Pleural cavity Diseases

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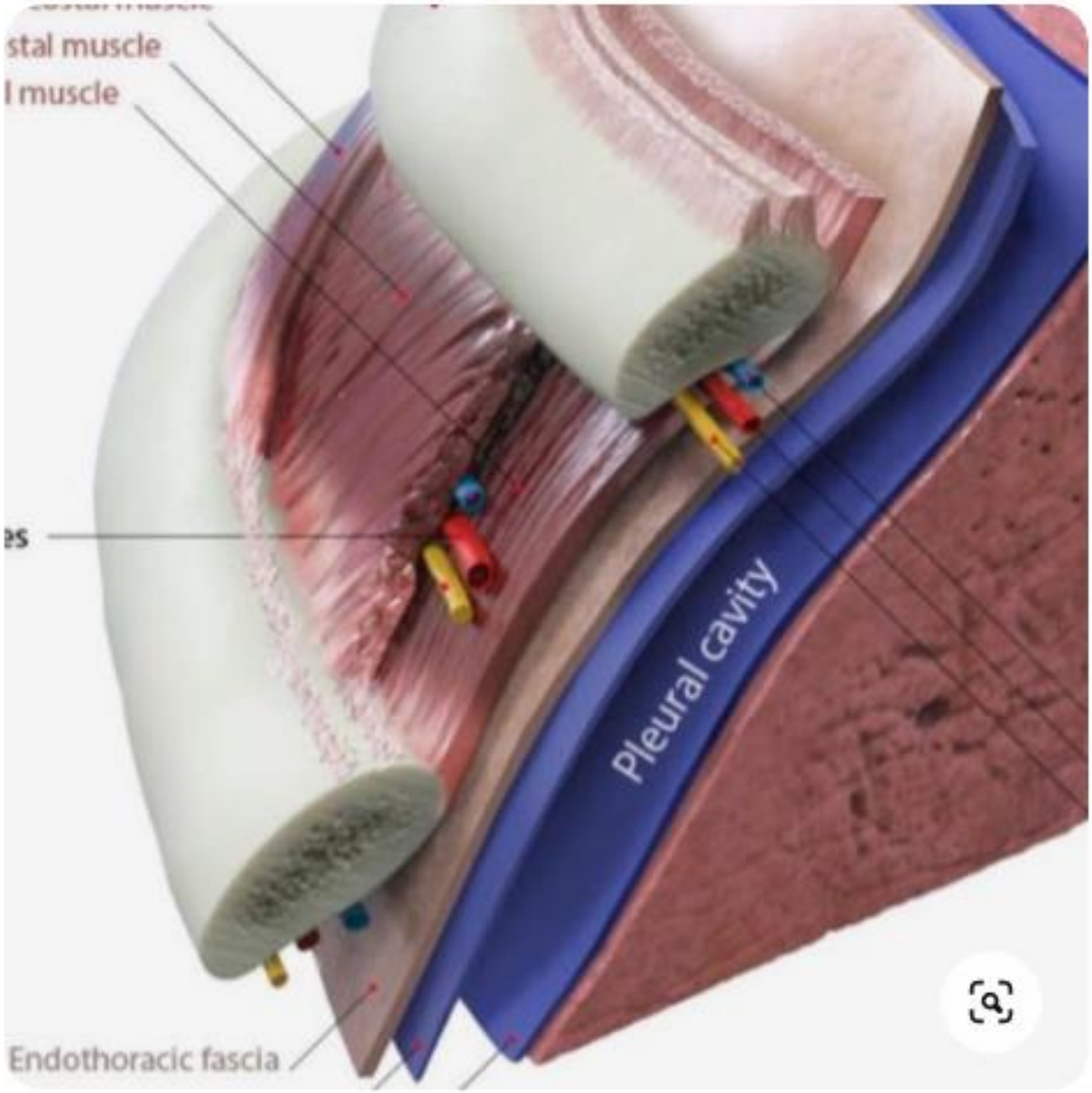
ANATOMY

- It is a serous membrane divided into visceral and parietal pleurae.
- Visceral pleura (VP) covers the lung parenchyma.
- Parietal pleura (PP) lines the inside of the thoracic cage, the diaphragm and the mediastinum.
- It is a single layer of metabolically active mesothelial cells that can absorb or secrete fluid.
- VP is devoid of somatic innervation, whereas PP is innervated through a rich network of somatic, sympathetic, and parasympathetic fibers.


stal muscle
l muscle
es
Pleural cavity
Endothoracic fascia

PHYSIOLOGY

- If the thorax is opened to atmospheric pressure, the lungs decrease in volume because of their elastic recoil, while at the same time, the thorax enlarges.
- The volume of the thoracic cavity is approximately 55% of the vital capacity, whereas the volume of the lung is below its RV.
- With the chest closed and the patient relaxed, the respiratory system is at its (FRC), which is approximately 35% of the total lung capacity.

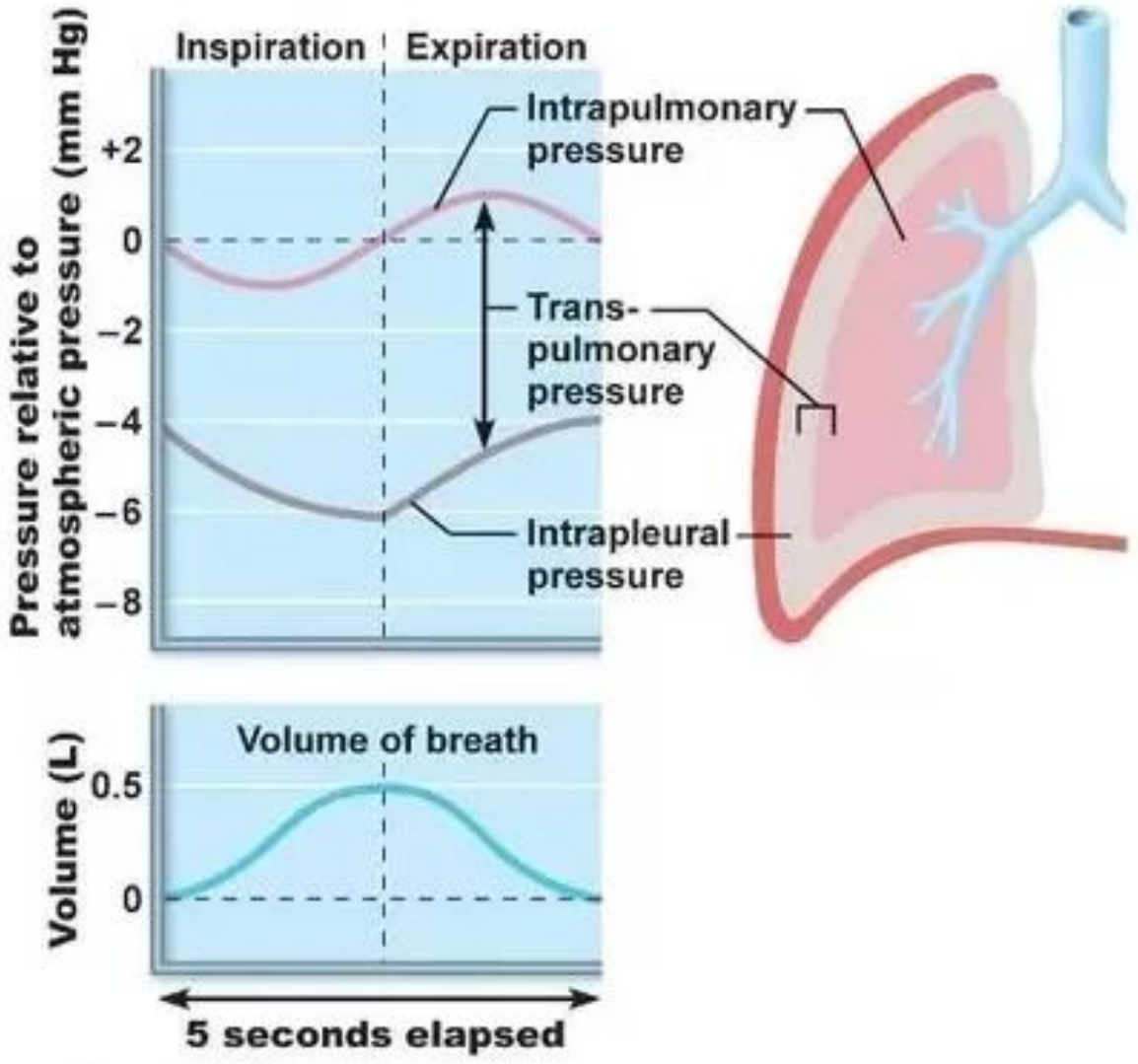
PHYSIOLOGY

- At FRC, the opposing elastic forces of the chest wall and lung produce a negative pressure between the visceral and the parietal pleura, which is called the pleural pressure Pp.
- This pressure surrounds the lung and is the primary determinant of the volume of the lung.
- Pp represents the balance between the outward pull of the thoracic cavity and the inward pull of the lung

Intrapulmonary pressure. Pressure inside lung decreases as lung volume increases during inspiration; pressure increases during expiration.

Intrapleural pressure. Pleural cavity pressure becomes more negative as chest wall expands during inspiration. Returns to initial value as chest wall recoils.

Volume of breath. During each breath, the pressure gradients move 0.5 liter of air into and out of the lungs.



PLEURAL FLUID PHYSIOLOGY

- Pleural fluid is constantly secreted, mostly by filtration from the microvessels in the parietal pleura resulting in 0.26 ml/kg volume in each side.
- The resorption of pleural fluid may be through lymphatic stomata in the parietal pleura rather than through the visceral pleura.
- The pleural pressure, which is subatmospheric, is proportional to the pressure developed within the lung.

PATHOPHYSIOLOGY OF PLEURAL EFFUSION

- **Fluid movement, or flux, in or out of any anatomic space, including the pleural cavity, is determined by :**
  1. **The relationship described by Starling between hydrostatic and oncotic pressures (the Starling forces) on each side of the membrane separating the space from the tissues.**
  2. **By the permeability of the membrane to fluid and macromolecules.**
  3. **The efficiency of lymphatic drainage of the space.**
- **When equilibrium is reached, the amount of fluid in the anatomic space is constant.**
- **Changes in any of these causes fluid movement in or out of the space to occur.**

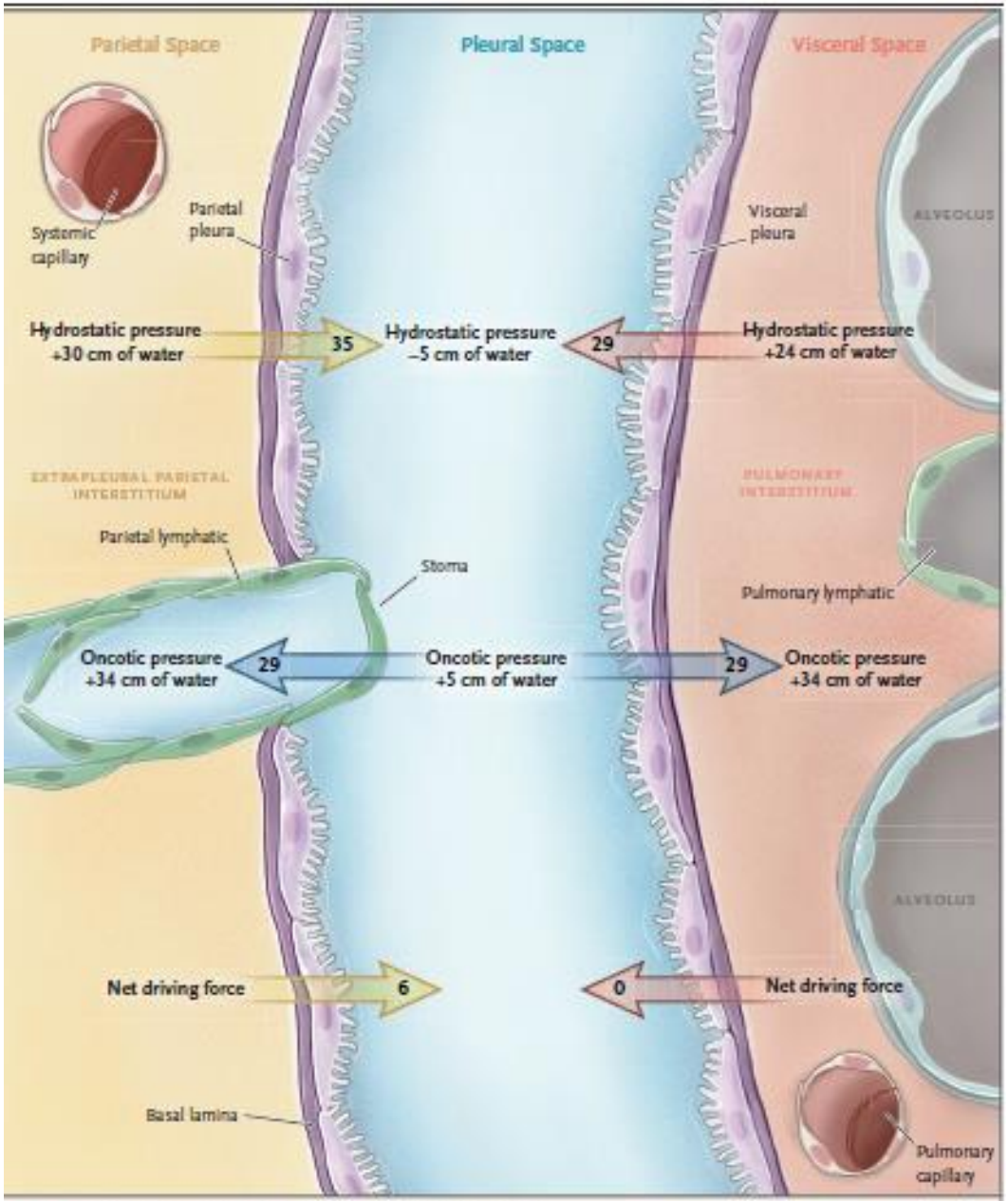
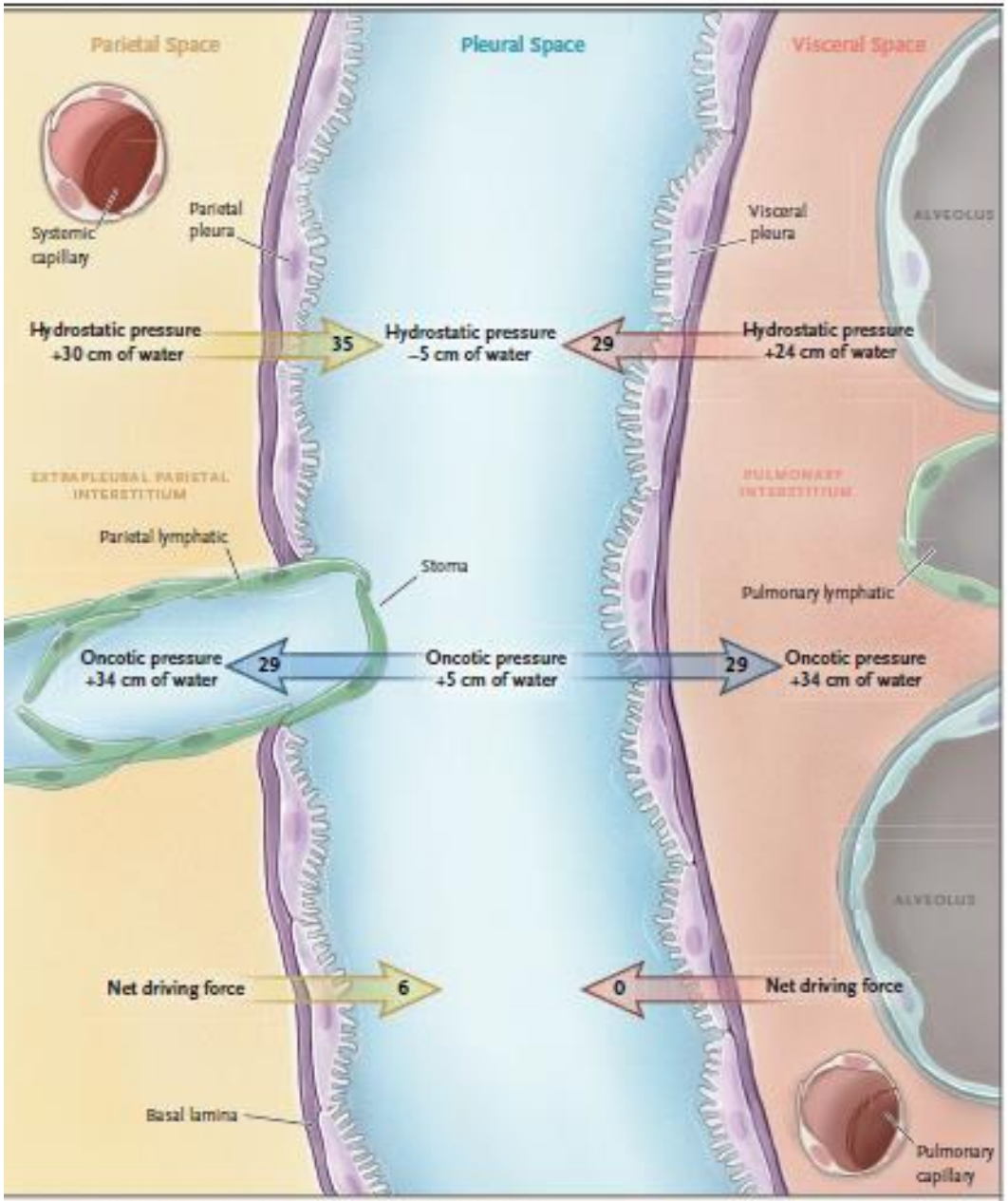


Figure 1. Balance of Forces Regulating Pleural Fluid Formation.

The amount of fluid in the pleural space is dependent on the balance of hydrostatic and oncotic pressures between the parietal and visceral pleura and the pleural space. Because hydrostatic pressures are higher on the parietal pleura than on the visceral pleura and the oncotic pressures are equivalent, pleural fluid is primarily produced from the parietal pleura. Likewise, the lymphatic vessels on the parietal pleura are responsible for the majority of pleural fluid resorption.



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Filteration

Drainage

Pathophysiology of Pleural Effusion

Potential causes of pleural effusion include:

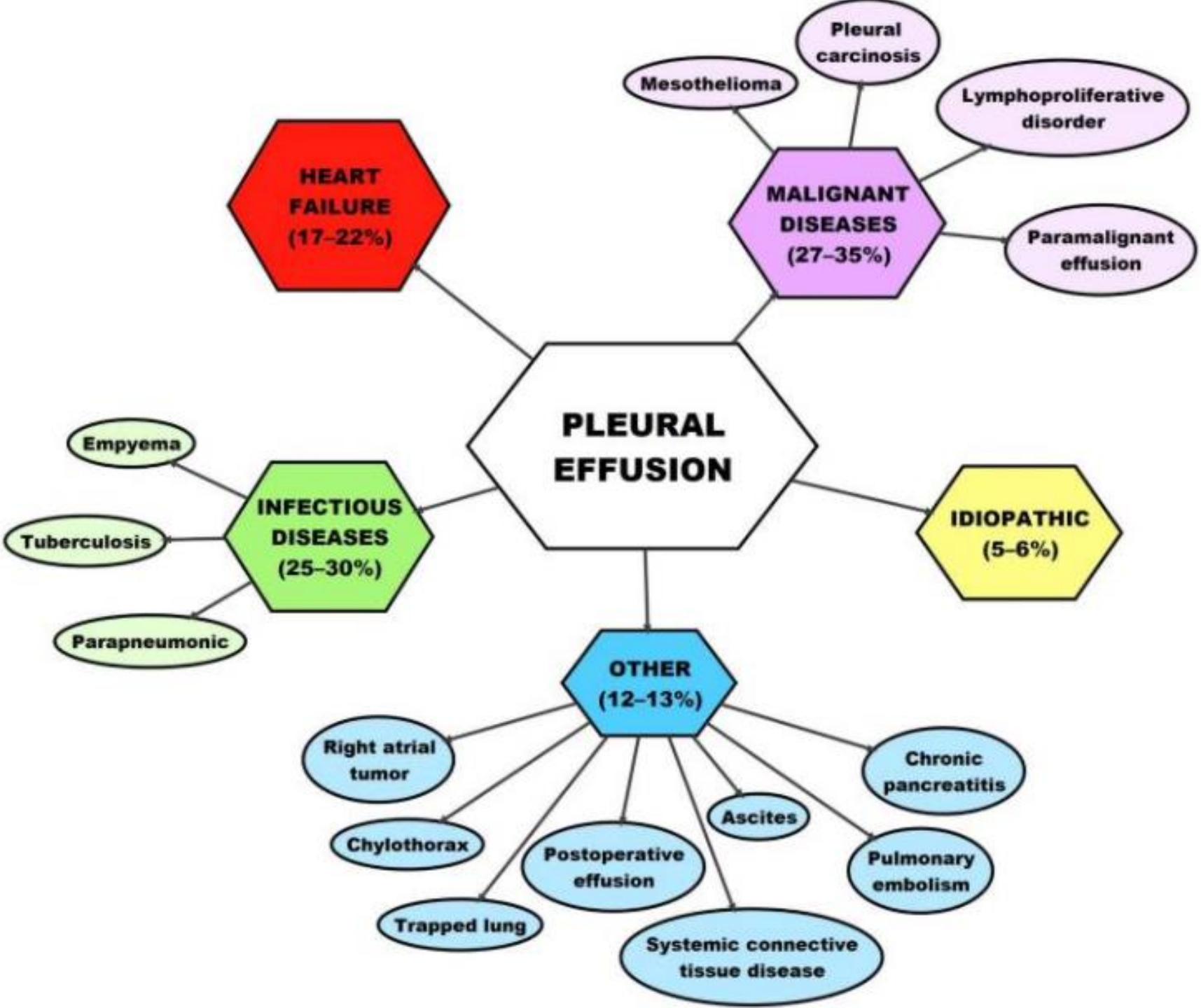
- **Increased hydrostatic pressure from heart failure.**
- **Decreased intravascular oncotic pressure from hypoalbuminemia.**
- **Decreased intrapleural pressure from atelectasis of the lung.**
- **Inefficient pleural lymphatic drainage because of obstructing mediastinal tumor.**
- **Increased capillary permeability from inflammation of or tumor implants on either the visceral or the parietal pleura.**

PATHOPHYSIOLOGY

- Normally between 1 and 2 liters of pleural fluid flow daily from the parietal through the visceral or mediastinal pleura into mediastinal lymphatics and ultimately the systemic venous circulation.
- The pleural space contains 5-10 mL of fluid.
- Increases in this amount constitute a pleural effusion, which can be classified as an exudate or a transudate.

PATHOPHYSIOLOGY OF PLEURAL EFFUSION

- ***Transudative effusion:*** is the result of increased formation or decreased absorption of pleural fluid caused by changes in the Starling forces.
- ***Exudative effusion :*** results from inflammatory or malignant alterations or diseases of the pleura itself. If analysis shows at least ***ONE*** of the following according to light's criteria:
  - ***Pleural fluid protein/serum protein > 0.5***
  - ***Pleural fluid LDH/serum LDH > 0.6***
  - ***Pleural fluid LDH >2/3 of the upper limit of normal for the serum LDH.***


PLEURAL EFFUSION
HEART FAILURE (17–22%)
MALIGNANT DISEASES (27–35%)
Pleural carcinosis
Mesothelioma
Lymphoproliferative disorder
Paramalignant effusion
INFECTIOUS DISEASES (25–30%)
Empyema
Tuberculosis
Parapneumonic
IDIOPATHIC (5–6%)
OTHER (12–13%)
Right atrial tumor
Chylothorax
Trapped lung
Postoperative effusion
Ascites
Systemic connective tissue disease
Chronic pancreatitis
Pulmonary embolism

Malignant Pleural Effusion (MPE)

- MPE complicates the care and worsens the quality of life of many cancer patients.
- The most typical pathogenetic situation is involvement of parietal or visceral pleura with metastatic deposits.
- Hypoalbuminemia, and metastatic tumor blockage of mediastinal lymphatic pathways can contribute to or even independently cause MPE

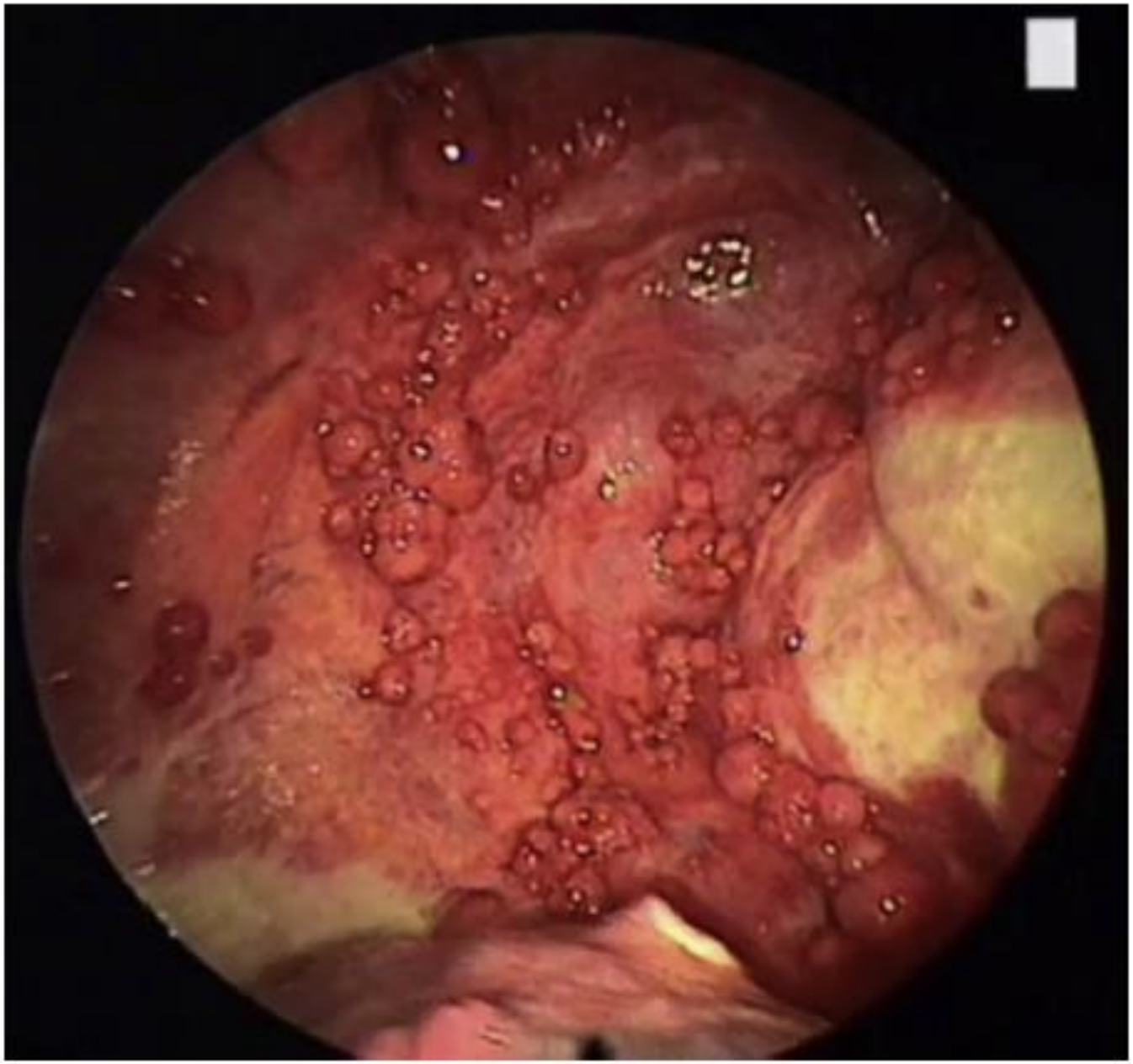
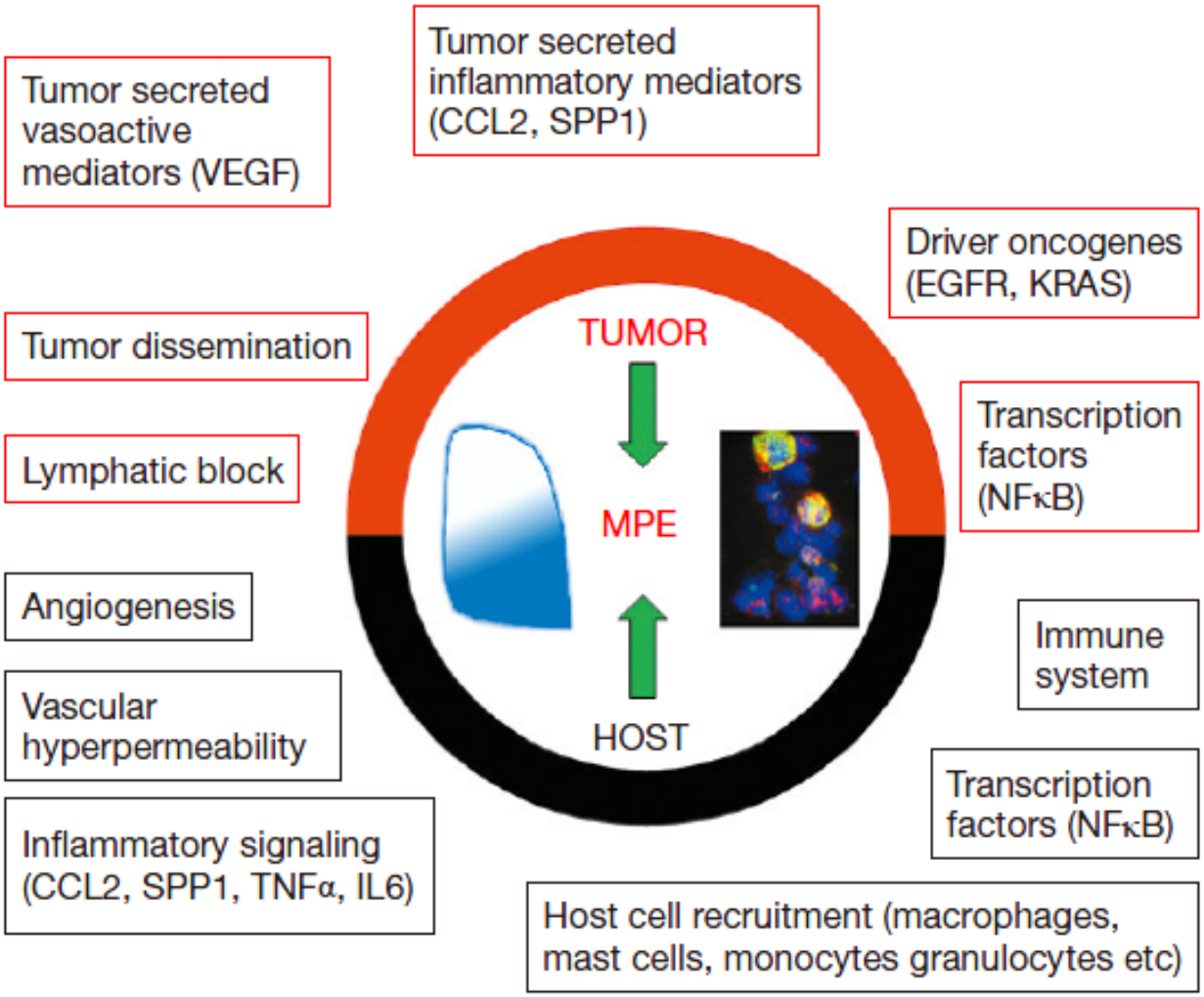


Fig. 6. Apical view showing pleural nodularity at thoracoscopy.

Pathophysiology of MPE



Malignant Pleural Effusion (MPE)

- 40% of significant pleural effusions are caused by malignancy with the most common types being lung cancer, breast cancer, and lymphoma.
- There is a difference in tumor types between men and women.
- The majority of malignant effusions are symptomatic. (>2/3)
- Massive pleural effusions are most commonly due to malignancy.

Etiology of Malignant Pleural Effusion

Men

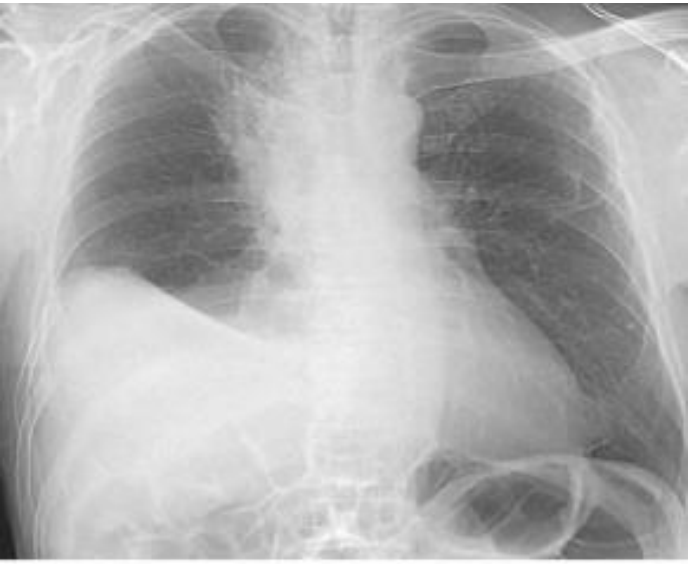
1° Tumor	Incidence (%)
Lung	49.1
Hematological	21.1
Gastrointestinal	7.0
Other	21.8

Women

1°Tumor	Incidence (%)
Breast	37.4
Genital tract	20.3
Lung	15.0
Hematological	8.0
Other	19.3

DIAGNOSIS

- Hx and P/E. (SOB is the most common symptom)
- Radiological evaluation (CXR, Thoracic US, Chest CT)
- Thoracentesis:
  - Chemical analysis: exudate vs transudate.
  - Cytological evaluation: +ve in 60%
- Closed pleural biopsy: +ve in 45%, diagnostic in 7%
- Video-Assisted Thoracic Surgery (VATS):
  - Sensitivety: 80-100%
  - Specificity: 100%



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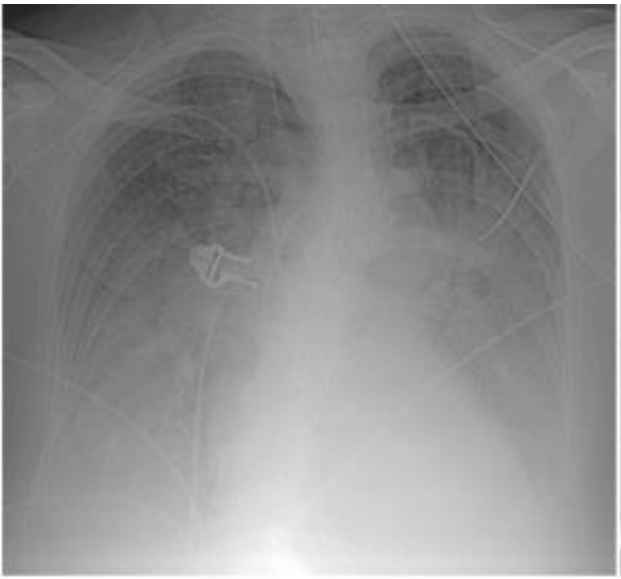

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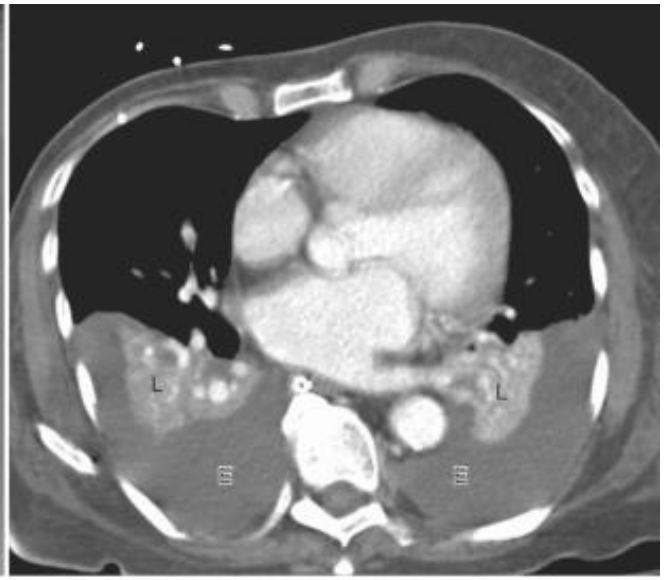

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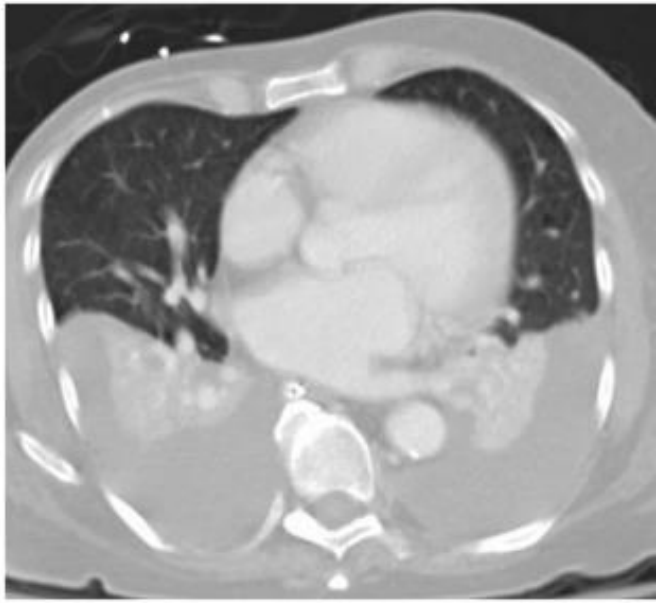

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TREATMENT

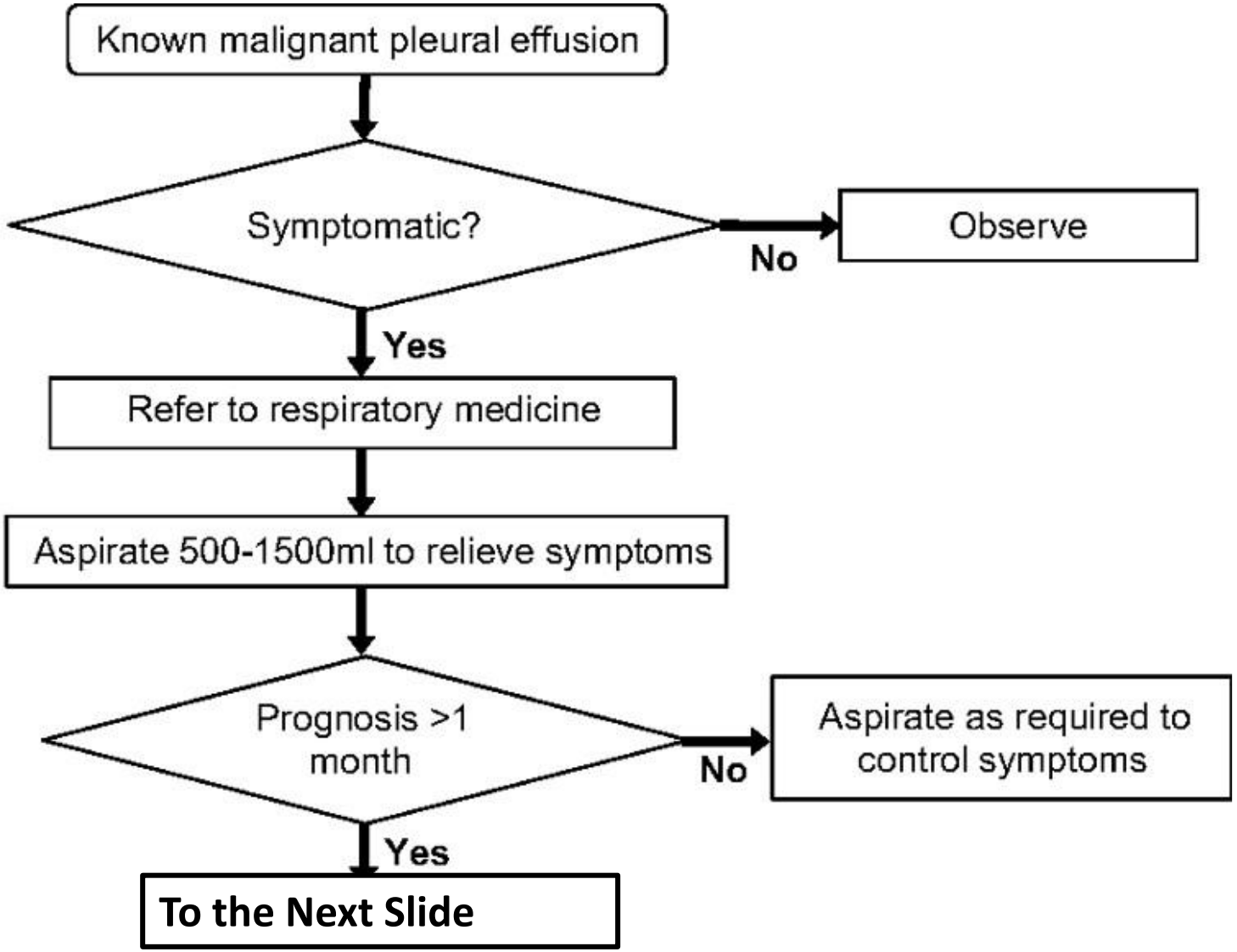
The aggressiveness with which treatment should be pursued is dependent upon:

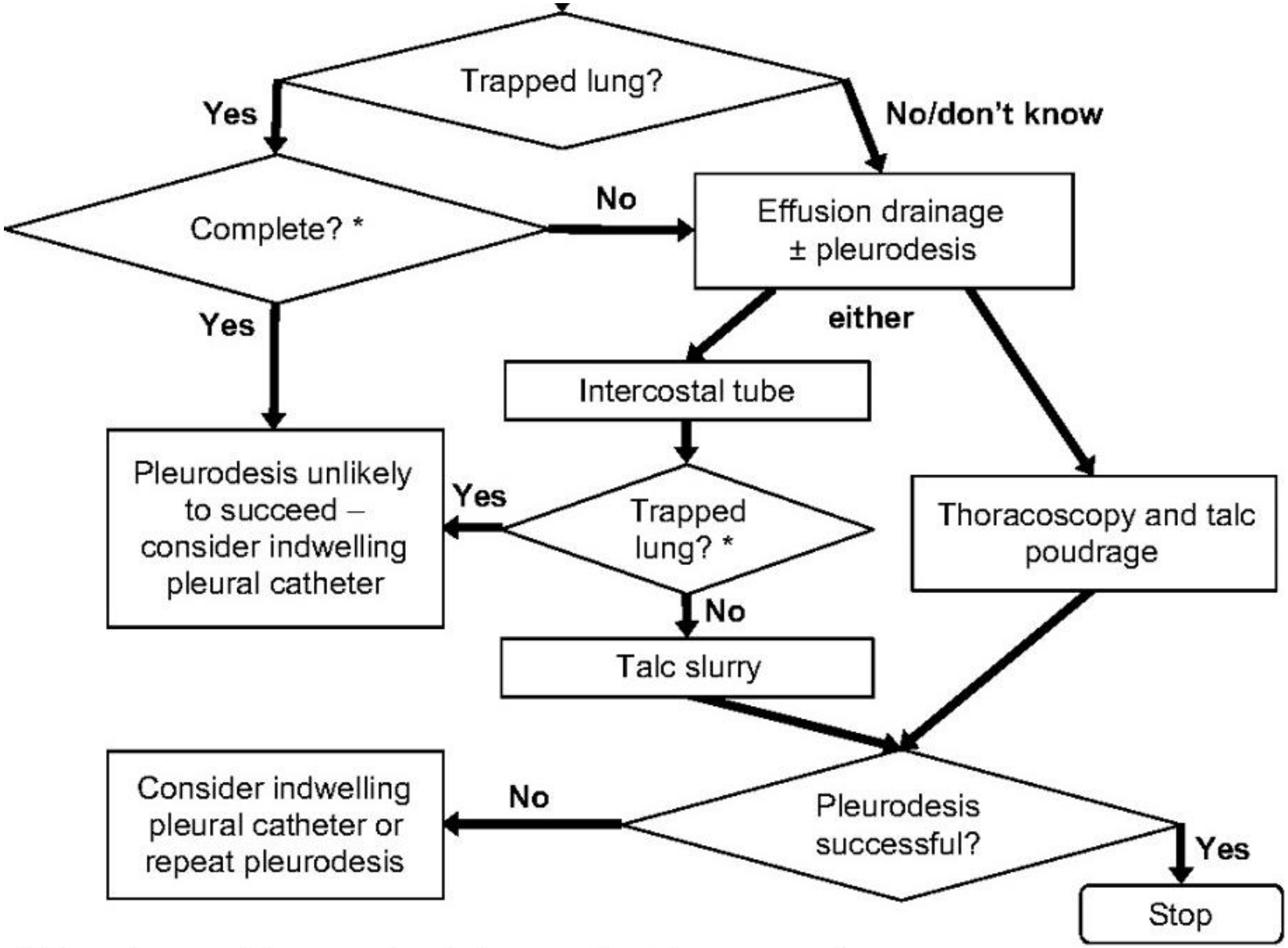
- **The extent to which a malignant pleural effusion produces respiratory symptoms**
- **The patient's performance status.**

Treatment Options:

- Observation
- Thoracentesis: pleural aspiration
- Thoracostomy and Pleurodesis (chemical e.g Talc, Bleomycin….etc.)
- VATS and Pleurodesis: chemical or mechanical
- Pleurectomy
- Long-term ambulatory indwelling pleural catheter drainage
- Shunts and Catheters (Pleuroperitoneal or pleurovenous)

MANAGEMENT ALGORITHM FOR MALIGNANT PLEURAL EFFUSION

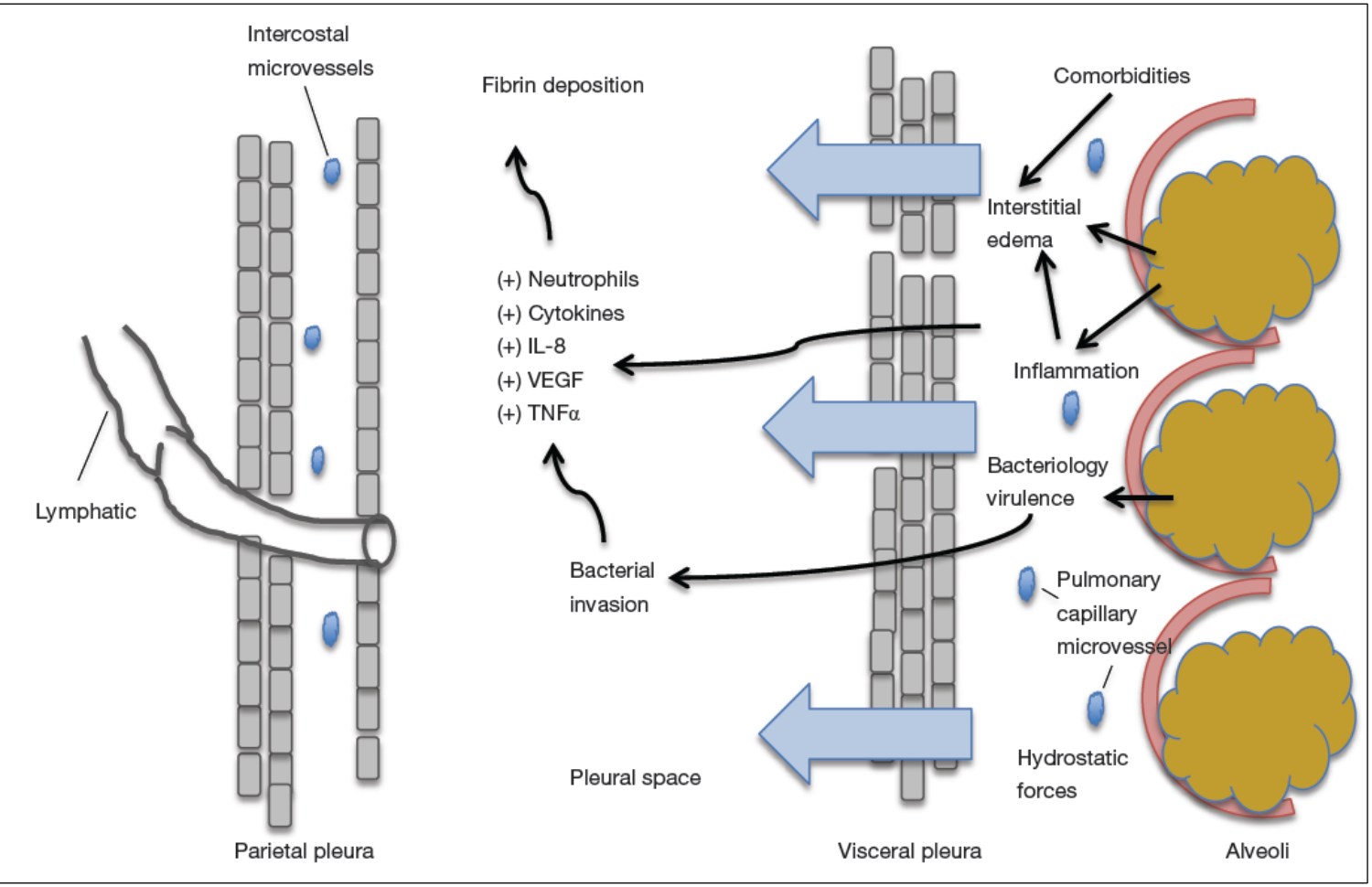




* There is no evidence as to what proportion of unapposed pleura prevents pleurodesis. We suggest that <50% pleural apposition is unlikely to lead to successful pleurodesis

PLEURAL SPACE INFECTIONS

- A parapneumonic effusion PPE is any pleural effusion associated with pneumonia.
- PPE can be classified as:
  - ***Simple effusions***: **uninfected, free-flowing fluid collections**
  - ***Complicated effusions***: **early infected fluid collections**
  - ***Empyemas:*** **well-established collections of pus within the pleural cavity.**


Intercostal
microvessels
Fibrin deposition
Comorbidities
Interstitial
edema
(+) Neutrophils
(+) Cytokines
(+) IL-8
(+) VEGF
(+) TNFα
Inflammation
Lymphatic
Bacteriology
virulence
Bacterial
invasion
Pulmonary
capillary
microvessel
Hydrostatic
forces
Pleural space
Parietal pleura
Visceral pleura
Alveoli

PLEURAL SPACE INFECTIONS

- PPE develops in 36 - 57% of patients with pneumonia.
- <5% of these PPE progress to empyema.
- *Causes of empyema thoracis:*
  - Parapneumonic effusion (40-60%)
  - Prior thoracic surgery (15-30%)
  - Thoracic trauma (10%)

BACTERIOLOGY

	Common organisms
Community-acquired	Streptococcus spp. (~52%)
	S milleri, S pneumonia, S intermedius
	Staphylococcus aureus (11%)
	Gram-negative aerobes (9%)
	Enterobacteriaceae, Escherichia coli
	Anaerobes (20%)
	▶ Fusobacterium spp.
	▶ Bacteroides spp.
	▶ Peptostreptococcus spp.
	▶ Mixed
Hospital-acquired	Staphylococci
	▶ Methicillin-resistant S aureus (MRSA) (25%)
	▶ S aureus (10%)
	Gram-negative aerobes (17%)
	E coli, Pseudomonas aeruginosa…etc
	Anaerobes (8%)

PLEURAL SPACE INFECTIONS

- The morbidity and mortality of patients with PPE is higher than in patients with pneumonia alone due in part to the need for management of the PPE.
- ***Prognosis is worse in:***
  - The elderly
  - Coexistent cardiac, pulmonary, or renal disease.
  - Patients with hospital-acquired or culture-positive empyema, especially those involving gram-negative bacteria or multiple pathogens.

CLINICAL PRESENTATION

- Can range from an absence of symptoms to a severe febrile illness with toxemia and shock.
- Include: fever, dyspnea, chest pain, and cough with mucopurulent sputum.
- ***Aerobic organisms*** usually manifest acutely, whereas in **anaerobic infections** the course is usually more protracted.
- Failure of response or worsening of the clinical condition despite adequate antibiotic therapy.
- A sudden expectoration of a large amount of purulent sputum or hemoptysis.

CLINICAL PRESENTATION

Physical examination often reveals:

- Decreased breaths sounds
- Dull percussion note.
- Restricted respiratory excursions.
- Rales or crepitations ,pleural friction rub
- With chronicity, an empyema can erode the chest wall and present as a spontaneously draining subcutaneous abscess known as empyema necessitatis.
- Other manifestations of Chronic empyema includes chondritis and osteomyelitis of the ribs, pericarditis, mediastinal and vertebral abscesses, disseminated infection, and multiorgan failure.

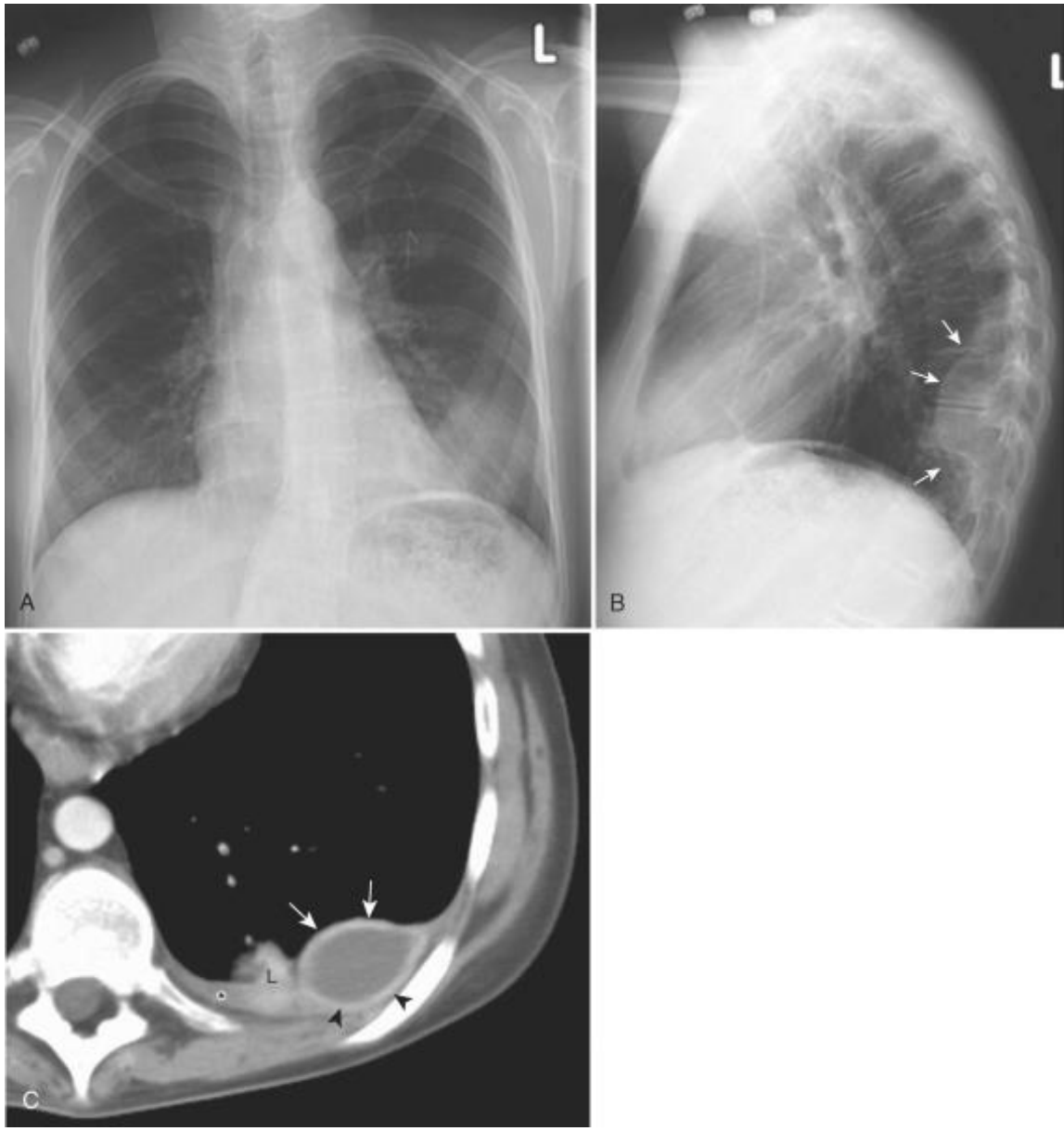
STAGES OF EMPYEMA THORACIS

- *Stage I:* exudative stage, “simple PPE”
- *Stage II:* fibrinopurulent or transitional stage, is characterized by infection of the pleural fluid. The fluid is turbid and contains bacteria and cellular debris “complicated PPE”
- *Stage III:* chronic or organizing stage, fibroblasts migrate and produce an inelastic membrane called the pleural peel or cortex, entrapping the lung and rendering it essentially functionless. ” complex empyema”

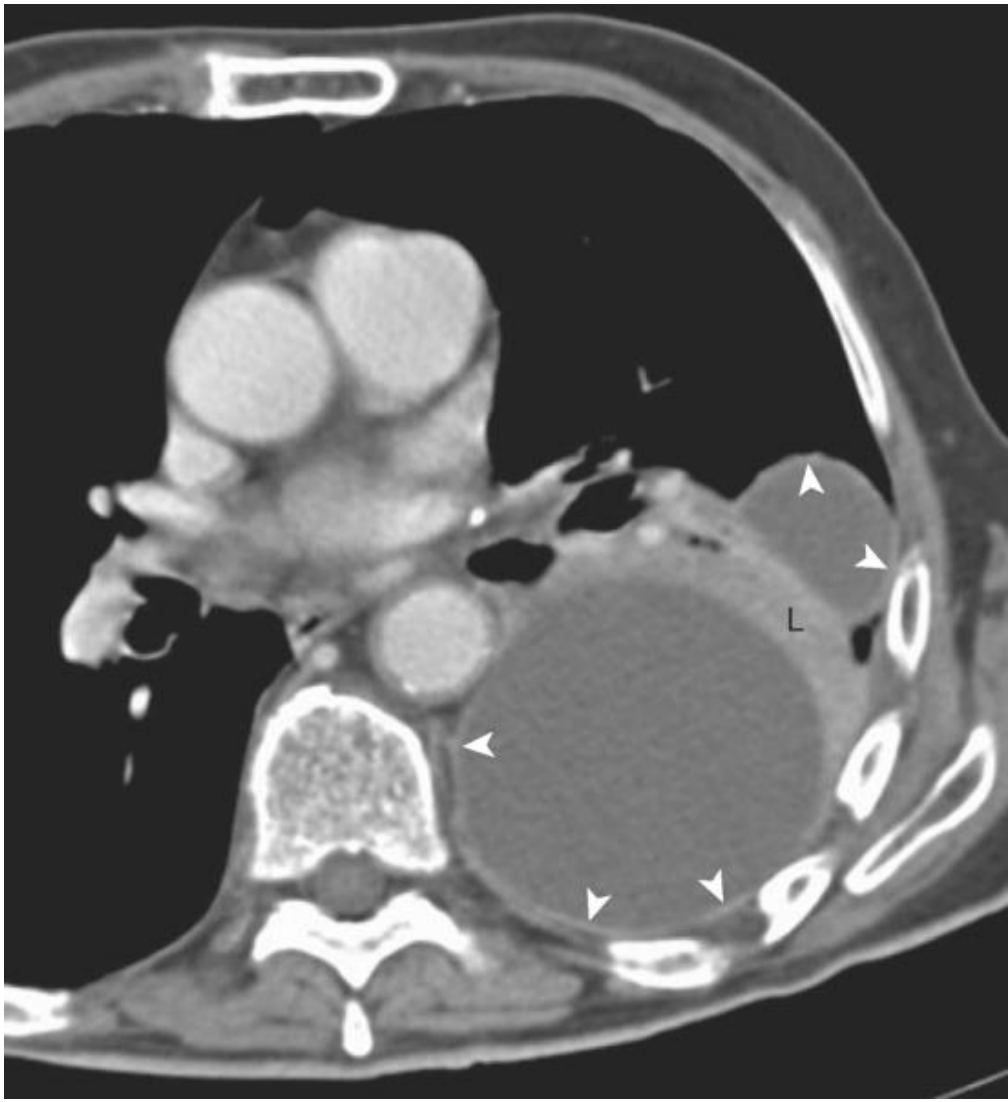
DIAGNOSIS

- Clinical findings
- Imaging studies and Lab studies:
  - ↑WBC and CRP
  - CXR
  - Thoracic US
  - Chest CT
  - Thoracentesis (pH, Ptn, Glu, LDH)
- Thoracoscopy
- Video-Assisted Thoracoscpic Surgery “VATS”

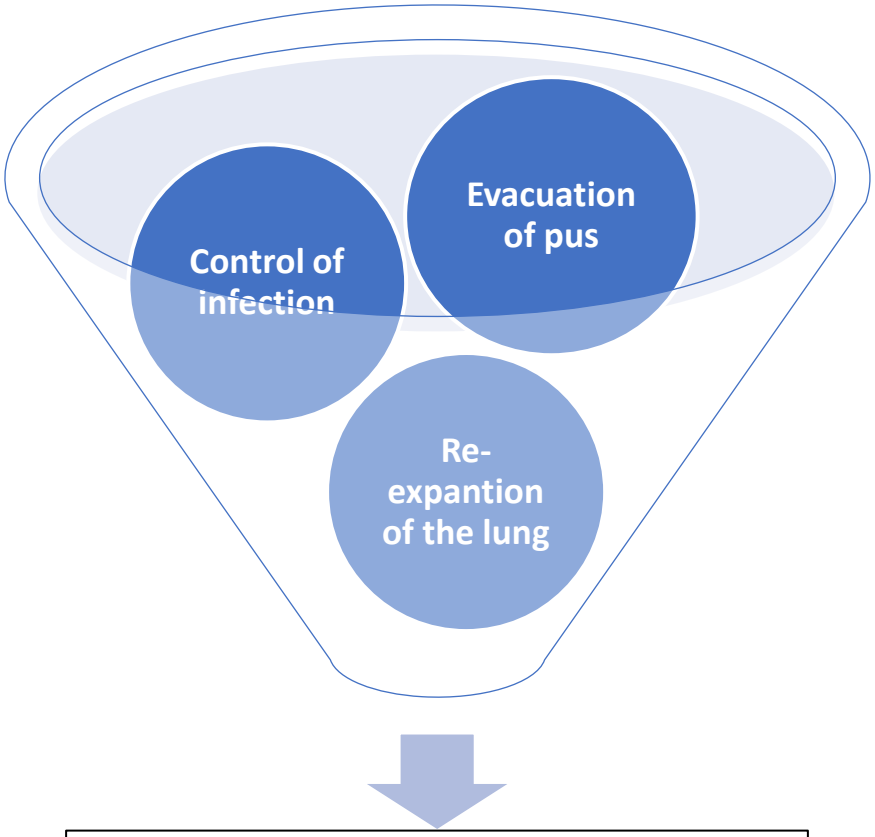
A:PA X-Ray reveals a poorly defined opacity projecting over the left lung base.
B: Lateral radiograph shows this to be a posterior, pleural-based, lenticular opacity suspicious for a pleural process.
C: CT scan shows a loculated pleural fluid collection with thickening and enhancement of visceral and parietal pleura (split pleura sign), representing an empyema. Note enhancing adjacent atelectatic lung and a small amount of free pleural fluid.



CT scan in a patient with an empyema shows a multiloculated fluid collection with pleural enhancement (*arrowheads)* compressing the adjacent enhancing, atelectatic lung.



AIM OF THE TREATMENT

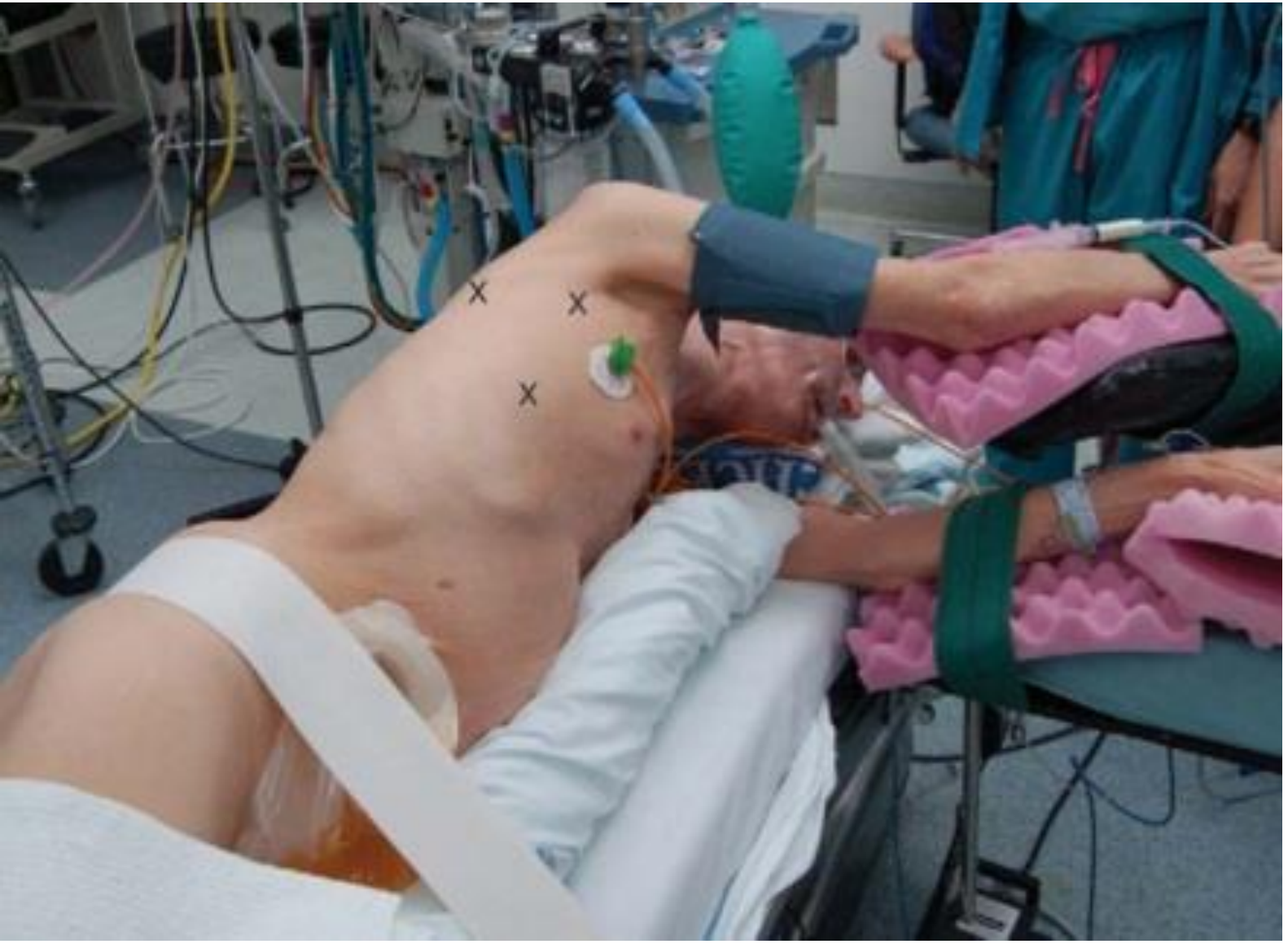


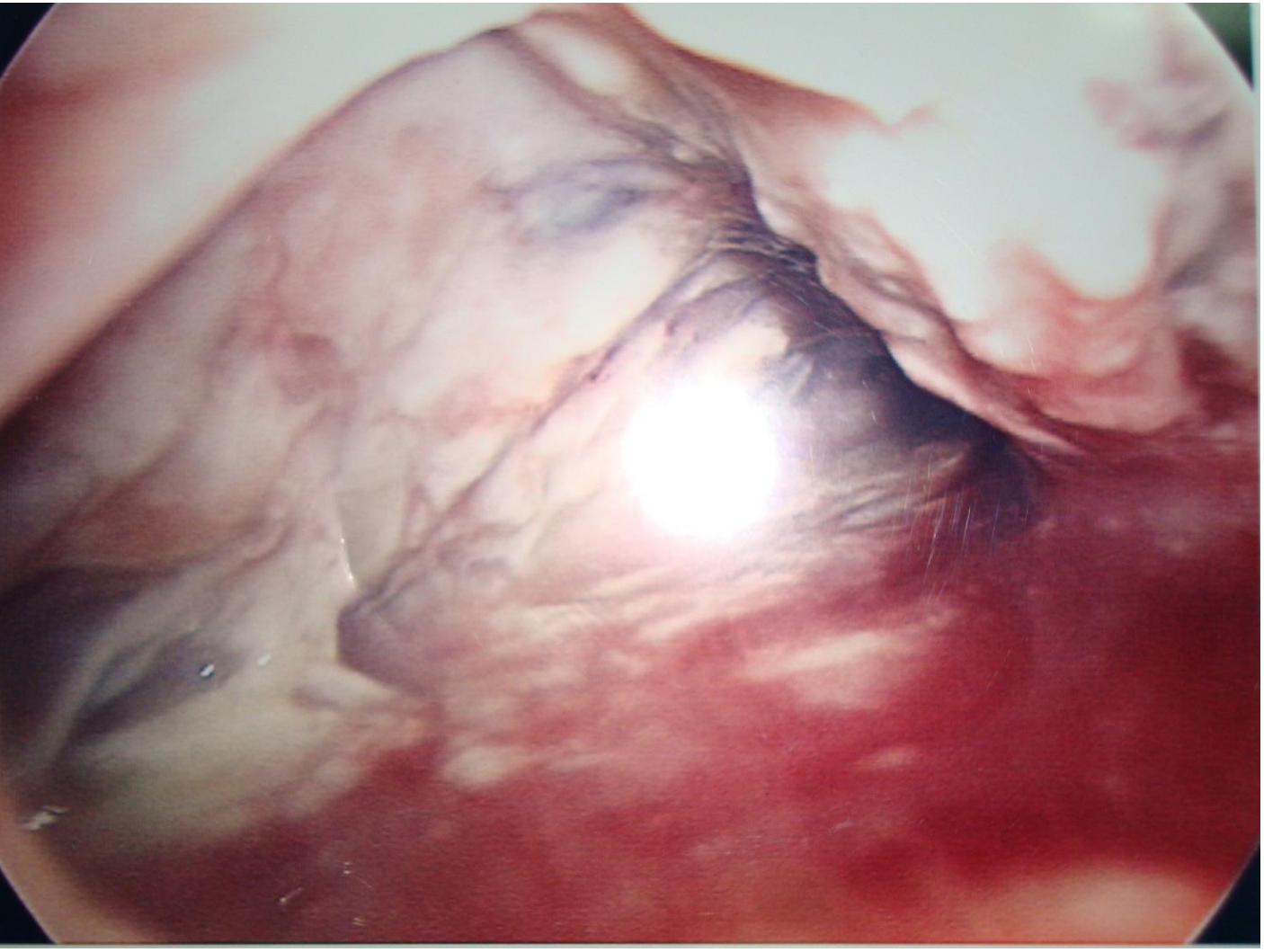
TREATMENT

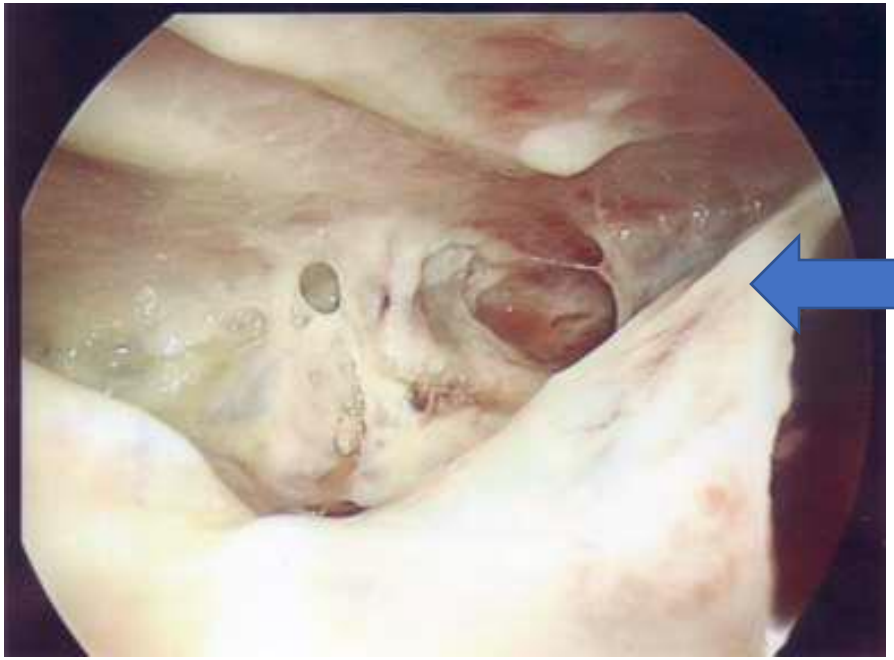
- **Stage I**
  - Antibiotics
  - Antibiotics +Thoracentesis
- **Stage II**
  - Antibiotics +Thoracostomy tube
  - Antibiotics +Thoracostomy tube ± fibrinolytics therapy ± VATS
- **Stage III**
  - VATS or Thoracotomy + Antibiotics
  - Decortication + Antibiotics

Classification and Treatment Scheme for Parapneumonic Effusions and Empyema

Parapneumonic effusion Class	Characteristics and Treatment
Class I – Non significant	Small< 10 mm thick on decubitus x-ray No thoracentesis indicated
Class II – Typical parapneumonic	>10 mm thick Glucose >40 mg/dl, pH> 7.2 Gram stain and culture negative Antibiotics alone
Class III – Borderline complicated	7.00<pH<7.20 and/or LDH> 1000 and Glucose>40 mg/dl Gram stain and culture negative Antibiotics plus serial thoracentesis
Class IV – Simple complicated	pH<7.00 and/or Glucose <40mg/dl and/or Gram stain or culture positive Not loculated not frank pus Tube thoracostomy plus antibiotics
Class V – Complex complicated	pH<7.00 and/or Glucose <40mg/dl and/or Gram stain or culture positive Multiloculated Tube thoracostomy plus fibrinolytics (rarely require thoracoscopy or decortication)
Class VI – Simple empyema	Frank pus present Single locule or free flowing Tube thoracostomy± decortication
Class VII – Complex empyema	Frank pus present Multiple locules Tube thoracostomy + thrombolytics Often requires thoracoscopy or decortication

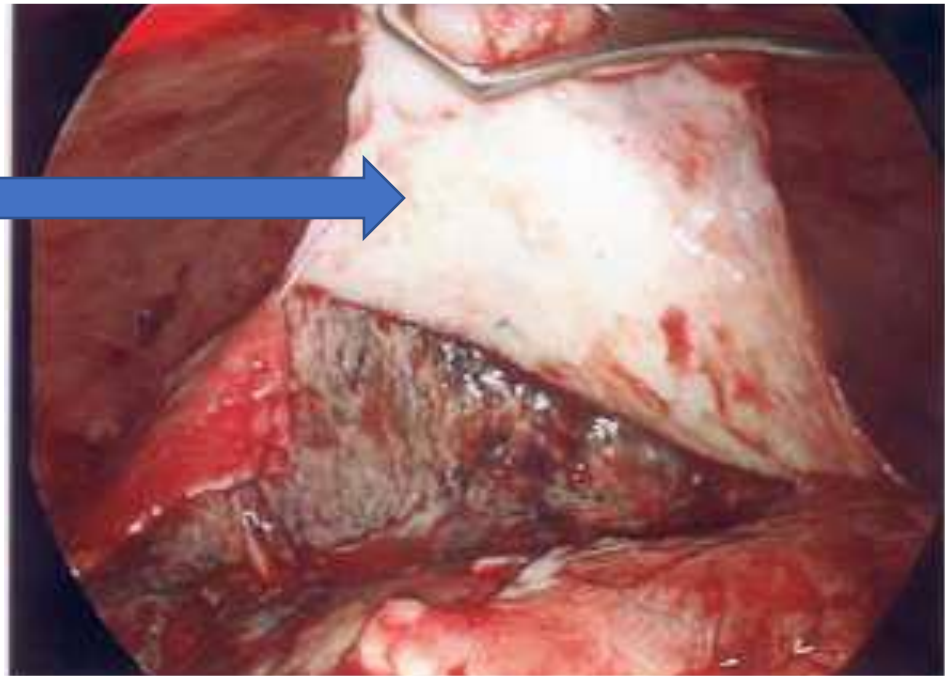






Fibro-purulent material

Fibrous peel that prevent lung expansion



CHYLOTHORAX

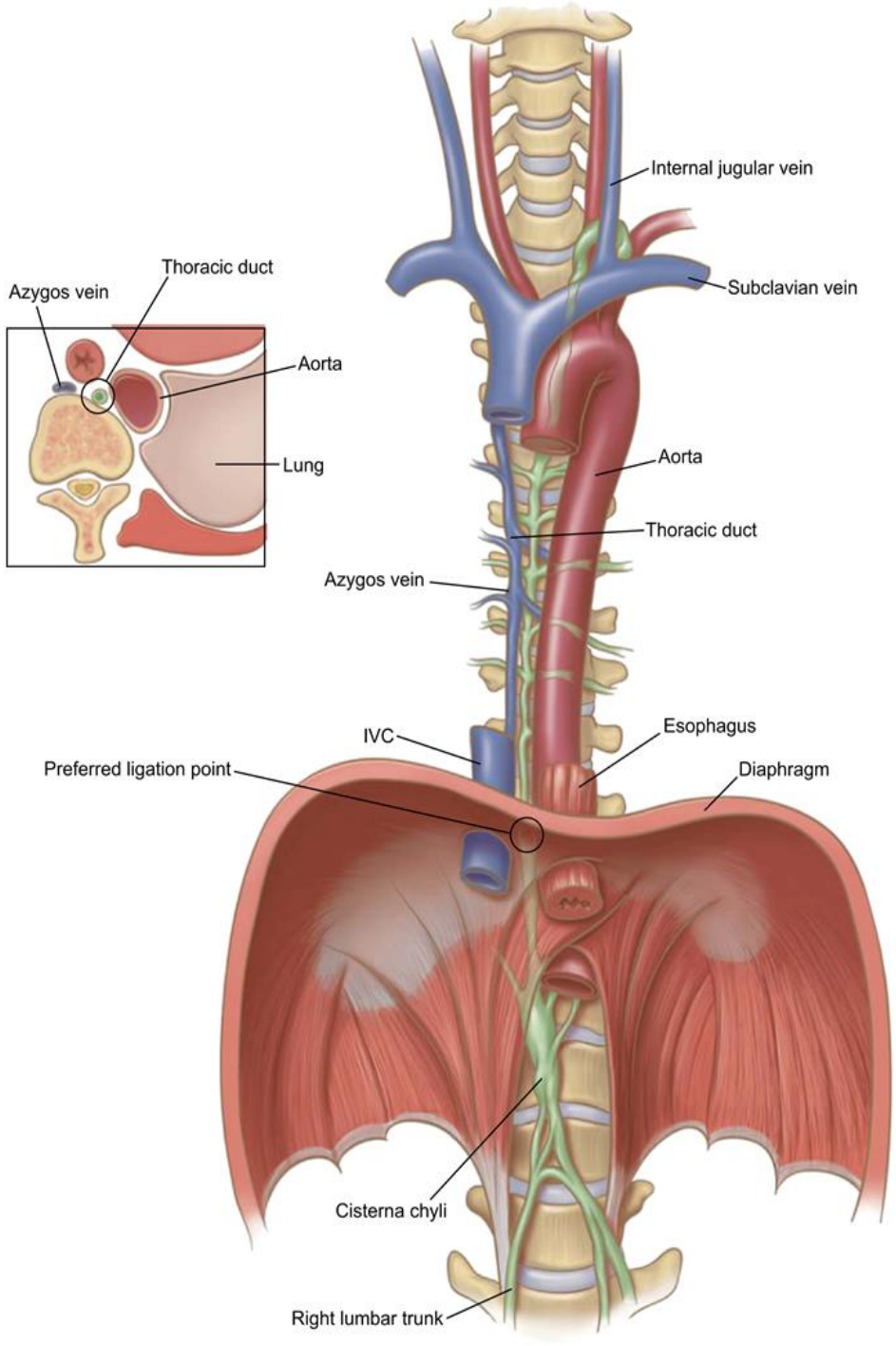
- Accumulation of lymph within the pleural space.
- The incidence may be increasing because the number of thoracic surgical procedures and chest traumas continues to rise.
- Characteristically is milky white fluid that contains a high concentration of emulsified fats (triglycerides, chylomicrons) and a lymphocytic predominance on cell count.

Chylothorax

- Chylothorax occurs when the contents of the thoracic duct empty into the pleural space.

- Due to the location of the thoracic duct, the right side is more common than the left, accounting for two-thirds of the total cases.

ANATOMY


Azygos vein
Thoracic duct
Aorta
Lung
Internal jugular vein
Subclavian vein
Aorta
Thoracic duct
Azygos vein
IVC
Esophagus
Preferred ligation point
Diaphragm
Cisterna chyli
Right lumbar trunk

CAUSES

- ***Acquired:***
  1. **Traumatic: (chest and neck); penetrating and blunt**
  2. **Iatrogenic: post surgical, post venous catheterization.**
  3. **Neoplasms: lymphoma, lung cancer, esophageal cancer**
  4. **Infectious: tuberculous lymphadenitis, mediastinitis**
  5. **Others**
- ***Congenital***

DIAGNOSIS

- Symptoms of chylothorax may:
  - Mimic the effects of a pleural effusion.
  - Be attributable to underlying disease (infectious or neoplastic causes).
  - Be the result of chronic metabolic (nutritional and immunologic) effects of a thoracic duct leak (loss of fat, protein, antibodies, and fat-soluble vitamins).
- Losses in fluid volume may be large (>3 L/day) and produce hemodynamic instability.
- Pleural Triglyceride level is >110 mg/100 mL

TREATMENT

- Tube thoracostomy drainage with complete lung re-expansion (1-2 wks)
- Supportive measures such as a low-fat or fat-free diet supplemented by medium-chain triglycerides and aggressive fluid, electrolyte, and nutritional replacement or correction.
- If the cause is malignancy, 1° treatment of the neoplasm may be necessary.
- Radiation therapy to the mediastinum has been useful in managing chylothorax 2° to lymphoma.

TREATMENT

- If the chylous effusion has not responded to conservative management, surgical intervention is indicated.
- ***The most common procedures are:***
  - ➢ Ligation of the thoracic duct:

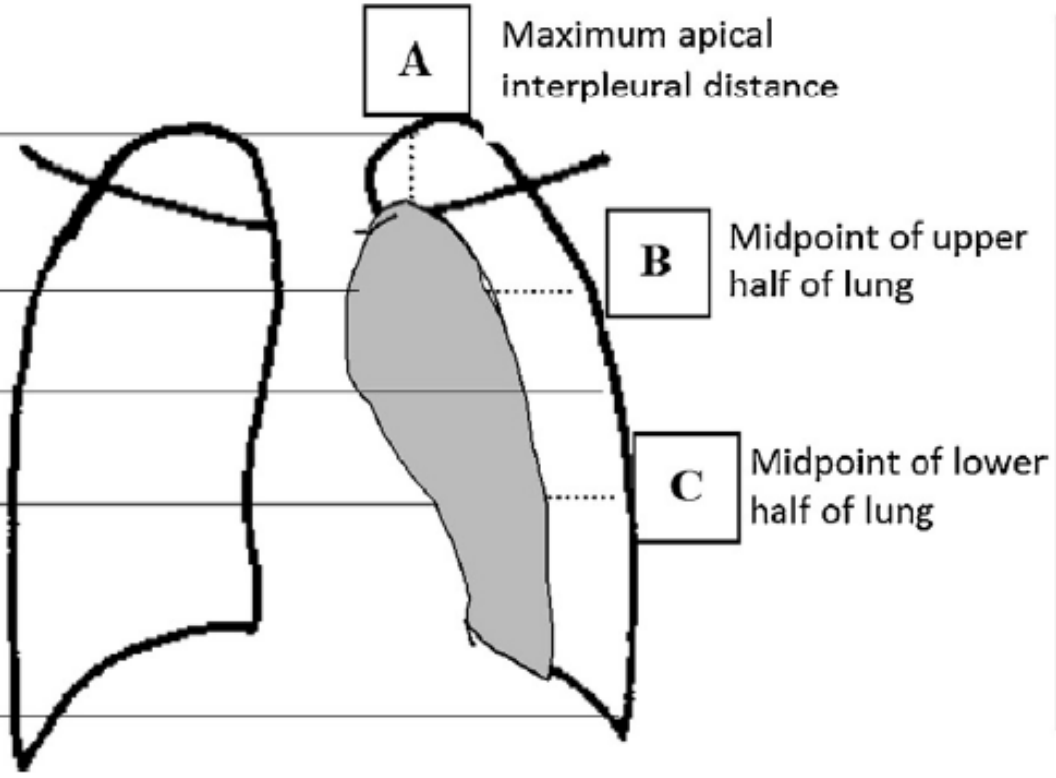
    Mass ligation of tissue at the diaphragmatic hiatus or direct closure of the duct injury.
  - ➢ Pleurectomy and pleurodesis
  - ➢ Thoracic duct obliteration through cisterna chyli cannulation or fenestration

PNEUMOTHORAX

- Accumulation of air within the pleural space.
- Pneumothoraces may be spontaneous or occur secondary to a traumatic, surgical, therapeutic, or disease-related event.
- A pneumothorax compresses lung tissue and reduces pulmonary compliance, ventilatory volumes, and diffusing capacity.
- These pathophysiologic consequences depend primarily on the size of the pneumothorax and condition of the underlying lung.

The size of pneumothorax

1. Measure the pneumothorax rim in centimeters in A,B,C
2. SID (sum interpleural distance) = A+B+C.
3. Read pneumothorax size from table PA or table AP



PA-projection (Rhea)
Formula:
Pn% = 4,95 + (8,8x SID/3)

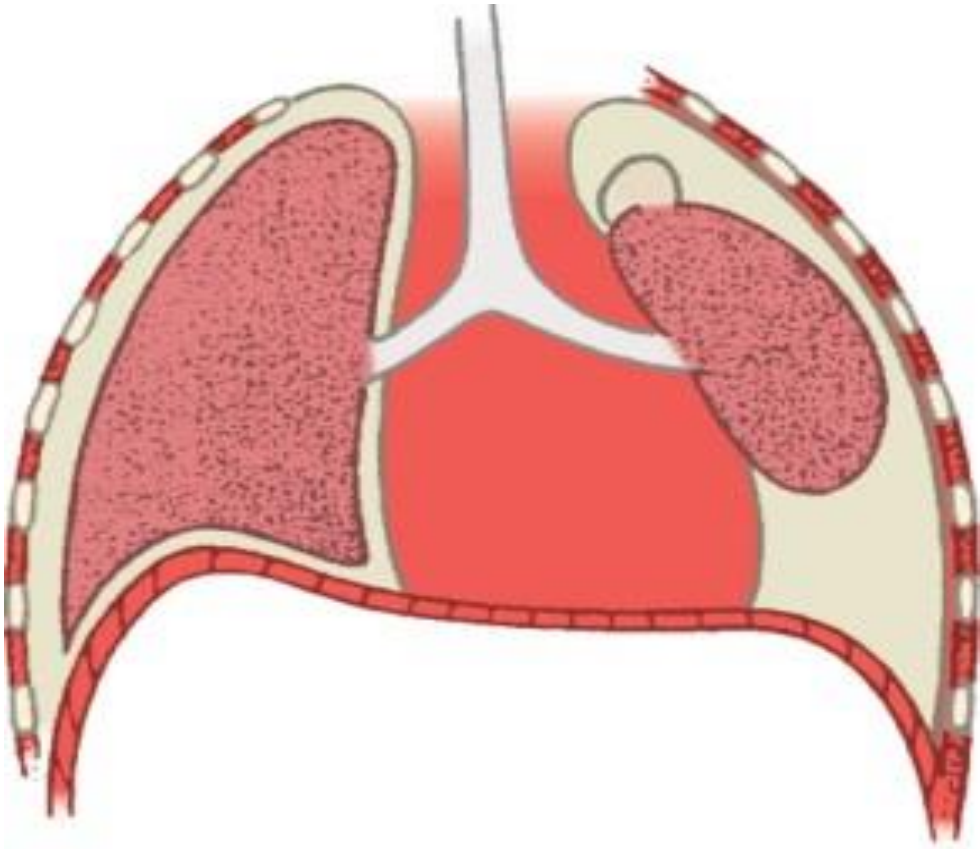
SID	2 cm equals	11 %
SID	4 cm ------	17 %
SID	**5,1 cm ------**	**20 %**
SID	6 cm ------	23 %
SID	**8,5 cm ------**	**30 %**
SID	10 cm ------	34 %
SID	12 cm ------	40 %
SID	14 cm ------	46 %
SID	16 cm ------	52 %
SID	18 cm ------	58 %
SID	20 cm ------	64 %
SID	22 cm ------	70 %
SID	24 cm ------	75 %
SID	26 cm ------	81 %
SID	28 cm ------	87 %
SID	30 cm ------	93 %
SID	32 cm ------	99 %

AP-projection (Choi)
Formula:
Pn% = 9 + (10x SID/3)

SID	2 cm equals	16 %
SID	**3,3 cm ------**	**20 %**
SID	4 cm ------	22 %
SID	5,1cm ------	26 %
SID	**6,3 cm ------**	**30 %**
SID	8 cm ------	35 %
SID	10 cm ------	42 %
SID	12 cm ------	49 %
SID	14 cm ------	56 %
SID	16 cm ------	62 %
SID	18 cm ------	69 %
SID	20 cm ------	75 %
SID	22 cm ------	82 %
SID	24 cm ------	89 %
SID	26 cm ------	96 %
SID	27 cm ------	99 %

Fig. 1. Tool for estimation of pneumothorax size in percent.

Some of the physiologic characteristics of simple spontaneous pneumothorax

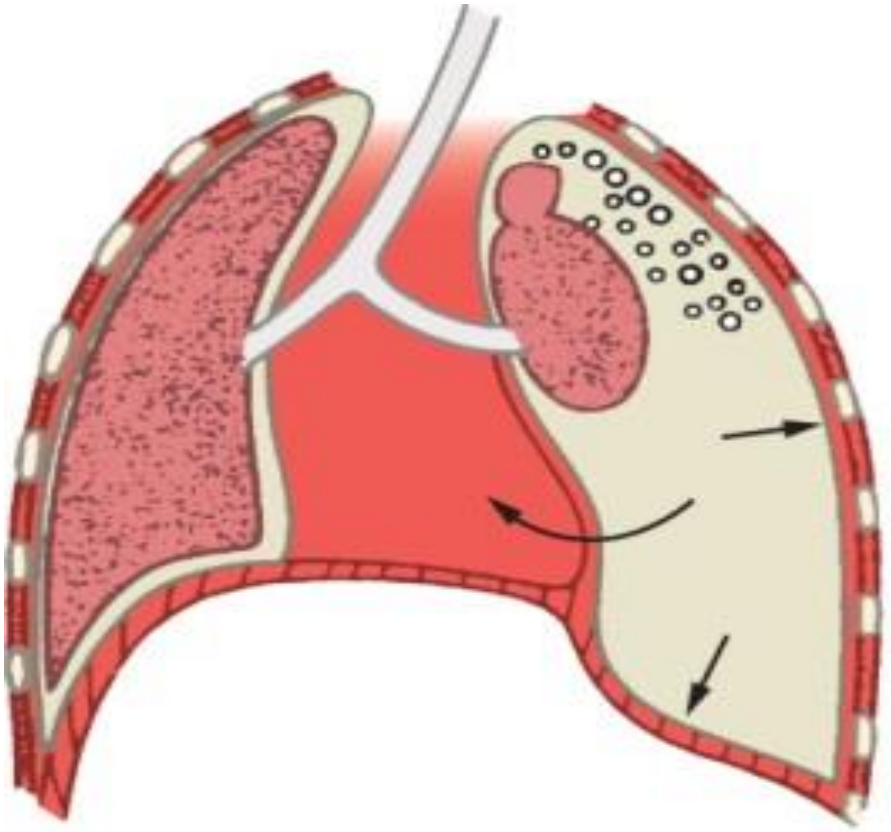


↑ Air flow until no pressure difference
↓ Apex to base pressure gradient
↓ Lung compliance
↓ Functional residual capacity
↓ Ventilation
↓ Oxygenation
Slight shunt

TENSION PNEUMOTHORAX

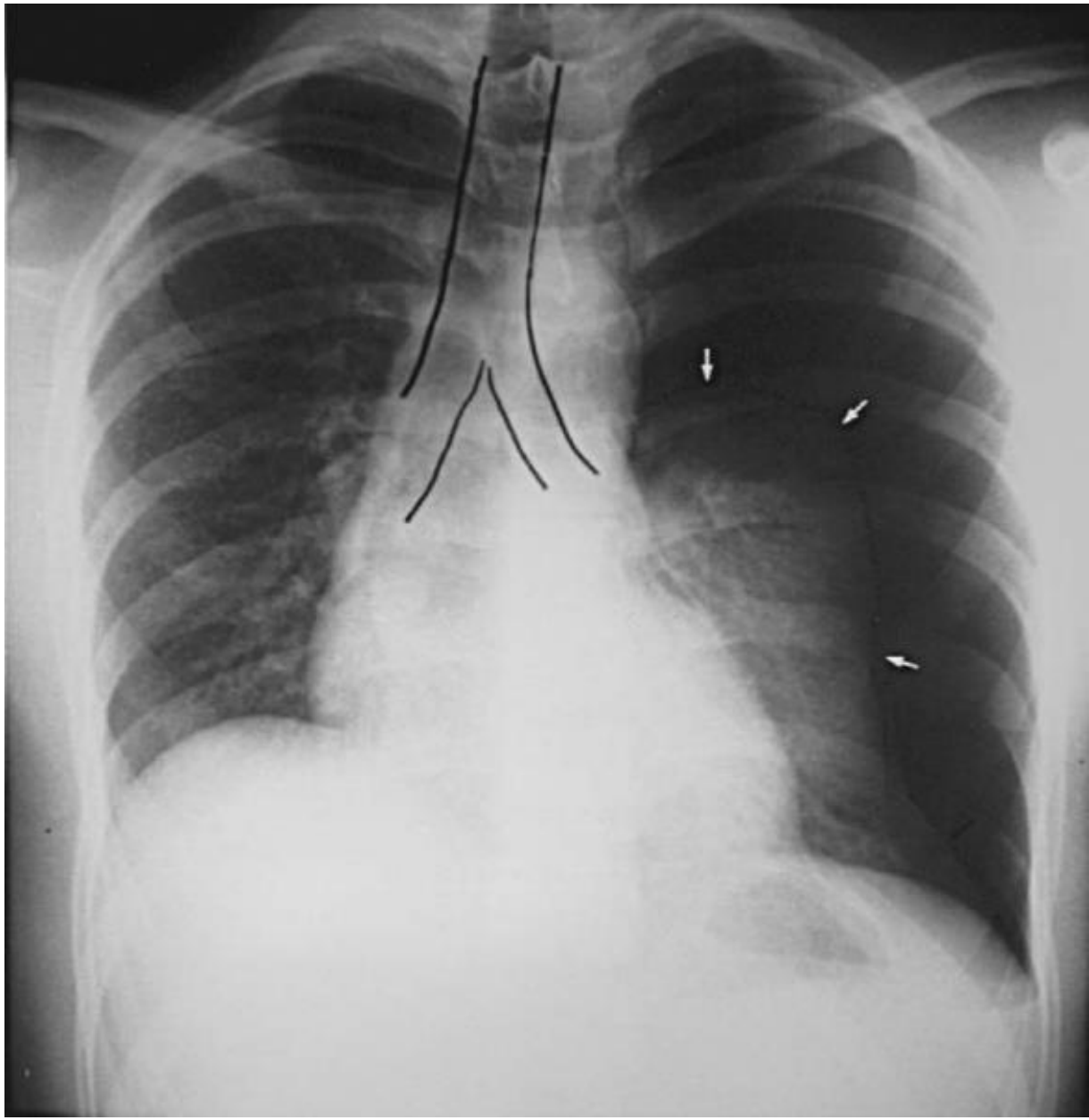
- If air enters the pleural space repeatedly (as with inspiration) and is unable to escape, positive pressure develops in the pleural space, causing compression or collapse of the entire lung, shifting of the mediastinum and heart away from the pneumothorax, and severe respiratory compromise with hemodynamic collapse.
- This situation is called a *tension pneumothorax* and requires immediate decompressive treatment. It may be the sequela of a pneumothorax from many causes.

Physiologic characteristics of a *tension pneumothorax*



↑ Continuous air flow (one-way valve)
↑ Intrapleural pressure
↑ Mediastinal shift (alteration of lung mechanics)
↓ Ventilation
↑ Shunt
↓ Oxygenation
↑ Cardiac stroke volume
↑ Heart rate

Chest radiograph showing a tension pneumothorax with mediastinal shift and deviated trachea. The trachea is outlined. *Arrows* indicate location of visceral pleura.



CLASSIFICATIONS OF PNEUMOTHORAX

- Spontaneous
  - **Primary**
  - **Secondary: COPD, Bullous disease, cystic fibrosis, *Pneumocystis*-related, congenital cysts, idiopathic pulmonary fibrosis (IPF), and pulmonary embolism.**
  - **Catamenial**
  - **Neonatal**
- Traumatic: Penetrating, Blunt
- Iatrogenic: Mechanical ventilation, thoracentesis, lung biopsy, venous catheterization and postsurgical
- Other: Esophageal perforation

CLINICAL MANIFESTATIONS

- Chest pain: It is often sharp and pleuritic and may lead to severe respiratory embarrassment or become dull and persistent.
- Dyspnea
- Nonproductive cough
- Orthopnea.
- Patients are often tall, thin men from 25 to 40 years of age.

CLINICAL MANIFESTATIONS

- Physical findings may be normal if the pneumothorax is less than 25%.
- Diminished chest excursion
- Hyperresonance on percussion of the affected side.
- Breath sounds are diminished to absent.
- Subcutaneous emphysema may be palpated.
- Signs of mediastinal shift.

Subcutaneous emphysema

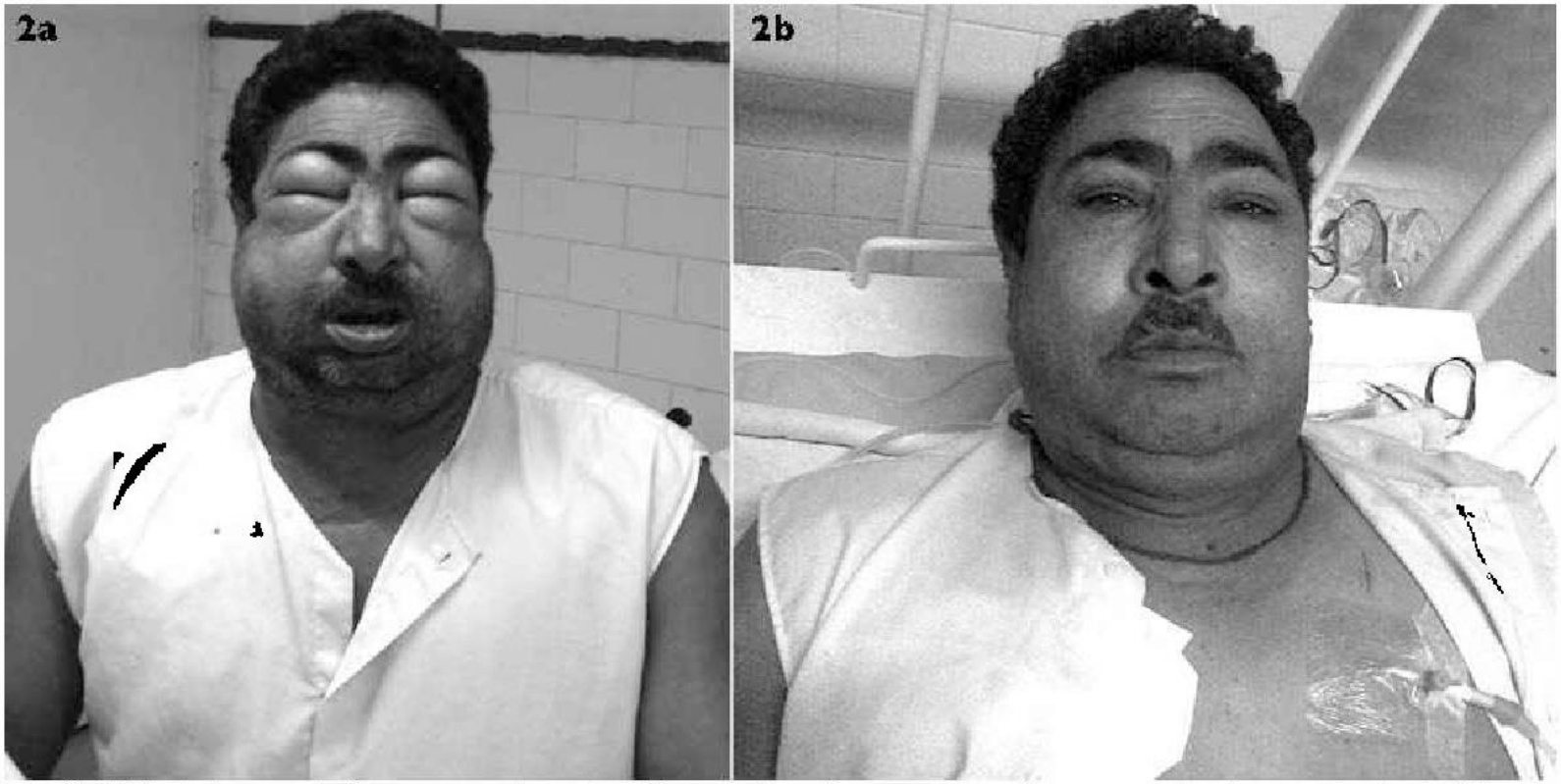


Fig. 2 Clinical photograph shows our patient with (a) extensive subcutaneous emphysema causing closure of palpebral fissure;

DIAGNOSIS

- History and physical examination
- CXR: PA (end inspiratory) and Lateral
- CT-Chest

TREATMENT

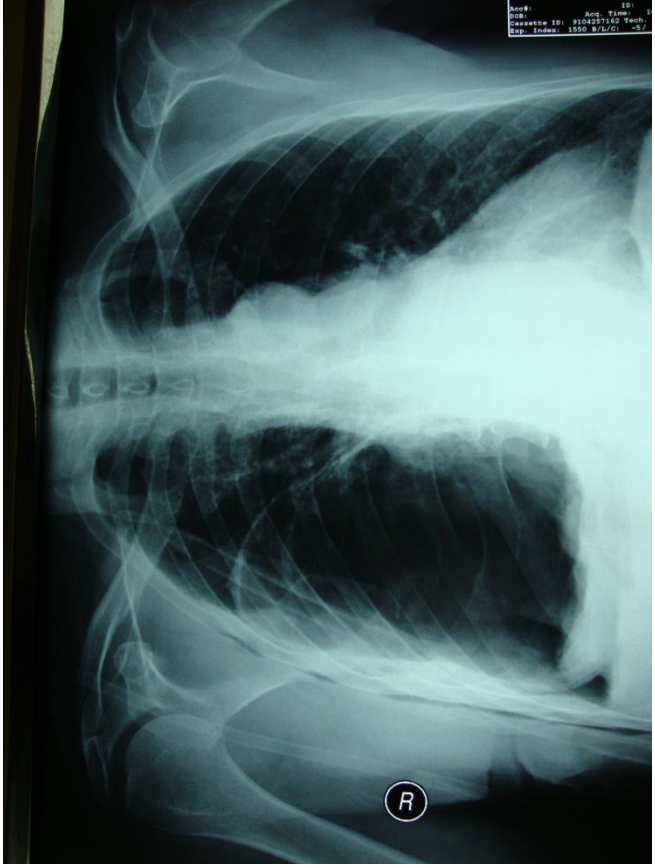
- Depends on the size, associated symptoms, and pulmonary disease history.
- Smoking cessation is advocated for all smokers.

Small pneumothoraces (<20%) that are stable may be monitored if the patient has few symptoms. Indications for intervention include progressive pneumothorax, delayed pulmonary expansion, or development of symptoms.

TREATMENT

Moderate (20%-40%) and ***large*** (>40%): nearly always require intervention.

- Simple needle aspiration: It provides excellent management of iatrogenic pneumothoraces after central venous access or lung needle biopsy. This approach conservatively treats a sealed pneumothorax and identifies those with an active air leak for chest tube insertion.
- Needle catheter and thoracic vent drainage systems
- Chest tube insertion
- Surgery (VATS or Thoracotomy)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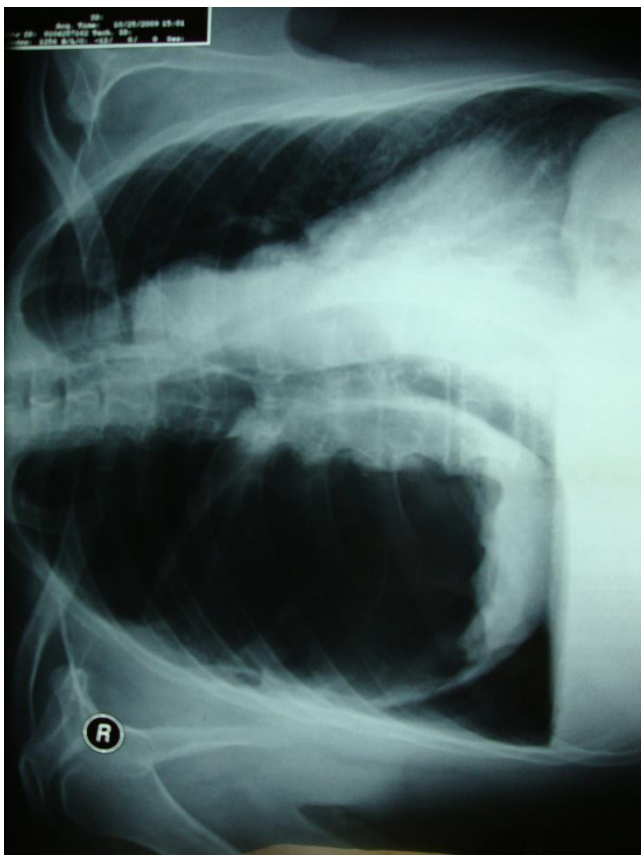
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Complications of Chest Tube Insertion

- Laceration of an intercostal vessel, the lung or any intrathoracic organ.
- Intrapulmonary or extrathoracic placement of the chest tube.
- Infection.
- Re-expansion pulmonary edema

  Although re-expansion pulmonary edema is thought to be secondary to a sudden increase in capillary permeability, the exact mechanism of this increased permeability is unknown. Most cases have been reported after rapid lung re-expansion.

INDICATIONS FOR SURGERY

Any episode:

- Persistent air leak for 5-7 days
- If the lung not completely re-expands.

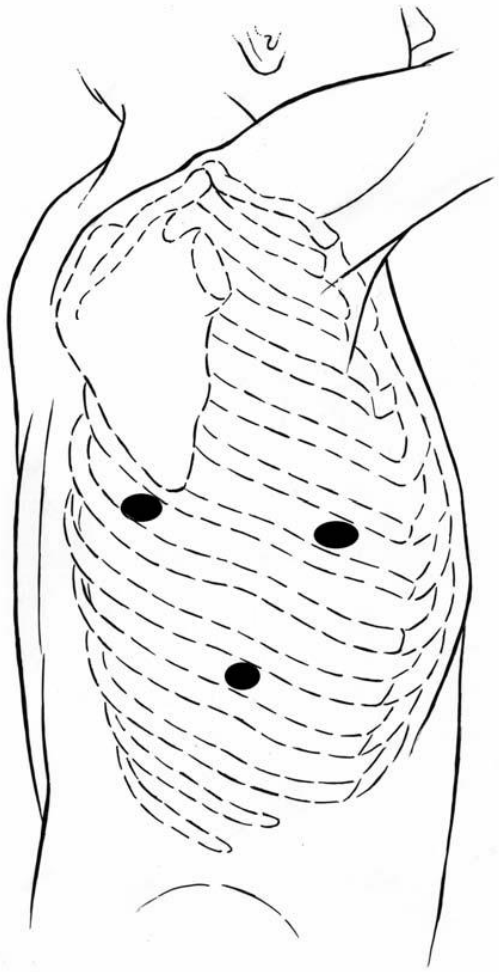
2nd episode:

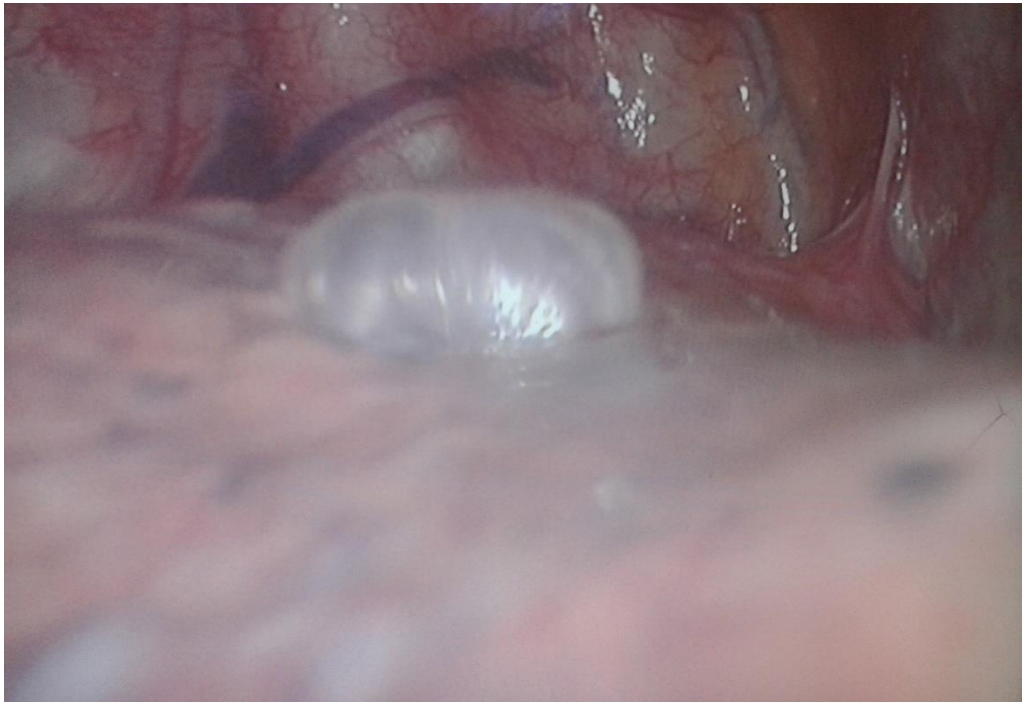
- Recurrence: The risk for first-time recurrence is about 25% to 30%, 2nd time 50%, and 3rd time 80%.
- Recurrence of a contralateral pneumothorax.

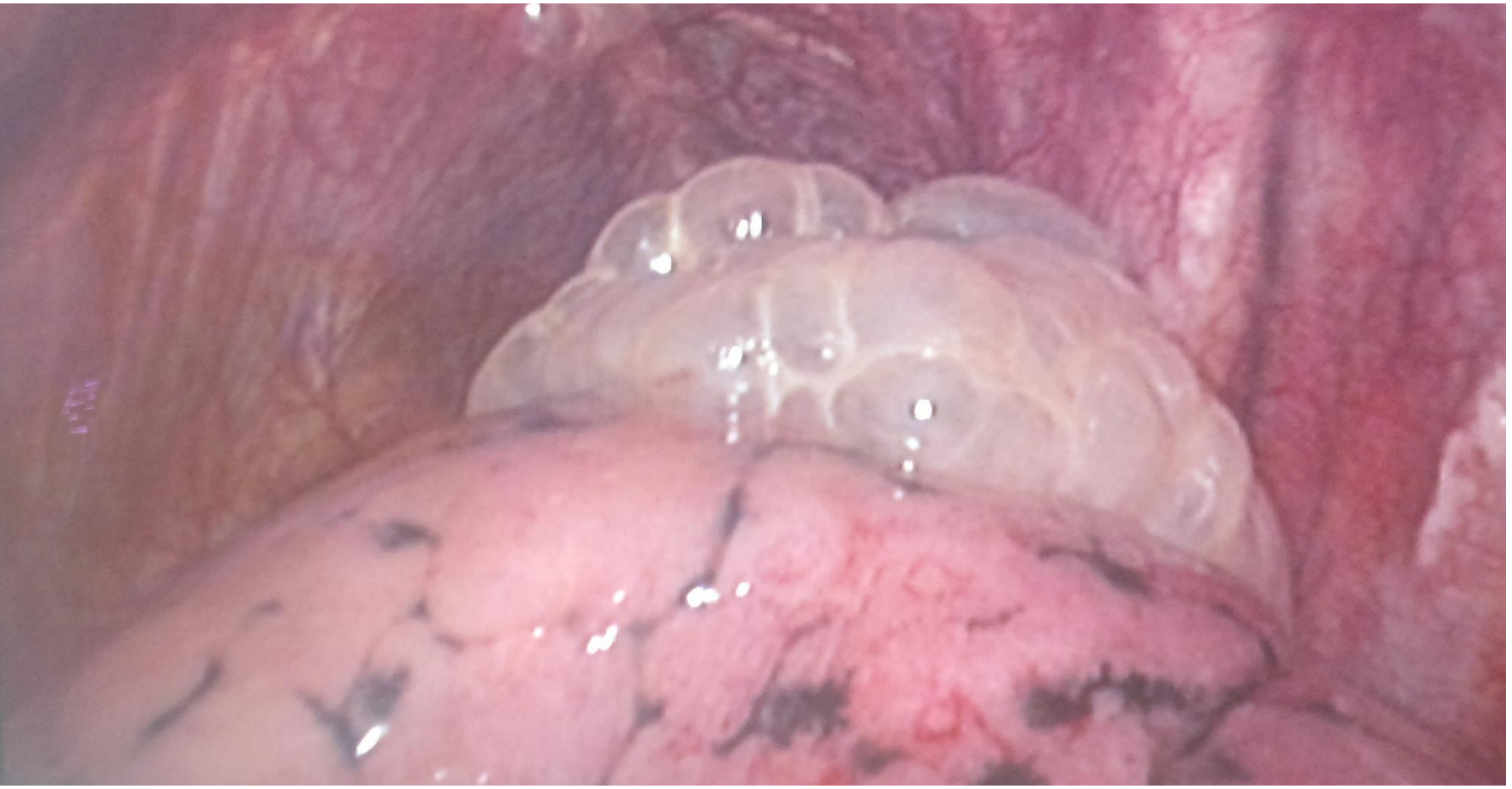
INDICATIONS FOR SURGERY

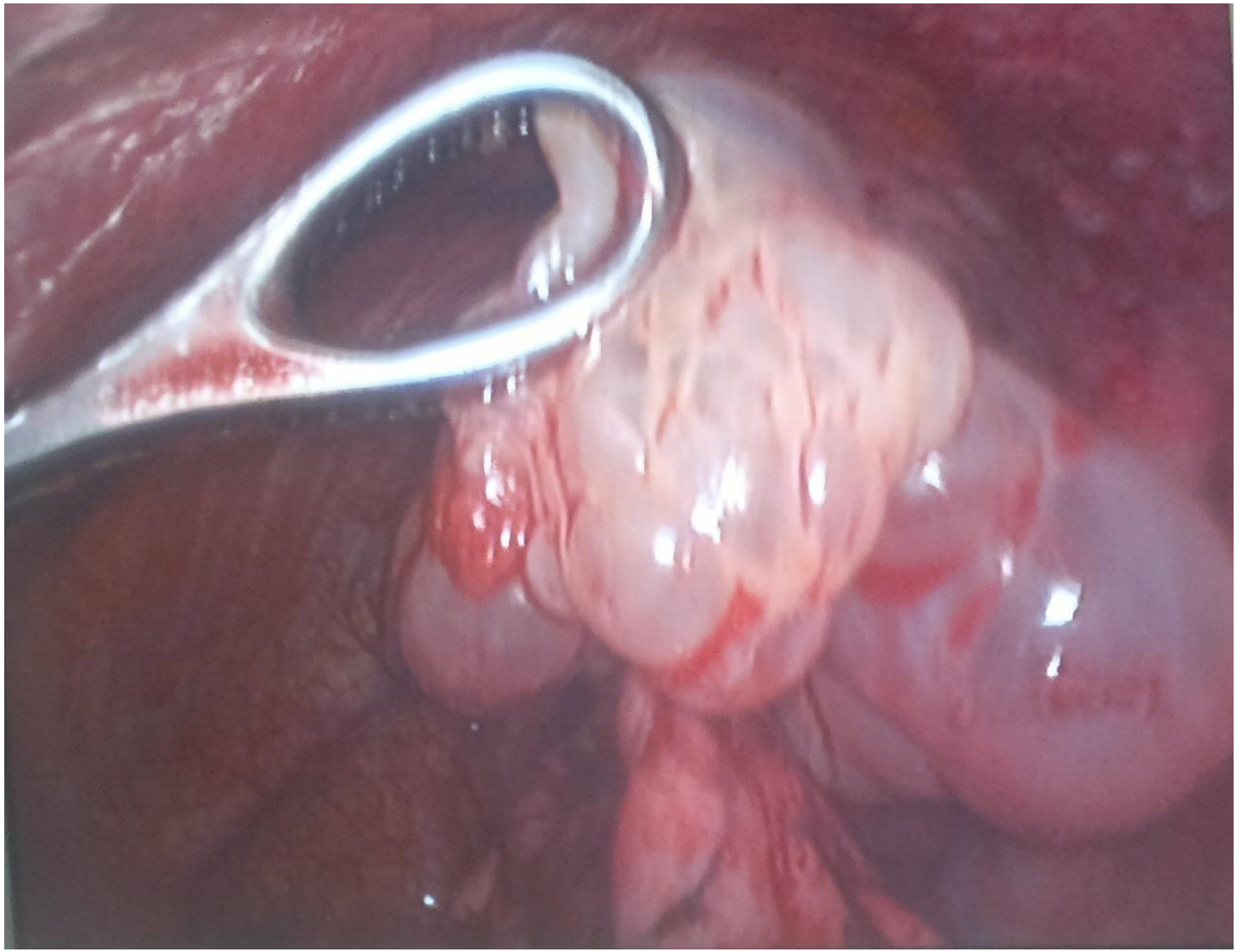
First episode

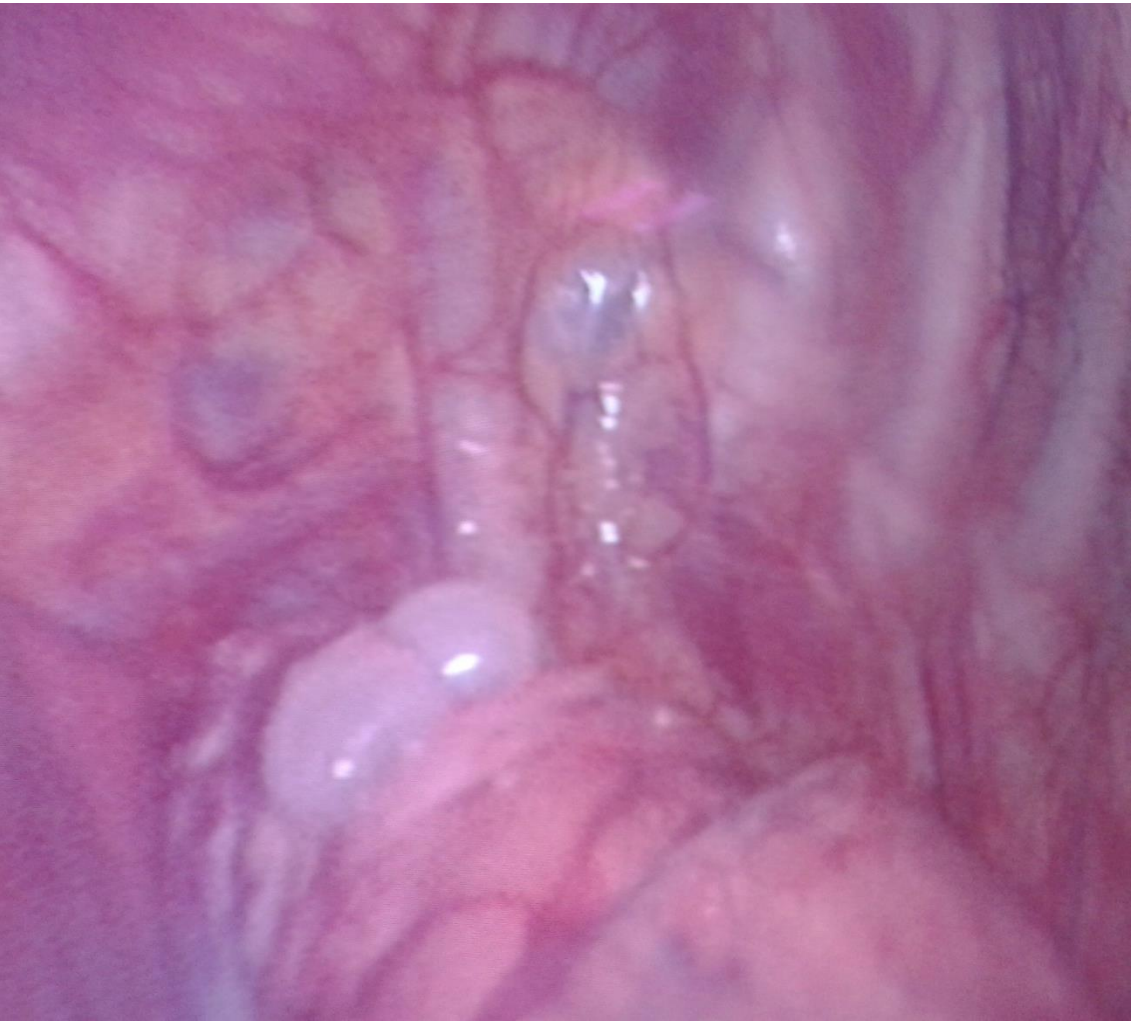
- Bilateral simultaneous pneumothoraces.
- Pneumothorax with contralateral pneumonectomy
- Complete (100%) pneumothorax
- Pneumothorax associated with tension or borderline cardiopulmonary reserve.
- In patients in high-risk professions or activities, such as pilots and scuba divers.
- Surgery for complications of pneumothorax (empyema, hemothorax, or chronic pneumothorax)

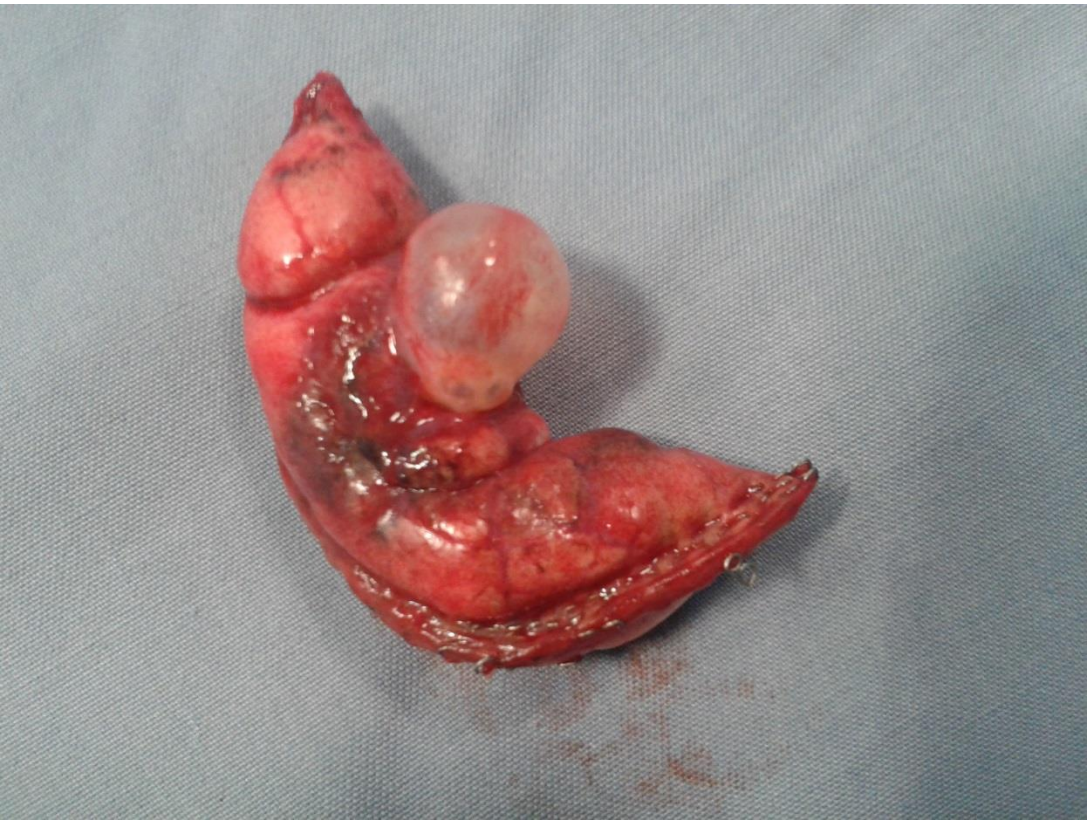












Thank You