

Pericardial Disease

Fluid accumulation · Inflammation · Fibrous constriction

Effusion

Hemodynamic consequences

Pericarditis

Exudative morphology

Constriction

Impaired diastolic filling

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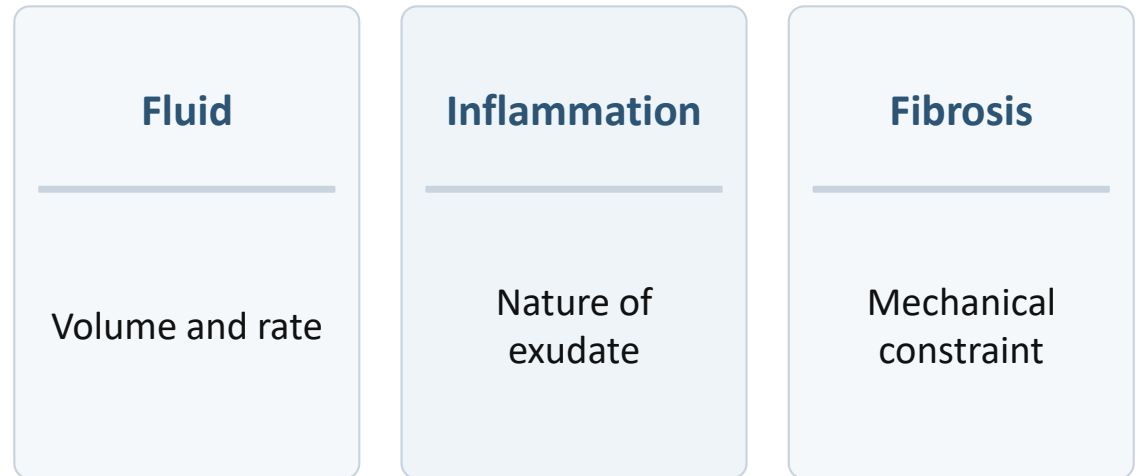
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Learning objectives

- Distinguish effusion, hemopericardium, and inflammatory exudates.
- Explain why rapid fluid accumulation causes tamponade.
- Recognize major acute pericarditis patterns and their causes.
- Relate chronic scarring to adhesive and constrictive disorders.

Three dominant forms of pericardial injury

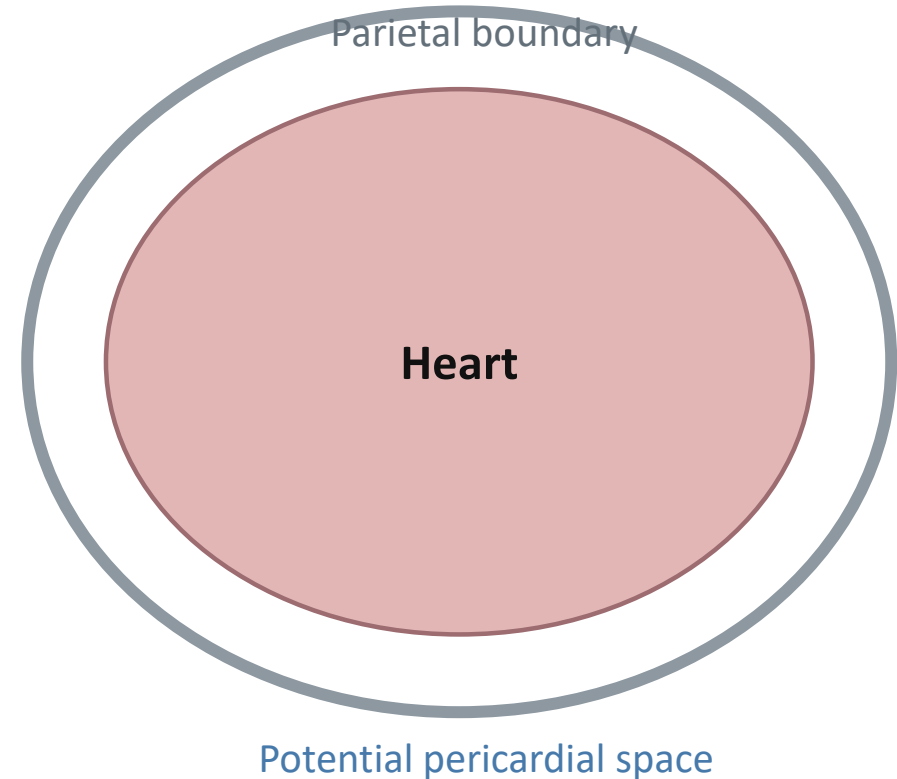
- Fluid may occupy and distend the pericardial sac.
- Inflammation alters serosal surfaces and exudate composition.
- Organization and fibrosis may tether or encase the heart.



Schematic

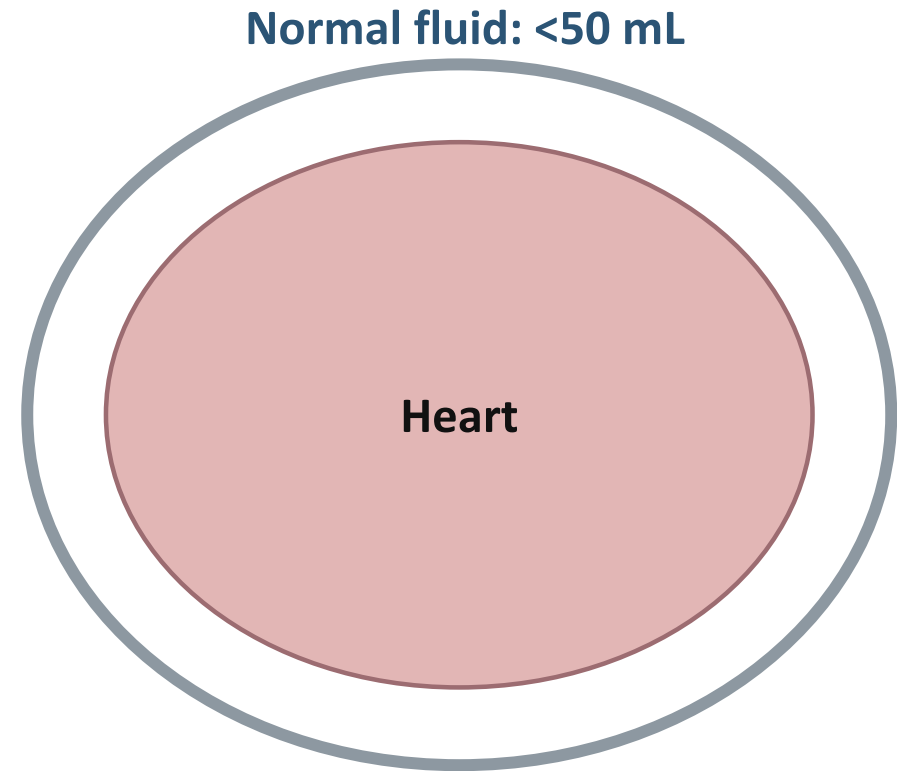
The pericardium: Functional compartments

- Visceral and parietal pericardial surfaces surround a potential space.
- The space normally contains only a small volume of fluid.
- Disease affects surface, space, or surrounding fibrous envelope.



Normal fluid is less than 50 mL

- Normal fluid is thin, clear, and straw-colored.
- Accumulation of extra serous fluid is termed effusion.
- Blood and pus define different pathologic contents.



Morphologic processes predict consequences

- An expanding effusion may compress cardiac chambers.
- Inflamed surfaces may produce pain and a friction rub.
- A dense scar can restrict filling even without free fluid.

Compression

Impaired filling

Friction

Pain or rub

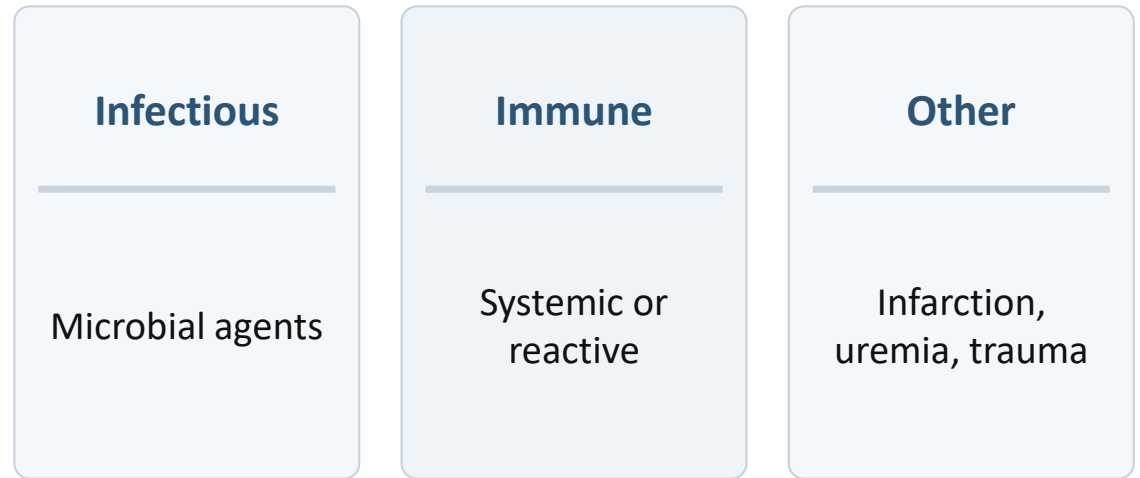
Constriction

Limited
expansion

Schematic

Etiology spans multiple clinical settings

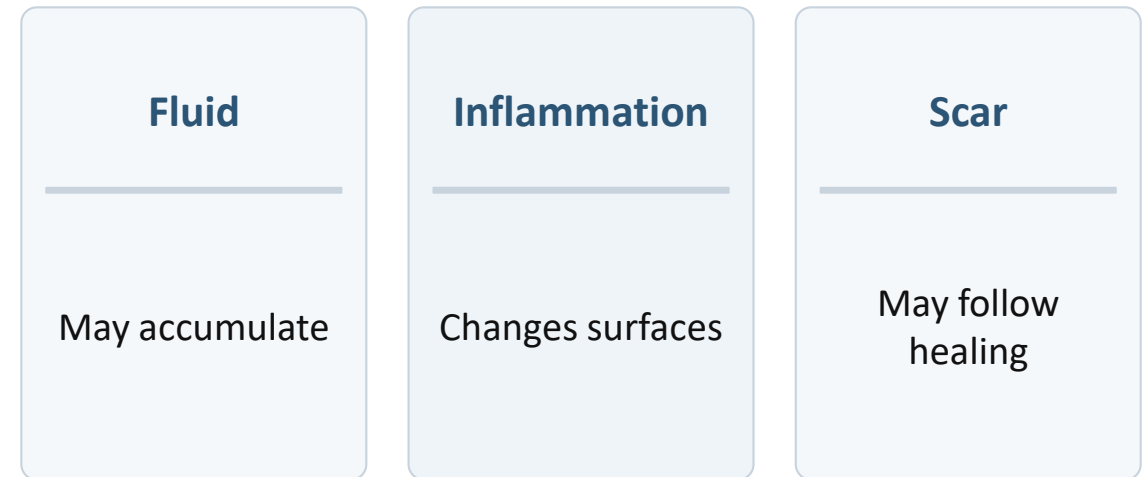
- Infection, immune reactions, and myocardial injury all contribute.
- Renal disease, malignancy, and trauma are additional contexts.
- Surgery and thoracic irradiation may initiate acute or chronic disease.



Schematic

The fluid, inflammation, and scar axes overlap

- Serous effusion may coexist with inflammatory pericarditis.
- Infection can produce purulent exudate followed by fibrosis.
- Hemorrhage can create a rapidly dangerous pressure load.



Schematic

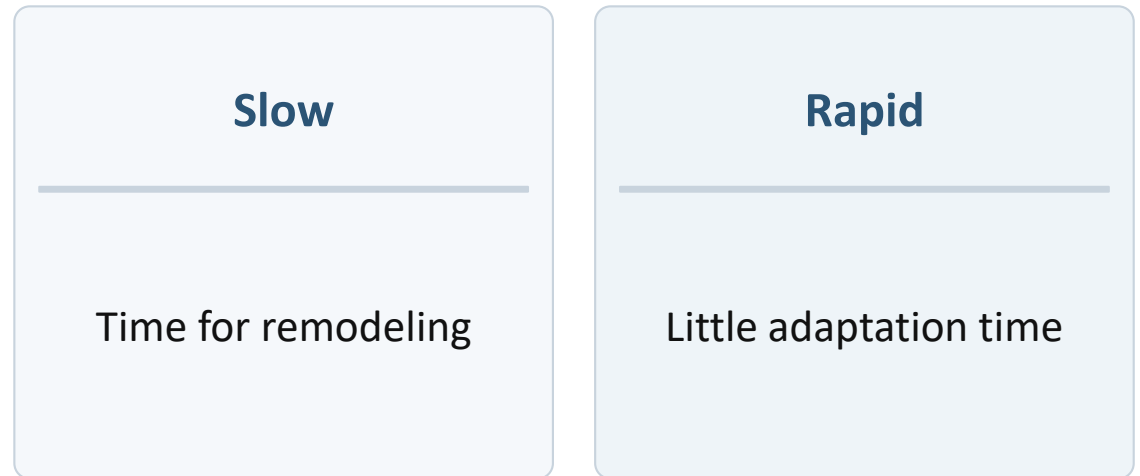
Classify a fluid-filled pericardial sac

- Serous fluid: Pericardial effusion.
- Blood: Hemopericardium.
- Pus: Purulent pericarditis, indicating an infectious process.

Serous	Blood	Pus
Thin fluid	Hemopericardium	Suppuration
Effusion	Potential tamponade	Infection

The rate of fluid accumulation matters

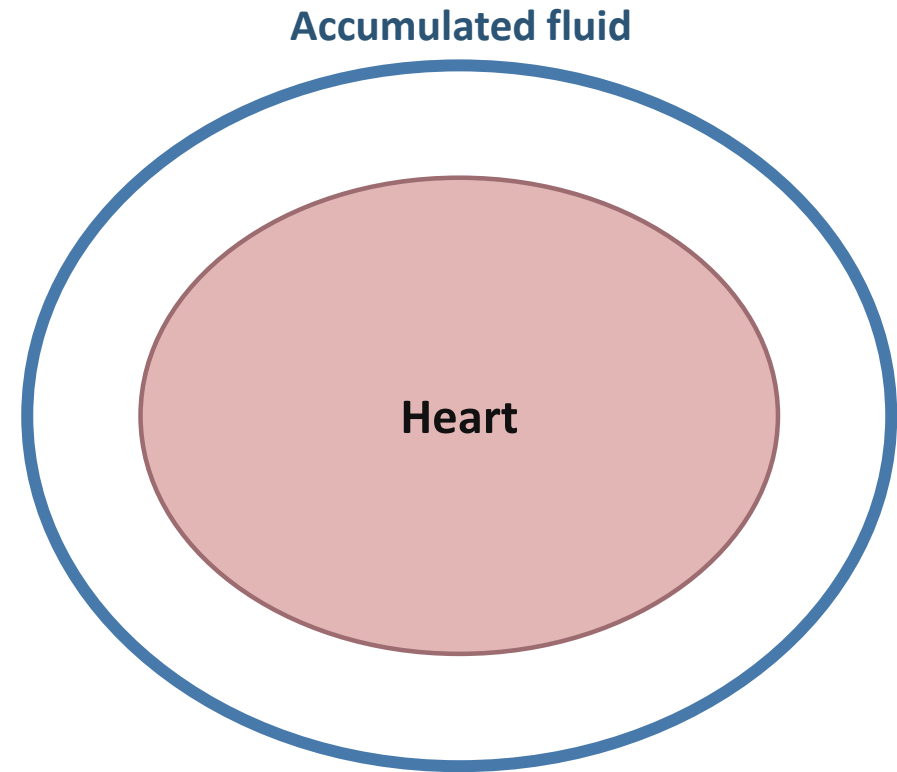
- Gradual distension permits time for pericardial remodeling.
- A rapidly growing collection lacks that adaptive interval.
- The same sac can tolerate different volumes at different rates.



Schematic

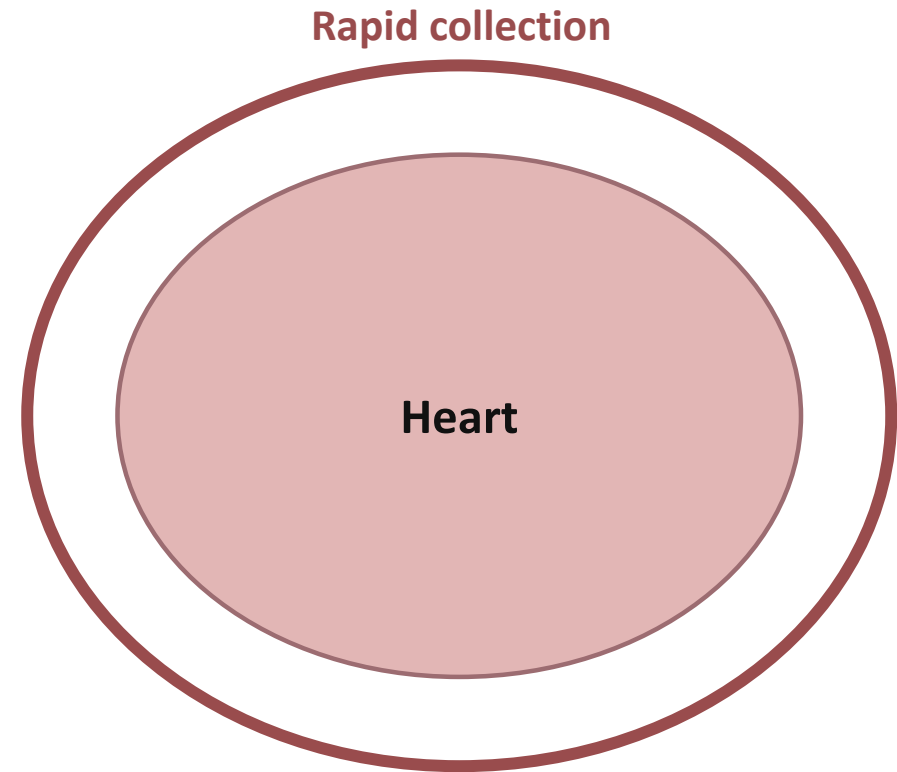
Slowly accumulating effusions permit adaptation

- Chronic enlargement expands the accommodating pericardial envelope.
- A substantial collection may have little hemodynamic impact.
- Clinical significance cannot be inferred from volume alone.



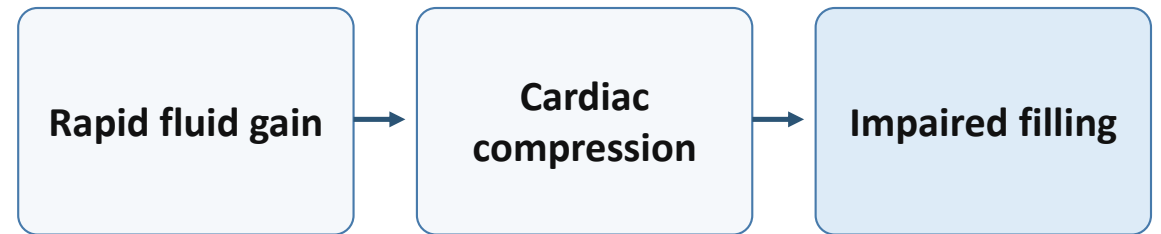
Rapid accumulation risks tamponade

- An acute collection of 200–300 mL may severely compromise filling.
- Examples include blood from a ruptured myocardial infarct or aortic dissection.
- Rapid compression can be fatal.



How tamponade develops

- Rapid pericardial volume expansion raises external cardiac constraint.
- Atria, venae cavae, and potentially ventricles are compressed.
- Restricted filling reduces effective cardiac output.



Schematic

Thin-walled structures are vulnerable

- The atria and venae cavae are specifically susceptible to pressure.
- Ventricular filling may also become compromised.
- The final common mechanism is impaired diastolic filling.

Early pressure targets

Atria / venae cavae

Advanced consequence

Ventricular filling
impaired

Schematic

Chronic effusion differs from acute hemopericardium

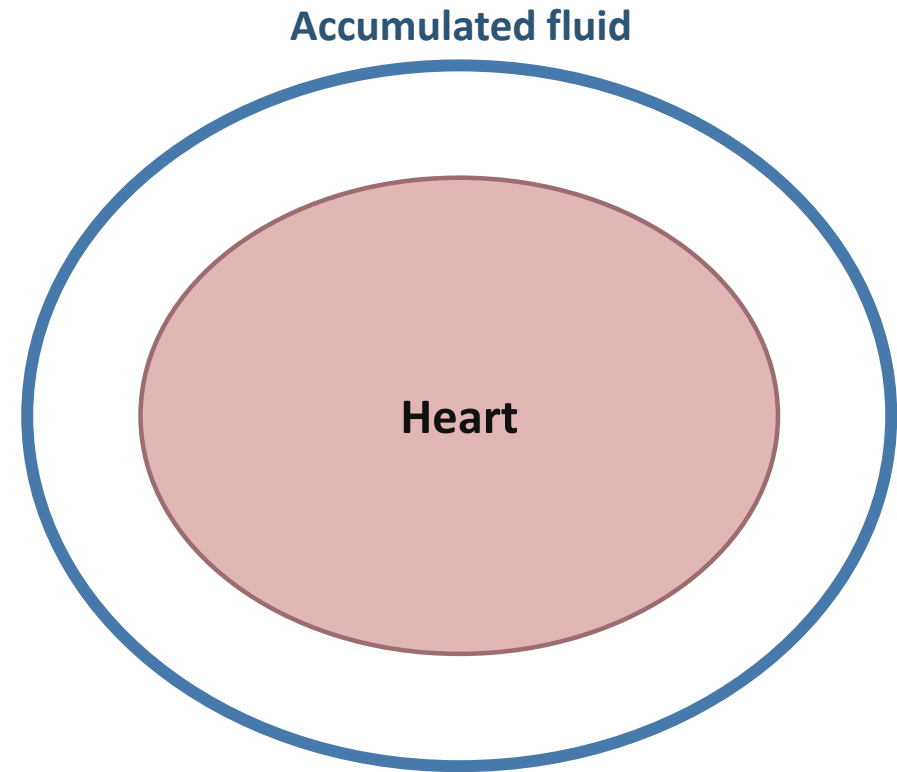
- Chronic effusion: Available remodeling time.
- Acute hemopericardium: Sudden pressure increase.
- Interpret the clinical threat through rate, volume, and filling effect.

Gradual	Acute
Remodeling possible	Minimal accommodation
Large volume tolerated	Tamponade at lower volume

Editable comparison

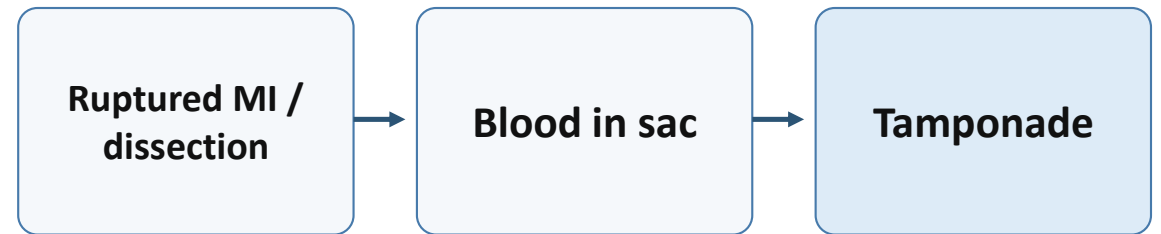
A globular heart shadow is a chronic clue

- The text describes a globular cardiac silhouette on chest radiographs.
- It may be the major finding with some chronic effusions below 500 mL.
- A radiographic silhouette does not, by itself, establish tamponade.



Hemopericardium from catastrophic injury

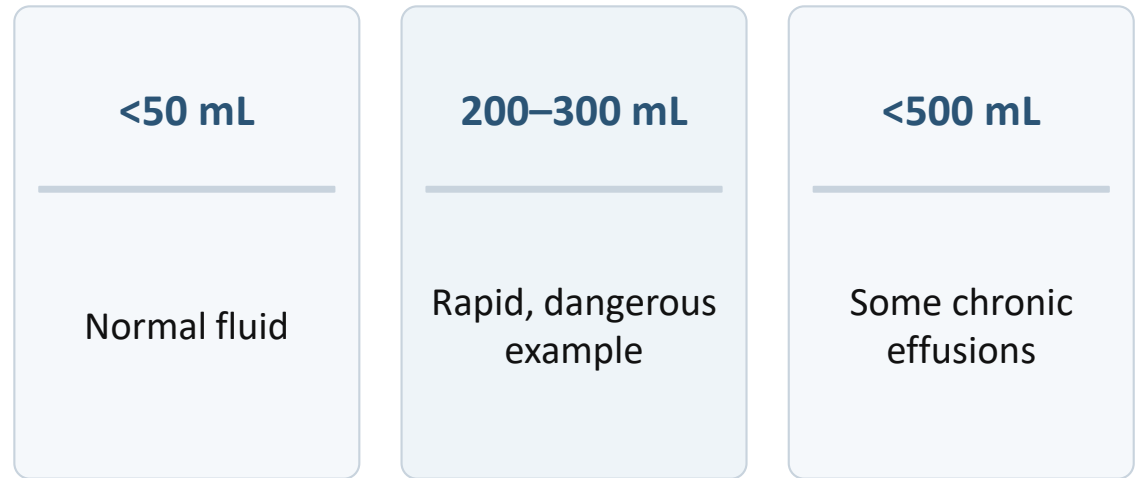
- Ruptured myocardial infarction and aortic dissection are cited examples.
- Blood can enter the pericardial space rapidly.
- The immediate danger is compression of the heart, not an inflammatory subtype.



Schematic

Volume thresholds require context

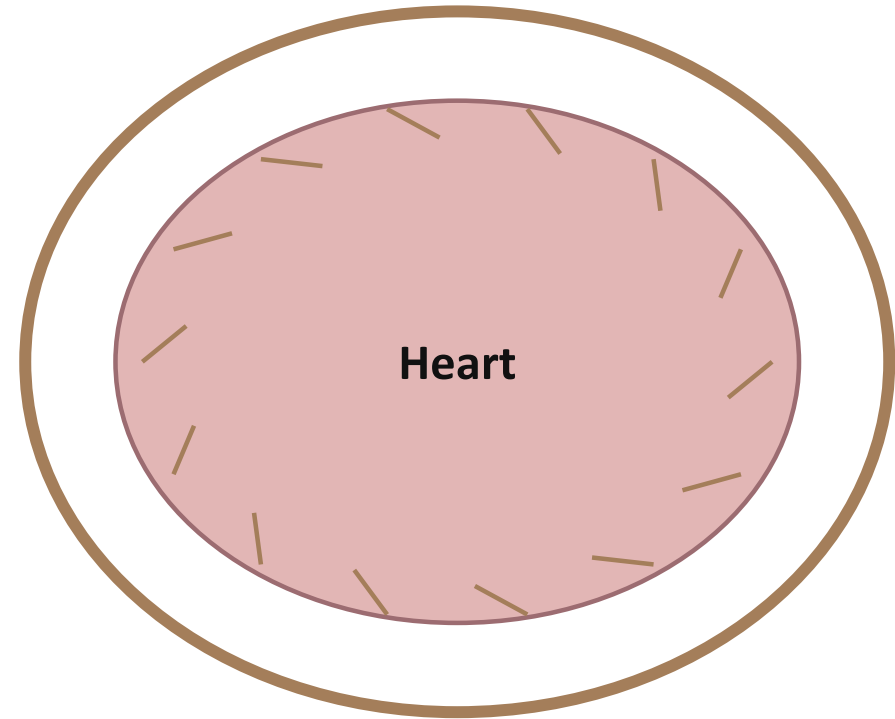
- Less than 50 mL describes normal pericardial fluid.
- 200–300 mL is an example of potentially fatal rapid accumulation.
- Less than 500 mL describes some slowly accumulating chronic effusions.



Schematic

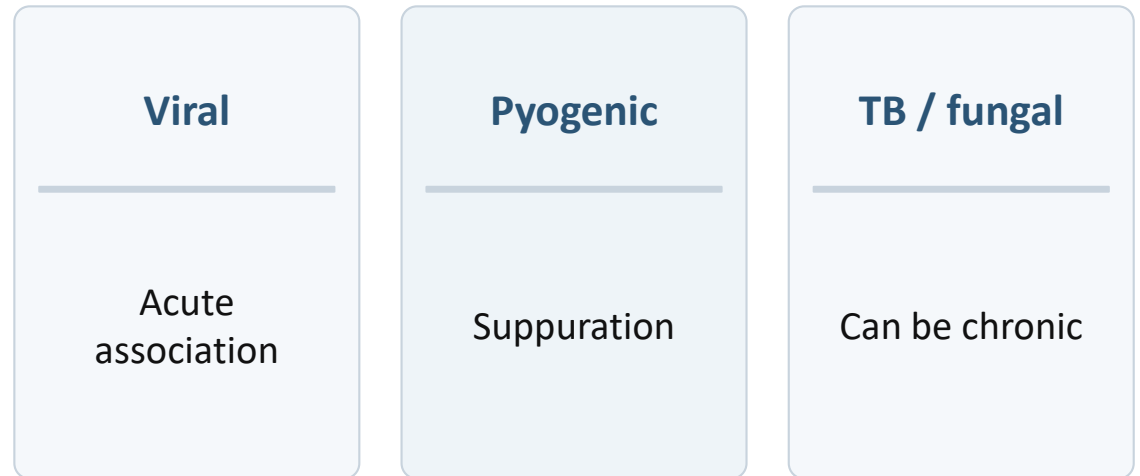
Pericarditis is inflammation of the sac

- Cardiac, thoracic, and systemic disorders may initiate inflammation.
- Direct neoplastic involvement and postoperative reactions also occur.
- The exudate and tissue response help distinguish subtypes.



Major infectious causes of pericarditis

- Viruses and pyogenic bacteria are listed infectious agents.
- Tuberculosis and fungal infections may yield chronic reactions.
- Other parasites also appear in the etiologic classification.



Schematic

Presumed immune-mediated settings

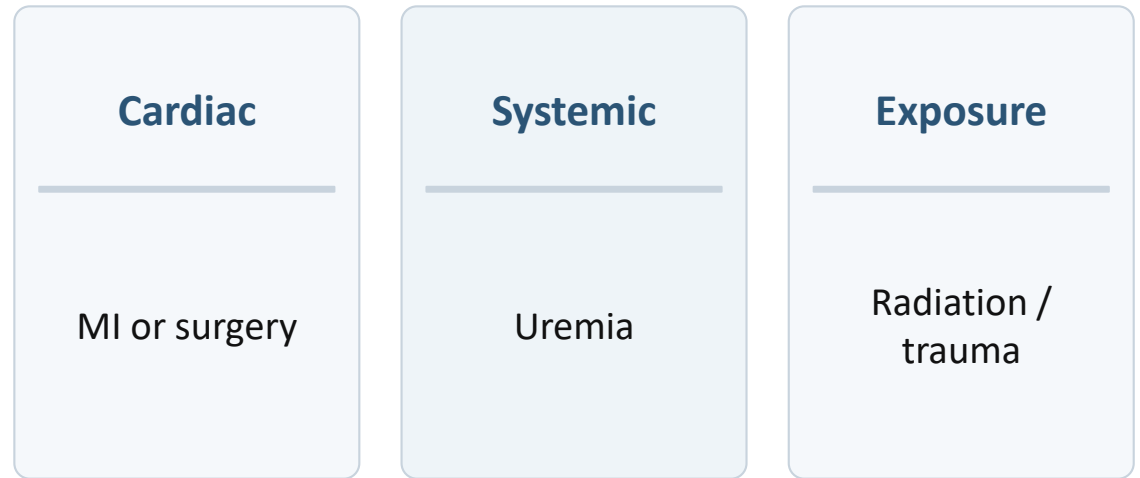
- Rheumatic fever, systemic lupus erythematosus, and scleroderma.
- Postcardiotomy and postinfarction Dressler syndrome.
- Drug hypersensitivity reactions.



Schematic

Other important etiologic contexts

- Myocardial infarction and uremia.
- Cardiac surgery, trauma, and radiation.
- Neoplasia involving the pericardium.



Schematic

Morphologic classification of acute pericarditis

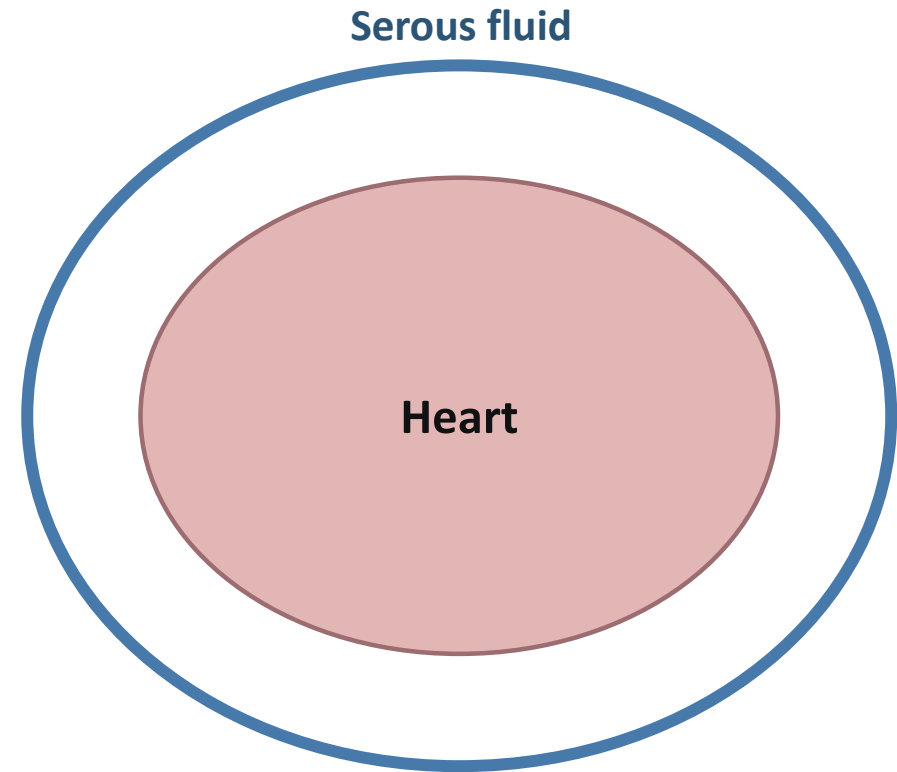
- Serous: Relatively thin fluid and mild inflammation.
- Fibrinous or serofibrinous: Fibrin admixed with exudate.
- Purulent: Pus; Hemorrhagic: Blood mixed with inflammatory exudate.



Schematic

Serous pericarditis often reflects systemic disease

- Rheumatic fever, SLE, and scleroderma are examples.
- Uremia and tumors may also be associated.
- The predominantly serous response may remain relatively mild.



An adjacent infection may begin as sterile serositis

- Inflammation in nearby tissues can irritate the parietal serosa.
- Bacterial pleuritis is an example of a contiguous source.
- A sterile serous response may progress toward fibrinous or suppurative change.



Schematic

Viral illness may precede pericarditis

- A respiratory, pulmonary, or parotid infection may be the initial focus.
- A primary viral pericarditis occurs less frequently.
- Associated myocarditis is possible: Myopericarditis.



Schematic

Tumors can cause serous pericarditis

- Lymphatic invasion may disrupt pericardial function.
- Direct contiguous spread is another mechanism.
- Effusions associated with metastases may contain neoplastic cells.



Schematic

Serous pericarditis: Microscopic response

- The epicardial fat may contain a mild inflammatory infiltrate.
- Lymphocytes predominate in the described histology.
- Prominent fibrous adhesions are unusual in this pattern.

Epipericardial fat

Mild lymphocytes

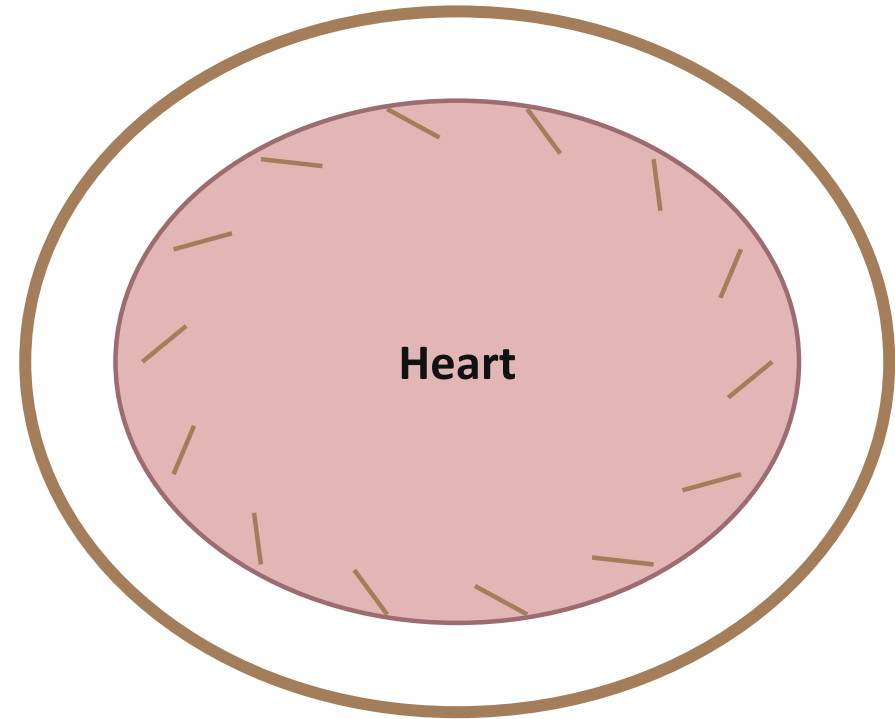
Pericardial space

Serous exudate

Schematic

Fibrinous patterns are the most frequent

- Fibrinous and serofibrinous pericarditis dominate the text's classification.
- Serous fluid is variably mixed with a fibrinous exudate.
- Inflammatory surfaces produce recognizable symptoms and signs.



Common causes of fibrinous pericarditis

- Acute myocardial infarction and postinfarction Dressler syndrome.
- Uremia, rheumatic fever, and systemic lupus erythematosus.
- Trauma and routine cardiac surgery; Radiation is another cause.

Myocardial

MI / Dressler

Systemic

Uremia / SLE

Iatrogenic

Surgery /
radiation

Schematic

Infarction-related mechanisms are distinct

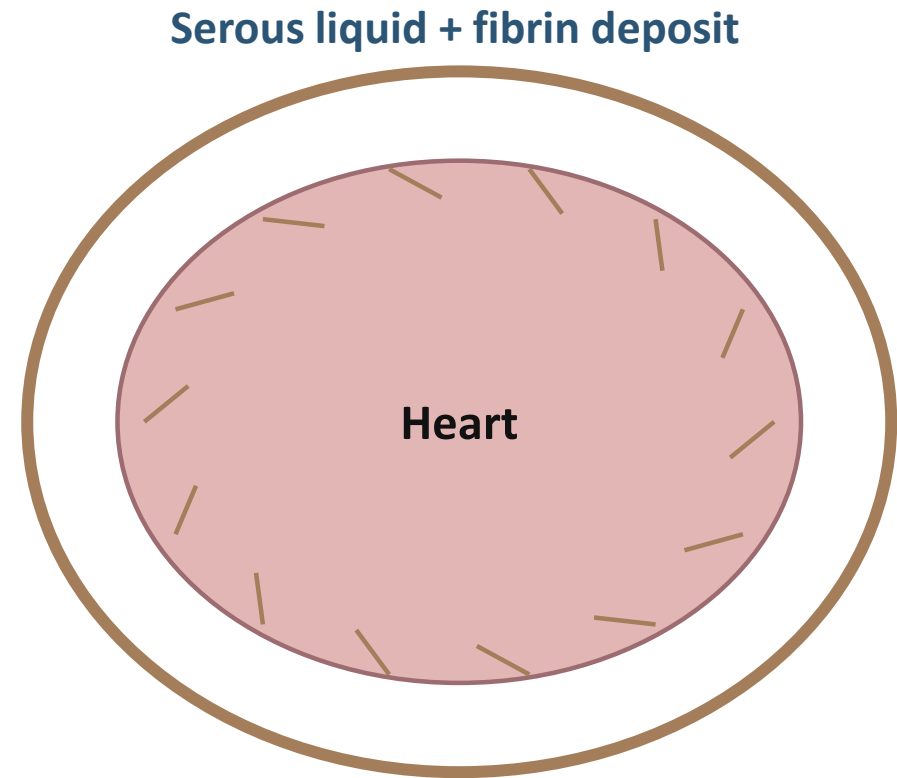
- Acute MI is a cause of fibrinous pericarditis.
- Dressler syndrome is a postinfarction autoimmune reaction.
- The text places Dressler syndrome weeks after infarction.

Acute MI	Dressler syndrome
Associated acute event	Autoimmune reaction
Fibrinous reaction	Weeks after MI

Editable comparison

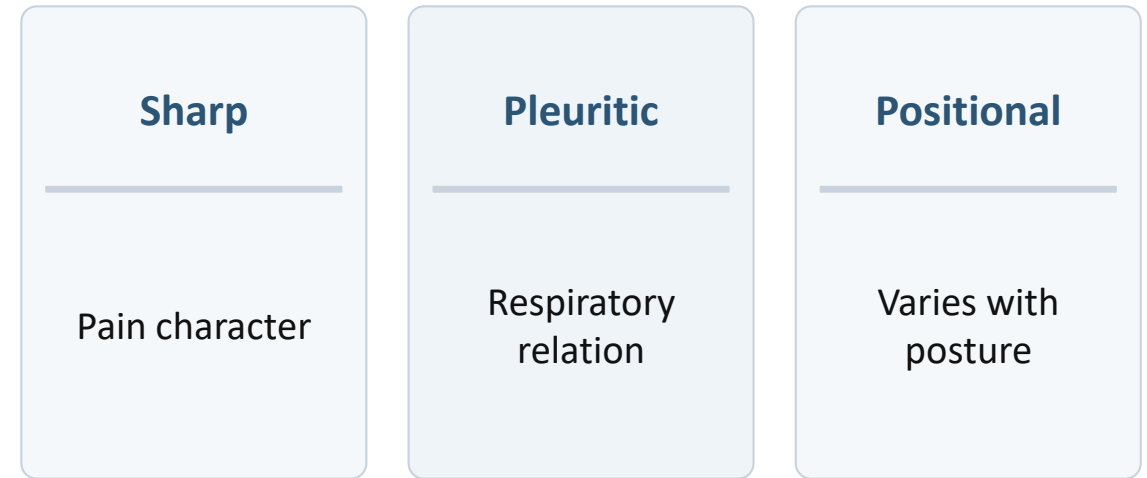
Serofibrinous exudate has two components

- Serous liquid separates or distends the pericardial layers.
- Fibrin is deposited on inflamed serosal surfaces.
- The relative proportions vary across cases.



Pain has a characteristic inflammatory pattern

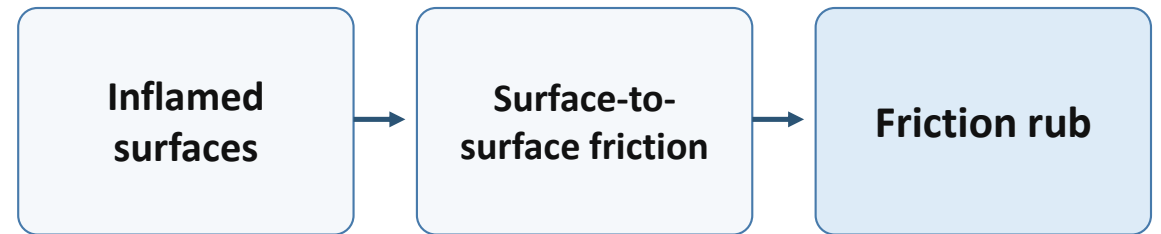
- Pain is often sharp and pleuritic.
- Its severity may vary with position.
- Fever and sometimes congestive failure accompany the process.



Schematic

A friction rub reflects opposing inflamed surfaces

- Inflamed surfaces can rub with cardiac movement.
- A loud pericardial friction rub may be the clearest clinical sign.
- This finding is most likely when the surfaces remain apposed.



Schematic

Fluid may silence an otherwise audible rub

- Additional effusion separates the opposing serosal layers.
- Mechanical contact decreases as separation increases.
- An absent rub therefore does not exclude pericarditis.

Surface contact	Layer separation
Rub may be heard	Rub may disappear
Less free fluid	More free fluid

Editable comparison

Radiation may affect several disease stages

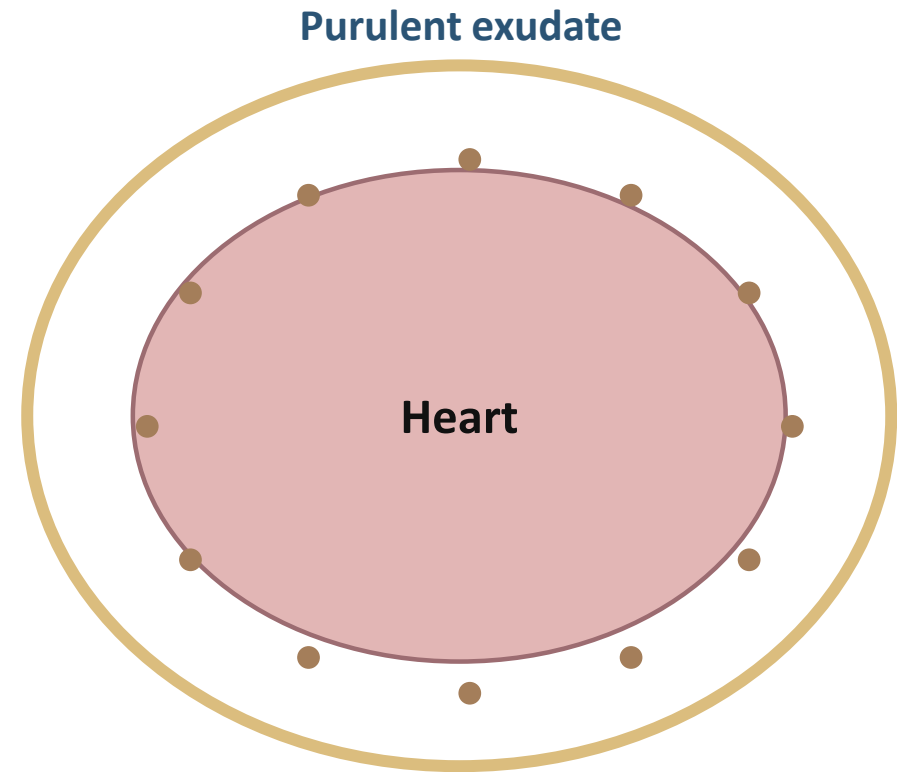
- Thoracic irradiation may cause acute pericarditis.
- It can also cause an effusion.
- Chronic pericardial damage and fibrosis may emerge later.



Schematic

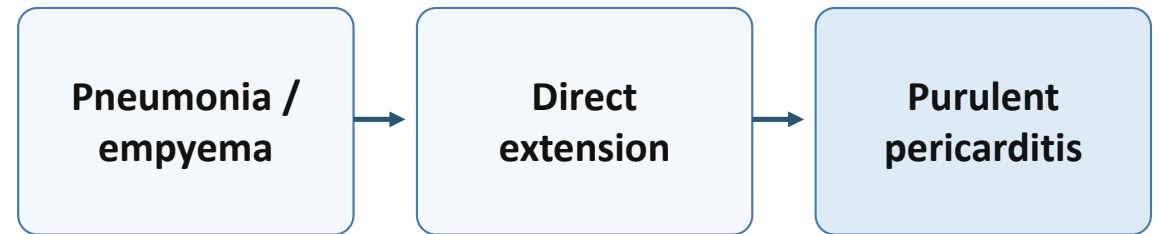
Purulent pericarditis requires microbial invasion

- Suppuration denotes active infection of the pericardial space.
- Infection may arrive by direct, blood-borne, lymphatic, or procedural routes.
- The process carries a strong propensity to organize into scar.



Contiguous spread is one route to suppuration

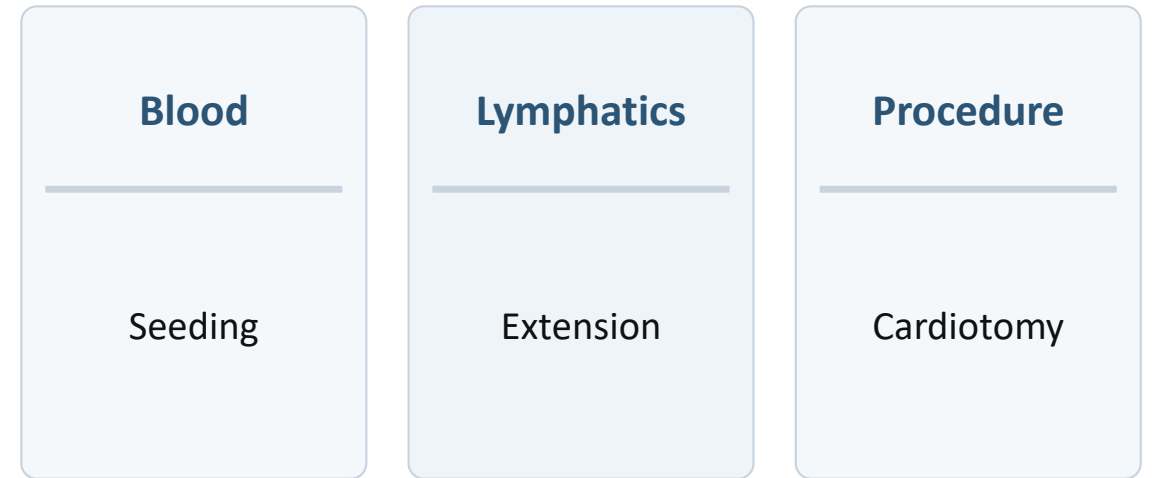
- Empyema or lobar pneumonia may extend toward the sac.
- Mediastinal infection is another source.
- A ring abscess can extend through myocardium or aortic root.



Schematic

Other routes introduce organisms

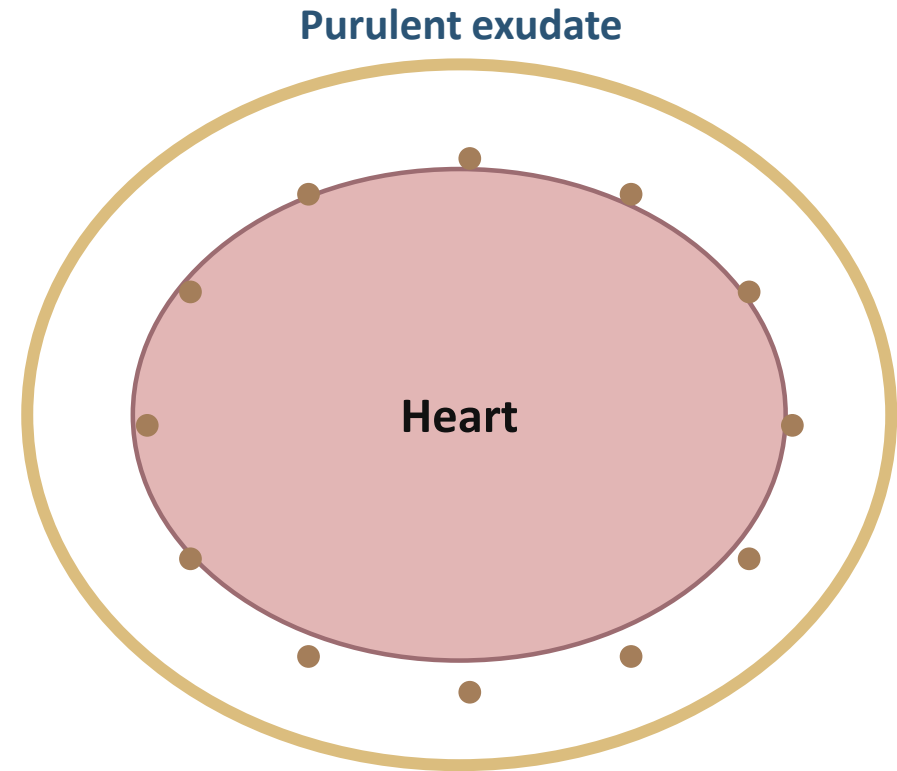
- Hematogenous seeding from the bloodstream.
- Lymphatic extension from another site.
- Direct introduction during cardiectomy.



Schematic

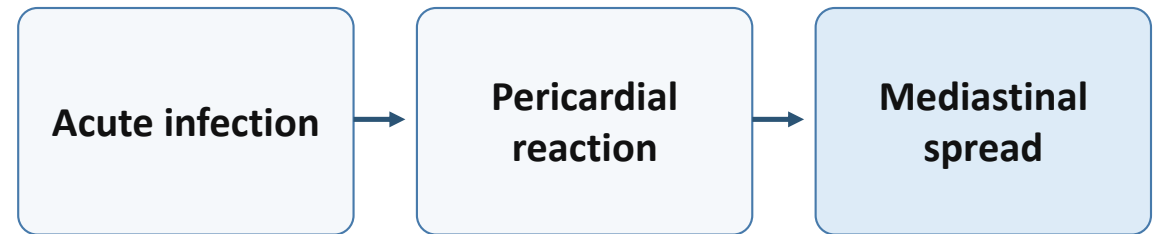
Purulent exudate ranges from cloudy fluid to pus

- The text describes cloudy liquid through thick purulent material.
- Reported volume may reach 500 mL.
- Serosal surfaces become reddened, granular, and exudate-coated.



Histologic inflammation may extend beyond the sac

- An acute inflammatory reaction characterizes suppurative pericarditis.
- Contiguous extension can involve surrounding mediastinal structures.
- This wider process is termed mediastinopericarditis.



Schematic

Suppurative disease causes prominent systemic illness

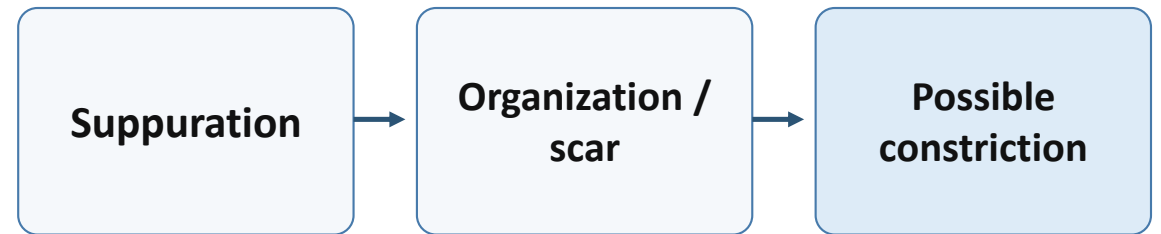
- The local pain and rub may resemble fibrinous pericarditis.
- Spiking fevers and rigors point toward active infection.
- Systemic toxicity often exceeds that of nonpurulent inflammation.

Fibrinous	Purulent
Sharp pleuritic pain	Local signs possible
Rub ± fever	Spiking fevers, rigors

Editable comparison

Purulent pericarditis commonly heals by scarring

- Complete resolution is described as infrequent.
- Organizing inflammation produces fibrous adhesions and contracture.
- This explains the association with later constrictive pericarditis.



Schematic

Tuberculosis may produce caseating foci

- Tuberculous pericarditis is included among infectious causes.
- Caseation may be present in the affected pericardium.
- Tuberculosis may follow a chronic rather than exclusively acute course.

Tuberculosis

Chronic course possible

Pericardium

Caseating foci may occur

Schematic

Hemorrhagic pericarditis contains mixed exudate

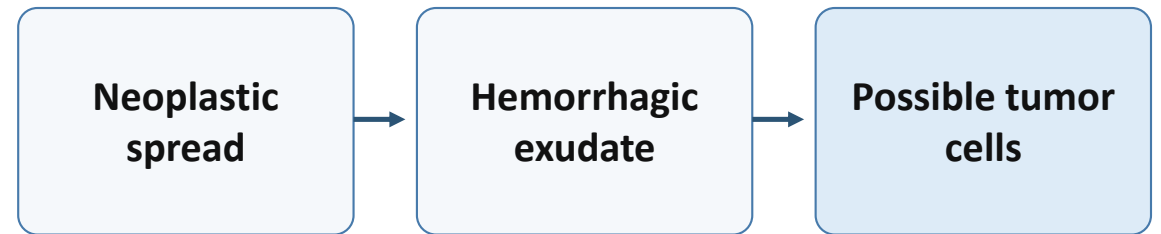
- Blood is admixed with fibrinous or suppurative material.
- This differs from hemopericardium described simply as blood in the sac.
- A hemorrhagic exudate should trigger etiologic investigation.

Hemopericardium	Hemorrhagic pericarditis
Blood in sac	Blood + exudate
May be rapidly fatal	Inflammatory process

Editable comparison

Malignancy is a leading cause of hemorrhagic pericarditis

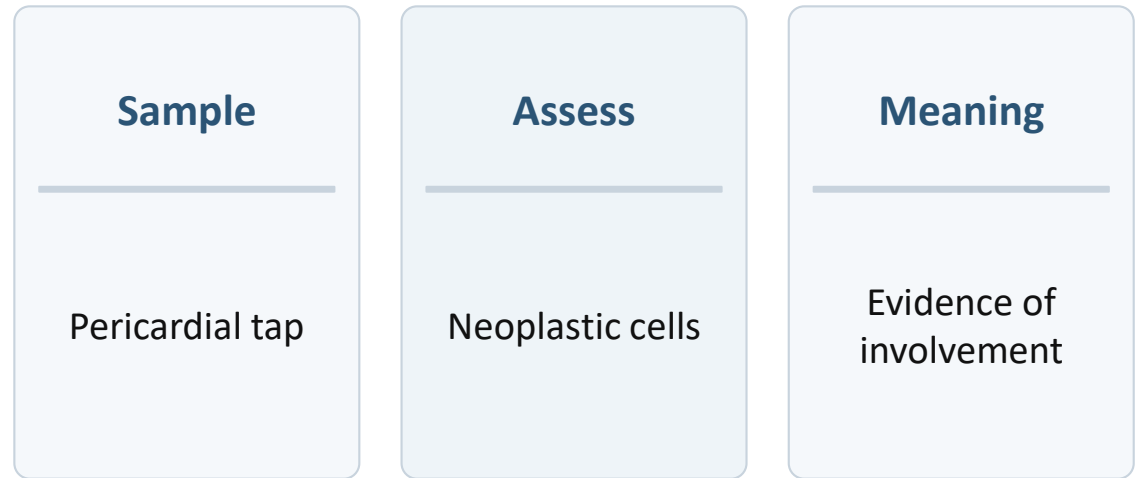
- Neoplastic spread into the pericardial space is a common context.
- Malignant cells may be found in the effusion.
- Underlying tumor biology determines the wider prognosis.



Schematic

Fluid cytology can reveal metastatic neoplasia

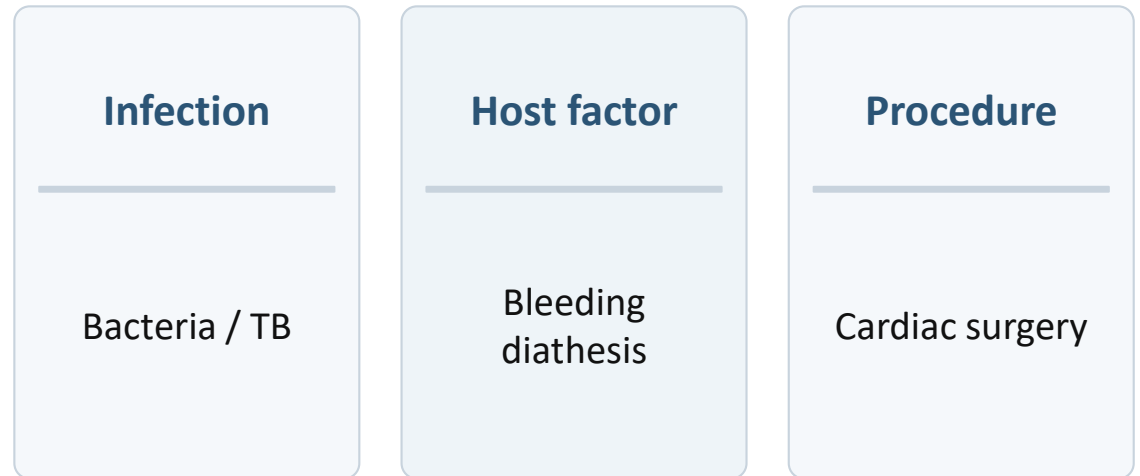
- Sampling fluid by pericardial tap can disclose neoplastic cells.
- Positive cytology provides etiologic evidence of malignant involvement.
- Interpret findings in the clinical setting of a suspected tumor.



Schematic

Hemorrhagic inflammation has additional causes

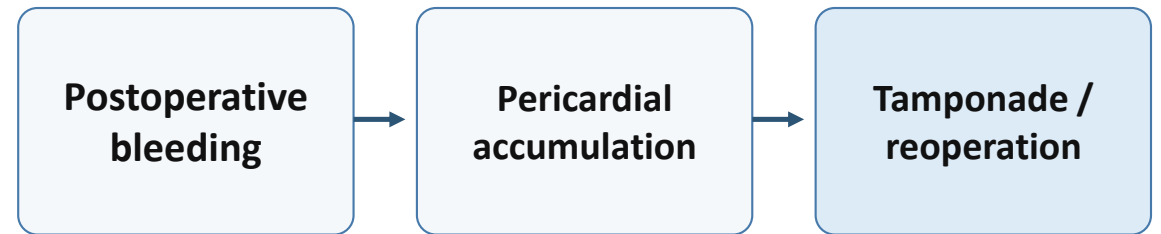
- Bacterial infection and tuberculosis are listed causes.
- An underlying bleeding diathesis can contribute.
- Hemorrhagic pericarditis may occur after cardiac surgery.



Schematic

Postoperative bleeding may cause tamponade

- Surgical hemorrhage can accumulate within the pericardial sac.
- Substantial blood loss or compression may necessitate reoperation.
- This is a clinically urgent complication rather than a benign expected effusion.



Schematic

Malignant hemorrhagic disease: Prognosis varies

- Pericardial spread of cancer generally indicates a poor prognosis.
- This is not absolute for every malignant neoplasm.
- Highly treatable cancers, including some non-Hodgkin lymphomas, are noted exceptions.

General pattern

Poor prognosis with
spread

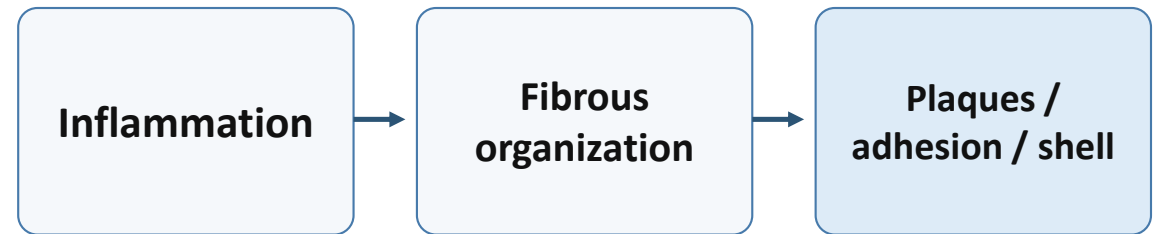
Important exception

Treatable lymphoma
possible

Schematic

Organization connects acute to chronic disease

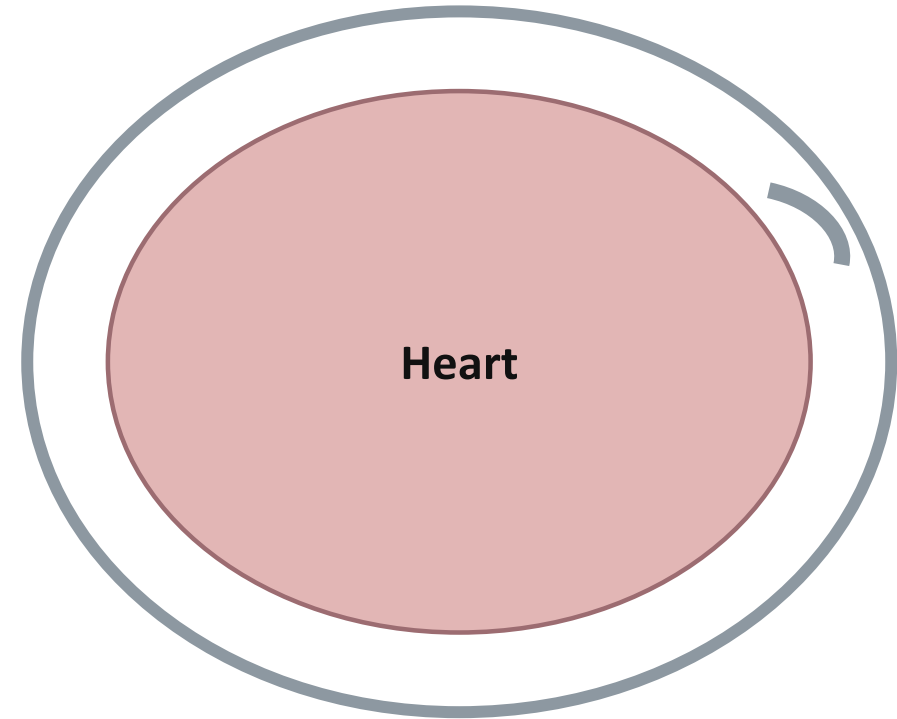
- Acute exudates may resolve or undergo organization.
- Healing can leave fibrous thickening or adhesions.
- The clinical effect depends on scar location, extent, and constraint.



Schematic

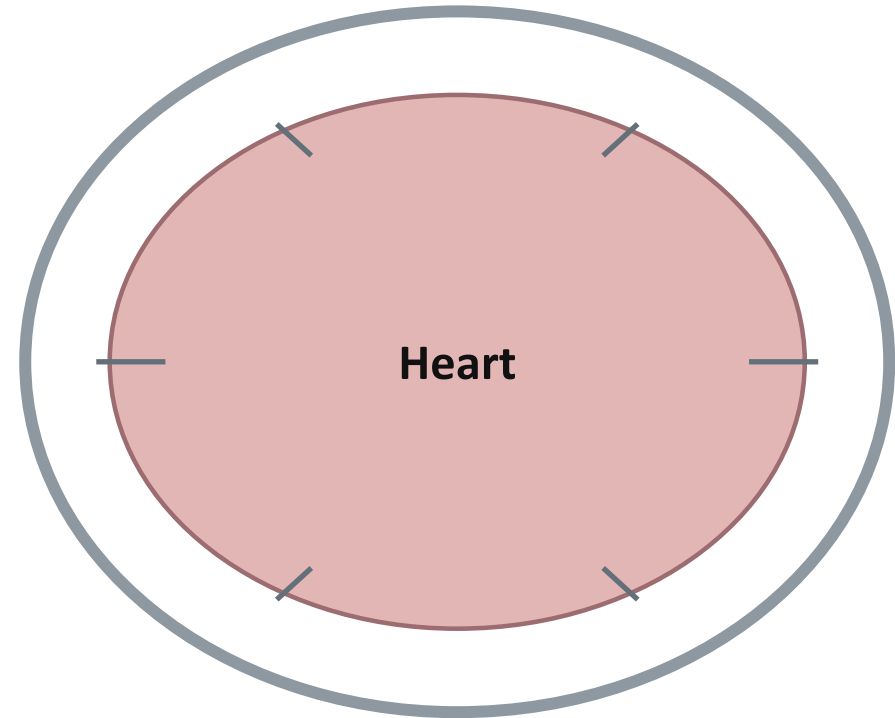
A soldiers' plaque may be incidental

- Plaque-like fibrous thickening can remain on a serosal surface.
- It is an organized outcome of prior inflammation.
- Such localized plaques rarely impair cardiac function.



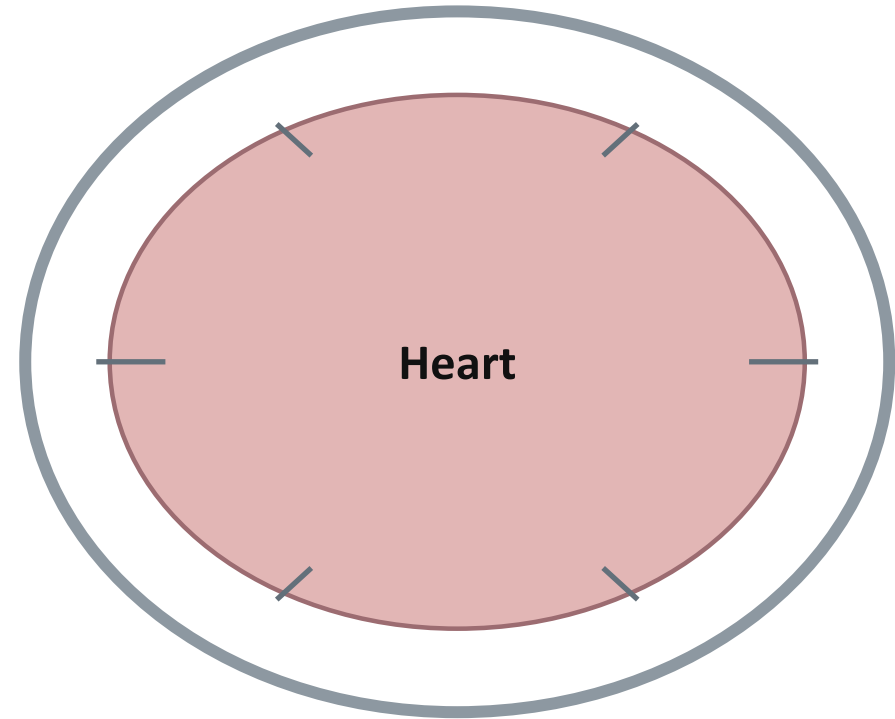
Delicate adhesions usually cause little dysfunction

- Thin, string-like attachments may persist after inflammation.
- Limited adhesions seldom alter hemodynamics.
- Scar presence alone does not establish constrictive physiology.



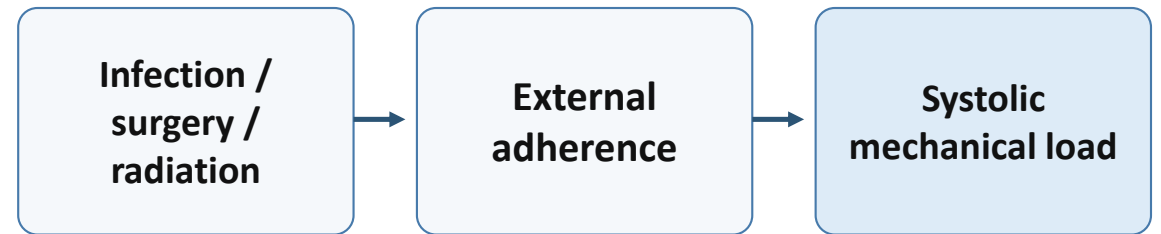
Adhesive pericarditis can obliterate the space

- Mesh-like fibrous adhesions bridge the two surfaces.
- The potential pericardial space may be largely lost.
- The text notes preserved cardiac function in most such cases.



Adhesive mediastinopericarditis fixes the sac externally

- It may follow infection, cardiac surgery, or mediastinal irradiation.
- Pericardium becomes adherent to neighboring structures.
- The external tethering places added load on cardiac contraction.



Schematic

External tethering changes systolic mechanics

- The contracting heart pulls on attached parietal pericardium.
- The rib cage and diaphragm can be retracted with systole.
- Pulsus paradoxus may be observed; Workload can cause dilation or hypertrophy.

Heart movement

Pulls attached structures

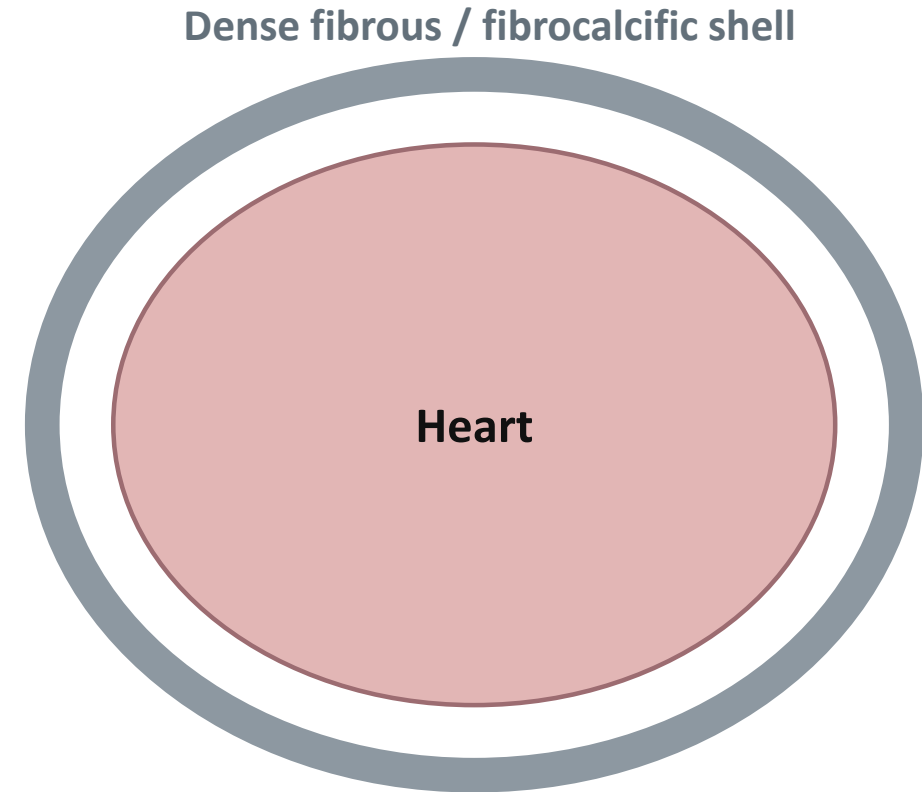
Possible effect

Hypertrophy / dilation

Schematic

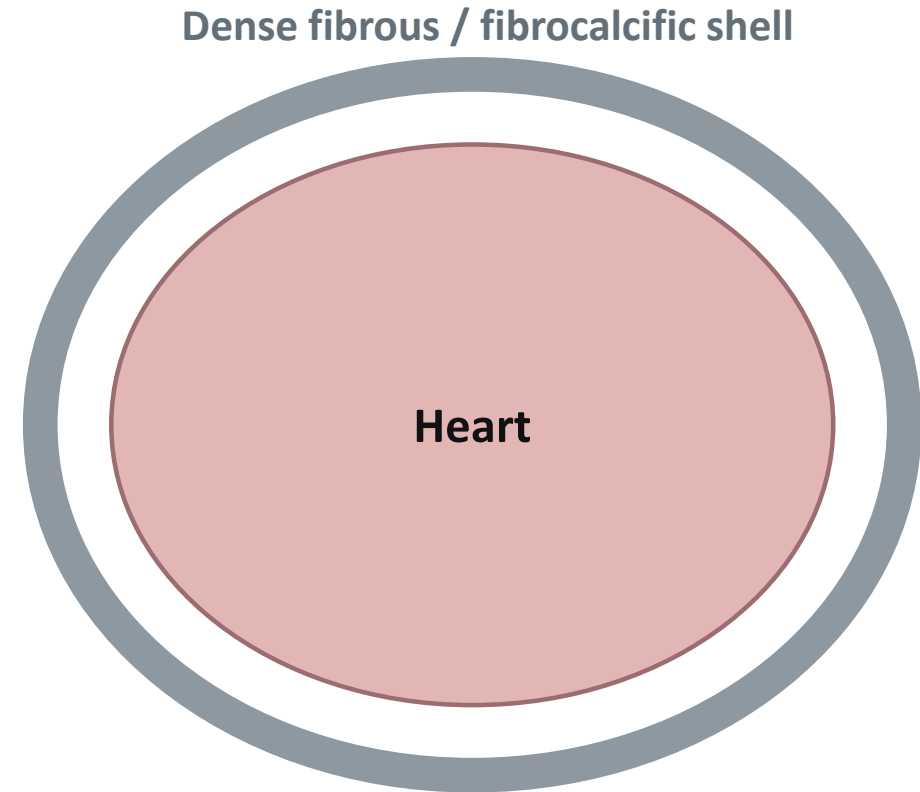
Constrictive pericarditis forms a rigid shell

- A dense fibrous or fibrocalcific scar encloses the heart.
- The pericardial space may be obliterated.
- An extreme rigid casing resembles a plaster mold: Concretio cordis.



The constricting scar can be markedly thick

- The text describes scar thickness up to 1 cm.
- Calcification may accompany dense fibrosis.
- An encasing wall limits the heart's capacity to enlarge.



Constrictive disease limits diastolic expansion

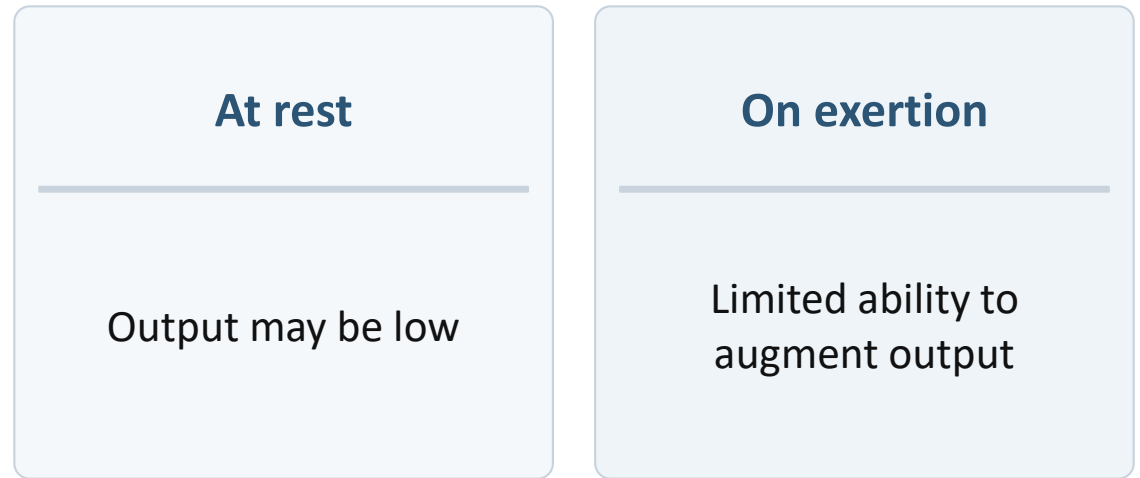
- A noncompliant shell restricts cardiac filling.
- The resulting decrease in output can mimic restrictive cardiomyopathy.
- The primary constraint is pericardial rather than intramyocardial.



Schematic

Cardiac reserve is especially impaired

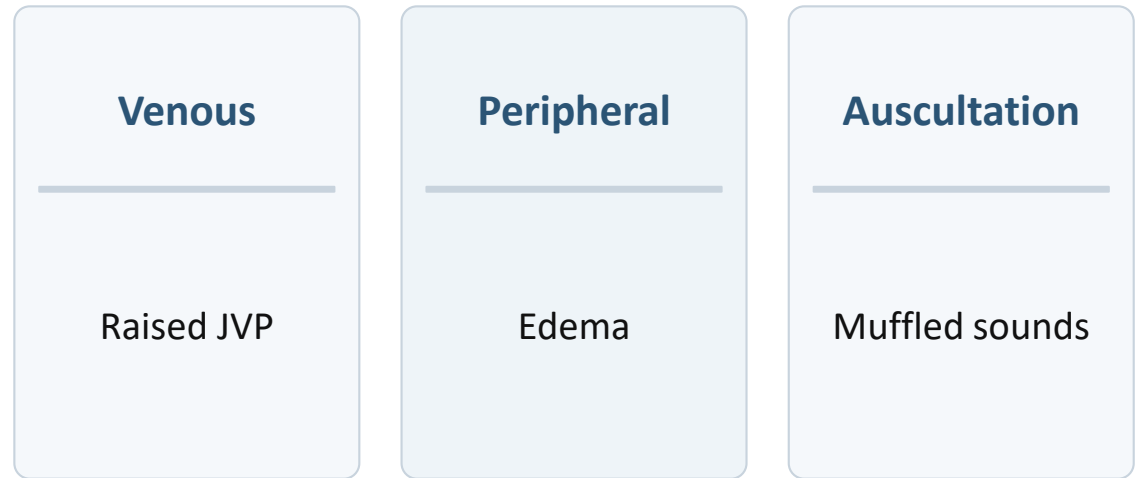
- Output may already be decreased at rest.
- The heart cannot substantially increase output with systemic demand.
- In contrast to external tethering, dense constriction prevents hypertrophy and dilation.



Schematic

Clinical signs reflect restricted cardiac filling

- Elevated jugular venous pressure suggests impaired venous return.
- Peripheral edema reflects congestive physiology.
- Heart sounds may be distant or muffled.



Schematic

Constrictive and restrictive disease can resemble

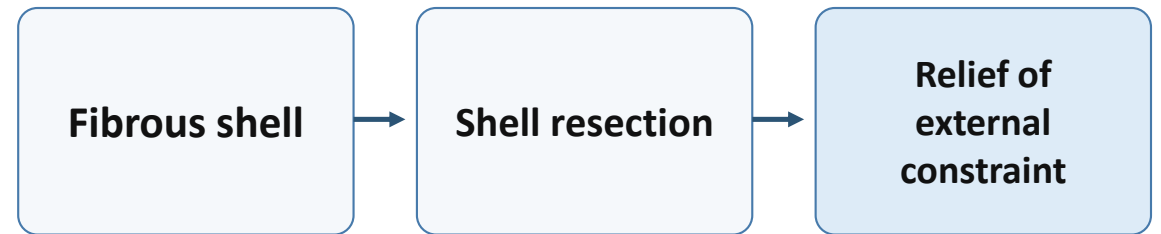
- Both impair cardiac filling and output.
- Constrictive disease involves an encasing pericardial scar.
- Restrictive cardiomyopathy involves altered myocardial compliance.

Constrictive	Restrictive
Encasing pericardial scar	Noncompliant myocardium
Extrinsic limitation	Intrinsic limitation

Editable comparison

Pericardiectomy addresses the mechanical cause

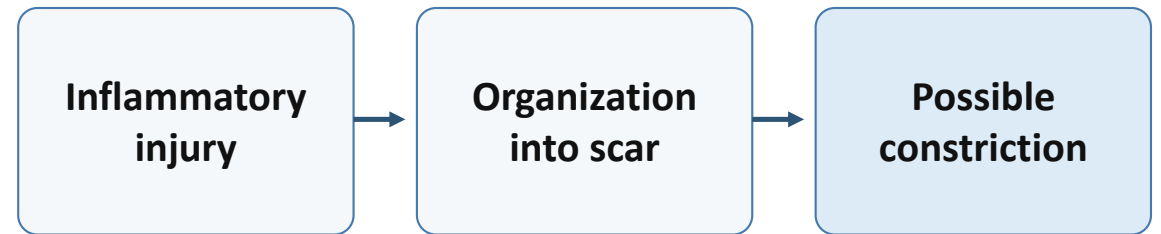
- Resection removes the constricting fibrous shell.
- The intended effect is relief of extrinsic diastolic restriction.
- Pericardiectomy as treatment for constrictive pericarditis.



Schematic

Pericardial pathology: Integrated sequence

- Inflammation may produce a serous, fibrinous, purulent, or hemorrhagic response.
- Some episodes resolve; Others organize into scar or adhesions.
- A dense encasing scar culminates in constrictive physiology.



Schematic

Different morphologies imply different threats

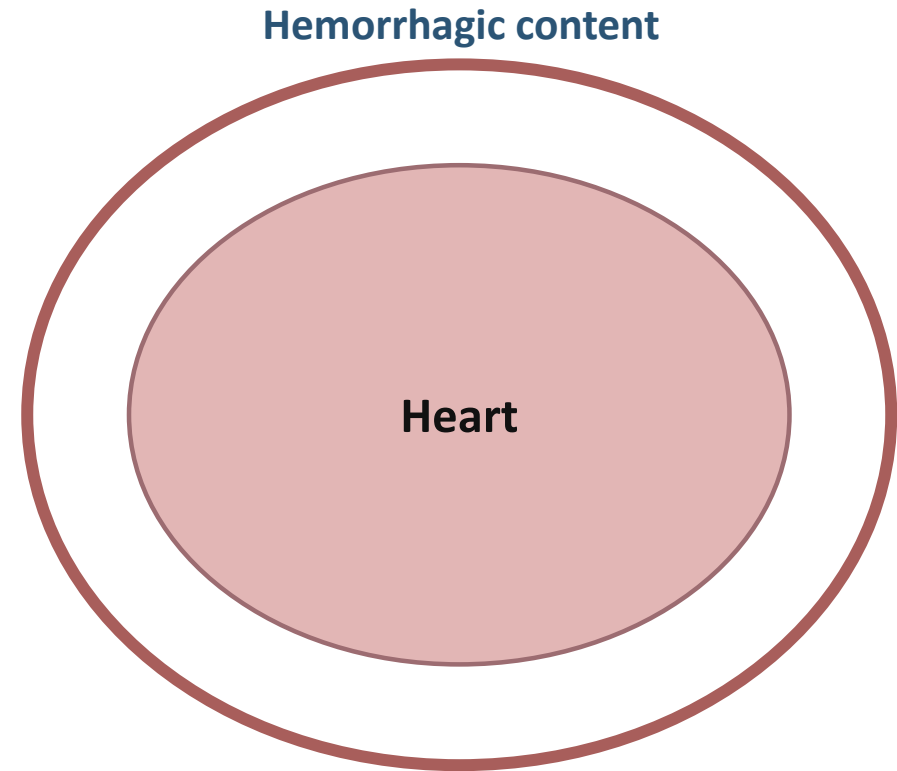
- Rapid blood accumulation: Acute filling compromise.
- Suppuration: Infection and subsequent fibrosis.
- Constrictive scar: Persistent restriction of diastolic expansion.

Hemorrhage	Suppuration	Constriction
Acute tamponade	Infection → fibrosis	Chronic filling restriction

Editable comparison

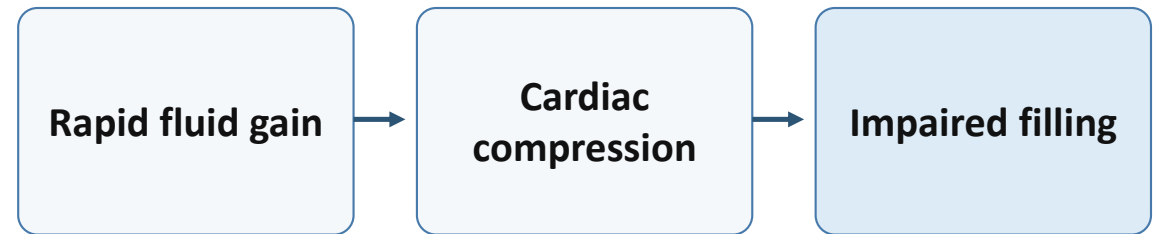
Case: Rapid hemodynamic deterioration

- A patient develops abrupt cardiac deterioration after myocardial rupture.
- A newly formed blood collection occupies the pericardial sac.
- Question: Which mechanism immediately threatens survival?



Case interpretation: Acute tamponade

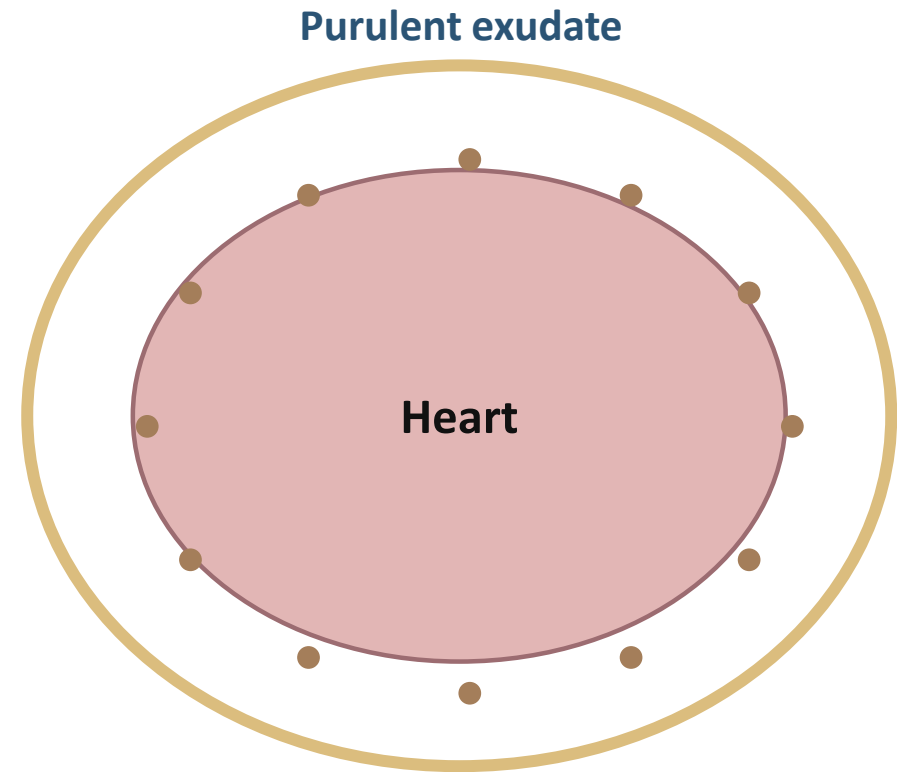
- Rapid hemopericardium leaves little time for sac remodeling.
- Compression primarily impairs filling of susceptible chambers.
- The rate of accumulation is essential to understanding severity.



Schematic

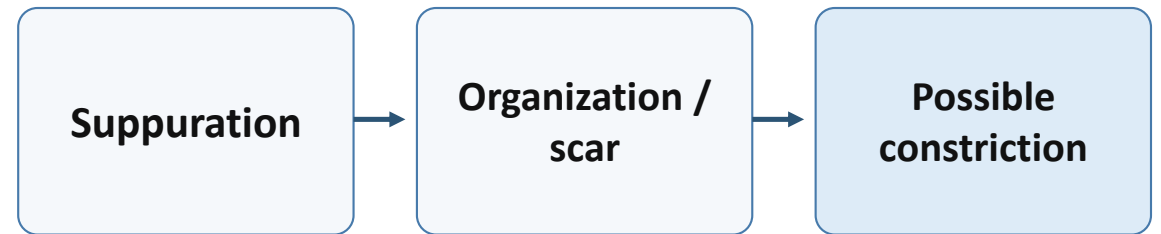
Case: Fever and an exudate-coated sac

- Lobar pneumonia is followed by spiking fever and rigors.
- The pericardial surfaces are red, granular, and coated with pus.
- Question: What is the route and likely healing consequence?



Case interpretation: Purulent extension

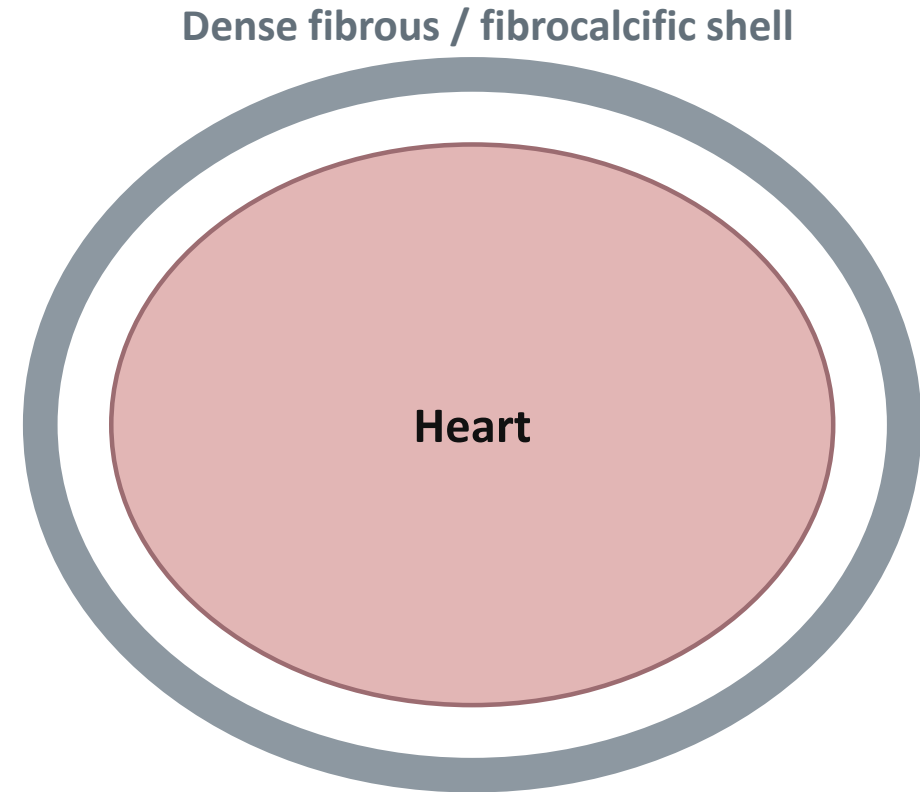
- Infection can spread directly from neighboring pneumonia.
- Acute suppurative inflammation tends to organize into scar.
- Constriction is therefore an important possible late consequence.



Schematic

Case: Elevated JVP and a rigid casing

- A patient has peripheral edema and muffled heart sounds.
- The heart is enclosed in a dense calcified pericardial scar.
- Question: Why is the ability to augment output reduced?



Case interpretation: Mechanical constriction

- The rigid pericardial shell limits diastolic expansion.
- The heart has very little capacity to increase output with demand.
- Removal of the constricting shell is the source-described treatment.



Schematic

Core distinctions to retain

- Effusion severity depends strongly on how quickly fluid accumulates.
- Acute exudate composition links morphology to etiology.
- The difference between incidental adhesion and constriction is functional.
- Pericardial disease can cause either an acute emergency or chronic limitation.

Primary reference and scope

- Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th edition (2026).
- Chapter 12, The Heart: pericardial disease and adjacent subheadings (printed pp. 525–526).
- Conceptual figures are original, editable schematics—not actual gross or microscopic specimens.