

Chronic or Healed Pericarditis

Morphology, mechanical consequences, and clinicopathologic interpretation

Anatomical pathology • clinician-oriented teaching

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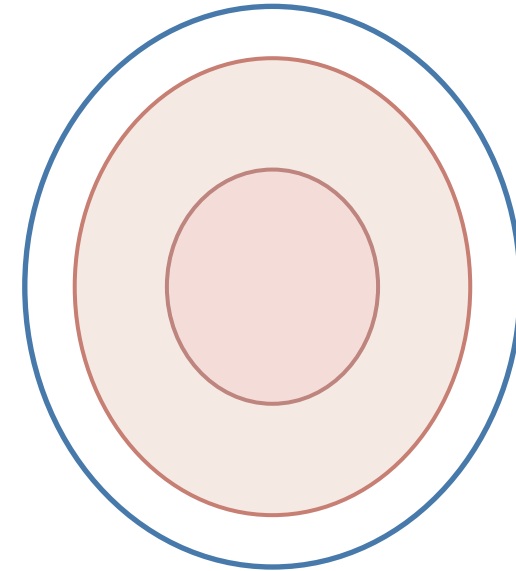
Learning goals

- Distinguish innocuous healed adhesions from hemodynamically important fibrosis.
- Explain how mediastinal tethering changes systolic motion and workload.
- Relate fibrocalcific encasement to limited diastolic filling.
- Connect gross morphology with clinical signs and pericardiectomy.

Pericardial disease: The major pathologic processes

- Fluid accumulation changes the mechanical environment around the heart.
- Inflammation can resolve or become organized into fibrous tissue.
- Dense fibrous constriction is a distinct, potentially serious endpoint.
- A combination of fluid, inflammation, and fibrosis can occur.

Simplified cross-sectional schematic

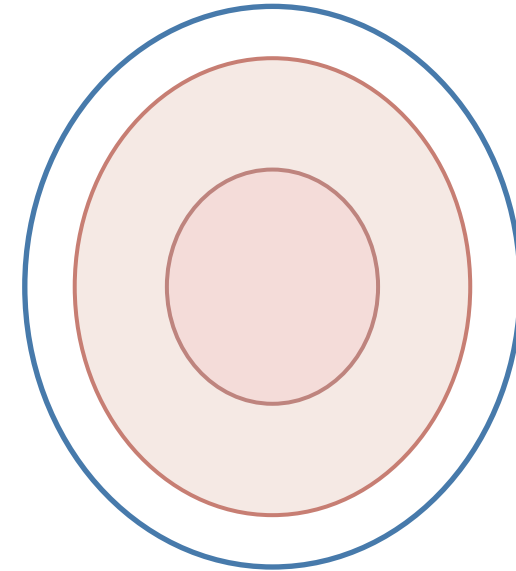


Sliding serosal layers

The pericardial cavity is normally a potential space

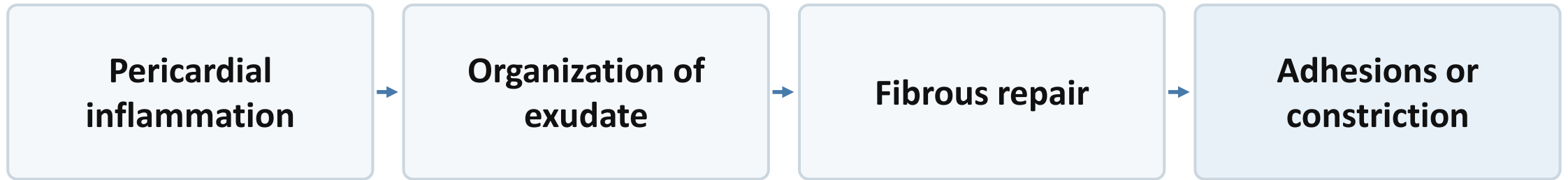
- Visceral and parietal serosal surfaces define the pericardial sac.
- The intervening virtual space permits relative movement of the layers.
- Scarring may bridge the layers without necessarily blocking filling.
- The mechanical effect depends on the configuration of the scar.

Simplified cross-sectional schematic



Sliding serosal layers

Resolution versus organization of pericardial inflammation



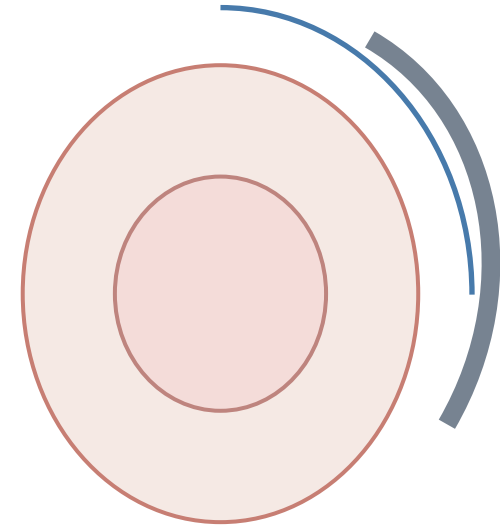
Repair may leave only small plaques, or may produce an extensive mechanical constraint.

Conceptual sequence; Timing and scale not implied

Plaque-like fibrous thickening: “soldier’s plaque”

- A localized plaque is a healed fibrous alteration of the serosa.
- The gross lesion may be unobtrusive despite an inflammatory history.
- It is generally not associated with important cardiac impairment.
- This illustrates that visible fibrosis does not always mean constriction.

Simplified cross-sectional schematic

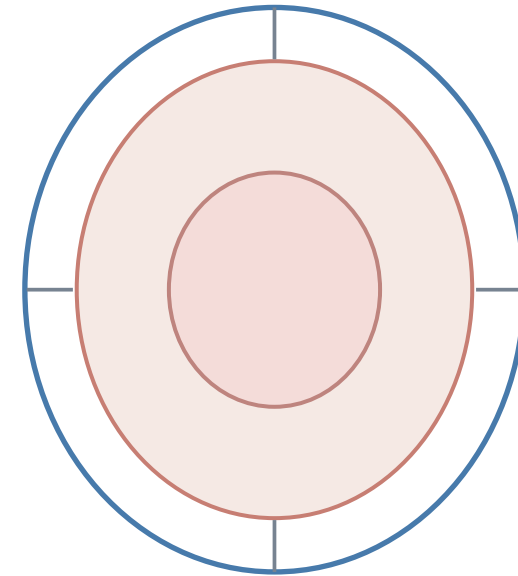


Localized fibrous plaque

Delicate fibrous adhesions are usually innocuous

- Thin adhesions may form between pericardial serosal surfaces.
- The bridges represent organization rather than active exudation.
- Most do not appreciably impair cardiac function.
- Recognition of the lesion requires separation of form from effect.

Simplified cross-sectional schematic

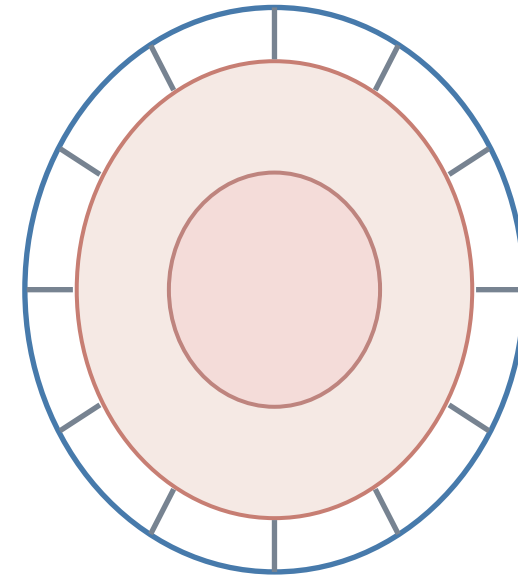


Delicate bridging adhesions

Mesh-like adhesions may obliterate the potential space

- Stringy strands can occupy much of the virtual pericardial cavity.
- The source calls this pattern adhesive pericarditis.
- Even substantial adhesions usually leave cardiac function intact.
- Obliteration alone is therefore not equivalent to constriction.

Simplified cross-sectional schematic



Mesh-like adhesions

Healed lesions: Morphology and usual consequence

Pattern	Pathologic appearance	Functional implication
Localized plaque	Discrete fibrous serosal thickening	Usually negligible
Delicate adhesions	Fine bridging fibrous strands	Rarely significant
Adhesive pericarditis	Mesh-like fibrous strands; Space may disappear	Most often no functional effect

The critical distinction is mechanical

The same reparative process can have very different consequences

Limited plaque

A localized change with little interference in cardiac motion.

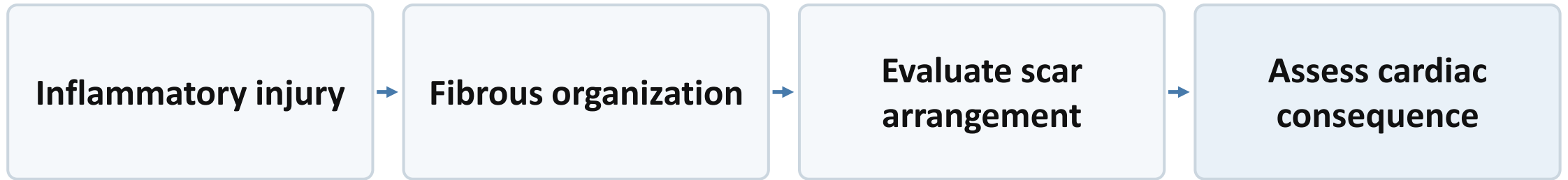
Adhesive fusion

Serosal bridges can eliminate the space without major dysfunction.

Rigid encasement

A continuous scar shell can impede ventricular expansion.

A reparative lesion is not necessarily a constricting lesion



Thickness, continuity, and attachment determine the interpretation more than the mere presence of fibrosis.

Conceptual sequence; Timing and scale not implied

Earlier inflammation can leave a late fibrous endpoint

- The healed lesion may be seen after the active inflammatory phase.
- Organized inflammation is the common pathologic substrate.
- The source does not require a clinically recognized antecedent episode.
- A chronic morphology cannot itself date the inflammatory event.

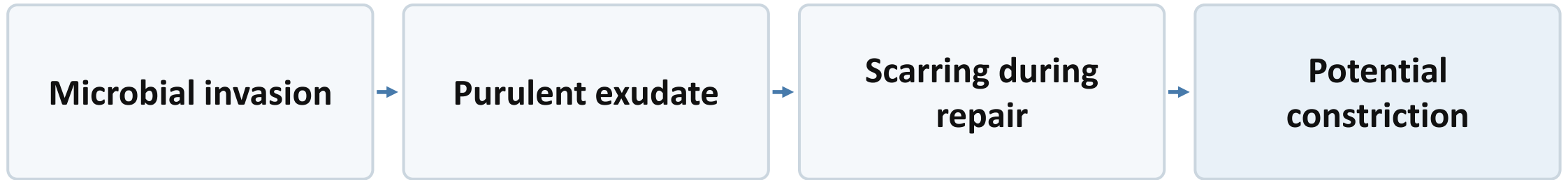
Pericarditis causes in the parent section

Group	Examples noted by Robbins
Infectious	Viruses, pyogenic bacteria, tuberculosis, fungi, parasites
Immune-mediated	Rheumatic fever, systemic lupus erythematosus, postcardiotomy, Dressler syndrome
Other	Uremia, infarction, cardiac surgery, tumors, trauma, radiation

Suppurative inflammation can heal by scarring

- Purulent pericarditis is an active microbial infection of the sac.
- The inflammatory exudate and subsequent tissue injury may be marked.
- Complete resolution is infrequent in the source description.
- Organization and scarring commonly follow; Constriction can result.

Suppurative pericarditis: From infection to fibrosis



A severe inflammatory antecedent can plausibly explain a later dense scar shell.

Conceptual sequence; Timing and scale not implied

Tuberculous pericarditis: A relevant antecedent

- Tuberculosis appears among infectious causes of pericarditis.
- The source notes that tuberculous involvement may show caseation.
- Healed pericardial fibrosis may follow inflammatory injury.
- Caseation is an inflammatory clue, not a requirement for a healed scar.

Previous cardiac surgery can precede adhesions

- Cardiac surgery is listed among causes of pericardial inflammation.
- A fibrinous reaction may follow routine cardiac operations.
- Adhesive mediastinopericarditis can follow previous surgery.
- A postsurgical history helps explain external pericardial attachments.

Mediastinal radiation can cause chronic pericardial damage

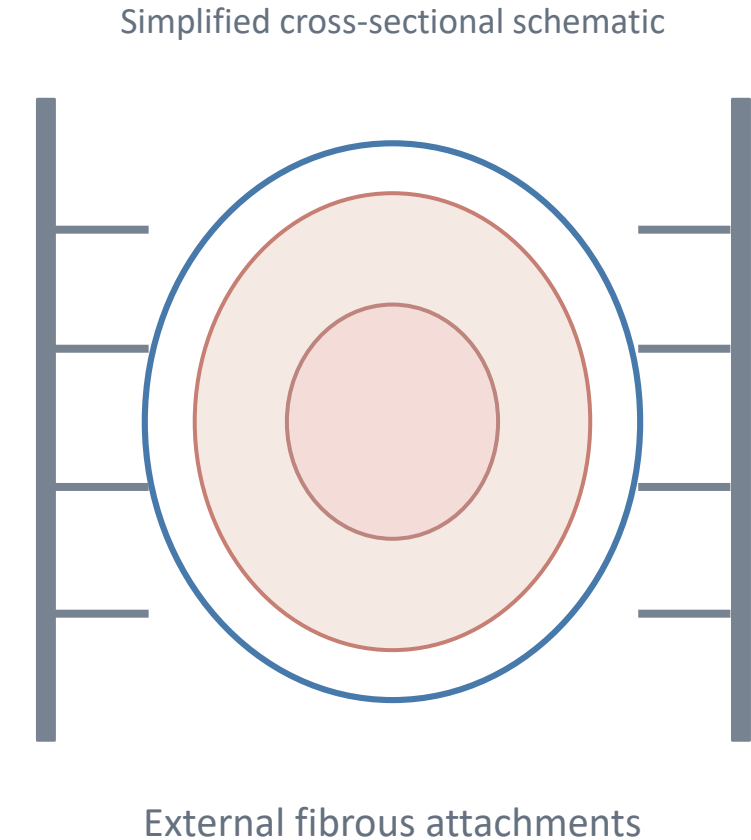
- Mediastinal irradiation is a stated antecedent of adhesive mediastinopericarditis.
- Radiation can induce pericarditis, fluid accumulation, and chronic disorders.
- The mechanism becomes important when repaired tissues adhere externally.
- The relevant exposure history may precede the presentation.

Antecedent clues and the resulting pathology

Antecedent history	Source-supported later concern
Prior infectious pericarditis	Adhesive mediastinopericarditis; Scarring after severe infection
Prior cardiac surgery	Fibrinous reaction and external adhesion
Mediastinal irradiation	Pericardial inflammation, effusion, chronic adhesive disease
No recalled pericarditis	Still compatible with constrictive pericarditis

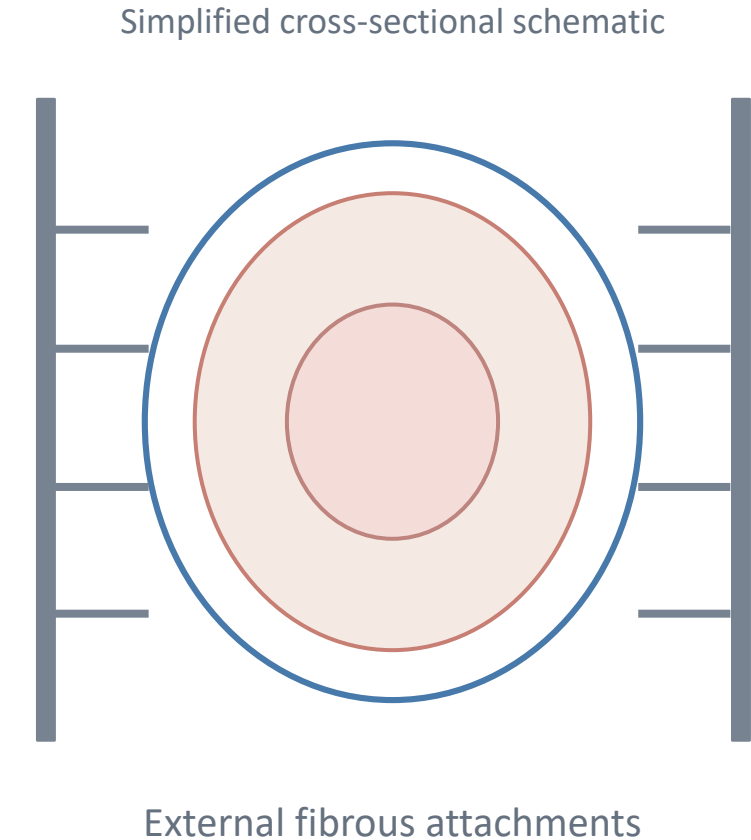
Adhesive mediastinopericarditis is external tethering

- The pericardial sac is obliterated by fibrous adhesion.
- The outer aspect of the parietal layer attaches to nearby structures.
- The scar restricts motion through mechanical tethering.
- This pattern is not simply a thick inner pericardial shell.

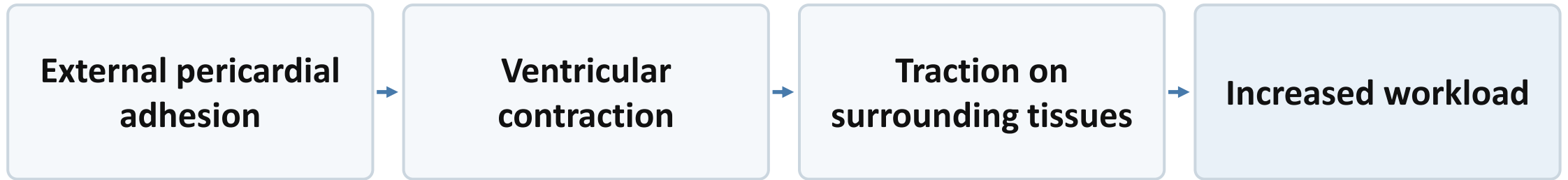


The site of adhesion determines the mechanical problem

- Internal adhesions connect pericardial serosal surfaces.
- External adhesions bind the parietal pericardium to the mediastinum.
- The contracting heart must move tissues attached to the sac.
- External connections explain characteristic systolic findings.



Adhesive mediastinopericarditis: Systolic mechanism



Mechanical work is expended against the attachments, not just to eject blood.

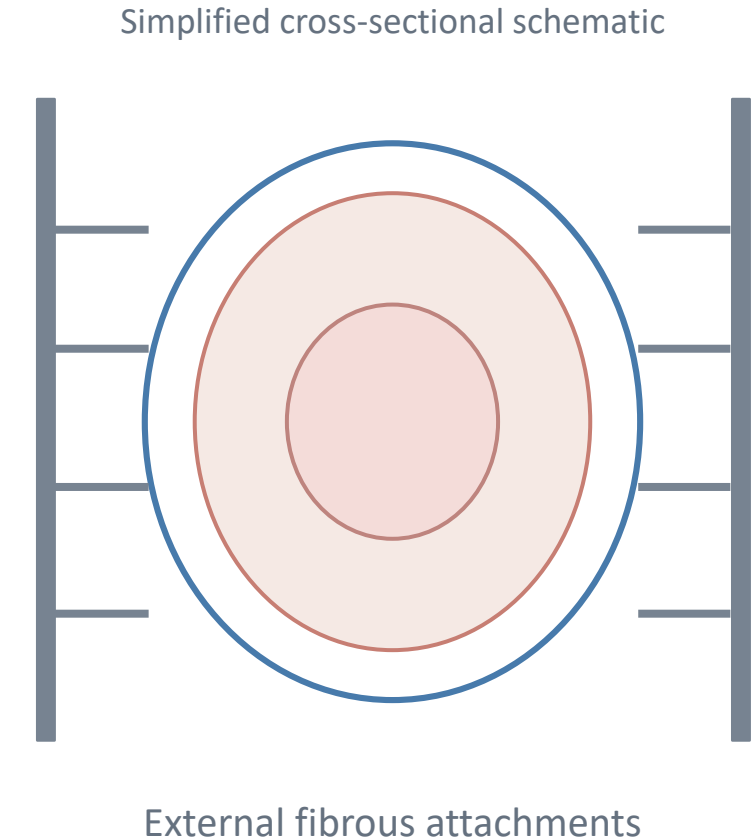
Conceptual sequence; Timing and scale not implied

Normal versus externally tethered systole

Feature	Untethered heart	External pericardial adhesions
Motion	Contraction without fibrous mediastinal coupling	Contraction pulls adjacent structures
Mechanical burden	No scar-related external traction	Additional work against attachments
Visible effects	No scar-induced chest retraction	Rib cage or diaphragm may retract

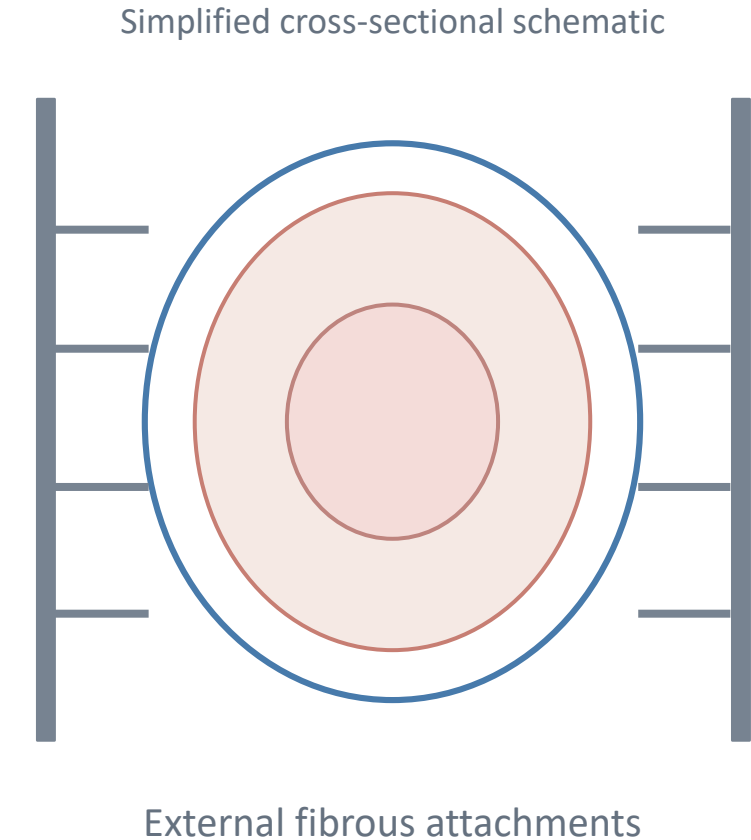
Systolic rib-cage retraction reflects traction

- The heart contracts while tethered to adjacent thoracic structures.
- Systolic traction can produce rib-cage retraction.
- This is a mechanical manifestation of mediastinal adhesions.
- The finding is possible, not obligatory, account.



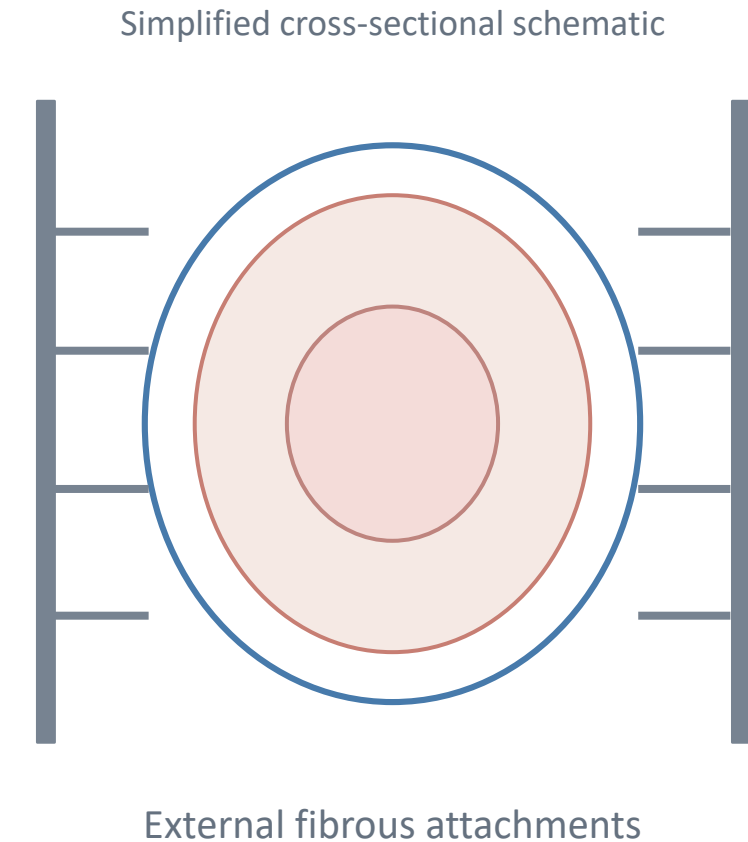
Systolic diaphragmatic retraction has the same basis

- The diaphragm may be linked mechanically to the adhered pericardium.
- Each systole can transmit movement into the diaphragm.
- This finding points to external attachment and abnormal traction.
- Absence of visible retraction does not disprove healed adhesions.



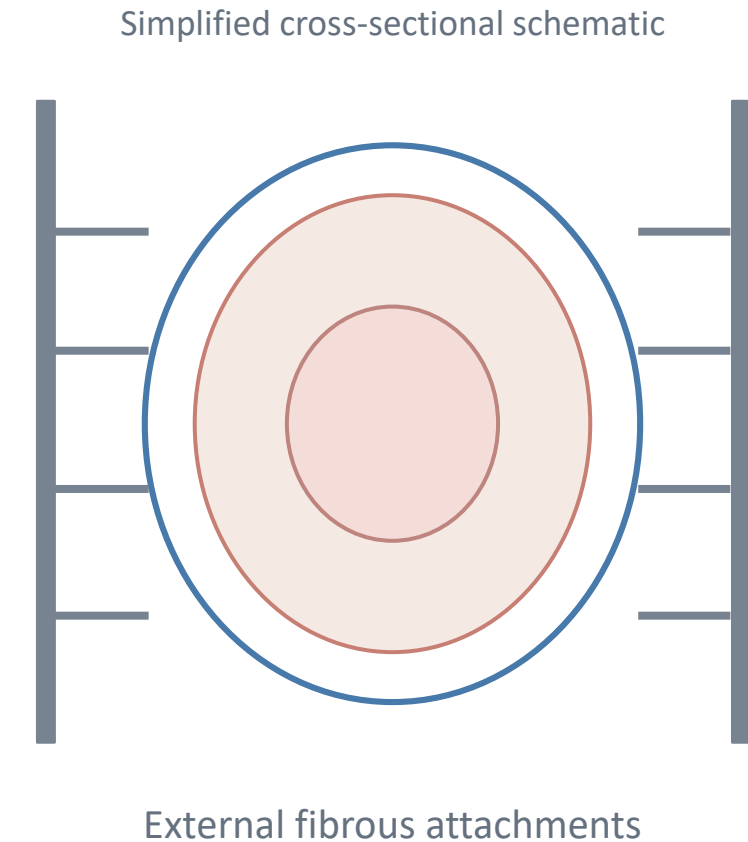
Pulsus paradoxus may accompany tethered disease

- Pulsus paradoxus among possible findings.
- It is presented as an observed clinical phenomenon, not a universal sign.
- It should be interpreted with other physical and pathologic features.
- The textbook excerpt does not assign a numerical threshold here.



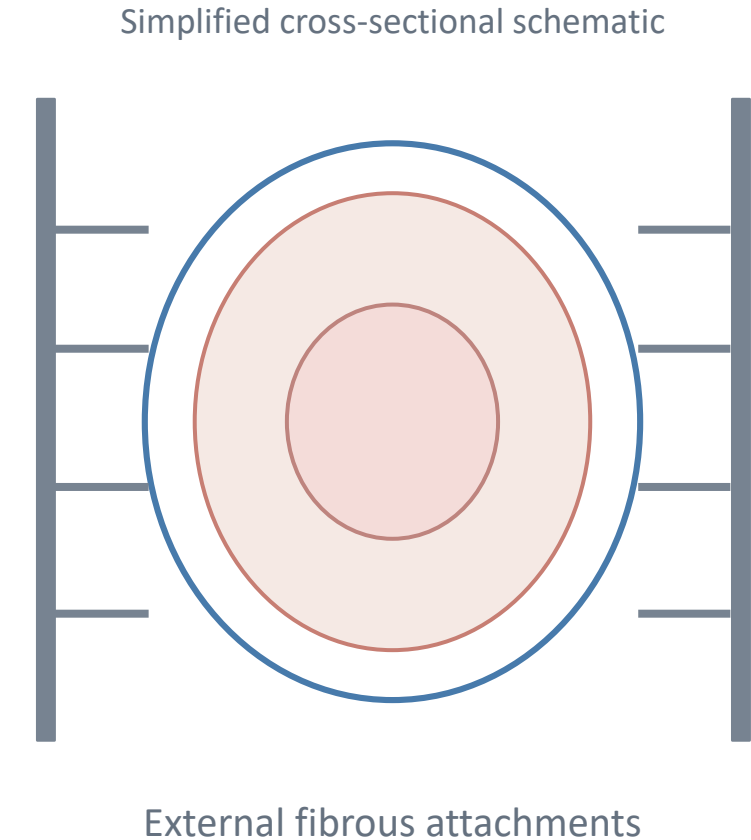
External traction can increase cardiac workload

- Each systolic contraction acts against attached surrounding tissue.
- The mechanical demand is added to the usual ejection task.
- Sustained increased workload may prompt cardiac remodeling.
- The source describes hypertrophy and dilation in some cases.



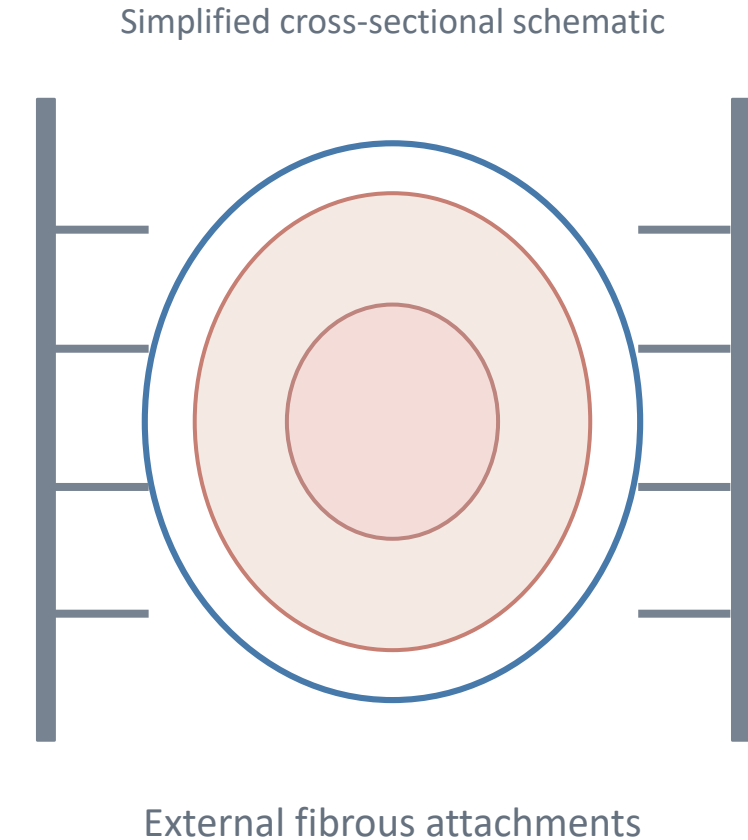
Hypertrophy in adhesive mediastinopericarditis

- Systolic tethering increases external mechanical load.
- Significant myocardial hypertrophy may occasionally develop.
- This is remodeling in response to the workload, not the scar itself.
- Hypertrophy is not claimed to occur in every adhesive case.



Dilation is also possible with external adhesions

- Some hearts develop dilation in addition to hypertrophy.
- The pericardium is attached externally rather than forming a rigid shell.
- This distinguishes it from classic fully constrictive pericarditis.
- The difference is a useful gross-pathologic discriminator.



Adhesive mediastinopericarditis: One causal unit

The defining event is abnormal systolic coupling of the heart to the mediastinum

Anatomic substrate

Obliterated sac with external fibrous attachments.

Mechanical effect

Systolic traction on rib cage and diaphragm; Added workload.

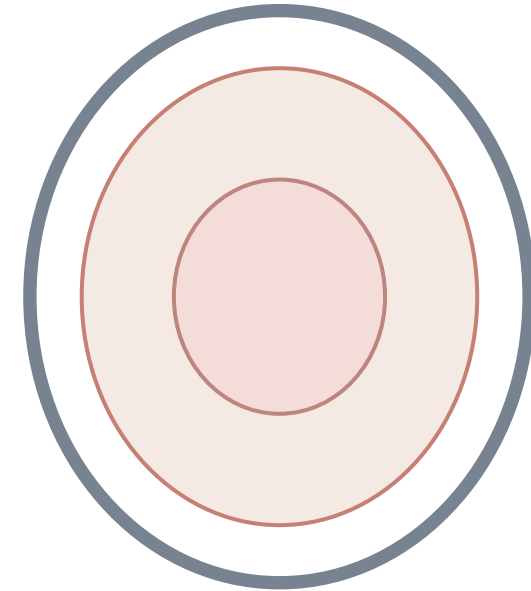
Possible consequence

Hypertrophy and dilation;
Pulsus paradoxus may be observed.

Constrictive pericarditis is an enclosing scar

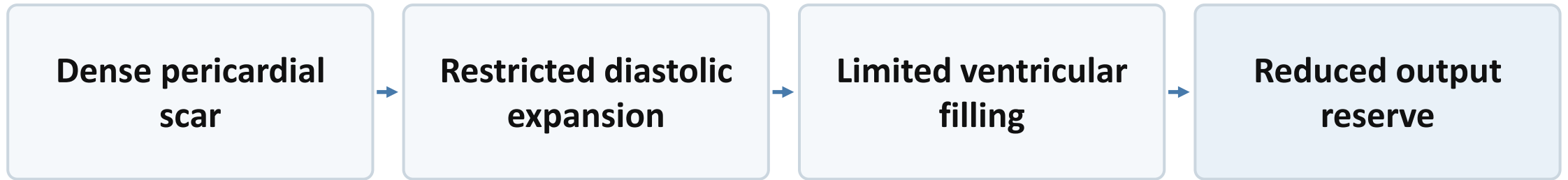
- The heart is encased within dense fibrous or fibrocalcific tissue.
- The scar limits diastolic expansion rather than merely tethering systole.
- Ventricular filling is restricted, with consequences for cardiac output.
- An identifiable previous attack of pericarditis is not required.

Simplified cross-sectional schematic



Constraining fibrous shell

The mechanical sequence of constriction



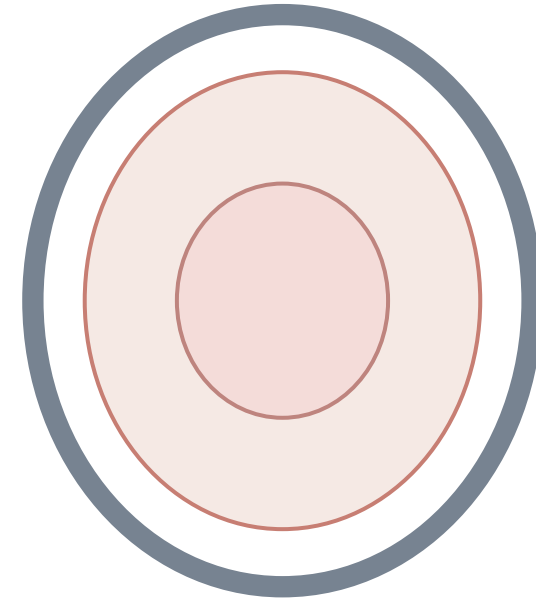
The enclosing scar imposes an external ceiling on the heart's ability to expand.

Conceptual sequence; Timing and scale not implied

Gross morphology: A dense circumferential shell

- The defining tissue is an enclosing fibrous scar.
- It may largely or completely eliminate the normal pericardial space.
- The encasement is more important than isolated thin adhesions.
- At severe extremes, the scar resembles a fitted mold.

Simplified cross-sectional schematic

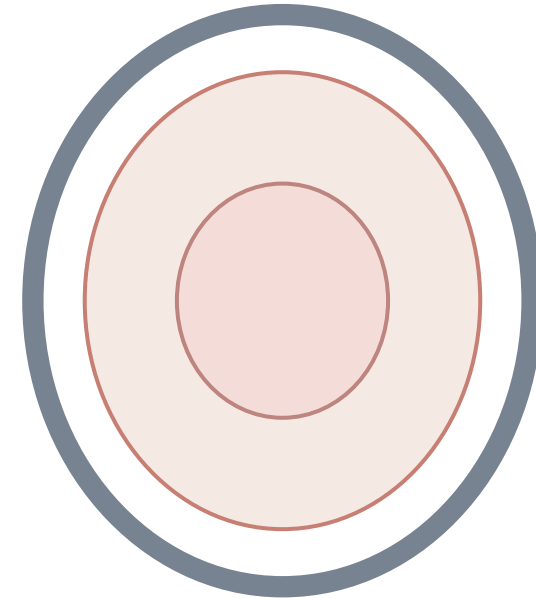


Thickened fibrous scar

The fibrous scar may be remarkably thick

- Scar thickness reaching as much as 1 cm.
- This is an upper descriptive value, not a diagnostic cutoff.
- The fibrous tissue may surround the heart extensively.
- Thickness must be interpreted with distribution and mechanical effect.

Simplified cross-sectional schematic

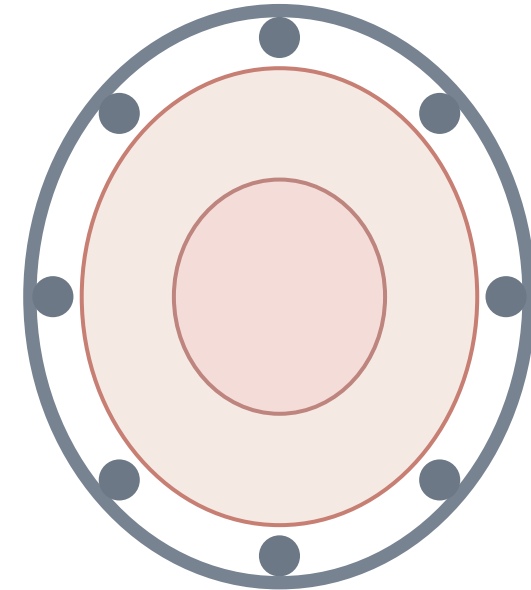


Thickened fibrous scar

Fibrocalcific pericardial scarring

- The constricting shell can include fibrous tissue and calcification.
- Calcification is an additional gross characteristic in some cases.
- The source does not require calcification for constrictive disease.
- A rigid enclosing scar is the critical functional lesion.

Simplified cross-sectional schematic

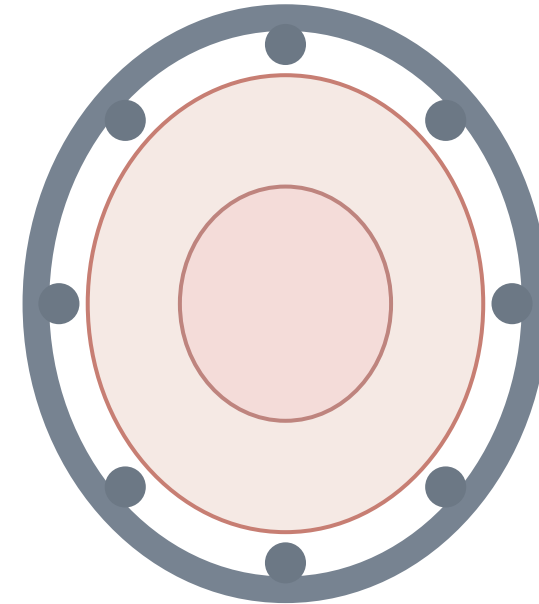


Fibrocalcific shell

Concretio cordis: The plaster-mold appearance

- Severe fibrocalcific encasement may resemble a plaster cast.
- This extreme configuration is termed concretio cordis.
- The scar closely surrounds the heart and impedes expansion.
- The term is a gross description, not a distinct etiology.

Simplified cross-sectional schematic

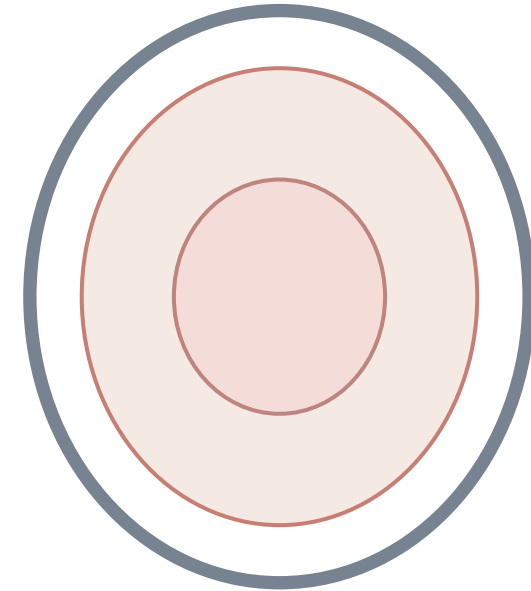


Dense plaster-mold scar

Obliteration of the pericardial space has two meanings

- Mesh-like adhesions can erase the space with little functional effect.
- Constrictive scar can also obliterate the space.
- In constriction, the scar creates a stiff circumferential boundary.
- Both morphology and function are needed for correct interpretation.

Simplified cross-sectional schematic



Constraining fibrous shell

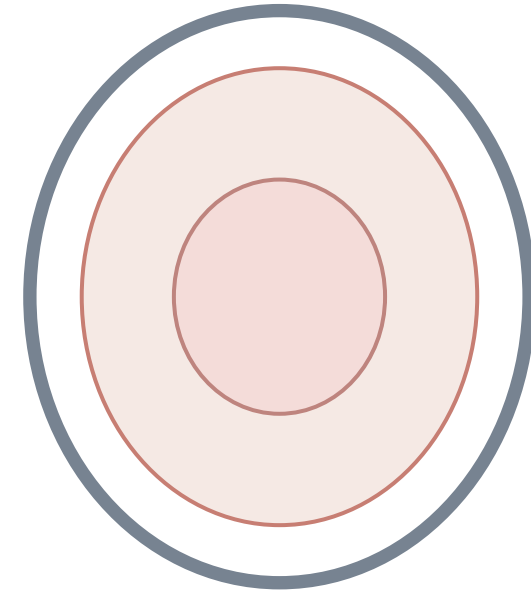
Adhesions are not synonymous with constriction

Observation	Adhesive pericarditis	Constrictive pericarditis
Main scar pattern	Fibrous bridges, potentially extensive	Dense enclosing scar shell
Pericardial space	Can be obliterated	Usually obliterated by scar
Mechanical result	Often no impairment	Limited diastolic expansion
Typical consequence	Usually clinically silent	Reduced filling and output reserve

Diastolic expansion is limited by the scar

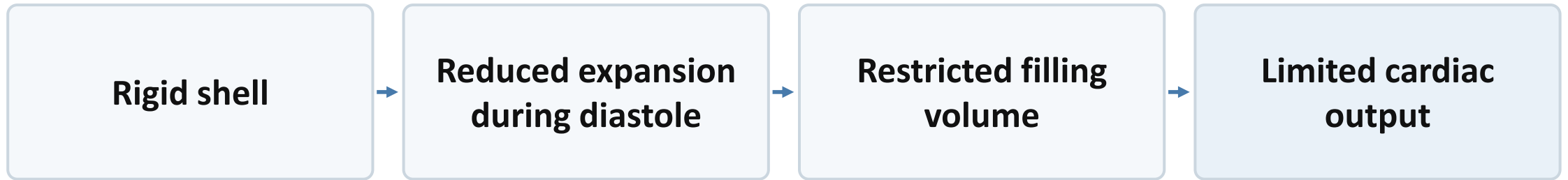
- Ventricles must expand during filling.
- The dense pericardial shell resists outward expansion.
- Filling is therefore externally constrained despite cardiac contraction.
- The result is impaired capacity to augment forward output.

Simplified cross-sectional schematic



Constraining fibrous shell

Constrictive physiology: Filling, not force generation



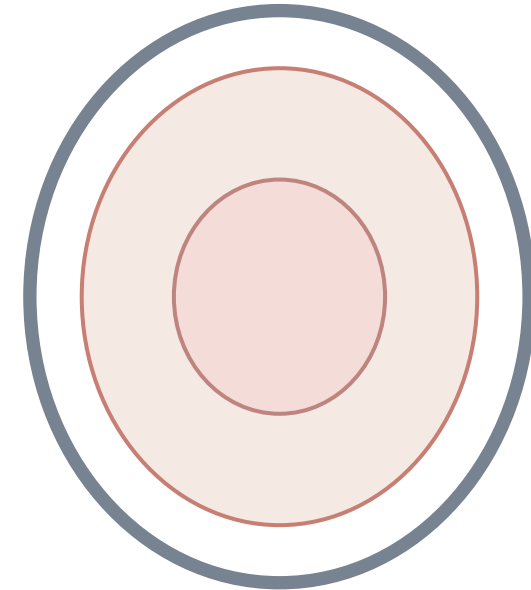
The primary lesion lies outside the myocardium, although clinical signs resemble myocardial restriction.

Conceptual sequence; Timing and scale not implied

Resting output may already be reduced

- Constrictive pericarditis can lower cardiac output at rest.
- Resting measurements do not capture the full functional problem.
- The scar also restricts the normal ability to respond to demand.
- Clinical burden is therefore partly a failure of physiologic reserve.

Simplified cross-sectional schematic

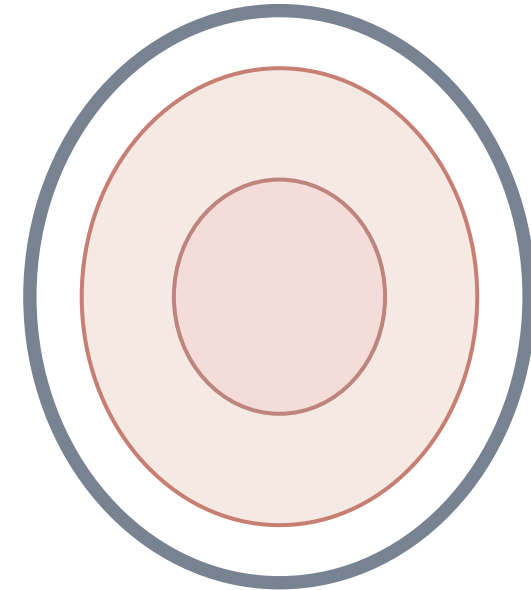


Constraining fibrous shell

Loss of cardiac-output reserve is particularly important

- Systemic demands require an increase in cardiac output.
- Rigid encasement prevents the heart from expanding adequately.
- The source emphasizes little or no capacity to augment output.
- Reduced reserve explains disproportionate limitation on exertion.

Simplified cross-sectional schematic



Constraining fibrous shell

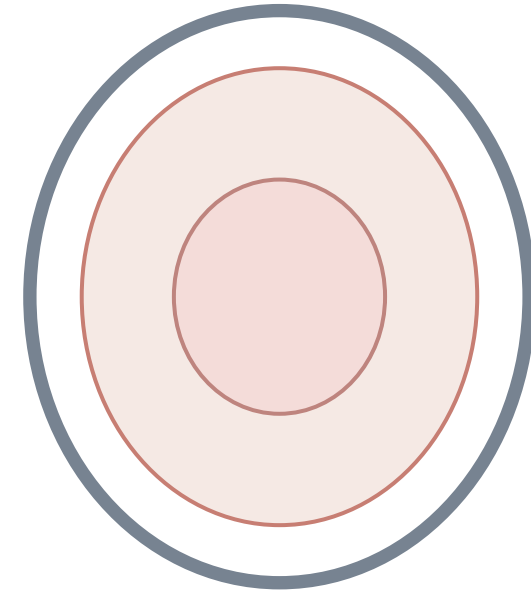
Resting versus increased systemic demand

Situation	What constriction permits	Teaching implication
Rest	Cardiac output may be low	Variable severity at baseline
Greater demand	Little or no increase in output	Major limitation in functional reserve

Why hypertrophy and dilation are absent in constriction

- A dense scar shell tightly encloses the heart.
- Hypertrophy and dilation cannot occur in this setting.
- This contrasts with the occasional remodeling of externally tethered hearts.
- The two diagnoses have fundamentally different mechanical boundaries.

Simplified cross-sectional schematic



Constraining fibrous shell

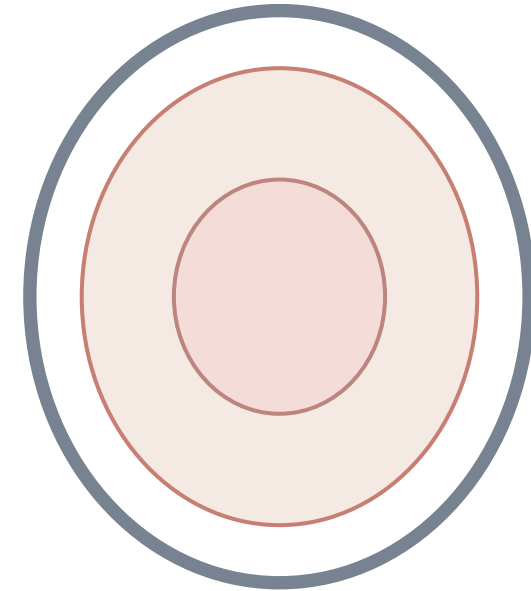
External tethering versus rigid encasement

Distinction	Mediastinopericarditis	Constrictive pericarditis
Principal restriction	Systolic movement against external structures	Diastolic expansion within a fixed scar
Workload	Can increase during contraction	Filling capacity is limited
Remodeling	Hypertrophy/dilation may occur	Prevented by the dense shell (Robbins)
Gross key	External parietal attachments	Enclosing fibrous/fibrocalcific scar

Constrictive disease can mimic restrictive cardiomyopathy

- Both can produce impaired filling and reduced cardiac output.
- Constrictive pericarditis is a disease of the external covering.
- Restrictive cardiomyopathy involves stiff, noncompliant myocardium.
- The tissue compartment causing stiffness is the discriminating concept.

Simplified cross-sectional schematic



Constraining fibrous shell

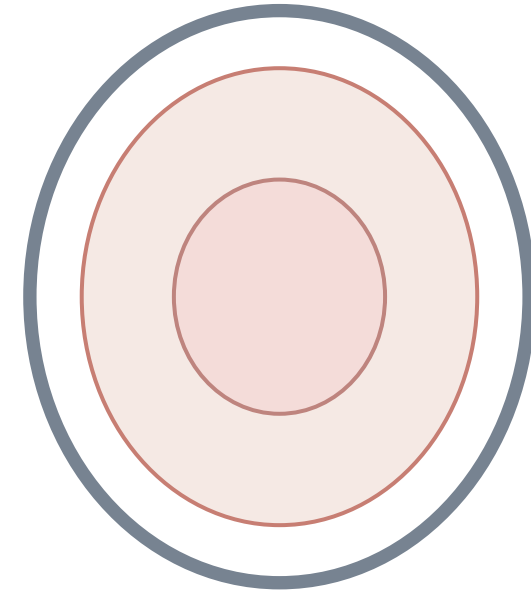
Constrictive pericarditis versus restrictive cardiomyopathy

Feature	Constrictive pericarditis	Restrictive cardiomyopathy
Site of lesion	Pericardial fibrous shell	Myocardial tissue
Structural basis	External encasement	Infiltration, fibrosis, or endomyocardial scar
Major problem	Restricted diastolic expansion	Noncompliant ventricular muscle
Clinical overlap	Restricted filling / low output	Restricted filling / low output

A prior clinical episode may be absent

- Robbins explicitly allows constriction with or without known pericarditis.
- The current gross lesion does not require a documented earlier diagnosis.
- The anatomic scar may be the dominant evidence of the process.
- Do not exclude constriction simply because the history is incomplete.

Simplified cross-sectional schematic

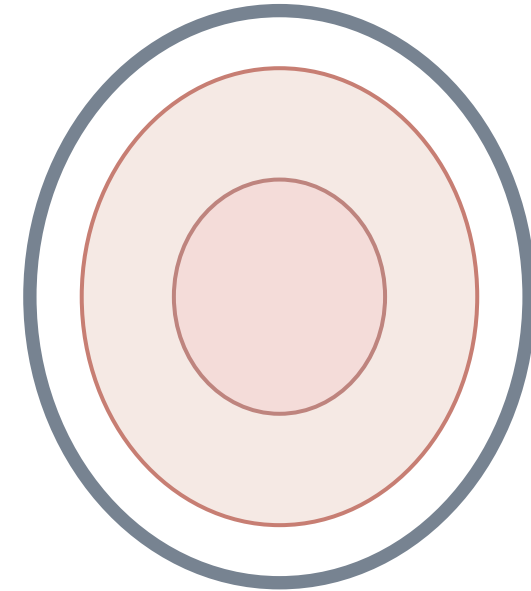


Constraining fibrous shell

Distant or muffled heart sounds

- Attenuated heart sounds are listed among signs of constriction.
- They should be interpreted alongside venous and systemic findings.
- The sign is supportive rather than pathognomonic.
- The relevant substrate is chronic pericardial encasement.

Simplified cross-sectional schematic

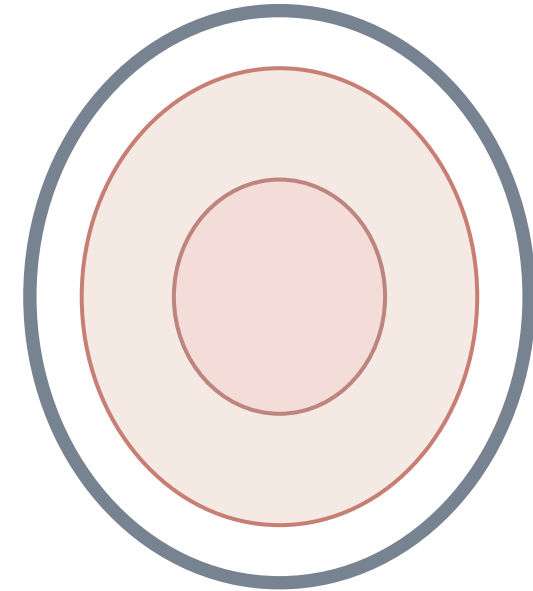


Constraining fibrous shell

Elevated jugular venous pressure

- Elevated jugular venous pressure is a stated clinical feature.
- The rigid pericardial envelope limits cardiac filling.
- This leads to the clinical picture of systemic venous congestion.
- The clinical observation and external scar are linked mechanistically.

Simplified cross-sectional schematic

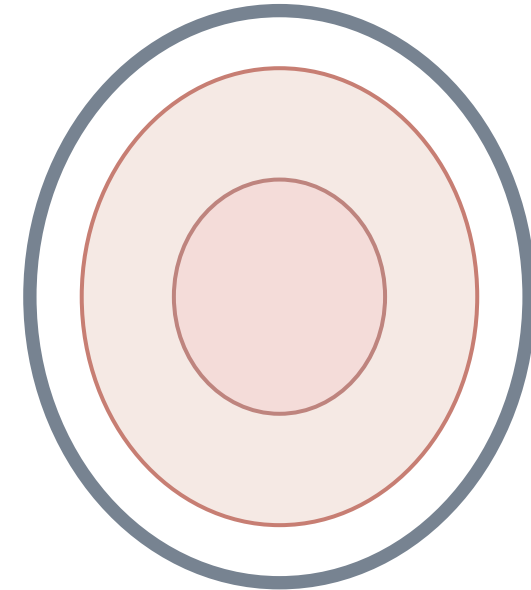


Constraining fibrous shell

Peripheral edema in constrictive disease

- Peripheral edema is another sign.
- Restricted cardiac filling accompanies impaired venous return handling.
- Edema is part of a broader congestive presentation.
- It should not be treated as specific for pericardial constriction.

Simplified cross-sectional schematic



Constraining fibrous shell

Clinical clues reflect the underlying encasement

Read each clinical sign together with the gross and physiologic lesion

Muffled sounds

A recognized physical finding with a diseased pericardium.

Elevated JVP

A venous-pressure clue in impaired filling.

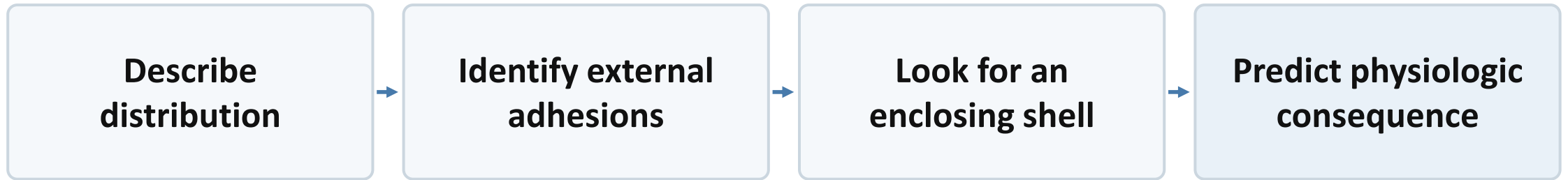
Peripheral edema

A systemic manifestation of congestion.

Three pathologic patterns at the bedside

Pattern	Primary mechanics	Expected effect
Simple healed adhesions	Fibrous attachments within sac	Usually no functional impairment
Adhesive mediastinopericarditis	External systolic tethering	Possible retraction, pulsus paradoxus, remodeling
Constrictive pericarditis	Dense shell limits diastolic expansion	Low output reserve, JVP elevation, edema

Reasoning from a chronic gross specimen



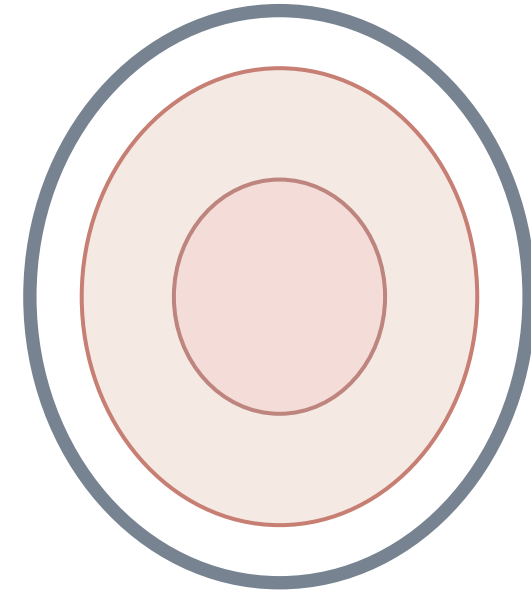
Morphology becomes clinically useful when a specific motion or filling mechanism is identified.

Conceptual sequence; Timing and scale not implied

Pericardiectomy addresses the mechanical lesion

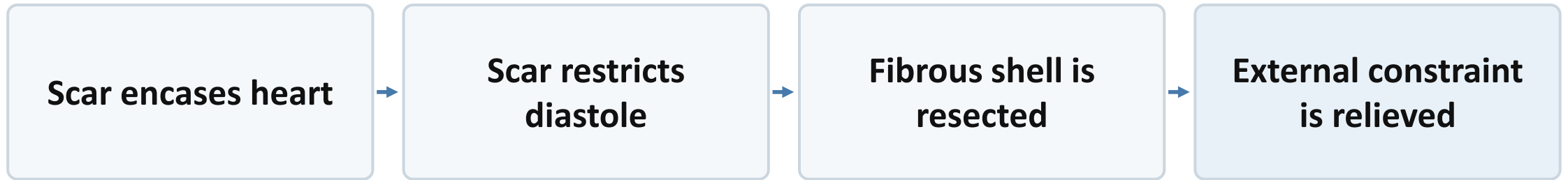
- Surgical resection of the constricting shell.
- The operation is termed pericardiectomy.
- The rationale is removal of the rigid barrier to diastolic expansion.
- The statement concerns constrictive disease, not every healed adhesion.

Simplified cross-sectional schematic



Constraining fibrous shell

The pathological target of pericardiectomy



The surgical target is the pathologic pericardium, not an intrinsic cardiomyopathy.

Conceptual sequence; Timing and scale not implied

Case 1: A gross finding without clinical impairment

A heart examined after death has several small, white plaque-like thickenings on the serosa. No ante-mortem cardiac dysfunction is documented.

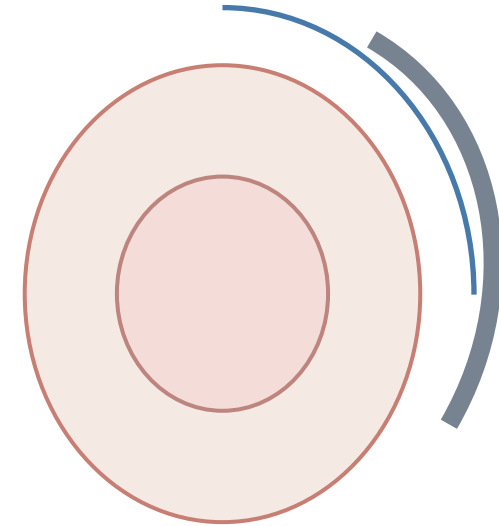
Diagnostic reasoning

- What pattern of healed pericardial injury is represented?
- Does fibrosis alone establish constrictive pericarditis?

Case 1: Interpretation of plaque-like fibrosis

- The pattern resembles the source-described “soldier’s plaque.”
- Small plaque-like thickening can be an innocuous healed lesion.
- No evidence here establishes a circumferential rigid shell.
- The morphologic diagnosis should not imply unproven clinical constriction.

Simplified cross-sectional schematic



Localized fibrous plaque

Case 2: A tethered heart after surgery

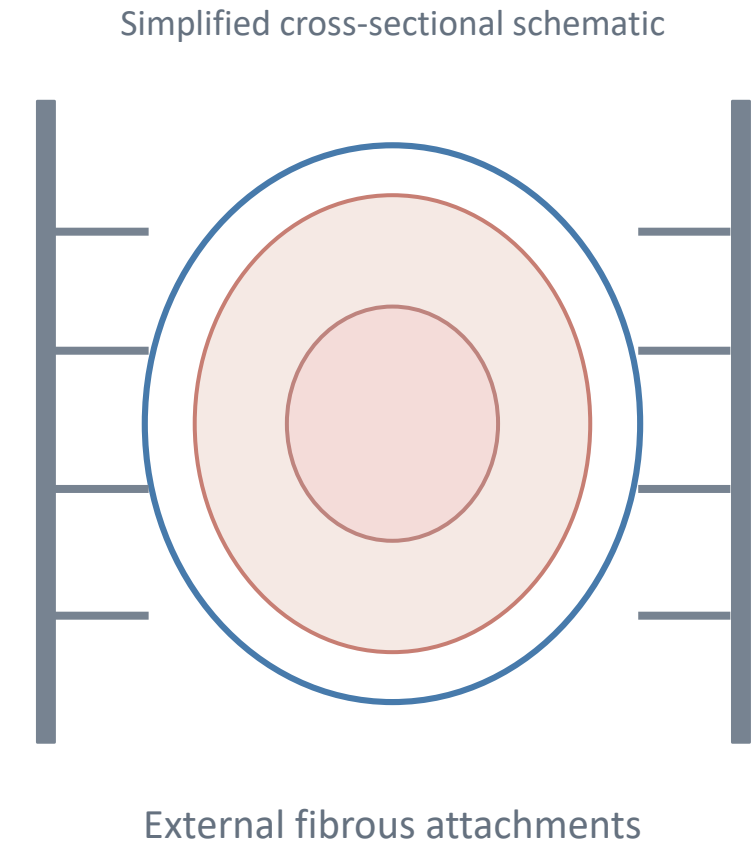
A patient with prior cardiac surgery has a largely obliterated sac and dense attachments from the parietal pericardium into adjacent mediastinal tissues. The rib cage retracts with systole.

Diagnostic reasoning

- What process best explains the retraction?
- Which phase of cardiac mechanics is directly strained?

Case 2: Interpretation of external tethering

- Adhesive mediastinopericarditis explains external attachments.
- The contracting heart pulls against adherent surrounding tissues.
- Systolic retraction is concordant with the described mechanism.
- Hypertrophy or dilation may occur from increased workload.



Case 3: A heart encased by a calcified shell

A patient has elevated jugular venous pressure, leg edema, and limited cardiac-output reserve. At operation, a thick fibrocalcific layer tightly envelops the heart.

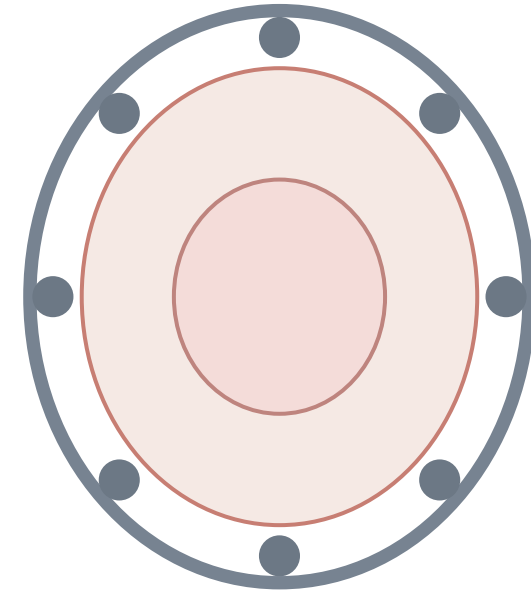
Diagnostic reasoning

- Identify the dominant pathologic lesion.
- Explain the link between the shell and impaired output.

Case 3: A mechanistic diagnosis of constriction

- The enclosing fibrocalcific scar supports constrictive pericarditis.
- The rigid shell limits diastolic expansion and ventricular filling.
- Venous congestion and low output reserve fit the mechanism.
- Pericardiectomy removes the constricting fibrous shell.

Simplified cross-sectional schematic



Fibrocalcific shell

Final morphology–mechanics matrix

Lesion	Anatomic signature	Physiologic meaning
Soldier's plaque	Discrete serosal fibrous thickening	Usually negligible
Adhesive pericarditis	Bridging/mesh-like fibrous adhesions	Usually negligible
Mediastinopericarditis	Parietal-to-mediastinal adhesions	Systolic tethering / excess workload
Constrictive pericarditis	Dense fibrous or fibrocalcific shell	Restricted diastolic expansion

Three rules to retain

Morphologic recognition should always be connected to the relevant mechanical consequence

Fibrosis $\neq$ constriction

Plaques and adhesions may be clinically silent.

External tethering

Adhesions to surrounding tissues burden systole.

Rigid encasement

A dense shell limits filling and cardiac-output reserve.

References and scope

- Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th edition (2026).
- Read the textbook sections on pericardial disease and its chronic healing patterns.
- Core reading: “Chronic or Healed Pericarditis.”
- All process figures are original editable schematics; not patient images.