

Inflammatory Pleural Effusions

Pathogenesis • Morphology • Empyema • Outcomes

Pleural disease and its consequences for lung expansion

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Learning objectives

- Distinguish inflammatory pleuritis from noninflammatory pleural fluid collections.
- Explain the causes and morphologic spectrum of serous, serofibrinous, and fibrinous pleuritis.
- Trace the routes of infection that produce empyema.
- Predict how fibrin organization and pleural fibrosis limit lung expansion.

Pleural pathology is commonly secondary

- Most pleural disorders complicate diseases arising elsewhere in the body.
- Secondary infections and inflammation are common findings at autopsy.
- Primary intrapleural bacterial infection can arise from bloodstream seeding.
- The underlying pulmonary or systemic disorder often directs interpretation.

Interpretation

Always investigate the associated lung, systemic, or adjacent-site process.

Normal pleural fluid: Small and relatively acellular



Simplified cross-section — schematic, not histology

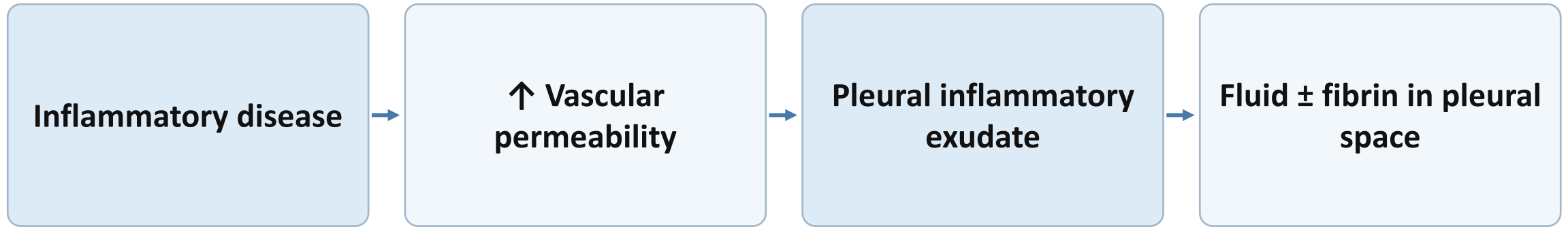
Normal volume

No more than ~15 mL of clear serous fluid lubricates pleural surfaces.

Five mechanisms can accumulate pleural fluid

Mechanism	Illustrative setting
↑ Hydrostatic pressure	Congestive heart failure
↑ Vascular permeability	Pneumonia
↓ Plasma oncotic pressure	Nephrotic syndrome
↑ Negative intrapleural pressure	Atelectasis
↓ Lymphatic drainage	Mediastinal carcinomatosis

Inflammation increases vascular permeability



Pathogenesis

Pneumonia is the source's illustrative condition for increased permeability.

Effusions: Inflammatory and noninflammatory

Feature	Inflammatory process	Noninflammatory collection
Primary concept	Pleuritis with exudate	Fluid accumulation without primary pleural inflammation
Representative form	Serous / serofibrinous / fibrinous	Hydrothorax
Example cause	Pneumonia	Heart failure
Important sequel	Fibrin organization	Related to underlying fluid imbalance

Pleuritis and pleural effusion are distinct

Pleuritis

Inflammatory reaction involving pleura

May yield fibrin deposition and/or an exudate

Can be mild without a large fluid volume

Pleural effusion

Accumulation of fluid in pleural space

May have inflammatory or noninflammatory causes

Large collections may compress lung tissue

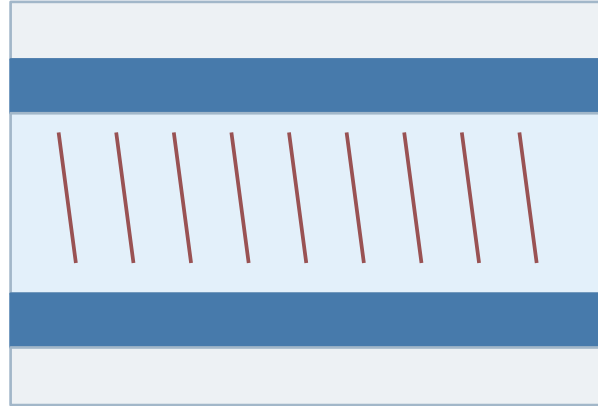
Inflammatory pleuritis: A morphologic spectrum

Serous



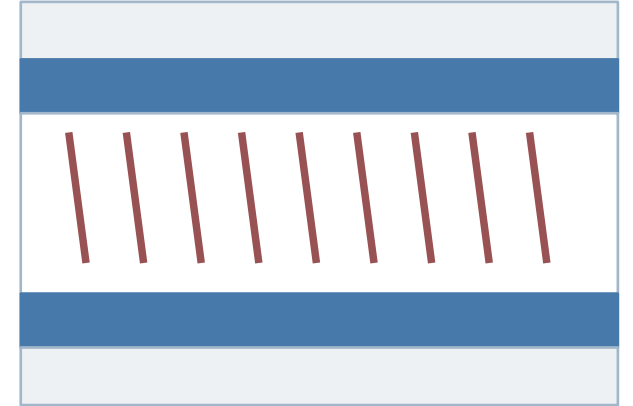
Fluid predominates

Serofibrinous



Fluid + fibrin

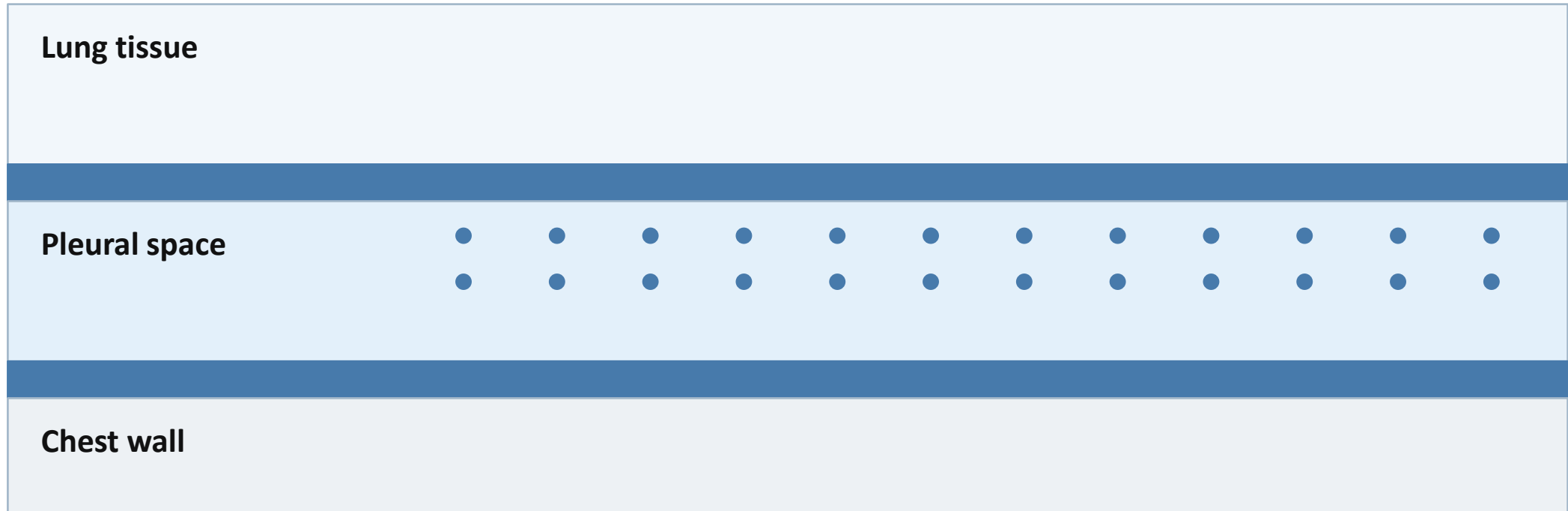
Fibrinous



Fibrin-rich surface

Conceptual spectrum; Categories reflect predominant exudate, not separate diseases

Serous pleuritis: Fluid predominates

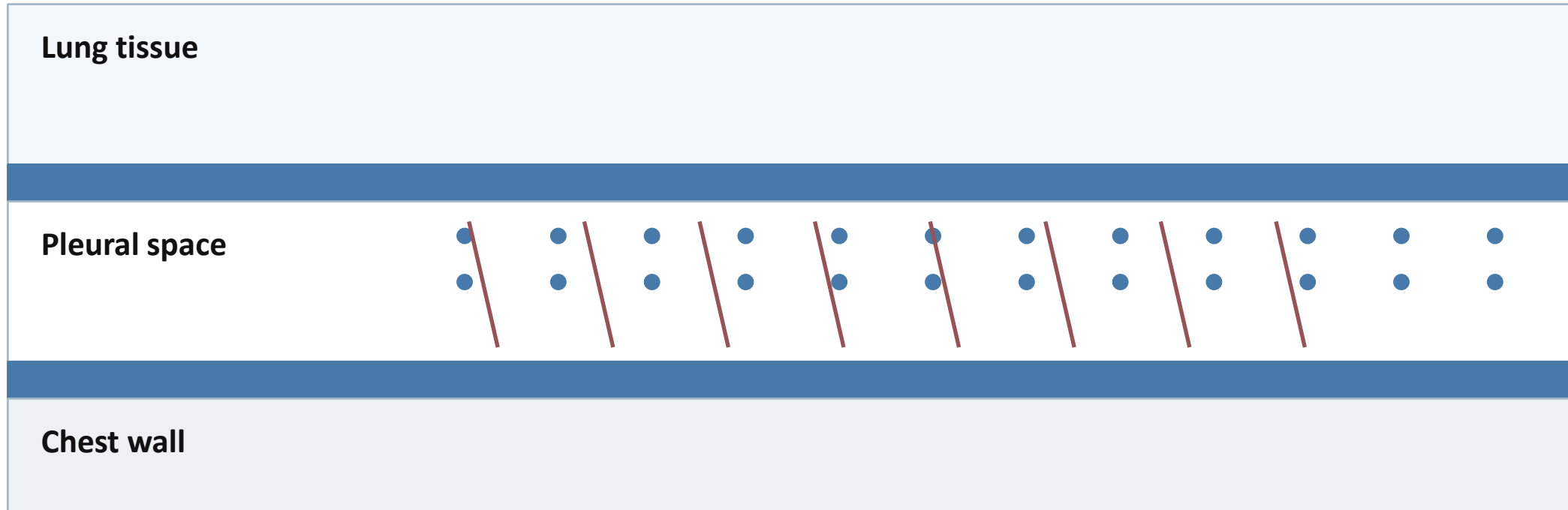


Simplified cross-section — schematic, not histology

Reading the label

Serous indicates the predominantly fluid character of the inflammatory reaction.

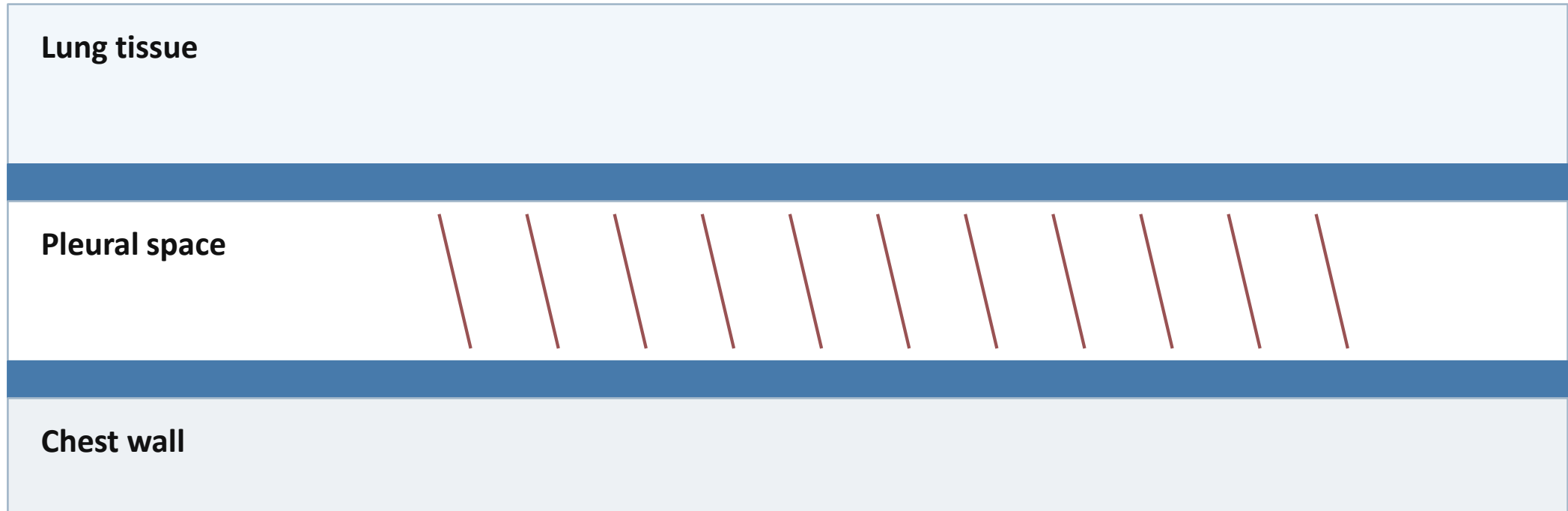
Serofibrinous pleuritis: Fluid with fibrin



Simplified cross-section — schematic, not histology

Morphologic idea A fluid exudate coexists with a fibrinous component.

Fibrinous pleuritis: Surface fibrin dominates



Simplified cross-section — schematic, not histology

Morphologic idea

The fibrinous reaction may resolve or progress to organization.

Intensity and duration shape pleural morphology

Inflammatory stimulus



**Variation in intensity and
duration**



**Serous ↔ serofibrinous ↔
fibrinous response**

Important

These three patterns share an inflammatory basis; They are not unrelated disorders.

Causes of inflammatory pleuritis: Overview

Pleural inflammation

```
graph TD; A[Pleural inflammation] --> B[Underlying lung disease]; A --> C[Systemic processes]; A --> D[Local injury / tumor]; B --> B1[Pneumonia, tuberculosis, infarction, abscess, bronchiectasis]; C --> C1[Rheumatoid arthritis, SLE, uremia, widespread infections]; D --> D1[Radiotherapy or metastatic pleural involvement];
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Underlying lung disease

Pneumonia, tuberculosis,
infarction, abscess,
bronchiectasis

Systemic processes

Rheumatoid arthritis, SLE,
uremia, widespread infections

Local injury / tumor

Radiotherapy or metastatic
pleural involvement

Pneumonia: Adjacent inflammation reaches pleura

Pneumonic consolidation near lung surface



Pleural fibrinous reaction



Possible inflammatory effusion

Clinical link

In pneumonia, pleuritis may coexist with pleuritic pain and a friction rub.

Tuberculosis can involve the pleural surface

- Tuberculosis is explicitly listed among the common underlying lung causes.
- Inflammation involving the underlying lung may produce pleuritis.
- The source also describes serous effusions, tuberculous empyema, and obliterative pleuritis in progressive tuberculosis.
- The pleural manifestation therefore spans fluid accumulation and fibrosing sequelae.

Pulmonary infarction is a pleuritic trigger

Lung infarction



**Inflammatory response near
pleura**



**Serous or fibrin-containing
pleuritis**

Differential

A vascular pulmonary insult belongs in the pleuritis differential—not only infection.

Lung abscess: Local source of pleural inflammation



Mechanism

Contiguous spread is the dominant route of empyema.

Bronchiectasis: Chronic diseased lung as a trigger

- Bronchiectasis appears's common pulmonary causes of pleuritis.
- The pleural response is secondary to inflammation in the underlying lung.
- An etiologic assessment should consider structural pulmonary disease.

Rheumatoid arthritis: Systemic cause of pleuritis

- Rheumatoid arthritis may produce serous or serofibrinous pleuritis.
- A pleural inflammatory exudate is not synonymous with bacterial infection.
- Extrapulmonary inflammatory disease is part of the differential.

Systemic lupus erythematosus and pleuritis

- Systemic lupus erythematosus is another listed cause.
- The source characterizes this association within serous or serofibrinous pleuritis.
- The pleural finding must be interpreted alongside systemic disease.

Uremia can produce inflammatory pleural disease

- Uremia is listed among causes of serous or serofibrinous pleuritis.
- Renal disease can also cause noninflammatory pleural fluid accumulation in generalized edema.
- Therefore, renal dysfunction alone does not establish the fluid's inflammatory status.

Diffuse systemic infection may inflame the pleura

- Diffuse systemic infections are listed among causes of serous or serofibrinous pleuritis.
- Pleural disease may coexist with illness extending beyond the thorax.
- Source identification requires attention to the systemic infectious context.

Radiotherapy can cause serofibrinous pleuritis



History

Prior thoracic radiotherapy changes etiologic interpretation.

Metastatic pleural involvement can cause exudate

- Metastatic pleural disease is listed among causes of pleuritis.
- Pleural metastases commonly produce serous or serosanguineous fluid.
- Neoplastic cells may be present, making cytologic examination valuable.
- Inflammatory appearance does not exclude tumor-related disease.

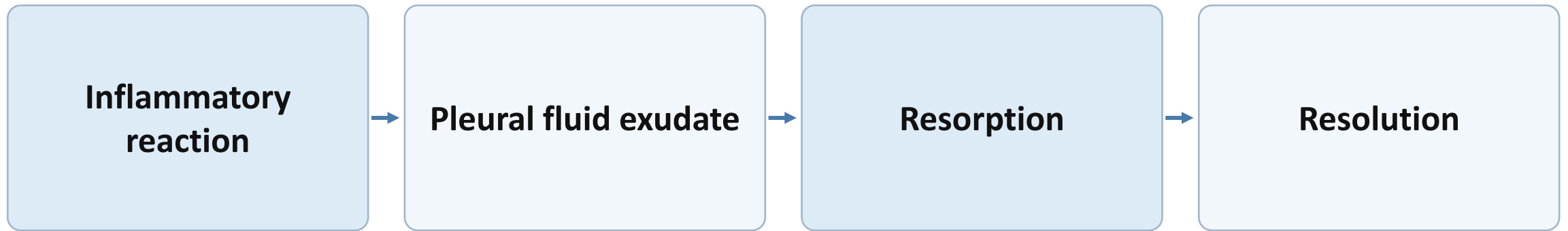
Interpret the etiologies by their source

Origin	Examples	Diagnostic reasoning
Pulmonary	Pneumonia, TB, abscess, bronchiectasis	Seek inflammation in underlying lung
Pulmonary vascular	Infarction	Consider tissue injury without direct infection
Systemic	RA, SLE, uremia, diffuse infection	Interpret alongside multi-organ disease
Iatrogenic / neoplastic	Radiotherapy, pleural metastasis	Review treatment and tumor history

Many inflammatory pleural reactions are limited

- In most listed conditions, the pleural inflammatory reaction is minimal.
- A fluid exudate may be resorbed rather than persisting.
- If fibrin is present, its fate determines whether adhesions remain.

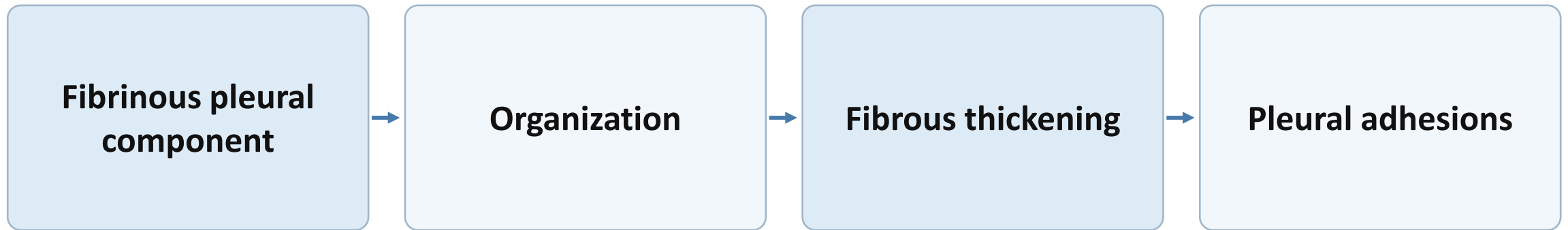
Fluid exudate may be resorbed



Outcome

Fibrin may follow a different path even when fluid resolves.

Fibrin can organize into fibrous adhesions



Clinical principle

Organization leaves persistent structural change after the active exudate wanes.

Pleural reaction: Minor or compressive

Limited reaction

Minimal pleural change

Exudate may be resorbed

Fibrin may resolve or organize

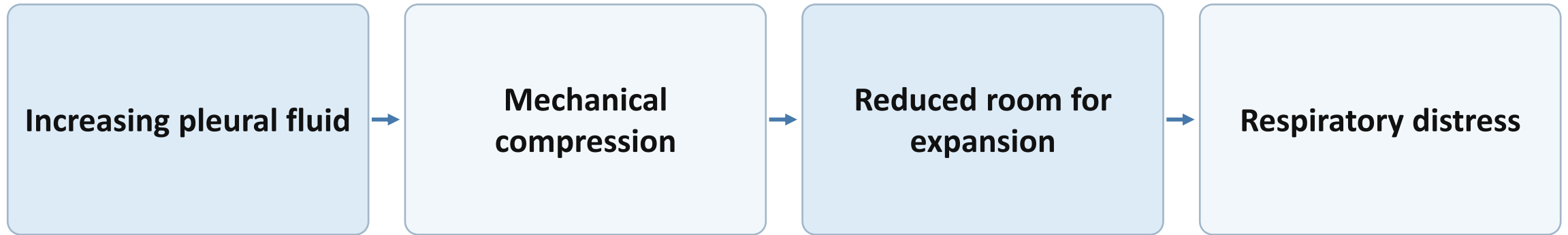
Large fluid collection

Increased pleural volume

Compression of underlying lung

Potential respiratory distress

Large effusions compress the lung



Mechanism

The direct problem is mechanical compromise, not simply presence of liquid.

Fluid burden and fibrous scarring are distinct

Consequence	Immediate substrate	Functional mechanism
Fluid-related restriction	Large pleural collection	Lung compression
Adhesion-related restriction	Organized pleural fibrin	Fibrous bridges limit pleural motion
Encasement-related restriction	Dense fibrous pleural covering	Impaired pulmonary expansion

Teaching distinction Both can cause respiratory compromise through different substrates.

Pleuritis may cause pain and a friction rub

- Pleuritic chest pain may accompany pleural inflammation in pneumonia.
- A pleural friction rub is another possible clinical manifestation.
- These signs indicate pleural irritation; They do not establish empyema.

Pneumonia at the pleural surface: Two pathways

Pneumonia reaching pleural surface

```
graph TD; A[Pneumonia reaching pleural surface] --> B[Pleuritis]; A --> C[Pleural infection]; B --> D[Fibrinous surface reaction may resolve or organize]; C --> E[Spread to the cavity can cause empyema];
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Pleuritis

Fibrinous surface reaction may resolve or organize

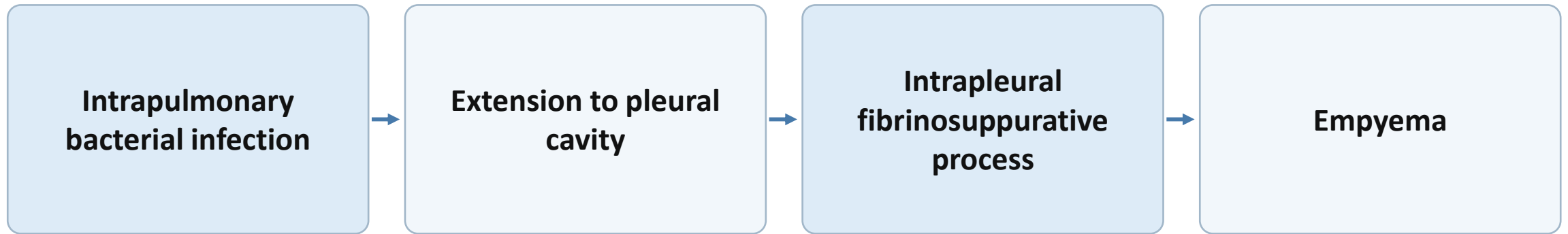
Pleural infection

Spread to the cavity can cause empyema

Principle

Do not equate a superficial fibrinous reaction with intrapleural pus.

Empyema is a recognized pneumonia complication



Morphologic basis Empyema is an infection-related purulent pleural exudate.

Empyema: Purulent pleural exudate

- Empyema is a collection of purulent inflammatory exudate in the pleural space.
- It usually results from seeding by bacteria or fungi.
- Its defining morphology is pus enriched in neutrophils and other leukocytes.

Microorganisms reach the pleura by several routes

Microbial seeding of pleural space

```
graph TD; A[Microbial seeding of pleural space] --> B[Contiguous spread]; A --> C[Hematogenous / lymphatic]; A --> D[Through diaphragm]; B --> B1[Infection in adjacent lung: Most common route]; C --> C1[Occasional distant dissemination]; D --> D1[Rare extension from subdiaphragmatic or liver abscess];
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Contiguous spread

Infection in adjacent lung: Most common route

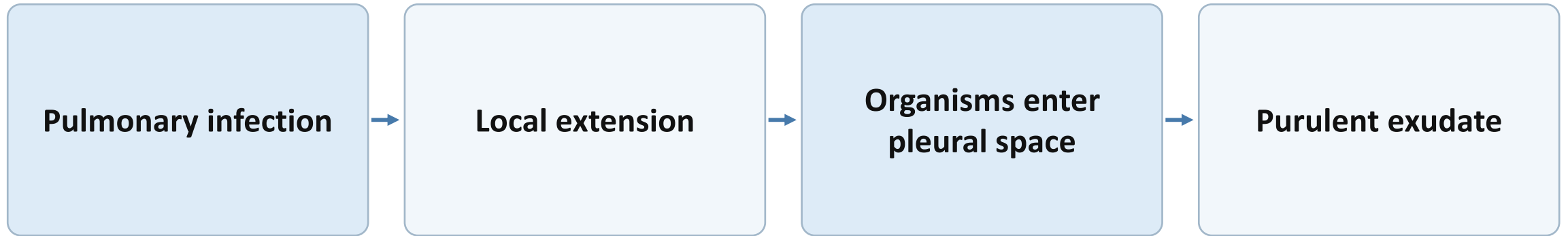
Hematogenous / lymphatic

Occasional distant dissemination

Through diaphragm

Rare extension from subdiaphragmatic or liver abscess

Contiguous spread is the dominant empyema route



Most common route A nearby intrapulmonary source is the principal pathway described.

Distant infectious seeding is less common

Hematogenous spread

Dissemination through blood

Infectious source can be remote

Seeding occurs in pleural cavity

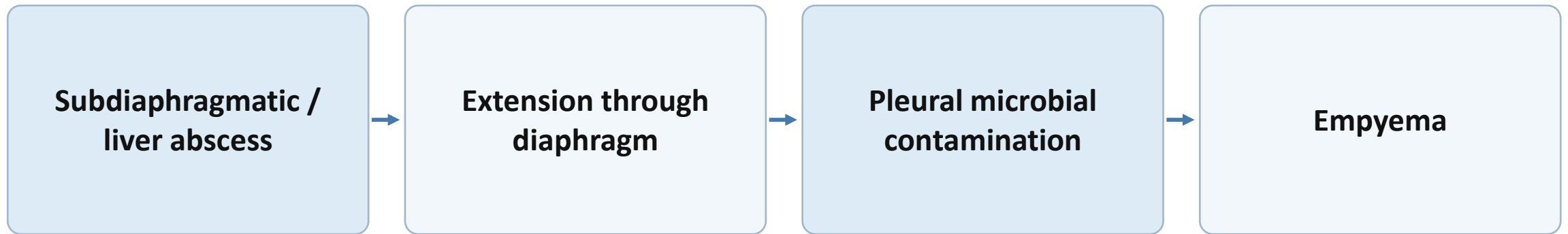
Lymphatic spread

Dissemination through lymphatic pathways

Source may be distant from pleura

Less frequent than local extension

Subdiaphragmatic infection can cross the diaphragm



Pattern

This rare pathway more often involves the right side.

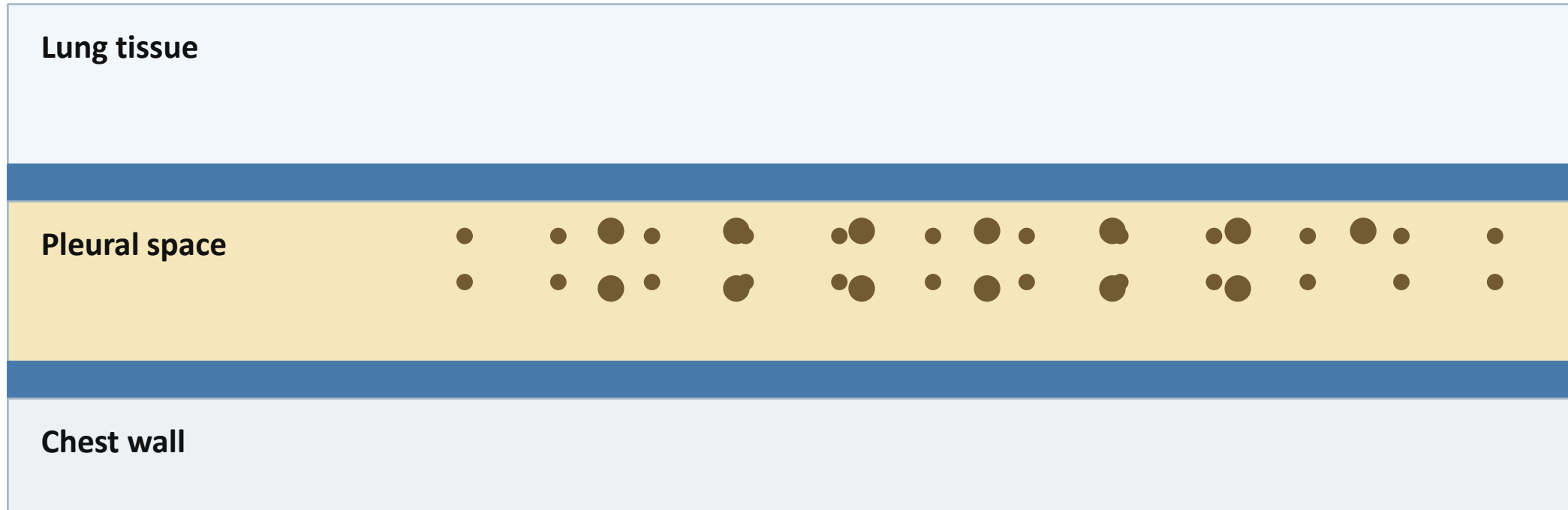
An anatomic clue: Right-sided transdiaphragmatic spread

- Rare extension from an abscess below the diaphragm can reach the pleural cavity.
- Subdiaphragmatic and liver abscesses as examples.
- Such spread occurs more often on the right side.
- A thoracic collection may therefore have an abdominal source.

Empyema pathways: Integrate the source

Route	Typical source context	Source frequency language
Contiguous	Intrapulmonary infection	Most commonly
Hematogenous	Infection at a distant site	Occasionally
Lymphatic	Infection at a distant site	Occasionally
Transdiaphragmatic	Subdiaphragmatic or liver abscess	Rarely; More often right-sided

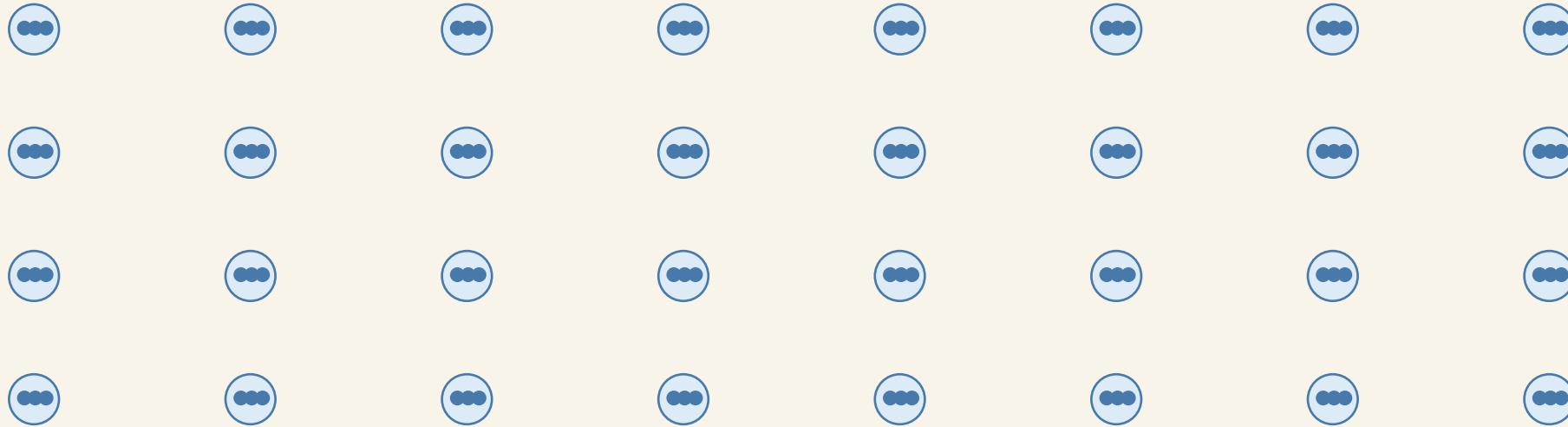
Empyema: Characteristic gross appearance



Simplified cross-section — schematic, not histology

Gross description Loculated, yellow-green, creamy pus is characteristic.

Purulent fluid is rich in neutrophils

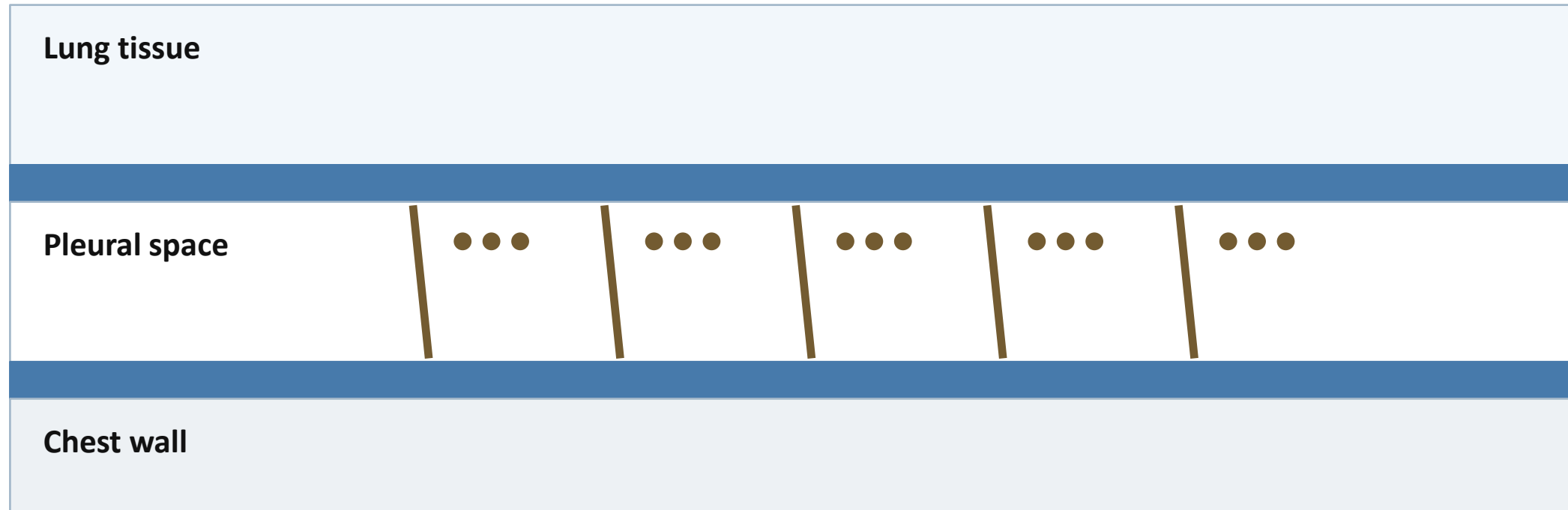


Conceptual leukocyte-rich purulent material — not a histologic image

Composition

Masses of neutrophils are admixed with other leukocytes.

Loculation partitions infected pleural material



Simplified cross-section — schematic, not histology

Spatial pattern

Empyema may become loculated and walled off by fibrosis.

Empyema volume: Often limited, sometimes large

- The volume of purulent exudate is usually relatively small.
- The textbook nevertheless describes accumulations reaching 500–1000 mL.
- A small loculated collection may still evolve into fibrotic pleural restriction.
- Volume alone does not capture the eventual degree of organization.

Empyema may resolve or organize

Purulent pleural exudate

```
graph TD; A[Purulent pleural exudate] --> B[Resolution]; A --> C[Organization]; B --> D[Infection and exudate may regress]; C --> E[Dense fibrous adhesions often develop];
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Resolution

Infection and exudate may regress

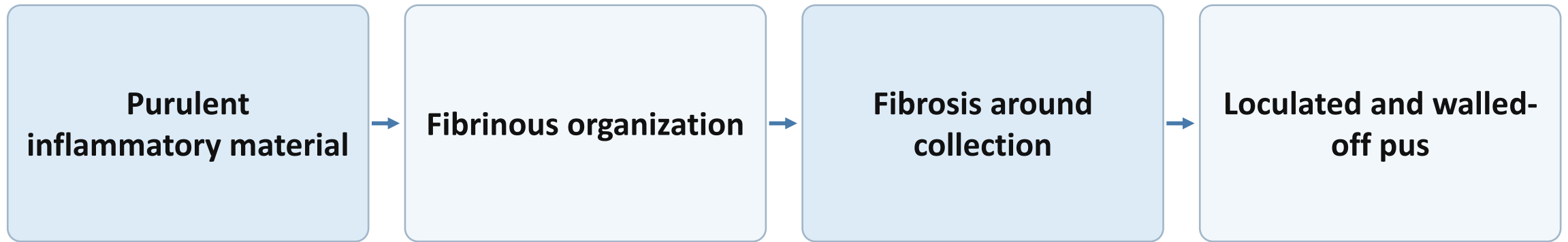
Organization

Dense fibrous adhesions often develop

Principle

The source emphasizes organization as a frequent outcome.

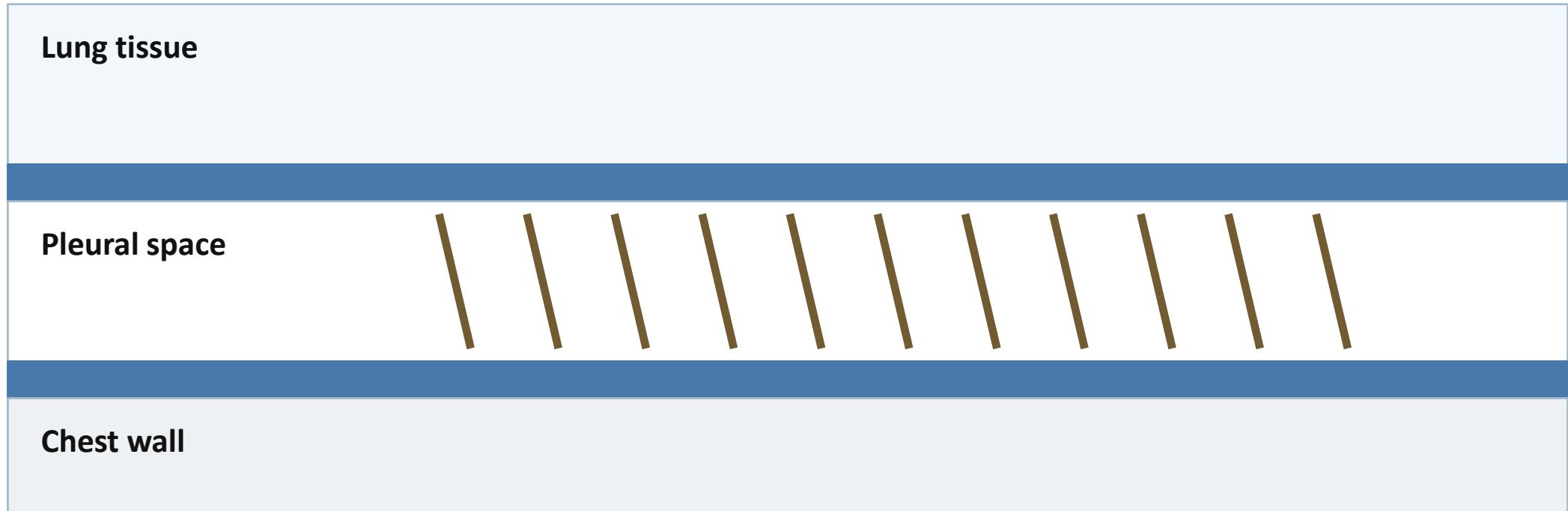
Fibrous organization walls off the pus



Morphology

A bounded pocket is not equivalent to a freely communicating effusion.

Organized empyema produces tough pleural adhesions

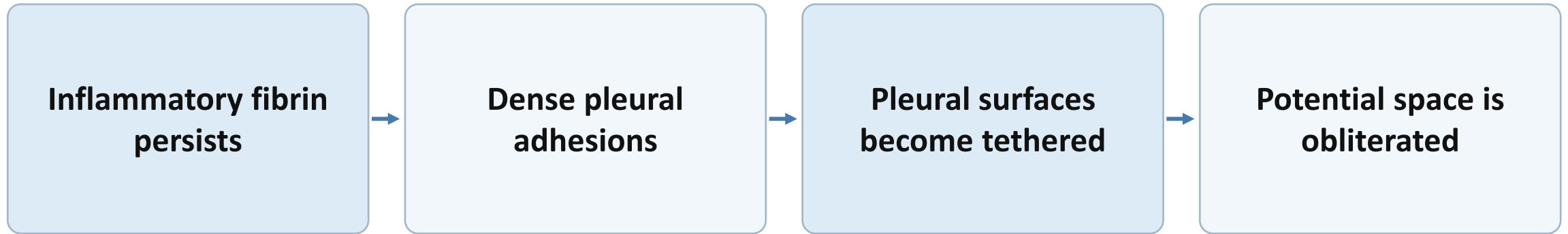


Simplified cross-section — schematic, not histology

Sequela

Dense, tough fibrous adhesions are emphasized in the source.

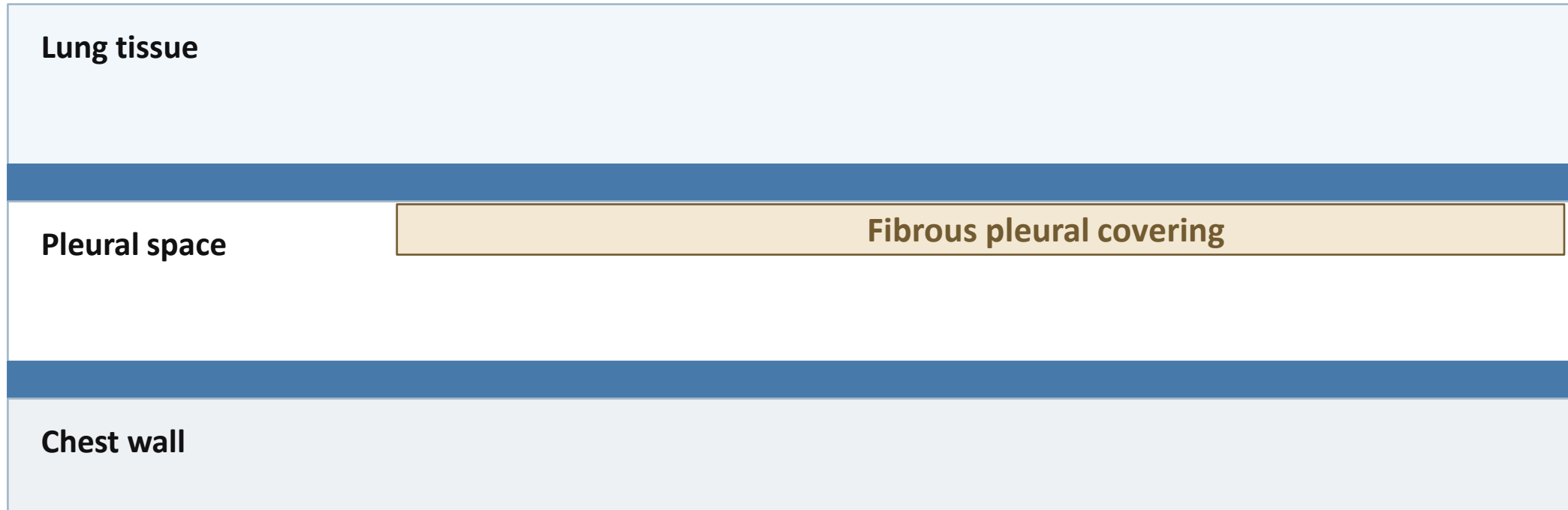
Adhesions may obliterate the pleural space



Anatomic change

The cavity may be lost to organized fibrous tissue.

Fibrous tissue can envelop the lung

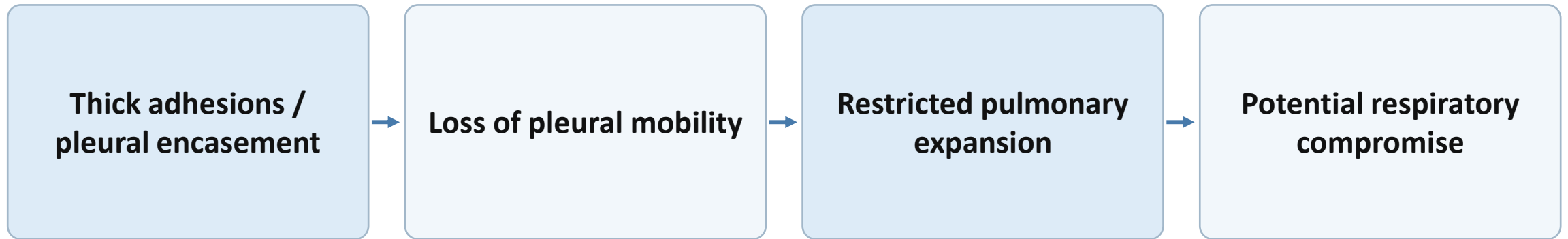


Simplified cross-section — schematic, not histology

Consequence

An encasing fibrous layer impairs the ability of the lung to expand.

Organized empyema restricts pulmonary expansion



Pathophysiology

Pleural space obliteration and lung encasement are distinct but related forms of restriction.

Serofibrinous pleuritis is not empyema

Feature	Serofibrinous pleuritis	Empyema
Principal material	Inflammatory fluid + fibrin	Purulent exudate
Typical context	Broad pulmonary or systemic inflammation	Microbial seeding (bacterial / fungal)
Gross description	Fluid with fibrinous component	Creamy yellow-green, often loculated pus
Outcome	Resolution or fibrin organization	Frequently dense adhesions / encasement

Pleural inflammation: Resolution versus fibrosis

Route of evolution	Morphologic outcome	Functional relevance
Exudate resorption	Loss of accumulated fluid	Potential return toward normal state
Fibrin organization	Pleural thickening / adhesions	Tethering of adjacent pleural surfaces
Dense organization after empyema	Space obliteration or lung encasement	Major restriction of lung expansion

Hemorrhagic pleuritis: Blood-tinged inflammatory exudate

- Sanguineous inflammatory exudates are comparatively infrequent.
- The source associates them with hemorrhagic diatheses, rickettsial infection, and neoplastic pleural involvement.
- The appearance requires distinction from blood accumulating as hemothorax.

Hemorrhagic pleuritis versus hemothorax

Question	Hemorrhagic pleuritis	Hemothorax
What is present?	Sanguineous inflammatory exudate	Blood within pleural cavity
Context in source	Diathesis, rickettsial infection, neoplasia	Trauma, surgery, ruptured aortic aneurysm
Key distinction	Inflammatory pleural reaction	Accumulation of escaped blood

Diagnostic warning Gross redness alone should not collapse these categories.

Blood-stained exudate: Consider neoplasia

- A careful search for tumor cells is advised in hemorrhagic pleuritis.
- Metastatic pleural implants can produce serous or serosanguineous fluid.
- Neoplastic cells are often present in metastatic effusion sediment.
- Careful cytologic examination has considerable diagnostic value.

Hydrothorax is noninflammatory serous fluid

- Hydrothorax is a noninflammatory collection of serous fluid.
- The fluid is described as clear and straw colored.
- Heart failure is the most common cause listed by Robbins.
- Other systemic causes include generalized edema in renal failure or cirrhosis.

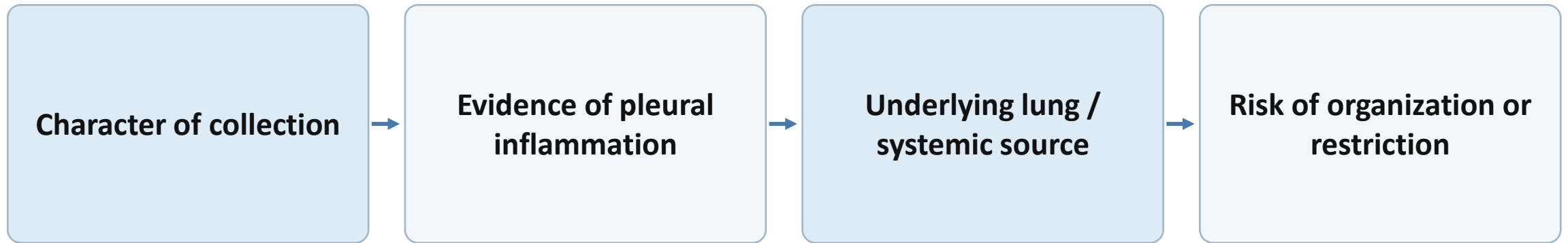
Chylothorax is a different pleural collection

- Chylothorax is an accumulation of milky fluid, usually of lymphatic origin.
- Finely emulsified fat accounts for its milky white appearance.
- The source emphasizes thoracic-duct trauma and lymphatic obstruction, often related to malignancy.
- It is not defined by the neutrophil-rich pus of empyema.

Pleural collection: Gross-pattern differential

Process	Dominant material	Source clue
Serous pleuritis	Inflammatory fluid	Underlying pulmonary or systemic inflammation
Serofibrinous / fibrinous	Fibrin $\pm$ inflammatory fluid	May organize into adhesions
Empyema	Purulent material rich in neutrophils	Bacterial or fungal pleural seeding
Hydrothorax	Clear straw-colored serous fluid	Heart failure / generalized edema
Hemothorax	Blood	Trauma / surgery / aortic rupture
Chylothorax	Milky lymphatic fluid	Lymphatic trauma or obstruction

Interpret pleural fluid within an etiologic framework



Source boundary

The excerpt provides morphology and causes; It does not establish a fluid-biochemistry cutoff algorithm.

Case: Pneumonia with pleuritic pain

Clinical vignette

A patient with lobar pneumonia develops sharp pleuritic pain and a friction rub. A small adjacent pleural exudate is present.

Questions

- Which tissue surface is reacting?
- Must there be pus?

Teaching point

Surface pleuritis can accompany pneumonia without established empyema.

Case: Infection beneath the diaphragm

Clinical vignette

A patient with a right-sided liver abscess subsequently develops a loculated, purulent collection in the right pleural space.

Questions

- What route connects the sites?
- Which side is more typical?

Teaching point

Rare transdiaphragmatic spread is a textbook-recognized route of empyema.

Case: Inflammatory disease without pneumonia

Clinical vignette

A patient with established systemic lupus erythematosus is found to have a serofibrinous pleural reaction without an identified adjacent lung abscess.

Questions

- Is infection the only cause?
- Which source category applies?

Teaching point

Autoimmune systemic disease is among the listed causes of serofibrinous pleuritis.

Case: Sanguineous pleural fluid

Clinical vignette

An adult with a known extrathoracic malignancy develops a serosanguineous pleural collection. The fluid is not described as frank whole blood.

Questions

- What alternate process is likely?
- What assessment is useful?

Teaching point

Consider metastatic pleural involvement; Careful sediment cytology is valuable.

Case: Chronic restriction after empyema

Clinical vignette

Following a pleural purulent infection, the gross pleural space becomes replaced by dense fibrous adhesions that partially encase the lung.

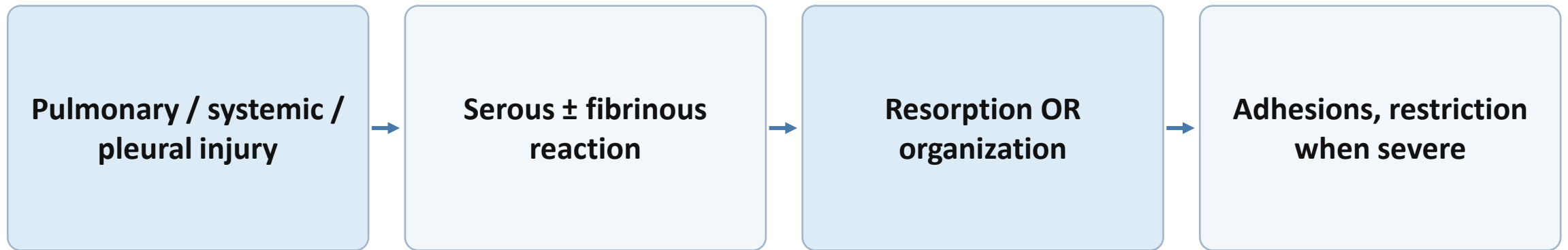
Questions

- What morphologic process occurred?
- Why is expansion restricted?

Teaching point

Organization of exudate can obliterate the pleural space and tether the lung.

Integrated model: Cause → exudate → outcome



- Empyema is the purulent form associated with microbial seeding.
- Dense pleural fibrosis, not fluid alone, can limit pulmonary expansion.

Reading and core references

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed. (2026). Chapter 15: The Lung.
- Pleura, Pleural Effusion, and Inflammatory Pleural Effusions.
- Related context: bacterial pneumonia morphology and pleural complications; noninflammatory pleural effusions; pleural tumors.
- Diagnostic numerical thresholds and management algorithms are outside the scope of the supplied excerpt.