
- Insufficiency permits regurgitation and creates volume overload.
- Both may produce secondary myocardial changes and progress to failure.

STENOSIS	INSUFFICIENCY
Pressure overload; Hypertrophy	Volume overload; Remodeling

Synthesis: Four steps to interpret a valve lesion

- Define the functional abnormality: Obstruction, regurgitation or both.
- Localize the anatomic substrate: Leaflets versus support structures.
- Characterize onset and expected compensatory response.
- Connect the flow disturbance to murmurs, abnormal loading and possible failure.



Core reference

- Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th edition (2026).